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Original Research

Contributors to Initial Success on the National Athletic Trainers’ Association Board of Certification Examination as Perceived by Candidate Sponsors: A Delphi Study
Mary Ann Erickson, PhD, ATC; Malissa Martin, EdD, ATC ..................................... 134

An Examination of Stress and Burnout in Certified Athletic Trainers at Division I-A Universities
Amy E. Hendrix, MA, ATC; Edmund O. Acevedo, PhD, FACSM; Edward Hebert, PhD ........................ 139

Optimal Burst Duration During a Facilitated Quadriceps Femoris Contraction
Todd A. McLoda, PhD, ATC; Jennifer A. Carmack ................................................................... 145

Patrick J. O’Connor, PhD; Robert M. Murphy, ATC, MEd; Ron W. Courson, ATC, PT, CSCS; Michael S. Ferrara, PhD, ATC .......................................................... 151

A Comparison of Sideline Versus Clinical Cognitive Test Performance in Collegiate Athletes
James A. Onate, MA, ATC; Kevin M. Gusiewicz, PhD, ATC; Bryan L. Riemann, MA, ATC; William E. Prentice, PhD, ATC, PT .................................................. 155

Injuries and Illnesses in the National Basketball Association: A 10-Year Perspective
Chad Starkey, PhD, ATC ........................................................................................................ 161

Literature Reviews

It’s Not “Just a Finger”
Jan A. Combs, MD, ATC, FACSM, FAAOS .......................................................... 168

Asthma Medications: Basic Pharmacology and Use in the Athlete
Joel E. Houglum, PhD ........................................................................................................ 179

Accreditation and Continuous Quality Improvement in Athletic Training Education
Kimberly S. Peer, MA, ATC/L; Jonathon S. Rakich, PhD .................................................. 188

The Effect of Spirituality on Health and Healing: A Critical Review for Athletic Trainers
Brian E. Udermann, PhD, ATC .......................................................................................... 194

Case Reports

An Acute, Traumatic Supraspinatus Lesion in an Intercollegiate Football Player: A Case Report
Keith M. Gorse, MEd, ATC; Joseph B. Myers, MA, ATC; Marirose Radelet, PT; Mary Lynn Scovazzo, MD; Donald D’Alessandro, MD .................................................. 198

Clinical Techniques

A Classification System for the Assessment of Lumbar Pain in Athletes
Jonathan F. Heck, MS, ATC; Jeana M. Sparano, MPT .................................................. 204

Communications

National Athletic Trainers’ Association Position Statement: Fluid Replacement for Athletes
Douglas J. Casa, PhD, ATC, CSCS; Lawrence E. Armstrong, PhD, FACSM; Susan K. Hillman, MS, MA, ATC, PT; Scott J. Montain, PhD, FACSM; Ralph V. Reiff, MEd, ATC; Brent S.E. Rich, MD, ATC; William O. Roberts, MD, MS, FACSM; Jennifer A. Stone, MS, ATC .................................................. 212

Contents Continued
Departments

Editorial: Promoting Diversity in Athletic Training
David H. Perrin, PhD, ATC ....................................................... 131

Letter to the Editor .................................................... 132

Media Reviews ....................................................... 225

CEU Quiz Notice ...................................................... 129

Requests for Research and Education Foundation Proposals ....................... 130

23rd Annual Student Writing Contest Notice ...................................... 129

1999 JAT Outstanding Manuscript Awards ....................................... 130

Authors’ Guide ....................................................... 230

Advertisers’ Index ..................................................... 232
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**First Runner-Up:** DeMont RG, Lephart SM, Giraldo JL, Swanik CB, Fu FH. Muscle preactivity of anterior cruciate ligament-deficient and -reconstructed females during functional activities. 2:115–120.

**Second Runner-Up:** Rozzi SL, Lephart SM, Fu FH. Effects of muscular fatigue on knee joint laxity and neuromuscular characteristics of male and female athletes. 2:106–114.

**1999 Journal of Athletic Training Clint Thompson Award for the Outstanding Non-Research Manuscript**


**Second Runner-Up:** Casa DI. Exercise in the heat, II, critical concepts in dehydration, exertional heat illnesses, and maximizing athletic performance. 3:253–262.

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**NATA Research & Education Foundation Requests For Proposals**

The NATA Research and Education Foundation currently has the following four (4) active Requests for Proposals. Detailed information regarding each RFP, as well as the application process, may be found at the NATA Foundation web site at www.natafoundation.org.

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The basic definition of diversity is the state of being different. At a more sophisticated level than the simple word definition, diversity means inclusiveness—being accepting of people who are different from you. To become more diverse as a profession is to become more inclusive and accepting of students and colleagues regardless of color, national origin, race, religion, sex, or sexual orientation. Diversity means not only increasing the numbers, but also creating a welcoming environment. Valuing diversity means unconditionally accepting others who may be different from ourselves, a belief we should all embrace in athletic training.

Either by design or happenstance, the membership of our profession has become more diverse with respect to sex. In 1950, there were essentially no women in our organization. Now, approximately 44% of the certified membership of the NATA is female. Yet we still have work to do with respect to cross-gender sport assignments, particularly in the professional sport and major university athletics settings.

Our record on diversity with regard to race and ethnicity is abysmal. For all categories of NATA membership, 78% are white, 1% are black, 2% are Hispanic, 1% are American Indian or Alaskan Native, and 2% are Asian or Pacific Islander (not all NATA members answered this question). This compares with national averages of 71.7% white, 12.2% black, 11.6% Hispanic, 0.7% American Indian or Eskimo, and 3.8% Asian or Pacific Islander. And, our racial and ethnic diversity should be greater than the national average, particularly with respect to African Americans, because of the highly diverse population of athletes for whom we provide health care.

Why should we have a more diverse membership? Diversity will enhance the character of our profession in comparison with other medical and allied health care professions. It will distinguish us from other professions that have not addressed this challenge. Our athletes and patients will realize that competent ATCs can be of any background, including one similar to their own, which can increase awareness of athletic training as a profession open to minorities. The education of our students will be enriched by studying with, and learning the perspective of, minority students. Our students will be better prepared to work with the diverse population of patients and athletes they will encounter in professional practice. Preconceived notions about those who are different with regard to race, ethnicity, religion, and sexual orientation “go out the window” when we learn and work in a diverse environment.

How can we become a more diverse profession? Some strategies are easy, and others are far more complex. The importance of support for increasing diversity from our leadership cannot be overstated. Diversity cannot be mandated by our leaders, but must be nurtured, defended, and rewarded. We should provide support and incentive to the districts and states to sponsor programs designed to attract minority students to our profession. We must project the diversity that does exist in our profession in all of our promotional and educational materials on the local and national levels. We should establish a speakers’ bureau of minority athletic trainers to promote and increase awareness of athletic training as a profession open to minorities at high school career days and in other appropriate venues.

Every member of our association can have an impact on increasing diversity. If you are an athletic trainer for a professional sport, contact the guidance department at an inner city high school, and offer to provide a field trip of your work setting for minority students interested in the medical and allied health professions. If you are a high school athletic trainer, recruit minority students in athletic training through workshops and career days. Counsel students to follow a program of study in high school that will strengthen their credentials for admission to an athletic training program in college. Advise them to attend a college or university that offers a Commission on Accreditation of Allied Health Education Programs-accredited athletic training program.

If you are a college or university curriculum director, work with your admissions office to expose prospective minority applicants to your program. Conduct a weekend or week-long summer workshop to introduce minority high school students in your area to the athletic training profession. If you are an undergraduate student athletic trainer, reach out to a minority classmate you think might be inclined to an allied health care profession like ours. Invite him or her to spend a day with you in the athletic training room. If your program graduated just 2 minority students each year, we would double the number of minority certified athletic trainers in our profession in just 4 years.

Let us not just tolerate but celebrate the diversity that presently exists in our profession and commit ourselves to beginning a dialogue and developing a plan to increase diversity among our students and colleagues. The mission of our Ethnic Diversity Advisory Council is to advocate sensitivity and understanding toward ethnic and cultural diversity and to enhance the growth and diversity of our profession. Let us all embrace this mission and dedicate ourselves to becoming passionate, rather than passive, about increasing diversity in athletic training.
Letter to the Editor

The authors of the article “Effect of Microcurrent Stimulation on Delayed-Onset Muscle Soreness: A Double-Blind Comparison,” (J Athl Train. 1999;34:334–337) are to be congratulated for choosing to research a treatment modality that needs further investigation. Although we do not disagree with their conclusion that “At the parameters selected for this experiment, microcurrent stimulation was not effective...,” we do have data and experience to suggest that, if applied in an appropriate fashion, positive results can be expected when using this modality on muscle soreness.

At the time of our group’s introduction to this instrumentation 15 years ago, it was clear that the information needed to obtain optimal clinical results was not available. Over a period of years, we embarked on a series of clinical studies to attempt to identify the most effective methods of application of microcurrent electrical neuromuscular stimulation (MENS). Based upon these studies and our clinical experiences, we believe a different result could have been obtained in this study.

MENS requires pad placement that facilitates current traveling through the involved tissues, as opposed to arcing along the superficial tissues (ie, anterior, posterior or medial, and lateral placement instead of all pads being on 1 body surface). The optimal placement for treating the biceps is medial and lateral. The positioning of pads on body surfaces opposite a large bone (eg, the humerus) will predictably result in reduction or elimination of a positive clinical result. In this study, a 1-channel set-up with 2 pads was used instead of 4 pads via either a 2-channel unit or splitters on a 1-channel instrument. A 4-pad application will produce a superior result, possibly because there is an impact on more neuroepidermal junctions.

Application of MENS via probes can produce a better response than application with pads. To treat the biceps with probes, the same medial and lateral approach is used. Results will vary depending on how many points on the arm are treated. A total treatment time of 10 minutes with probes used every 2.54 cm (1 in) along the muscle will not produce as good a result as probes used every 1.27 cm (0.5 in) along the muscle. The every 1.27-cm (0.5-in) application possibly is more effective because of the impact on more neuroepidermal junctions.

Selection of treatment time, frequency, output, and wave slope will also affect the outcome. Optimal treatment time varies with the individual response to the treatment. The total treatment time of 20 minutes used in this study is less than optimal. The 30-Hz and 0.3-Hz frequencies are commonly used and produce predictable results if the other treatment parameters are correctly chosen. Better choices are 100 μa and 40 μa, given the results of several studies suggesting that, for this type of electrotherapy, less current tends to be more productive. If the meter on the unit shows that the circuit is not 100% conductive, then the output must be turned down until 100% conductivity is achieved.

The paper does not state the type of wave slope used in this study. The selection of wave slope has a distinct impact on pain reduction. A square wave tends to have an immediate impact on pain relief, but selection of a more gradually ascending sloped curve will positively affect pain status 24 hours later.

Patient positioning during treatment, presence or absence of movement during treatment, and clinician attitude are all variables that will influence outcome.

Based upon a review of 1531 consecutive cases in 1 of our clinics, we have established treatment goals using MENS. The average of these cases was a 52% reduction in pain during the first visit, 33% carryover to the next visit, and 3.8 visits to pain-free status. Results obtained with MENS are very consistent, regardless of use on extremities, spine, joints, muscles, or tendons. Some differences in results occur in that the more vascular tissues respond better.

Certainly more double-blind studies are needed on this instrument and the multiple variables that need to be manipulated to achieve an optimal clinical result. We hope that the authors will continue to use the resources at their disposal for further investigation in this area.

Elizabeth Holtzheimer, PTA, BS
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Authors’ Response

We thank Dr. Wallace and colleagues for sharing their clinical observations regarding the application of microcurrent stimulation. Documented efficacy of MENS treatment is limited in comparison with other therapeutic modalities. Moreover, there is a dearth of experimentally derived treatment protocols in the scientific literature. As such, we relied on the manufacturer’s recommendations for the treatment parameters selected for our study.

We commend Wallace and colleagues for the diligent evaluation of their clinical cases and the documentation of their clinical results. In their commentary, they address a series of factors that are important to consider when designing a research protocol. They recommend treating the biceps brachii with a medial-lateral pad placement and 4-electrode application versus the anterior-posterior placement and a 2-electrode application in our study. Additionally, they suggest that...
increasing treatment time and altering treatment frequency and output may provide better treatment results. In our judgment, these are all appropriate suggestions for further study. The authors also noted that we did not describe the wave slope used in this study. For purposes of clarification for further study, we used a square wave.

Our goal in examining the treatment parameters selected for our experiment was to examine 1 of the many possible treatment methods that may be used for MENS. Although our study demonstrated no significant difference between the treatment and sham groups, this does not imply that MENS treatment will never be effective in the treatment of delayed-onset muscle soreness, but simply that it was not effective at the parameters we selected.

In summary, there are various clinical parameters and protocols using MENS that require scientific validation. We thank Dr. Wallace and colleagues for their comments and encourage them to submit their work to the rigor of peer review. Without scholarly discourse or peer-reviewed publication, it is often difficult for clinicians and researchers to access valuable and reproducible techniques and data. Continued investigation to establish treatment efficacy with this modality is certainly warranted, and it is our hope that dialogue such as this will encourage additional research.

Jennifer D. Allen, MEd, ATC, PT
Carl G. Mattacola, PhD, ATC
David H. Perrin, PhD, ATC
Contributors to Initial Success on the National Athletic Trainers’ Association Board of Certification Examination as Perceived by Candidate Sponsors: A Delphi Study

Mary Ann Erickson, PhD, ATC*; Malissa Martin, EdD, ATC†

*Fort Lewis College, Durango, CO; †Middle Tennessee State University, Murfreesboro, TN

**Objective:** To determine the factors that are perceived to contribute to first-time success on the National Athletic Trainers’ Association (NATA) Board of Certification Examination.

**Design and Setting:** We surveyed a panel of athletic training educators who sponsor candidates for the examination by means of the Delphi technique. The Delphi technique is a method of structuring the collective judgments of a group of experts, conducted through a series of sequential questionnaires, each containing summarized information from earlier responses.

**Subjects:** A total of 29 athletic training program directors whose programs are accredited by the Commission on Accreditation of Allied Health Education Programs or approved by the NATA.

**Measurements:** We used 3 questionnaires to solicit the opinions of experts and ultimately reach consensus. Each questionnaire was generated from the results of the previous questionnaire. The initial questionnaire asked respondents to list items that they perceived as contributing to first-time success. The second was generated from the results of the first and asked respondents to rate items using a Likert scale. The third questionnaire allowed respondents to change their answers based on the information presented. The study concluded with a consensus confirmation report that asked respondents to concur with the results of the study.

**Results:** The panel reached consensus on items that reflected clinical experiences, teaching qualities and practices of the clinical instructors, student knowledge, test-taking skills, and student characteristics. Of these consensus items, the items contributing significantly to initial examination success were “ability to interpret the question,” “knowledge of theories and techniques in rehabilitation and modalities,” “clinical assessment skills,” “clinical settings that allow students to take an active role,” and “instructors committed to providing a positive learning environment.”

**Conclusions:** We noted 5 different areas perceived as contributing to a student’s passing the examination on the first trial. These results can be used by program directors to enhance their curricular, didactic, and clinical structures to better prepare students for the examination. The results can also be used by the NATA Education Council in the development of educational programs for certified athletic trainers to become effective clinical instructors.

**Key Words:** education, examination success, testing, Delphi technique, predictors of success, certification examination

Today, unlike any other time in the history of higher education, the public holds institutions of higher learning accountable to properly prepare students to successfully compete in the employment arena. Academic programs are held accountable for students who pass the certification and licensing requirements that their educational programs prepare them to take. Athletic training education programs prepare students for the National Athletic Trainers’ Association Board of Certification (NATABOC) examination and employment as certified athletic trainers (ATCs). The purpose of the examination is to certify to the public that the ATC is qualified to practice athletic training. In the past 3 years, the percentage of students who passed all 3 sections of the NATABOC examination on their first attempt was 27% in 1996 (J. Henderson, oral communication, April 1997), 32% in 1997,1 and 31% in 1998.2 Physical therapy reports an initial passing rate of 85%.3 Nursing is similar, reporting a passing rate of 82%.4 This discrepancy may be an indication of the difficulty of the NATABOC examination when compared with other allied health fields. The NATABOC examination essentially requires first-time candidates to sit for 3 examinations. The physical therapy boards consist of only 1 examination, a written test. Another explanation is that athletic training students are not being adequately prepared for certification.

A positive relationship seems to exist between predictor variables and student success on certification examinations and academic achievement.5–10 Grade point average (GPA) appears to be a better predictor of success than grades in theory courses, American College Testing composite scores, and interview ratings. Although similar studies have been conducted in athletic training education,11–16 their inconclusive results give us an indication of the limited understanding...
educators have when trying to assist athletic training students prepare for certification as athletic trainers. The variables studied have been those that are easily quantifiable, and quantifiable measures do not always give a true indication of the ingredients necessary for success. Through the use of the Delphi technique and the solicitation of expert opinion, contributing factors that are common to all athletic training education programs and are essential for examination success can be made clear.

Our purpose was to determine the factors that athletic training educators throughout the United States perceived as contributing to first-time success on the NATABOC examination. By identifying those factors that contribute to examination success, schools gain some control over the academic success of their students, and they can develop intervention programs to assist students in achieving their educational goals.

METHODS

Selection of the Panel of Experts

By means of the Delphi technique, we surveyed a panel of athletic training program directors who sponsor candidates for the examination. Participants were selected based on 3 criteria: program director of a Commission on the Accreditation of Allied Health Education Programs (CAAHEP)-accredited or National Athletic Trainers' Association (NATA)-approved athletic training education program; the program must have been accredited or approved before 1995; and the director must have sponsored at least 10 candidates for the NATABOC examination. A total of 77 program directors met the criteria for inclusion in the initial round of the study.

We mailed round 1 of the study to all 77 qualified program directors. Thirty-five program directors (45%) returned the initial questionnaire and composed the panel of experts for round 2. Round 2 was mailed to the 35 panel members. Twenty-nine panel members (83%) returned the second questionnaire, thus composing the panel for round 3. All panelists who received questionnaire 3 participated in the consensus confirmation report asking them if they concurred with the results.

Survey Instrument

Three athletic trainers who had previously sponsored students for the NATABOC examination piloted the first-round questionnaire. The pilot verified that the initial question was clear and unambiguous and generated the intended information. These athletic trainers did not participate in subsequent rounds. Permission to conduct the study was obtained from the Institution’s Internal Review Board before initiating the study, and each subject signed an informed consent.

The study consisted of 3 sequential questionnaires and a consensus confirmation report. The first questionnaire asked the participants to list those factors that they perceived as contributing to first-time success on the NATABOC examination. Subsequent questionnaires were based on the results of the previous questionnaire.

The second questionnaire listed the results of the first questionnaire and asked the participants to rate each item using a 7-point Likert scale, indicating their judgment of its contribution to first-time success on the NATABOC examination. The third questionnaire contained the results of the second questionnaire, together with the group mean and standard deviation for each item, as well as individual results. Each participant was given the opportunity to change his or her score based upon the group data provided. A final consensus confirmation report asked the participants to agree or disagree with the final results of the study.

RESULTS

The initial open-ended question resulted in 221 items that program directors perceived as contributing to initial examination success. We compiled the items into 66 items to avoid redundancy on the second questionnaire. Participants were then asked to rate each item’s contribution to examination success using a 7-point Likert scale. Descriptive statistics (mean and standard deviation) for each of the items were calculated using the Statistical Package for the Social Sciences, version 7.5 for Windows (SPSS Inc, Chicago, IL).

The third questionnaire was created using responses on the second questionnaire with a mean greater than or equal to 2.0, as well as the mean and standard deviation of each response. This questionnaire contained participants’ individual scores and boxes for them to change their scores if they chose to do so. We also gave them the opportunity to make comments concerning the item if they desired. Responses to the third questionnaire were again analyzed using SPSS 7.5 for Windows. As is expected with the Delphi technique, changes tended to be toward the mean (Table). Items are listed in rank order by mean.

Of the original 66 items compiled from individual responses, 23 (35%) were retained as contributors to passing all 3 sections of the NATABOC examination on the first attempt. Items had a mean of 5.0 or higher, indicating that the panel considered the items important to examination success. They also reached a high degree of consensus (SD ≤ 1.0) on the selected items, indicating agreement on contributing factors to first-time examination success.

DISCUSSION

The panel believed it was important and strongly agreed that students must possess the ability to interpret the examination questions correctly (mean = 5.45, SD = 0.67). In preparation for the NATABOC examination, students must be exposed during their educational experience to multiple choice questioning, oral and practical examinations, and problem-solving situations. To expect athletic training students to enter the NATABOC examination qualified to pass on the first trial, athletic training educators and clinical instructors must be able to design testing experiences that mirror the format of the examination.

The written simulation portion of the NATABOC examination presents students with an initial problem and asks students to make choices based on the information given. The students must interpret the question correctly and make appropriate choices to increase their chances of passing this section of the examination. The other skill necessary to successfully complete this portion of the examination is problem solving. Students are instructed to answer the written simulation question in an efficient and proficient manner. Therefore, if the students are lacking in problem-solving skills, they may not be
able to deduce the proper set of responses. The panel identified possession of appropriate problem-solving skills as important to first-time examination success and agreed on its importance (mean = 5.23, SD = 0.74).

The panel strongly agreed (SD ≤ 0.68) that students need to possess knowledge of theories and techniques of rehabilitation and modalities, and they also need exposure to clinical settings where they take an active role in the care of the athlete and practice and perfect those clinical skills. Students, though requiring a didactic background for understanding, need a “hands-on” clinical environment in which to practice and perfect clinical skills to be effective in their delivery of care to athletes. Not only did the experts agree (SD = 0.68) on this concept, but they felt it was important (mean = 5.41) for examination success.

Participants strongly agreed and felt it was important (mean ≥ 5.37, SD ≤ 0.75) that students have knowledge of anatomy and physiology and knowledge of the prevention and evaluation of athletic injuries to pass the examination on their first trial. Again, these subjects are the foundation of the care the athletic trainer provides to athletes. Athletic trainers cannot properly evaluate an injury without a thorough understanding of anatomy. Questions addressing these areas are found on all 3 portions of the NATABOC examination.

When developing education programs in athletic training, program directors must demonstrate how the competencies in athletic training are being addressed in didactic courses, as well as in the clinical courses. The competencies are developed from the Role Delineation Study, from which the examination is developed. Although the panel considered it important for athletic training educators to cover these competencies in athletic training classes (mean = 5.31), it did not feel student familiarity with the competencies or the Role Delineation Study was important (mean = 4.43). The fact that this item did not reach consensus may be contradictory to educational theory. The Role Delineation Study is embedded in the competencies. Both documents give us a basis for establishing educational objectives and goals. Clearly identified objectives and goals allow students to recognize the skills and competencies they will be expected to understand and perform at the completion of a particular educational experience. Clearly stated educational objectives drive the educational process.

Inherent in the profession of education is the need for instructors who are committed to providing a positive learning environment in which students can learn and develop their skills. This premise was supported by the data. The item “Clinical instructors committed to providing a positive learning environment in which students can learn and develop their skills” reached consensus (mean = 5.31, SD = 0.68). This item is an example of a teaching component that has been identified as important to examination success.

### Means and Standard Deviations of Questionnaires 2 and 3 in Rank Order by Mean of Questionnaire 3

<table>
<thead>
<tr>
<th>Item</th>
<th>Mean #2</th>
<th>SD #2</th>
<th>Mean #3</th>
<th>SD #3</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. The student’s ability to interpret the question correctly.</td>
<td>5.41</td>
<td>0.72</td>
<td>*5.45</td>
<td>*0.67</td>
</tr>
<tr>
<td>2. Clinical assessment skills.</td>
<td>5.41</td>
<td>0.82</td>
<td>*5.45</td>
<td>*0.74</td>
</tr>
<tr>
<td>3. Knowledge of theories and techniques in rehabilitation and modalities.</td>
<td>5.41</td>
<td>0.68</td>
<td>*5.45</td>
<td>*0.69</td>
</tr>
<tr>
<td>4. Clinical instructors committed to providing a positive learning environment that stresses educating the student on athletic training skills.</td>
<td>5.30</td>
<td>0.82</td>
<td>*5.41</td>
<td>*0.68</td>
</tr>
<tr>
<td>5. Exposure to clinical settings that allow students to take an active role in the overall care of the athlete.</td>
<td>5.41</td>
<td>0.68</td>
<td>5.41</td>
<td>0.68</td>
</tr>
<tr>
<td>6. Students demonstrate a high level of genuine inquisitiveness and take initiative in their own learning.</td>
<td>5.38</td>
<td>0.82</td>
<td>*5.41</td>
<td>*0.78</td>
</tr>
<tr>
<td>7. Knowledge of anatomy and physiology.</td>
<td>5.40</td>
<td>0.75</td>
<td>5.40</td>
<td>0.75</td>
</tr>
<tr>
<td>8. Knowledge in the prevention and evaluation of athletic injuries.</td>
<td>5.37</td>
<td>0.68</td>
<td>5.37</td>
<td>0.68</td>
</tr>
<tr>
<td>9. Integration of cognitive, psychomotor, and affective competencies and skills into daily clinical experience.</td>
<td>5.30</td>
<td>0.82</td>
<td>*5.33</td>
<td>*0.78</td>
</tr>
<tr>
<td>10. Ensuring all NATABOC competencies are being covered in our athletic training courses.</td>
<td>5.30</td>
<td>0.93</td>
<td>*5.31</td>
<td>*0.89</td>
</tr>
<tr>
<td>11. Courses that place a demand on the students to perform optimally.</td>
<td>5.26</td>
<td>0.81</td>
<td>*5.29</td>
<td>*0.77</td>
</tr>
<tr>
<td>12. High level of common sense combined with the ability to adapt to situations perhaps not covered or covered differently in undergraduate courses.</td>
<td>5.26</td>
<td>0.83</td>
<td>*5.29</td>
<td>*0.79</td>
</tr>
<tr>
<td>13. The ability of the student to think critically.</td>
<td>5.22</td>
<td>0.85</td>
<td>*5.25</td>
<td>*0.77</td>
</tr>
<tr>
<td>14. Quality clinical experiences.</td>
<td>5.31</td>
<td>0.81</td>
<td>*5.25</td>
<td>*0.79</td>
</tr>
<tr>
<td>15. Possession of appropriate problem-solving skills.</td>
<td>5.23</td>
<td>0.74</td>
<td>5.23</td>
<td>0.74</td>
</tr>
<tr>
<td>16. Decision-making skills.</td>
<td>5.16</td>
<td>0.77</td>
<td>*5.22</td>
<td>*0.75</td>
</tr>
<tr>
<td>17. Students’ willingness to review and seek out further information in an identified area of weakness.</td>
<td>5.17</td>
<td>0.76</td>
<td>5.17</td>
<td>0.76</td>
</tr>
<tr>
<td>18. A well-structured, positive learning environment where all athletic trainers frequently evaluate the students on their skills and where everyone has a stake in the success of the students.</td>
<td>5.14</td>
<td>0.79</td>
<td>5.14</td>
<td>0.79</td>
</tr>
<tr>
<td>19. Ability of the student to focus.</td>
<td>5.12</td>
<td>1.03</td>
<td>5.12</td>
<td>1.03</td>
</tr>
<tr>
<td>20. Leadership and mentoring by a variety of clinical instructors that is challenging but also supportive.</td>
<td>5.09</td>
<td>0.87</td>
<td>5.09</td>
<td>0.87</td>
</tr>
<tr>
<td>21. Instructors who possess a pedagogic background and are committed to the education of the student.</td>
<td>5.09</td>
<td>0.91</td>
<td>5.09</td>
<td>0.91</td>
</tr>
<tr>
<td>22. Completion of an accredited curriculum.</td>
<td>5.03</td>
<td>1.05</td>
<td>*5.07</td>
<td>*0.96</td>
</tr>
<tr>
<td>23. Integration of appropriate supporting materials (textbooks, current research) throughout curriculum.</td>
<td>5.03</td>
<td>0.87</td>
<td>5.03</td>
<td>0.87</td>
</tr>
</tbody>
</table>

*Indicates a change from round 2 to round 3.
environment that stresses educating the student on athletic training skills" was believed to be important (mean = 5.41), and the panel strongly agreed on its importance (SD = 0.68). They indicated that courses should place a demand on the student to perform optimally and thus contribute to examination success. They also felt that the learning environment should be well structured and positive for the student to succeed (mean = 5.10, SD = 0.79). Creating this environment in the clinical setting is difficult in the normal course of the athletic training student’s education. Most education takes place in the athletic training room, where athletes are in various stages of injury, evaluation, and treatment. As 1 panel member stated, “The traditional athletic training room is generally not a ‘well-structured positive learning environment’ and really needs to be for the student’s sake.” Didactic education of the student, on the other hand, can and should be made to be positive and structured. Students should receive frequent feedback on their academic performance and progress throughout the duration of the course.

The panel perceived proper mentorship and leadership by a variety of instructors as contributing to examination success (mean = 5.09). Athletic training students work side by side with certified athletic trainers. Mentorship would indicate that these certified athletic trainers “guide” the students through the clinical experience. As students gain more experience in the clinical setting, they should be given more and more responsibility. Clinical instructors who are aware of pedagogic principles gradually allow students to assume more responsibility. These instructors are the leaders who facilitate the education of the students, rather than the bosses who dictate to the students what, how, and when to do something.

Clinical instructors who possess a pedagogic background also contributed to examination success (mean = 5.09). One comment by a panel member addressed this issue: “Most clinical supervisors are interested in 1) athletic coverage, and 2) student education. This puts the student in a work situation rather than an educational situation. Placing the students in a situation (practical) in which they have no classroom preparation is pedagogically incorrect.” The theory behind the rehabilitation and modality techniques must be integrated into the clinical education of the student. Athletic training educators, both clinical and didactic, must teach “what” and “how,” but more importantly “why.” Erudition of pedagogic principles allows the clinical instructor to construct an effective educational experience for the student.

Athletic training educators on the panel were of the opinion that those students who possessed self-confidence, inquisitiveness, common sense, the ability to think critically, and decision-making skills (mean ≥ 5.22, SD ≤ 0.75) were more likely to pass their examination on the first attempt. Athletic training is a profession that frequently presents stressful, nontextbook situations. The better students can adjust to these ever-changing situations, the more successful they will be.

Athletic training students must possess adequate clinical assessment skills to be prepared for the oral-practical section of the NATABOC examination. Participants concurred, rating this factor as important (mean = 5.45) and agreeing on its importance (SD = 0.74). When it comes to the oral-practical examination, students with poor assessment skills frequently flounder and fail to address the posed questions. The clinical education of students must be designed to allow students adequate practice of assessment skills under the direct supervision of the clinical supervisor. This direct supervision allows the clinical supervisor to intercede and carefully redirect students while they are perfecting their assessment skills.

Athletic training educators are currently discussing the clinical-hour requirement necessary to sit for the NATABOC examination. The NATA requires that students obtain a minimum of 800 clinical hours before becoming eligible to sit for the examination. CAAHEP, the accrediting body for athletic training educational programs, does not require a minimum number of clinical hours for students graduating from the same programs. The quantity and quality of the clinical hours has been left to the discretion of the individual school providing the athletic training education program. The discrepancy arises from the quality versus the quantity of the hours students obtain. The panel participants considered a quality clinical experience (mean = 5.25, SD = 0.79) important to examination success. Quality clinical experiences should involve active student participation. As 1 panel member stated, “This is very important to back up didactic learning.” Active participation by students must be built into an effective athletic training educational program. We cannot expect students to graduate and practice athletic training after watching certified athletic trainers evaluate injuries for 3 to 4 years. Students must be exposed to situations that force them into independent decision making.

Although the panel perceived obtaining hours in excess of the 800 required as important to examination success (mean = 4.73), that factor was perceived as less important than the quality of the clinical hours. Interestingly, the panel believed that exposing the student to a variety of clinical experiences was not highly important to examination success (mean = 4.36, SD = 1.08) and was fairly strong in its agreement. As one panel member put it, “a sprained ankle is a sprained ankle.”

The literature in physical therapy and nursing5-10 is fairly consistent in demonstrating GPA as a predictor of success. The experts on this panel disagreed with that premise and did not feel that overall GPA (mean = 4.31) was as important as athletic training GPA (mean = 4.58), and they were in fairly strong agreement (SD = 0.85). GPA seems to be an issue that higher education is struggling with in current educational practice.

Researchers in allied health fields have selected quantitative variables for predicting examination success.5-10 Our results demonstrate that athletic training educators believe there is more to passing a certification examination than good grades. Many of these contributing factors (problem solving, interpretation of test questions, providing a quality clinical experience, learning environment) originate in the educational environment of the student. The design of the educational experience must address these variables so that students are prepared for the NATABOC examination and for employment as ATCs.

As the Education Council develops the courses for the Clinical Instructor Educator, the above-mentioned variables must be taken into consideration. The skills of problem solving and test-question interpretation are not acquired from a textbook. The quality of the clinical experience and a positive learning environment reflect the overall structure of the educational experience. Our results indicate that athletic training educators must carefully review both didactic and clinical instruction in athletic training education programs. Athletic training educators must begin to carefully construct our students’ clinical experiences and take formal control of the educational programs.
ACKNOWLEDGMENTS

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REFERENCES

An Examination of Stress and Burnout in Certified Athletic Trainers at Division I-A Universities

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*Southeastern Louisiana University, Hammond, LA; †University of Mississippi, University, MS

Objective: A growing body of knowledge indicates that too much stress can negatively influence psychological and physical health. A model proposed by Smith to explore personal and situational variables, stress appraisal, and burnout has led to significant understanding of burnout of individuals working in service professions. We examined the relationship of hardness, social support, and work-related issues relevant to athletic trainers to perceived stress and the relationship of perceived stress to burnout.

Design and Setting: Correlational analyses were performed to examine the relationships predicted by Smith’s model. In addition, we conducted stepwise multiple regression analyses to assess the relative contributions of the personal and situational variables to perceived stress and to examine the relative impact of perceived stress on 3 burnout factors (emotional exhaustion, personal accomplishment, and depersonalization).

Subjects: One hundred eighteen certified athletic trainers working in National Collegiate Athletic Association Division I-A intercollegiate settings that maintain a football program.

Measurements: We assessed personal and situational variables using the Hardiness Test, the Social Support Questionnaire, and the Athletic Training Issues Survey, adapted for this study. The Perceived Stress Scale was used to assess stress appraisal, and the Maslach Burnout Inventory was used to assess 3 dimensions of burnout.

Results: Our results were in support of Smith’s theoretical model of stress and burnout. Athletic trainers who scored lower on hardness and social support and higher on athletic training issues tended to have higher levels of perceived stress. Furthermore, higher perceived stress scores were related to higher emotional exhaustion and depersonalization and lower levels of personal accomplishment.

Conclusions: Our findings examining burnout in Division I athletic trainers were similar to those of other studies investigating coaches and coach-teachers and in support of Smith’s theoretical model of stress and burnout.

Key Words: hardness, social support, athletic training issues, sport psychology

High levels of perceived stress and burnout have been reported in a variety of service professions, including social workers, physicians, psychologists, police officers, lawyers, and counselors. Stress and burnout are constructs that can have a negative influence on those working in human service or helping professions. The detrimental effects of stress and burnout have been documented, and the importance of managing stress has been established by the medical community. However, the limited literature on burnout in sport has focused on coaches and sport officials and coach-teachers. Burnout has been defined as a reaction to chronic stress that involves negative interactions between environmental and personal characteristics. Burnout has also been characterized as a chronic condition that develops when one is working too hard for too long in a high-pressure situation. Furthermore, burnout is conceptualized as uncontrollable, negatively perceived events occurring over a period of time that lead to 3 negative psychological responses: depersonalization, emotional exhaustion, and a lack of personal accomplishment.

Few theoretical models of burnout have been proposed and investigated. Additionally, theories by Cherniss and Golembiewski and Munzenrieder have been criticized for theoretical and practical limitations that have prevented empirical progress from continuing. More recently, Smith’s proposed theoretical framework has received substantial support and has been used as the basis for much of the contemporary research on burnout.

Smith’s model asserts that personal and situational characteristics influence perception of stress, and, in turn, perception of stress influences the level of burnout. Personal and situational characteristics such as hardness, social support, motivation, and work-related issues compose the first component of the model. The second component reflects the cognitive stress appraisal of individuals, and the third component represents the coping strategies employed by individuals (ie, level of burnout). With poor coping skills, an individual is at higher risk for burnout and the deleterious consequences that accompany the phenomenon. Research with coaches and coach-teachers has demonstrated significant relationships between the personal and situational variables and stress appraisal and between stress appraisal and dimensions of burnout. In addition, this research has demonstrated distinctions among professions.
Factors influencing burnout in athletic trainers. 

Attrition is a concern facing the athletic training profession. Just as athletes retire before reaching their physical or psychological "prime," athletic trainers lose the compassion and excitement that initially drew them to the profession. To date, 2 published studies have examined burnout in athletic trainers. Higher frequency and intensity levels of burnout were associated with role conflict, role ambiguity, a greater number of athletes to care for, a decreased resource base, and a greater number of hours needed to provide for the athletes. Athletic trainers are subject to a unique set of stressors as health service professionals. These stressors can include high athlete to athletic trainer ratio, minimal financial support, and dual role responsibilities such as head athletic trainer and curriculum director or clinical instructor. Other stressors that athletic trainers experience are associated with relationships to athletes, parents, coaches, administrators, and physicians. These factors can lead to high stress levels, which in turn can influence burnout.

The purpose of our study was to explore the utility of Smith's model to predict stress appraisal and burnout in certified athletic trainers in the National Collegiate Athletic Association (NCAA) Division I-A intercollegiate setting. More specifically, we investigated the ability of hardiness, social support, and athletic training issues to predict stress appraisal and the ability of stress appraisal to predict burnout (conceptually defined as emotional exhaustion, depersonalization, and personal accomplishment). The Figure presents Smith's model of burnout, including the constructs examined in this study.

A greater understanding of the factors that influence burnout in athletic trainers will provide direction in our efforts to alleviate stress and burnout. We expected that athletic trainers who report lower levels of hardiness and social support and higher levels of athletic training issues would report higher levels of perceived stress. In addition, we predicted that athletic trainers reporting higher levels of perceived stress would also indicate higher levels of emotional exhaustion and depersonalization and lower levels of personal accomplishment.

METHODS

Subjects

The subjects for this study were 118 certified athletic trainers working at NCAA Division I-A universities that sponsored football.

Instrumentation

We assessed the personal and situational variables of hardiness, social support, athletic training issues, and perceived stress. The psychological tools included 5 instruments that have endured psychometric testing and have been deemed valid and reliable.

Hardiness

Hardiness is a personality construct that reflects control (the tendency to believe in one's ability to influence the course of events and to act as if one has power in the face of various life circumstances), commitment (facing life with an eager curiosity, a sense of purpose, and a willingness to commit oneself to relationships), and challenge (believing that change rather than stability in life is the norm and that changes are interesting, positive, and the stimulus for growth). The Hardiness Test is a 30-item instrument designed to measure this construct. Items include, "In general, I tend to be a bit critical, pessimistic, and cynical about most things in work and life" and "I am committed to my work, the school, and the activities that I am currently pursuing." A 5-point Likert scale ranges from "strongly agree" (1) to "strongly disagree" (5). Estimates of internal consistency have yielded coefficient alphas in the 0.90s for total hardiness score and in the 0.70s for commitment, control, and challenge scores, with test-retest correlations in the 0.60s for a 6-week time interval.

Social Support

Social support was measured using a modified version of the Social Support Questionnaire (SSQ), a 6-item tool designed to assess the number of perceived social support network members and the degree of satisfaction with that support. A sample item is "How satisfied are you with those whom you feel really appreciate you as a person?" In an attempt to limit the time required to complete the packet and increase the response rate, we administered the scaled items and did not use the open-ended component of the scale. Responses to the items were indicated on a 6-point scale of "very dissatisfied" (0) to "very satisfied" (6). Validity and reliability have been documented for the general population. Test-retest correlations have shown social support satisfaction to be quite stable for 2-month (0.86) and 5-month (0.67) time intervals.

Athletic Training Issues

To assess situational variables that can be viewed as stressful, we used the Athletic Training Issues Survey (ATIS), which is an adapted version of the Coaching Issues Survey (CIS). The CIS is a 30-item survey based on specific events that occur in the coaching profession. Modifications were made to specific items so that the questions were relevant to athletic training. Changes to the CIS were made with consent from the author. The author of the CIS evaluated the ATIS for face validity and determined that it was appropriate. For example, subjects are asked to respond on a 5-point Likert scale from "no stress" to "extreme stress" to items like "Not having time to myself," "Personality conflicts with the individuals I supervise," "Budget limitations hampering improvements, growth, and development," and "Inadequate travel budget for professional development." Although we modified the CIS, several items were somewhat less pertinent to athletic trainers, such as "Momentum turning against our school teams in contest" and "Handling defeat when our teams lose." Kelley and Gill and
Kelley have documented that CIS scores were correlated with perceived stress and a measure of teacher-role conflict (0.44).

**Perceived Stress**

Perceived stress was assessed using the Perceived Stress Scale (PSS). This 14-item scale measures thoughts and feelings about stressful events, control, overload, coping, and experienced stress, as well as how often the individual felt or thought in a stressful manner. The scale also allows for determining risk factors in behavioral disorders or disease, providing a closer look at the processes of stressor and pathology relationships, and may be implemented as an outcome variable. One such item is “In the last month how often have you dealt with irritating life hassles?” Responses are provided on a 5-point Likert scale anchored by “never” (0) and “very often” (4). Coefficient alpha reliability for the PSS was 0.84, 0.85, and 0.86 in 3 individual samples tested.

**Burnout**

The Maslach Burnout Inventory (MBI) is a widely accepted and widely used method to quantify burnout in the helping professions. It incorporates 3 subscales: emotional exhaustion, depersonalization, and personal accomplishment. The first, emotional exhaustion (EE), illustrates feelings of being emotionally overextended and exhausted by work. The second subscale, depersonalization (DP), describes a loss of concern for the people with whom one is working and an impersonal and unfeeling response toward them. The third scale is personal achievement (PA), which defines the feelings of accomplishment and a sense of competence about one’s job and a sense of self-appreciation for the successes achieved. Examples of items are “I feel emotionally drained at work” and “I feel frustrated by my job.” The assessment tool is based on a 7-point Likert scale from “never” (0) to “every day” (6). This inventory is scored by summing the items in each of the subscales; overall burnout scores are not calculated.

**Procedures**

After receiving approval from the Southeastern Louisiana University Institutional Review Board, we sent packets to 2 certified athletic trainers, chosen from the National Athletic Trainers’ Association 1998 Directory, at all 112 NCAA Division I-A universities that sponsored football (addresses obtained from the 1998–1999 NCAA Athletic Directory) (n = 224). Packets were addressed to the first full-time athletic trainer listed in the NCAA Athletic Directory and mailed in mid-November. Included within the packet were (1) a cover letter explaining the study, (2) a demographic sheet including specific items relevant to the athletic trainer’s years of service and workload, (3) the Hardiness Test, (4) the SSQ, (5) the ATIS, (6) the PSS, and (7) the MBI. The cover letter and the demographic sheet were presented first in the packet, with the remaining surveys in random order. Questionnaires were coded to identify the university only, and the confidentiality of each participant was assured in the cover letter. A self-addressed, stamped envelope was included in the packet in an attempt to increase response rate. Approximately 3 weeks after the mailing, we sent an e-mail message to the Athletic Training Listserv to increase response rate.

**Statistical Analysis**

To determine which personal or situational variables would predict stress appraisal, we computed Pearson product-moment correlations between perceived stress and the personal and situational variables of hardiness, social support, and athletic training issues. Pearson correlations were also computed to examine the relationships between perceived stress and the 3 burnout scores. To determine the relative contribution of the personal and situational variables to stress and the relative contribution of the 3 burnout dimensions to stress, we performed 2 stepwise multiple regressions.

**RESULTS**

**Descriptive Analyses**

Of the 224 certified athletic trainers who received a packet, 118 (52%) returned completed surveys ready for data analysis. Of this sample, 57% indicated that football was their primary sport (men, 49%; women, 8%), and 43% were primarily involved in other sports (men, 21%; women, 22%). The average age was 38.7 years for the football athletic trainers and 31.0 years for the nonfootball athletic trainers. Most of the athletic trainers had been employed for 2 to 5 years (45%); 14% had been employed for 6 to 10 years; 12%, for 11 to 15 years; and 11%, for 21 to 25 years.

Table 1 illustrates the means and standard deviations for hardiness, perceived stress, and the 3 dimensions of burnout for this sample of athletic trainers, along with previously reported data from other allied health professionals, coaches, and teachers. The athletic trainers tended to score higher on the hardiness scale than coaches and lower on perceived stress than coaches and coach-teachers. Emotional exhaustion scores were midrange for all occupations; however, personal accomplishment for athletic trainers appeared to score lower than for most other occupations. Depersonalization had the most variance among the occupations listed.

**Correlations Among Variables in the Proposed Model**

Pearson product-moment correlations demonstrated significant relationships among the variables measured. Individuals with higher levels of hardiness tended to have lower perceived stress ($r = -0.63$). The ATIS scores were positively related to perceived stress ($r = 0.45$), while the SSQ scores were negatively related to perceived stress ($r = -0.41$). Perceived stress was related to emotional exhaustion ($r = 0.59$) and depersonalization ($r = 0.43$) and negatively related to personal accomplishment ($r = -0.27$). These relationships supported the hypothesis developed from Smith’s model.

**Stepwise Multiple Regression Analyses**

The multiple $r$ provides an indication of the amount of variance in 1 set of variables that is accounted for by another set of variables. A minimum variance of 10% is considered significant and meaningful. We conducted 2 stepwise regression analyses. In the first, perceived stress was the criterion variable, and the predictor variables were hardiness, athletic training issues, social support, and sport (football or nonfootball). A significant overall multivariate effect was obtained:
Table 1. Means (Standard Deviations) of Related Populations on Hardiness, Perceived Stress, and Burnout

<table>
<thead>
<tr>
<th>Population</th>
<th>Hardiness Test</th>
<th>Perceived Stress Scale</th>
<th>Emotional Exhaustion</th>
<th>Depersonalization</th>
<th>Personal Achievement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Div. I-A ATCs</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Football</td>
<td>94.4 (9.6)</td>
<td>24.6 (6.2)</td>
<td>20.24 (9.03)</td>
<td>10.93 (6.24)</td>
<td>37.15 (6.16)</td>
</tr>
<tr>
<td>Nonfootball</td>
<td>94.3 (9.5)</td>
<td>23.8 (6.1)</td>
<td>20.06 (8.71)</td>
<td>8.44 (5.20)</td>
<td>38.82 (4.60)</td>
</tr>
<tr>
<td>Coaches*</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
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</tbody>
</table>

*Not determined.

A significant amount of perceived stress was explained by the personal and situational variables (Table 2). Hardiness was the first variable entered and explained 40% of the variance, followed by athletic training issues, which accounted for an additional 7% of the variance ($P < 0.0001$). Social support was the final construct to enter the equation, and the variance was raised 5% to a cumulative $r^2$ of 52% ($P < 0.0001$).

A significant amount of perceived stress was explained by the personal and situational variables. More specifically, the perception of more social support, limited athletic training issues, and higher levels of hardiness was associated with lower levels of perceived stress. However, perceived stress was not predicted by whether the athletic trainer was involved with football.

The second stepwise multiple regression analysis used the stress-appraisal component of perceived stress as the criterion variable and the burnout dimensions of emotional exhaustion, depersonalization, and personal accomplishment as the predictor variables (Table 2). The multiple $r$ was significant: $r = 0.59$, $F_{2,111} = 36.04$. Emotional exhaustion entered into the equation first and accounted for 35% of the variance, followed by personal accomplishment, which added 5% ($P < 0.005$). Depersonalization was not a significant variable in this analysis.

Table 2. Results of Stepwise Multiple Regression Analyses

<table>
<thead>
<tr>
<th>Personal/Situational Variables Predicting Perceived Stress</th>
<th>Variables</th>
<th>Multiple $r$</th>
<th>$r^2$</th>
<th>Beta</th>
<th>$F$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 1</td>
<td>Hardiness</td>
<td>0.638</td>
<td>0.408</td>
<td>-0.638</td>
<td>78.409*</td>
</tr>
<tr>
<td>Step 2</td>
<td>Athletic training issues</td>
<td>0.687</td>
<td>0.471</td>
<td>0.268</td>
<td>50.398*</td>
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<tr>
<td>Step 3</td>
<td>Social support</td>
<td>0.722</td>
<td>0.521</td>
<td>-0.233</td>
<td>40.565*</td>
</tr>
</tbody>
</table>

* $P < 0.0001$.

<table>
<thead>
<tr>
<th>Burnout Variables Predicting Perceived Stress</th>
<th>Variables</th>
<th>Multiple $r$</th>
<th>$r^2$</th>
<th>Beta</th>
<th>$F$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 1</td>
<td>Emotional exhaustion</td>
<td>0.588</td>
<td>0.346</td>
<td>0.588</td>
<td>60.261*</td>
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<tr>
<td>Step 2</td>
<td>Personal accomplishment</td>
<td>0.624</td>
<td>0.389</td>
<td>-0.210</td>
<td>36.040*</td>
</tr>
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</table>

* $P < 0.005$.

DISCUSSION

Historically, empirical progress has occurred when theoretical models guide research and the development of hypotheses. Our investigation was based upon the theoretical model of stress and burnout proposed by Smith to predict psychological burnout in the helping professions. Similar to other studies examining coaches and coach-teachers, we found support for Smith's theoretical model of stress and burnout in examining burnout in NCAA Division I athletic trainers.

Variables Related to Perceived Stress

The personal and situational variables we explored were hardiness, social support, athletic training issues, and working football or nonfootball sports. Our hypotheses proposed that individuals who reported lower levels of hardiness and social support and higher levels of athletic training issues would report higher levels of stress appraisal. These hypotheses were supported. Correlational analyses indicated significant rela-
tionships between these personal and situational variables and perceived stress.

Similar to the findings in other professions, hardiness was a significant predictor of perceived stress for athletic trainers. It is also a positive health influence for stressful experiences. Hardy individuals tend to have lower levels of stress and seem to thrive on stressful challenges, possibly because they have learned to cope effectively with stressors. Athletic trainers in this study tended to score higher on hardiness than coaches (Table 1). This suggests that athletic trainers tend to believe they have some control over their work situations and are likely to view problems as challenging rather than threatening. Although hardiness is a personal characteristic that varies with each individual, cognitive behavioral-intervention techniques may help individuals recognize the aspects of their work or life that they directly influence with their actions and give them a sense of purpose or direction. Goal-setting strategies can also be used to increase one’s level of hardiness.

Social support was negatively related to perceived stress, a finding that parallels previous research. Individuals reporting low social support tended to report higher levels of perceived stress, whereas athletic trainers with higher scores in social support, demonstrating satisfaction with their social support network, tended to report lower perceived stress scores. In similar studies involving coach-teachers, baseball and softball coaches, and teachers, those who reported low social support scores tended to develop poor lifestyle behaviors. Taylor et al have suggested that these individuals seek out new potential social resources and learn to effectively manage their stress. Camaraderie between teachers or coworkers has been shown to decrease the level of burnout one feels, and an active social life outside of the work setting may prevent or delay the onset of burnout.

Athletic trainers deal with a multitude of job responsibilities and relationships on a daily basis. The ATIS is designed to reflect these issues. In our study, the greater the number of issues an athletic trainer could identify with, the higher the stress reported. An occupational survey is exclusive to the sample of professionals it examines, and similar research using an issues survey has demonstrated support for the positive relationship between relevant professional issues and perceived stress. In research involving coaches, those concerned with coaching issues scored higher than those not influenced by that set of problems. Additionally, scores on the CIS were significantly related to perceived stress. Because of the unorthodox medical setting of athletic training, an examination of difficult professional issues could direct attempts at resolving the problems and providing a better work atmosphere.

Variables Related to Burnout Dimensions

The relationships between perceived stress and the dimensions of burnout has been investigated by multiple researchers. Several of these studies have examined the 3 dimensions of burnout and shown significant relationships between perceived stress and the 3 subscales in a wide array of professions. Doctors and mental health staff who scored high in emotional exhaustion were more likely to want to avoid people and evaluated clients more negatively over time, which is a precursor to depersonalization. Furthermore, when working in direct contact with students, teachers reported higher levels of emotional exhaustion than when some of their time is spent performing administrative duties. Athletic trainers can develop close friendships with their athletes and coaches and become emotionally involved in their lives.

We found perceived stress to be a significant predictor of emotional exhaustion. Emotional exhaustion has consistently been reported as a primary factor in burnout because of feelings of despair, isolation, exhaustion, and being overwhelmed. Interestingly, emotional exhaustion has been reported to be higher in older individuals and in females. Personal accomplishment was the other burnout dimension demonstrating a significant relationship to perceived stress. Athletic trainers in this study appeared to have scored slightly higher than many other professionals, demonstrating a relatively high sense of personal accomplishment in their work. Depersonalization was also significantly related to perceived stress. Athletic trainers appeared to score higher on this dimension of burnout than teachers in higher education, doctors, and nurses, but only slightly higher than coaches or coach-teachers (Table 1). This may occur because of the quantity of athletes athletic trainers are in contact with daily, the number of hours spent in the athletic training room, and the various professional relationships involved in the occupation (ie, with administrators, coaches, doctors, students, and colleagues).

Future Directions

Our study supports the theoretical model proposed by Smith and the hypothesized relationships between personal and situational variables and stress appraisal and stress appraisal and burnout. Other studies have also demonstrated support, however, the physiologic aspects of Smith's model have often been overlooked. Research has illustrated the detrimental effects of stress on the body, and future studies should address these effects as a component of burnout. A further examination of the physiologic symptoms may assist in detecting the stages of burnout so that intervention techniques can be implemented to prevent severe consequences. Most importantly, based upon the results of this study, we recommend that future research examining athletic trainers also use this theoretical framework to investigate the variables relating to stress and burnout. Such research will allow for more accurate comparisons between the samples.

Another recommendation for future research is to identify the potential stressor variables that athletic trainers at smaller colleges and universities encounter to better understand the stress and burnout at various collegiate levels. The sample of Division I-A institutions that sponsor football surveyed for this study are typically financially able to provide certified athletic trainers to service all the athletic teams the university sponsors. The smaller universities may expect the athletic training staff to serve the athletes without the necessary financial support.

A variable not addressed in this study is sex. Research has found that females and older individuals score higher in emotional exhaustion because they tend to be more nurturing toward those with whom they work. However, the respondents in this study who scored high in emotional exhaustion were male (70%) and middle aged (35.3 years old), contradicting responses seen in other studies. Previous research has also demonstrated that men tend to report slightly higher scores on depersonalization than women. Future studies should attempt to distinguish differences between male and female athletic trainers in all sports.
It has been suggested\(^8\),\(^9\),\(^{25}\) that the level of burnout in coaches and athletic trainers varies at different times of the season. Longitudinal studies need to be conducted across seasons to determine variances in burnout in athletic trainers. We attempted to survey athletic trainers at the end of the fall sport season, which can be a stressful time.

The anxieties of daily living can be overwhelming. Stress can have a negative effect on job performance, personal relationships, and individual personalities. Therefore, stress management is the key to coping with stress and the detrimental effects that accompany it. One’s level of hardiness, the satisfaction with a social support network, and the number of pertinent athletic training issues are just a few variables that predict stress appraisal, which then predicts burnout. Further research to understand burnout and effective coping strategies in athletic trainers is warranted.

ACKNOWLEDGMENTS

We thank Drs. Ronnie Harper and Linda Synovitz for their excellent comments on preliminary reviews of this manuscript. This study was funded in part by a “Mini-grant” from Southeastern Louisiana University, Hammond, LA. This study was conducted as part of the first author’s master’s thesis under the direction of the second author.

REFERENCES

Objective: To determine the most efficient burst duty cycle for eliciting an involuntary quadriceps femoris contraction in healthy subjects. This information will allow clinicians to make an informed decision about the optimal burst duty cycle based upon a specific treatment goal. The logical goal for such a treatment choice is to enhance motor unit recruitment in an effort to maintain postoperative or postinjury strength, when voluntary contractions may be less effective.

Design and Setting: Single-group and 5-measures design. All tests were performed in a university laboratory.

Subjects: Forty-eight healthy subjects (27 men and 21 women; mean age, 26.4 ± 8.5 years) performed a maximal voluntary isometric contraction (MVIC) on an isokinetic device and received neuromuscular electrical stimulation (NMES) at 5 different burst duty cycles.

Measurements: Subjects first performed an MVIC for knee extension on an isokinetic dynamometer at 60° of knee flexion. NMES surface electrodes were applied to the quadriceps muscle of each subject’s dominant leg. The values for the NMES were 2500-Hz carrier frequency, 50-bursts-per-second treatment frequency, amplitude increased to maximum tolerance, and burst duty cycle set to each of the 5 comparison values. The peak isometric force generated by each of the 5 nonvolitional contractions was recorded, along with the maximal charge per burst tolerated by each subject. Force generated was described as percentage of MVIC. Efficiency was the amount of force per burst charge.

Results: The mean MVIC achieved by the subjects was 553.8 newtons (N). The average force per burst charge generated at 10% burst duty cycle was 132.9 N; at 30%, 104.2 N; at 50%, 93.1 N; at 70%; 52.9 N; and at 90%, 41.3 N. The average efficiency (force per milliampere) at 10% burst duty cycle was the highest at 6.49 N/mC and at 90% was the lowest at 1.85 N/mC.

Conclusions: A burst duty cycle of 10% was the most efficient ratio of burst duration to interburst interval duration for eliciting the strongest muscle contraction. Use of this preferred duty cycle enabled only 8 of 48 subjects (16.6%) to achieve 60% MVIC within 1 trial in 1 session of NMES. Twenty-five subjects (52.0%) were able to achieve at least 10% of the MVIC in the same trial period.

Key Words: electrical neuromuscular stimulation, Russian NMES, burst duty cycle, quadriceps femoris, visual analog scale, MVIC
the tissue. Starkey,13 asserted that high duty cycles cause premature muscle fatigue due to the increased employment of the phosphocreatine energy system in the muscle. We expected that a high burst duty cycle would deliver increased charge per burst and would, therefore, evoke a stronger contraction and consequently fatigue the muscle more quickly than a low burst duty cycle.

We hypothesized that comparing 5 different burst duty cycles, ranging from 10% to 90%, as they induce a muscle contraction would best determine efficiency. That is, the burst duty cycle able to cause a strong contraction with the least amount of charge per burst delivered to the subject would be the most efficient. We selected the range of 10% to 90% to cover the available settings of many therapeutic stimulation generators currently in use or commercially available.

METHODS

Subjects

Subjects were 27 men and 21 women volunteers, with age ranging from 18 to 48 years (mean = 26.4 ± 8.6 years). No subjects had a history of dominant-leg hip or knee injury or contraindications to NMES. The dominant leg was determined by asking the subject which leg would be used to kick a ball. The research methodology was explained to all subjects, and each participant and a witness signed a consent form. At the time of the study, the university did not have an institutional review board; however, the study was reviewed and accepted by the research grant selection committee.

Instrumentation

We used the Kin-Com AP isokinetic dynamometer (Chattanooga Corp, Hixson, TN) to collect data. Patient-positioning techniques as provided by the manufacturer were strictly followed. The backrest angle was set at 78° and the seat angle, at 15°. The trunk-thigh depth was set at approximately 18 cm, as determined by femur length and knee joint axis alignment with the dynamometer. The subject’s hips were secured with a lap belt, and the dominant lower leg was secured to the double shin pad 3 cm proximal to the ankle joint. (This measurement ranged from 28 to 32 cm on the lever arm, depending on the subject’s tibia length.) The Kin-Com was set to isometric mode for purposes of data observation. Each subject was seated on the Kin-Com with the hips at 90° of flexion and the knees at 60° of flexion.6,11 The Kin-Com was set to the isometric mode for purposes of data observation. First, subjects completed 2 maximal voluntary isometric contractions (MVICs) for a duration of 5 seconds, each separated by 10 seconds of rest. The higher of the 2 values was recorded as the MVIC. This allowed for the calculation of 10% and 60% of the MVIC for the purpose of comparing the nonvolitional force to the voluntary force.

The sensations characteristic of Russian NMES, such as light tingling, pinpricking, and crushing pain, were then described to each subject. A sensory dose of current was delivered to provide increased understanding of electric stimulation and to minimize the apprehension of the subject. Next, the first of the 5 burst duty cycles was adjusted, the Intelect 900 was reset to 0 milliamps, and the subject was instructed to allow for a maximal involuntary contraction. A 0 to 5 visual analog pain scale (VAS) with descriptives ranging from “no pain” to “excruciating pain” was introduced to each subject. The subject was instructed to notify the investigator when he or she felt that the stimulation caused a perception of pain of at least 4 of 5 (“horrible”) on the VAS for each of the 5 burst duty cycles.

The amplitude of the first burst duty cycle was increased quickly while 1 investigator monitored each subject’s reaction and recorded the maximal amplitude of the stimulation from the stimulator readout. A second investigator monitored the Kin-Com display and recorded the peak force generated by the electrically induced contraction. Once the subject stated that a 4 of 5 on the VAS was achieved, current to the muscle was interrupted. The subject then rested for 10 seconds before the second burst duty cycle. This sequence was followed for each subject for each of the 5 burst duty cycles.

Data Analysis

The peak current reading of the Intelect 900 was converted from milliamps to milliCoulombs (mC) to compare charge per burst administered to the subjects rather than the effective root mean square (RMS) value obtained from the machine. This was accomplished through the use of formulas provided by Chattanooga Corp to be certain that calculations coincided with the programming of the Intelect 900. It should be noted that the value on the display of the Intelect 900 is the actual RMS current with the duty cycle taken into account. A summary of the formulas used to make the conversions follows:

\[
\frac{[\text{Intelect 900 meter reading}] \times \sqrt{\text{(duty cycle selected)}}}{\sqrt{2}} = \text{peak current of waveform} \quad (1)
\]

\[
[2 \times \text{(peak current of waveform)} \times \text{(duty cycle selected)}}]/\pi = \text{average current at the selected duty cycle}
\]

(also the total current in mC/s) \quad (2)
average current at selected duty cycle/number of bps

\[ = \text{charge per burst} \quad (3) \]

From the values calculated for charge per burst and the recorded MVIC figures, we determined the mean efficiency for each burst duty cycle by dividing the peak force generated by the charge per burst. Paired t tests were used to analyze whether a significant change occurred in NMES efficiency between each pair of burst duty cycles. Ten t tests in all were performed, so the confidence level was adjusted to \( \alpha = 0.005 \) to reduce the likelihood of type I error. In addition, a multivariate analysis of variance (MANOVA) was performed to determine whether a treatment effect was caused by the sequencing of burst duty cycles randomly assigned to the subjects.

RESULTS

The MANOVA revealed that no significant treatment effect occurred with relation to the 3 sequences (\( F_8 = 1.14, P \geq 0.342 \)). Observation of the mean efficiencies for each burst duty cycle indicated that the lowest burst duty cycle (10%) was the most efficient (strongest nonvolitional muscle contraction per mC). The 30% burst duty cycle was next, and so on, with the 90% burst duty cycle being least efficient. Table 1 displays the mean charges per burst, nonvolitional forces, and the percentage of the average MVIC for each burst duty cycle. Table 2 lists the mean efficiencies and standard deviations by burst duty cycle.

Significant differences between the means of the efficiencies occurred between each pair of burst duty cycles, except for 30% and 50% (\( P \approx 0.006 \)) and 70% and 90% (\( P \approx 0.043 \)) burst duty cycles.

For each burst duty cycle, the subjects who were able to tolerate an acceptable force were identified. An acceptable force was defined, for these purposes, as enough NMES to tolerate an acceptable force were identified. An acceptable force was defined, for these purposes, as enough NMES to achieve a nonvolitional force equal to 10% of the recorded MVIC. The number of subjects to achieve 60% of the MVIC was also calculated. Twenty-five subjects (52.0%) were able to reach 10% of the MVIC, while only 8 subjects (16.6%) achieved 60% of the MVIC at a burst duty cycle of 10%. Further results are summarized in Table 3.

DISCUSSION

The various values adjusted for this investigation include carrier frequency, treatment frequency, phase duration, amplitude, and burst duty cycle. For the purposes of this investigation, we considered only alternating current: current that changes phases from positive to negative cyclically. A phase is a given period of time in which the current is either positive or negative. A pulse accounts for 1 positive phase coupled with 1 negative phase and may also be referred to as 1 cycle. Frequency relates to the number of pulses or cycles that occur in a given period of time, usually 1 second. Phase duration is the period of time that the individual pulse is in the positive or the negative phase. This value, sometimes erroneously referred to as width, is a critical factor related to the physiologic effect of the stimulation and the comfort of the patient. Some alternating currents may be further divided into a carrier frequency and a treatment frequency. In such currents, a treatment frequency includes a series of pulses, combined by the stimulator device into what may be described as a burst of pulses separated by periods of no stimulation (interburst intervals). This effectively reduces the RMS of the stimulation and allows for a higher peak amplitude, or intensity, of the stimulus. In a given unit of time, there may be numerous bursts of pulses. The total number of pulses occurring per unit of time is the carrier frequency, and the total number of bursts per unit of time is the treatment frequency. Figure 1 provides a labeled representation of a Russian waveform incorporating a carrier and a treatment frequency.

Researchers and clinicians have attempted to adjust the various settings to control disuse atrophy and maintain muscle strength without overstimulating sensory fibers in the cutaneous layers. A fine line exists between selecting values that evoke a strong muscle contraction and those that minimize pain to the patient. Various stimulation waveforms have been developed in an attempt to fulfill both of these goals. One such waveform includes a symmetric, sinusoidal current with a 2500-Hz carrier frequency. The reciprocal of the carrier frequency determines the pulse duration and, in the case of a 2500-Hz waveform, the pulse duration is equal to 400 microseconds with a corresponding phase duration of 200 microseconds. The stimulation is modulated into 50 bursts (treatment frequency), each containing 50 pulses. Researchers have indicated that the optimal setting for the treatment frequency is similar to the pulse rate for other NMES devices (50 bps). Subsequent bursts are separated by interburst intervals with timing determined by the burst duty cycle. The interburst...

### Table 1. Charge Per Burst, Force, and MVIC % for Each Burst Duty Cycle*

<table>
<thead>
<tr>
<th>Burst Duty Cycle (%)</th>
<th>Average Charge Per Burst (mC)</th>
<th>Average Force Generated (N)</th>
<th>Percentage of Average MVIC (%)</th>
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<tr>
<td>10</td>
<td>5.0 (1.55)</td>
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<td>30</td>
<td>12.6 (4.08)</td>
<td>104.2 (144.17)</td>
<td>19</td>
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<td>50</td>
<td>20.1 (5.94)</td>
<td>93.1 (111.07)</td>
<td>17</td>
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<tr>
<td>70</td>
<td>26.5 (7.04)</td>
<td>52.9 (77.22)</td>
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<tr>
<td>90</td>
<td>33.1 (7.58)</td>
<td>41.3 (54.59)</td>
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*Data are given as mean (SD).

### Table 2. Average Efficiency* for Each Burst Duty Cycle

<table>
<thead>
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<th>Burst Duty Cycle (%)</th>
<th>Duty Cycle Efficiency (N generated per mC)</th>
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<tr>
<td>50</td>
<td>2.56 (2.68)</td>
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<tr>
<td>70</td>
<td>1.42 (1.90)</td>
</tr>
<tr>
<td>90</td>
<td>1.05 (1.44)</td>
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</table>

*Data are given as mean (SD).

### Table 3. Summary of Number of Subjects Able to Achieve 10% or 60% of MVIC at Each Selected Duty Cycle

<table>
<thead>
<tr>
<th>Burst Duty Cycle (%)</th>
<th>10% MVIC*</th>
<th>60% MVIC*</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>25 (52.0%)</td>
<td>8 (16.6%)</td>
</tr>
<tr>
<td>30</td>
<td>24 (50.0%)</td>
<td>3 (6.3%)</td>
</tr>
<tr>
<td>50</td>
<td>24 (50.0%)</td>
<td>1 (2.0%)</td>
</tr>
<tr>
<td>70</td>
<td>14 (29.1%)</td>
<td>0</td>
</tr>
<tr>
<td>90</td>
<td>15 (31.2%)</td>
<td>0</td>
</tr>
</tbody>
</table>

*Data are given as number (percentage of subjects).
Carrier frequency = 2500 pps, burst frequency = 50 bps

Figure 1a. Depiction of Effective Reduction of Root Mean Square of Burst Charges

Figure 1b. Waveform Components

Figure 1. Russian waveform.

intervals cause an overall reduction in the RMS of the current, allowing for a higher peak and subsequently a stronger muscle contraction.\textsuperscript{10} (Figure 2). The interval is too short for muscle relaxation, but does serve to alter the perception of the stimulus.\textsuperscript{10}

Such manipulation of the current is frequently termed “Russian” stimulation because of its development in the mid 1970s by Dr. Yakov Kots,\textsuperscript{14} who attempted to enhance the training of athletes in the Soviet Union. Additional development of the Russian stimulators gave rise to a clinician-selected burst duty cycle, which allows for further manipulation of the RMS of the stimulus delivered in a given treatment. The burst duty cycle is, simply stated, a form of current modulation that is a ratio of 1 time to the total cycle time.\textsuperscript{13} Figure 2 provides diagrams of the burst duty cycle. Including interburst intervals and using the duty cycle would seem to allow for a high peak current with less patient discomfort. However, as the current amplitude is further increased, the more powerful muscle contraction will eventually be perceived as painful.

During a review of the literature, we did not find a study similar to ours. Other researchers studying the use of NMES as a strengthening tool have recommended values for many of the settings, but have not indicated the optimal value for burst duty cycle for the application of Russian NMES (Table 4). This type of current is capable of producing a strong nonvolitional contraction with low to moderate discomfort.\textsuperscript{8,15} It was important to note the relative strength of the nonvolitional contractions and to select a baseline at which weaker contractions are not beneficial and stronger contractions are inefficient. Kots\textsuperscript{14} reported that subjects in his study were generating nonvolitional contractions in excess of 100% of the MVIC; however, those stimulation values have not been published. With the goal of increasing muscle strength, it is common practice to elicit the most powerful contraction possible within the comfort level of the athlete. Several researchers\textsuperscript{8,15} have identified 60% of the MVIC as the goal for such a contraction. However, Snyder-Mackler et al\textsuperscript{16} determined that training contraction intensities should not drop below 10% of the MVIC of the uninvolved quadriceps femoris muscles that is necessary to elicit a training effect that increases muscle force production. This statement provides further insight beyond the findings of Kots.\textsuperscript{14} The finding by Snyder-Mackler et al\textsuperscript{16} suggests that nonvolitional training is more useful than previously thought because attaining a lower MVIC is acceptable for gaining quadriceps femoris muscle strength.

Electrical stimulation is similarly applied at lower intensities for the retardation of disuse atrophy.\textsuperscript{17} It should be noted, however, that Snyder-Mackler et al\textsuperscript{18} stated in an earlier study that atrophy is not the major cause of diminished quadriceps femoris strength after anterior cruciate ligament rupture. Rather, the weakness is due to altered motor unit utilization.\textsuperscript{18} This would seem to change the logical treatment goal after anterior cruciate ligament repair from reducing disuse atrophy to increasing muscle strength and facilitating optimal motor unit recruitment. Consequently, if it is necessary to obtain a nonvolitional contraction force equal to or in excess of 60% of the MVIC to facilitate muscle strengthening, then some value other than burst duty cycle would need adjustment to cause such a force in athletes. Yet if a force generation of only 10% of the MVIC is needed, the values given are sufficient for
Figure 2. The 5 burst duty cycles. *IBI, Interburst interval.

Table 4. Suggested Values for Russian NMES for the Goal of Quadriceps Femoris Strengthening

<table>
<thead>
<tr>
<th>Selected Value</th>
<th>Suggested Setting</th>
<th>Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carrier frequency⁶,¹⁴</td>
<td>2500 Hz</td>
<td>Possibly blocks the superficial sensory fibers while stimulating the</td>
</tr>
<tr>
<td></td>
<td></td>
<td>deeper motor (and sensory) fibers</td>
</tr>
<tr>
<td>Burst frequency⁹,¹²</td>
<td>30 to 50 bps</td>
<td>Maintains a smooth tetanic contraction</td>
</tr>
<tr>
<td>Burst duty cycle</td>
<td>10%</td>
<td>Strong contraction with low total charge per burst</td>
</tr>
<tr>
<td>Pulse duration¹⁰,¹³</td>
<td>400 μs*</td>
<td>Reduces capacitative skin resistance; motor stimulation occurs at</td>
</tr>
<tr>
<td></td>
<td></td>
<td>lower amplitude</td>
</tr>
<tr>
<td>Amplitude¹²,¹³</td>
<td>Maximum tolerable (strong</td>
<td>Simulates the resulting contraction of volitional strengthening methods</td>
</tr>
<tr>
<td></td>
<td>visible contraction</td>
<td>except for type of fiber stimulated</td>
</tr>
<tr>
<td>Electrode placement¹¹,¹²</td>
<td>Over desired motor points</td>
<td>Produces optimal MVIC; improves comfort</td>
</tr>
<tr>
<td>Cycle time (on:off)⁹,¹²</td>
<td>10:50 s</td>
<td>Reduces the onset of fatigue or force failure from the carrier frequency</td>
</tr>
<tr>
<td>Treatment time⁸,¹²,¹⁷</td>
<td>10 cycles or fatigue</td>
<td>Effectively works muscle group to fatigue</td>
</tr>
</tbody>
</table>

*Pulse duration in Russian NMES is actually determined by the carrier frequency.

muscle strengthening. This assumes that healthy muscle will respond the same as muscle tissue after trauma or surgery.

An additional finding associated with the study related to the tolerance for NMES among the participants. Subjects who understood the basis for the clinical use of NMES were able to generate a higher percentage of the MVIC. Such participants included student athletes and athletic trainers, as well as those who more thoroughly questioned the basis of the investigation. This finding is supported in research conducted by Delitto et al.¹⁹,²⁰ who indicated that knowledge of the procedure increased subject cooperation and tolerance of the stimulation. Finally, it is worth noting that many of the participants reported symptoms of delayed-onset muscle soreness within 48 hours of completing the study. Alon¹⁰ suggested that this may indicate overstimulation. If the amplitude levels used in this investigation to achieve maximal approximation of the MVIC were so high as to produce microscopic tissue damage, then it may not be practical to rely on Russian NMES for muscle strengthening. We did not consider whether I particular burst duty cycle setting caused delayed-onset muscle soreness to result or if this effect was caused by an accumulation of intensities, as all of the burst duty cycles were tested consec-
utively. To correct for this factor, the burst duty cycles could be tested individually with a 3-day recovery period between test sessions. Alternatively, perhaps the treatment amplitude could be adjusted for consecutive treatments over several days to allow the muscle to adapt to the nonvolitional stimulation.

**Clinical Implications**

Our results suggest that the optimal selection for the burst duty cycle when using Russian NMES for muscle strengthening is 10% for healthy tissue. Of course, other settings must be considered if the clinician is to use the optimal settings for amplitude, pulse duration, carrier frequency, and electrode size and placement. Suggested values for Russian NMES with the goal of quadriiceps femoris strengthening are summarized in Table 4.

Admittedly, volitional contractions are likely more efficient at increasing strength and reducing atrophy than the use of external stimuli. Additionally, training isometrically at limited angles will not likely be as functional as training isotonically. This is due to the specificity principle, which implies that to gain isotonic strength, one must train isotonically. The data cited promote the use of nonvolitional contractions (NMES) during the inflammatory and fibroblastic phases of trauma or postsurgery, when the collagen matrix of the healing tissues is frail and vulnerable to reinjury, thereby necessitating an alternative approach for increasing strength or reducing atrophy.

**CONCLUSIONS**

Within the limitations of this study, we conclude that a burst duty cycle of 10% is the optimal setting in Russian NMES when the treatment goal is muscle strengthening. We acknowledge that differences exist between healthy subjects and those who are recovering from injury or surgery. We also note that this information may not be useful if exceeding 60% of the MVIC is absolutely necessary to achieve muscle strengthening. A recent investigation, however, supports the belief that only 10% of the MVIC is necessary to strengthen muscle. While 52% of our subjects achieved at least 10% of the MVIC, and 16% of them were able to reach 60% of the MVIC at 10% burst duty cycle, the amplitude was much higher than normal treatment levels and would be perceived as uncomfortable to most, if not all, athletes. With the existing knowledge of how to administer Russian NMES most effectively, the reader has useful information regarding the adjustment of the burst duty cycle to a value that allows for the closest approximation of the MVIC, thereby promoting efficiency and athlete safety.

**ACKNOWLEDGMENTS**

We acknowledge the duPont Summer Scholars Program at Wingate University for its generous support of the research project. In addition, we express gratitude to David Bley and Steve Brown of the Chattanooga Corp (Hixson, TN) and Stan Kowalski of TheraQuip (Greensboro, NC) for their technical assistance. Finally, we thank Dr. J.C. Andersen for his review of the initial manuscript.

**REFERENCES**


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**Objective:** To examine, evaluate, and summarize the techniques used to assess pain in all the Original Research articles published in the *Journal of Athletic Training* from 1992 through 1998. A second objective was to determine whether any of the Original Research investigations that did not assess pain were on topics that included a pain component. A third purpose was to make recommendations for assessing pain in a clinical athletic training setting.

**Data Sources:** Every Original Research article published from 1992 through 1998 was reviewed independently by 2 of the authors to determine whether a pain assessment was included in the investigation and, if so, to evaluate the pain assessment technique used.

**Data Synthesis:** A total of 23 (12.5%) of the 184 Original Research articles included some type of pain assessment. Most of these articles addressed the topics of delayed-onset muscle pain (43.5%), knee pain (17.4%), or pain resulting from cryotherapeutic procedures (17.4%). Most of the articles that included some type of pain measurement focused on the assessment of pain intensity using a category scale (17/23, 73.9%). In a substantial percentage of studies, a pain assessment tool that either lacked published supportive validity evidence (8/23, 34.8%) or was poorly constructed (because pain affect and pain intensity were confounded within a single scale) (7/23, 30.4%) was used. In a small number of articles on a topic directly relevant to pain (4/184, 2.2%), pain was not assessed, even though it could have provided useful information.

**Conclusions/Recommendations:** Pain is a construct of interest to those conducting athletic training research. Pain measures were included in approximately 1 of every 8 Original Research articles published in the *Journal of Athletic Training*. However, investigators have too frequently measured pain in a limited fashion, often focusing only on pain intensity. Measuring other components of pain could provide additional opportunities for learning more about the relationships between pain and athletic training procedures. We recommend that athletic trainers involved in research, as well as those engaged in clinical practice, consider systematically employing valid, multidimensional measures of pain to better understand the relationships between pain and athletic training outcomes.

**Key Words:** athletes, emotions, injury, pain, pain measurement

**Pain** is the principal symptom prompting athletes to seek medical attention. Reports about pain are a primary tool that athletic trainers use to assess injuries and monitor rehabilitation. Thus, it is important for athletic trainers to understand basic concepts about pain, to measure pain adequately, and to keep abreast of the sports medicine literature on pain, such as the recent studies documenting analgesia during and after both vigorous exercise and athletic competition.1–3

Adequate treatment and monitoring of rehabilitation require that pain be quantified. The measurement of pain seems simple at first: just ask the athlete where and how much it hurts. However, there is an emerging consensus that beyond pain intensity, useful information can be obtained in clinical settings by measuring pain in a multidimensional manner. This means, for example, obtaining reports not just about the location and intensity of the pain, but also about the cognitive and affective components of the pain. The most commonly used multidimensional pain measures, such as the McGill Pain Questionnaire4 or the Brief Pain Inventory,5 were designed primarily for use with chronic pain patients, not for athletes suffering from acute injuries. Also, the McGill Pain Questionnaire and the Brief Pain Inventory may be too lengthy to be of practical use by athletic trainers in a busy athletic training room or sports medicine clinic.

Since there appears to be no current consensus on the best method for measuring pain in collegiate athletic training settings, we thought that a useful first step in deciding what measures to consider using would be to systematically examine what athletic trainers conducting original research use to measure pain. To that end, we examined pain assessment methods reported in Original Research articles published in the *Journal of Athletic Training* from 1992 through 1998. Thus, 1 purpose of our present study was to summarize and evaluate the techniques used to assess pain in all the Original Research articles published in the *Journal of Athletic Training* from 1992 to 1998. A second purpose was to determine whether any of the Original Research investigations that did not assess pain were on topics that included a pain component. A third purpose was to recommend valid pain measurement tools for use in a clinical athletic training setting.
METHODS

Two of the authors (P.J.O. and R.M.M.) completed an independent, systematic search of the Methods and Results sections of every Original Research article published during the 7-year period from 1992 through 1998. Discrepancies about which papers were “related to pain but did not measure pain” emerged in the 2 independent search results. Subsequently, the raters jointly decided which papers best fit this categorization (ie, were related to pain), and cases with any uncertainty were removed from this category (ie, a conservative approach was taken).

RESULTS

A total of 23 (12.5%) of the 184 articles published in the Original Research section of the Journal of Athletic Training included some type of pain assessment. Most of these articles concerned the topics of delayed-onset muscle pain (43.5%), knee pain (17.4%), or pain resulting from cryotherapeutic procedures (17.4%). Two articles included a description of a method to measure pain, but then failed to report any pain data.6,7

Most of the studies that included some type of pain measurement focused on the assessment of pain intensity using a category scale (17/23, 73.9%). In a substantial percentage of the studies that included some type of pain measurement, a pain assessment tool that either lacked published supportive validity evidence (8/23, 34.8%) or was poorly constructed (eg, pain affect and pain intensity were confounded within a single scale or pain intensity and exercise behavior were confounded within a single scale) (7/23, 30.4%) was used. Pain assessment techniques with published supportive validity evidence that were used included the McGill Pain Questionnaire (4/23, 17.4%), the visual analogue scale (3/23, 13%), and pain ratings made in response to pressure algometry (1/23, 4.4%).

Investigators usually did not report in detail the instructions given, and in only a few instances were instructions on the use of the various pain scales described well enough to be replicated by others. In 1 case, pain data appeared to be misinterpreted by the authors: Byerly et al18 interpreted higher pain scores for a nonadherent group as reflecting more pain in the Discussion section, but Table 1 indicated that higher scores were indicative of a rehabilitation program being less painful.

A small percentage (4/184, 2.2%) of the total number of published Original Research articles were judged to be on a topic with direct relevance to pain (eg, patellofemoral syndrome or temperature changes with modalities such as ultrasound or cryotherapy), but did not include a measure of pain.

DISCUSSION

Our primary finding is that investigators publishing Original Research in the Journal of Athletic Training frequently measured pain in a noncomprehensive or inadequate way. One concern is the uncertain validity of some of the pain measures employed8-10; while some of these measures may possess face validity, such evidence is recognized as inadequate for establishing the validity of an instrument in a compelling way. A number of methods for assessing pain are available that have substantial published evidence to support their validity,10-13 however, many of the reviewed studies either did not use techniques with established validity or failed to provide a reference to such evidence.

A second concern is the over-reliance on measures of pain intensity alone. Pain experts have reached a consensus that pain involves more than the experience of a sensory intensity, and it also includes affective and cognitive components. The implication of this consensus is that pain should be measured in a multidimensional way.10-13 A simple approach involves adding a measure of pain affect (ie, how unpleasant the pain makes a patient feel or how much it bothers the patient). The distinction between pain intensity and pain affect is potentially important since some treatments (eg, antianxiety medications, such as diazepam, or hypnosis) influence affective responses to pain to a greater extent than they do pain intensity.10 Athletic trainers who obtain information about the affective component of pain might find athletes who report a low pain intensity, but who also indicate that the pain is highly unpleasant or bothersome. In other clinical settings, pain affect scores have been found to be better than pain intensity scores at identifying patients with comorbid psychological or psychiatric problems.14 Hence, the additional information about pain affect may help athletic trainers with certain treatment decisions; for example, whether it would be useful to refer an athlete for counseling to assist in the psychological adjustment to the injury. Moreover, it is possible that some athletic training procedures initially result in a rapid and dramatic improvement in the affective component of pain without a large change in pain intensity. This type of beneficial outcome may go unrecognized if pain affect is not measured. The affective component of the pain experience may be an important determinant of an individual athlete's rehabilitation behavior and outcome, and collecting data on this aspect of pain may help athletic trainers learn which aspects of pain are most important to monitor during rehabilitation. Most of the reviewed studies (73.9%) failed to use a multidimensional pain measure.

A third observation was that a small percentage (2.2%) of articles were on topics that, in our admittedly subjective yet purposefully conservative judgement, included a pain component, but the investigators failed to actually assess the pain component. In our view, an opportunity to gather potentially useful information is being lost in those few cases in which pain reports are not obtained in studies that address pain-related questions.

Although the most direct implication of our results is that athletic training researchers should consider including valid multidimensional pain measures as dependent variables in their research, the findings do have several clinical implications. The primary clinical implication is that it is useful for athletic trainers to measure pain in field settings as comprehensively and accurately as possible to provide additional information related to each athlete's injury. These data will provide a baseline from which to assess the efficacy of various interventions employed in the treatment of a variety of injuries and provide additional information with which to estimate the severity of injury. Also, by obtaining multiple measures of pain during the injury-healing process, the athletic trainer could evaluate treatment effectiveness and thus choose and modify modalities and other therapeutic measures used in the treatment program. Moreover, during the rehabilitation process, assessment of pain could lead practitioners to be more or less aggressive in their rehabilitation procedures. Thus, an opportunity to document the progress and effectiveness of athletic training treatments is being missed when athletic trainers do not include valid pain assessments in their documentation.
RECOMMENDATIONS

Based on this review, we recommend that investigators conducting athletic training research with a pain component (a) employ valid measures of pain in their experiments, (b) provide details about the instructions given so that others can replicate the work, and (c) consider obtaining more than a simple pain intensity measure. Some widely used research instruments that have published evidence supporting their validity as multidimensional measures of pain include the McGill Pain Questionnaire, the Brief Pain Inventory, and the Descriptor Differential Scale. Comprehensive texts are also available to assist investigators in deciding on the appropriate pain assessment tool for research purposes. Also, we recommend that athletic trainers in clinical practice consider always quantifying the location, intensity, and affective components of pain to monitor and document the efficacy of athletic training rehabilitation procedures. We present a suggested method for measuring these aspects of pain in a clinical athletic training setting in the Appendix.

APPENDIX

Suggested Pain Assessment Tools for Clinical Athletic Training Settings

Rationale. Pain is an internal event that cannot be directly observed. Consequently, assessment of pain is based on self-reports. The 3 most commonly measured aspects of pain are pain location, pain intensity, and pain affect. All 3 pain components are potentially useful to athletic trainers who wish to document treatment effectiveness. Strengths and limits are associated with any pain measure, and the following tools are suggested both because they possess published supportive evidence of validity and because of their simplicity and ease of use and scoring.

Pain Location. It is recommended that pain drawings be used to document the sensory distribution of the pain. One template for a pain drawing is illustrated (right), and athletic trainers can devise more detailed drawing of body areas, such as the shoulder or the knee, to suit their particular clinical needs. Regardless, patients are instructed to shade in the areas of their body that are “in pain.” Research with the drawing illustrated has shown that scores equal to the total number of regions shaded by patients are correlated with key pain-related behaviors such as medication use and time spent being inactive. These scores also have been shown to be independent of ratings of pain intensity and affect. More information about the drawings illustrated here can be obtained from Margolis et al.

Pain Intensity. The 0 to 10 (11-point) Numerical Graphical Rating Scale is recommended to assess pain intensity. Athletes should be instructed to rate the intensity of their pain on a 10-centimeter horizontal scale as illustrated (right). Athletes should be instructed that 0 represents “no pain” and that 10 represents the “highest possible pain intensity.” For use in clinical athletic training settings, we recommend this intensity scale over the commonly used visual analogue scale because the numerical format makes it easier for athletes to use and athletic trainers to score. More information about this scale can be obtained from Turk and Melzack.

Pain Affect. The 0 to 10 (11-point) Numerical Graphical Rating Scale is also recommended to assess pain affect. Athletes should be instructed to rate the degree to which their pain is unpleasant on a 10-centimeter horizontal scale as illustrated (below). Athletes should be instructed that a score of 0 means that the pain is “not unpleasant” and that a score of 10 means that the pain is “as unpleasant as possible.” If athletes have difficulty distinguishing between pain intensity and unpleasantness, they can be told that the task is similar to listening to music and reporting both how loud the music is (ie, how intense) and how it makes them feel (ie, good or bad, pleasant or unpleasant). More information about this scale can be obtained from Turk and Melzack.

Clinical Pain Assessment Tool

Where Does It Hurt? Draw on the figure to indicate the locations on your body where you currently feel pain.


How Much Does It Hurt? Use the scale below to indicate the intensity of the pain you are feeling. A score of 0 represents “no pain” and a score of 10 represents the “highest possible pain intensity” that you can imagine.

How Unpleasant Is The Pain? Use the scale below to indicate how much the pain is bothering you. A score of 0 represents “no unpleasantness” and a score of 10 represents a pain that is “as unpleasant as possible.”
REFERENCES


A Comparison of Sideline Versus Clinical Cognitive Test Performance in Collegiate Athletes

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Objective: To test whether performance on 5 cognitive tests administered in a controlled clinical environment differed compared with administration in an uncontrolled sideline environment. Additionally, we investigated the effect of testing environment on the learning effect for each cognitive test.

Design and Setting: Athletes were assessed on 2 test occasions (8 ± 2 days apart), once in a sports medicine research laboratory and once on a lacrosse practice field site.

Subjects: A total of 59 Division I collegiate student-athletes participated in this study.

Measurements: Normative data were collected on 5 cognitive tests (Stroop Test, Trail-Making Test part A, Trail-Making Test part B, Wechsler Digit-Span Forward Test, and Digit-Span Backward Test).

Results: An independent-samples t test for environment difference on test day 1 revealed no significant differences between tests performed in the controlled environment and those performed in the uncontrolled environment. A repeated-measures analysis of variance test revealed a significant learning effect for all 5 tests, as subjects tended to improve approximately 11 points on the Stroop Test, 3 seconds on the Trail-Making A Test, 7 seconds on the Trail-Making B Test, and 1 point each on the Wechsler Digit Span Forward and Backward Tests. A paired-samples t test using delta scores (first test minus second test), sorted by order of testing environment, revealed a significant difference for the Stroop Test, but not for the remaining cognitive tests.

Conclusions: There appears to be no difference in cognitive testing performance completed in a controlled clinical environment versus that performed in an uncontrolled sideline environment. This finding suggests that clinicians can administer cognitive tests to athletes with mild head injuries in uncontrolled sideline environments and expect valid results. Thus, clinicians can more thoroughly evaluate mildly head-injured athletes during the most crucial period after injury so that a safe return-to-play decision can be based on quantifiable, objective data.

Key Words: concussion, mild head injury, neuropsychological testing

Athletic trainers and other sports medicine personnel are constantly faced with the challenge of deciding when an injured athlete should return to competition, and perhaps the toughest situation involves an athlete with a mild head injury (MHI). The incidence of repeated concussions and the long-term sequelae that follow have been topics of considerable debate in the sports medicine literature.1-6 The National Athletic Trainers’ Association studies1,5 of high schools in 1986–1988 and 1995–1997 revealed national estimates of approximately 40 000 concussions in football players annually, while another study6 of the incidence of MHI in collegiate and high school football showed a 5.1% incidence rate and a 14.7% recurrence rate in the same seasons. However, these statistics do not include the number of head injuries that go unrecognized or unreported; thus, the clinician performing the initial evaluation must always be aware that an individual may have had a previous MHI that went undetected, resulting in increased susceptibility to serious complications from MHI.7

MHI assessment presents a unique situation because of the difficulty in gathering quantifiable, objective information during an immediate sideline evaluation. The sideline management of mild head injuries has long relied on subjective information such as headache, dizziness, and blurred vision. Unfortunately, these symptoms are often not reported by the athlete; therefore, this method of evaluation has been criticized for lacking objectivity. The use of neuropsychological cognitive testing to objectively assess an athlete with MHI has recently come to the forefront, yet the focus of this testing has been on the follow-up evaluation for return to competition, ie, 1 day, 1 week, or 1 month postinjury.3,8-22

The typical sideline evaluation consists of assessing orientation to time, place, person, situation, and simple memory and concentration tests.1-3,17-19 The fact that normative baselines may not be established for each individual athlete or for entire groups of athletes makes rating difficult. Deciding when an athlete who has possibly sustained an MHI should return to competition is normally a judgment decision made by sports medicine personnel. If normative cognitive baselines are established for individuals and groups of athletes, a more objective decision can be made, and athletes can be returned safely to competition.

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Baseline neuropsychological testing is becoming increasingly popular among high school, collegiate, and professional sports medicine personnel. This type of preseason quantifiable data may aid the clinician in making return-to-play decisions after MHI. Yet neuropsychological tests are normally given in a quiet, controlled, clinical environment, while the typical MHI evaluation occurs on the sideline, during an athletic practice or contest. The need for normative sideline neuropsychological baselines may be imperative because the effects of fatigue, motivation to return to competition, noise, and other distractions may alter an athlete’s neuropsychological test performance. The comparison of sideline scores to clinical scores may result in a normal individual’s being labeled as deficient in cognitive capacity, which may be the result of testing environment conflicts rather than actual cognitive impairment. Thus, it is important to investigate the effect of environment on normative baseline neuropsychological testing so that the certified athletic trainer and team physician can use this information in making a sideline decision regarding immediate return to play.

The main purpose of our study was to investigate if there was a significant difference between the scores of cognitive tests administered to subjects in a controlled clinical environment to scores administered in an uncontrolled sideline environment. We assessed the need to establish different normative baseline values to be used for clinical testing (controlled environments) versus sideline testing (uncontrolled environments). Additionally, we looked at the effect of testing environment order on the learning effect for each cognitive test.

METHODS

A total of 59 Division I college student-athletes from the University of North Carolina at Chapel Hill were recruited from the men’s and women’s lacrosse teams: 39 men (age = 19.80 ± 1.20 years) and 23 women (age = 19.30 ± 1.29 years). Student-athletes who had sustained a head injury within the last 6 months were excluded from this study. Also, any athlete who had undergone any type of neuropsychological testing within the last 6 months or had a known learning disability, color-vision disorder, or color blindness was excluded from this study. We obtained advance permission from each of the team’s head coaches and thoroughly discussed the procedures for the clinical and sideline evaluations. All subjects were informed of the testing procedures and were asked to sign an informed consent form in accordance with the Human Subjects Committee at the University of North Carolina at Chapel Hill. The study was approved by the Academic Affairs Institutional Review Board at the University.

Half the subjects on each team were randomly placed in group 1 (controlled clinical environment first), while the other half were placed in group 2 (uncontrolled sideline environment first). Subjects scheduled a time to take the test battery and were assigned, then retested 8 ± 2 days later in the other environment. The controlled clinical environment tests were performed in the Sports Medicine Research Laboratory, with only the researcher and the subject present in the room. All outside noises, distractions, and interferences were kept to a minimum. The uncontrolled sideline environment involved testing subjects on the sideline during practice; no attempt was made to control for noises, distractions, or interferences.

During each test session, subjects were asked to complete trial each of 5 neuropsychological tests, lasting approximately 10 minutes total. The tests were administered in a set order to minimize the effects of testing fatigue on the individual: Stroop Test (cognitive flexibility and attention span), Trail-Making Tests A and B (orientation, concentration, visual-spatial capacity, and problem-solving abilities), Wechsler Digit-Span Forward and Backward Tests (WDSFT and WDSBT, respectively; attention span, concentration, distractibility, and immediate memory recall), Scoring for the Trail-Making Tests A and B was modified slightly. This test is traditionally scored by recording the total time taken to complete each test, and separate time totals and error totals are then calculated. We added 1 second per error (eg, not touching the circled item or connecting the wrong sequence). The purpose of this modification was to combat the extreme competitiveness of the athletes, who tended to finish quickly and to disregard the directions to properly touch the circled items or to carry on a wrong sequence to achieve a better time.

RESULTS

Mean test scores, standard deviations, and ranges were calculated for all 5 tests on each of the testing sessions. An independent-samples t test was performed with scores for all tests taken during the controlled clinical environment and the uncontrolled sideline environment to evaluate for a significant difference between testing environments. We performed a repeated-measures analysis of variance for each test to detect any significant differences between the learning effects for the 2 groups. Additionally, a paired-samples t test using delta scores (first test minus second test), sorted by order of testing environment, was performed to determine if a significant change in the learning effect occurred as a result of the environment order. Data were organized and analyzed with SPSS for Windows, version 6.1 (SPSS Inc, Chicago, IL).

Mean values and standard deviations were calculated for each of the 5 cognitive tests when sorted by testing order (Table 1) and by test day (Table 2). An independent-samples t test for environment difference on test day 1, with an alpha level of 0.05, was carried out. None of the analyses revealed a significant difference in cognitive testing compared with those performed in the uncontrolled environment (P > .05).

A repeated-measures analysis of variance revealed a significant learning effect (P < .05) for all 5 scores: subjects tended to improve approximately 11 points on the Stroop Test, 3 seconds on the Trail-Making Test A, 7 seconds on the Trail-Making Test B, and 1 point each on the WDSFT and WDSBT (Table 3).

We performed an additional analysis to determine if a change in the learning effect occurred as a result of the environment order. A paired-samples t test using delta scores revealed a significant difference only for the Stroop Test (P < .05) (Table 4). No significant differences were seen with any of the other cognitive tests. As for the Stroop Test, subjects tested first in the controlled environment demonstrated a significant difference in improvement between tests compared with subjects tested first in the uncontrolled environment (Figure).
Table 1. Mean Test Scores (SDs) for the 5 Cognitive Tests on Test Days 1 and 2 Sorted by Group

<table>
<thead>
<tr>
<th>Group</th>
<th>Day</th>
<th>Stroop*</th>
<th>Trail A*</th>
<th>Trail B*</th>
<th>WDSFT*</th>
<th>WDSBT*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controlled</td>
<td>1</td>
<td>236.15 (27.99)</td>
<td>23.42 (5.12)</td>
<td>47.85 (12.35)</td>
<td>9.73 (1.48)</td>
<td>7.77 (1.99)</td>
</tr>
<tr>
<td>Clinical Environment First</td>
<td>2</td>
<td>256.35 (30.40)</td>
<td>19.57 (4.49)</td>
<td>42.64 (12.47)</td>
<td>10.38 (1.36)</td>
<td>8.12 (2.10)</td>
</tr>
<tr>
<td>Uncontrolled</td>
<td>Delta</td>
<td>20.19 (12.17)</td>
<td>3.85 (5.11)</td>
<td>5.21 (13.17)</td>
<td>0.65 (1.44)</td>
<td>0.35 (2.02)</td>
</tr>
<tr>
<td>Sideline Environment First</td>
<td>1</td>
<td>247.52 (26.85)</td>
<td>21.91 (5.03)</td>
<td>51.10 (17.69)</td>
<td>9.48 (1.52)</td>
<td>7.42 (1.89)</td>
</tr>
<tr>
<td></td>
<td>Delta</td>
<td>250.79 (33.71)</td>
<td>20.02 (5.08)</td>
<td>42.34 (15.06)</td>
<td>10.03 (1.63)</td>
<td>8.70 (2.11)</td>
</tr>
</tbody>
</table>

* Mean represents the total number of correct responses in 3 45-second trials.
† Mean represents the total number of seconds to complete the task.
‡ Mean represents the total number of correct sequences of digits recalled.

Table 2. Mean Test Scores (SDs) for the 5 Cognitive Tests on Test Days 1 and 2 Sorted by Day (Environments Combined)

<table>
<thead>
<tr>
<th>Test</th>
<th>Test Day</th>
<th>Stroop*</th>
<th>Trail A*</th>
<th>Trail B*</th>
<th>WDSFT*</th>
<th>WDSBT*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stroop</td>
<td>Day 1</td>
<td>242.51 (27.71)</td>
<td>22.57 (5.09)</td>
<td>49.67 (15.53)</td>
<td>9.59 (1.50)</td>
<td>7.58 (1.92)</td>
</tr>
<tr>
<td></td>
<td>Day 2</td>
<td>253.24 (32.15)</td>
<td>19.82 (4.79)</td>
<td>42.47 (13.86)</td>
<td>10.19 (1.51)</td>
<td>8.44 (2.11)</td>
</tr>
</tbody>
</table>

* Mean represents the total number of correct responses in 3 45-second trials.
† Mean represents the total number of seconds to complete the task.
‡ Mean represents the total number of correct sequences of digits recalled.

Table 3. Repeated-Measures Analysis of Variance F Values Indicating Significant Learning Effects Between Test Days

<table>
<thead>
<tr>
<th>Test</th>
<th>F_{1,56} Value</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stroop</td>
<td>24.14</td>
<td>0.000*</td>
</tr>
<tr>
<td>Trail-Making A</td>
<td>21.09</td>
<td>0.000*</td>
</tr>
<tr>
<td>Trail-Making B</td>
<td>21.33</td>
<td>0.000*</td>
</tr>
<tr>
<td>WDSFT</td>
<td>10.36</td>
<td>0.002*</td>
</tr>
<tr>
<td>WDSBT</td>
<td>12.12</td>
<td>0.001*</td>
</tr>
</tbody>
</table>

* Values of P < 0.05 were considered significant.

DISCUSSION

The return-to-play decision after an MHI is one of the most difficult decisions for a certified athletic trainer or team physician. Sports neuropsychological testing has come to the forefront of MHI research in terms of looking at preseason baseline normative values to quantifiably compare data, yet the results may be skewed in an on-the-field sideline evaluation situation due to testing environment circumstances.14 The purpose of our study was to determine whether scores on 5 neuropsychological tests (Stroop Test, Trail-Making Tests A and B, and Wechsler Digit-Span Forward and Backward Tests) administered in a controlled clinical environment differed when compared with tests administered in an uncontrolled sideline environment. Our most important finding was that there appears to be no significant difference in cognitive testing performance completed in a controlled clinical environment versus that performed in an uncontrolled sideline environment. This finding may be invaluable for future MHI evaluation, since we found no adverse effect of environment on cognitive testing abilities, which may allow clinicians to administer cognitive tests (traditionally administered in controlled laboratory environments) in uncontrolled sideline environments with more confidence.

The establishment of preseason baseline data for each athlete is needed to make a sound judgment regarding cognitive status, since people often vary in cognitive abilities.14,29 The establishment of preseason data allows clinicians to have quantifiable baselines as reference points so that sound judgments can be made during the return-to-play decision-making process. We found no differences in 5 cognitive tests performed in controlled clinical situations as opposed to uncontrolled sideline situations; thus, there is no apparent need to establish
Another finding of this study was the occurrence of a significant learning effect for each of the 5 cognitive tests across test days. A repeated-measures analysis of variance revealed a significant \( P < 0.05 \) learning effect 8 days later for all 5 scores, as subjects tended to improve approximately 11 points on the Stroop Test, 3 seconds on the Trail-Making Test A, 7 seconds on the Trail-Making Test B, and 1 point each on the WDSFT and WDSBT. This finding, which is consistent with previously reported research, offers valuable information, since MHI assessment requires constant re-evaluation. This finding allows clinicians who use cognitive testing to assess normal function to expect learning or improvement during follow-up assessment. The question still remains as to how much time between tests would negate this learning effect. Clinicians should suspect lingering pathology if there is no improvement following initial testing.

We performed further analysis on the learning effect to determine if a change had occurred as a result of the testing environment order. A paired-samples \( t \) test using delta scores (first test minus second test), sorted by order of testing environment, revealed a significant difference \( P < 0.05 \) only for the Stroop Test. The learning effect for the Stroop Test remained normal for the subjects tested first in the controlled clinical environment, yet there was less of an observed learning effect for those subjects tested first in the uncontrolled sideline environment. Table 1 reveals that the Stroop Test for day 1 scores for both groups are comparable with those found in Oliaro et al, but a comparison of day 2 scores revealed a significantly lower comparison than in Oliaro et al’s study, which was performed on a similar population group in a controlled clinical environment only. Oliaro et al reported that an improvement of approximately 20 points can be expected from day 1 to day 2 scoring on the Stroop Test, consistent with our group that tested first in the controlled clinical environment. However, the group that tested first in the uncontrolled sideline environment revealed only a 3-point average improvement on the Stroop Test. We found that a learning effect is still present 8 ± 2 days later, but that effect may be significantly decreased on the Stroop Test if subjects are tested first in an uncontrolled sideline environment. A possible explanation for this is that the learning effect in the uncontrolled environment may not be as drastic as that in the controlled environment for the Stroop Test because of possible deleterious effects caused by the environment when first taking the test in a distraction-based situation. A better learning strategy for the Stroop Test most likely occurs in the controlled clinical environment that allows the athlete first tested in the controlled environment to perform subsequent follow-up tests better than the athlete first tested in the uncontrolled environment. Perhaps this is due to the fact that environmental distractions may cause a difference in the Stroop Test learning strategy. This additional finding regarding environment testing order concerning the Stroop Test may result in caution when evaluating the learning effect of the Stroop Test when first performed in an uncontrolled sideline situation or a controlled clinical situation. Future testing should focus on the Stroop Test and whether this environment learning phenomenon occurs consistently.

Future research should also be directed at collecting data on a larger number of collegiate athletes in the preseason and then comparing established baselines with those in athletes with recent MHI episodes. The use of cognitive
tests in preseason screening has already been established by numerous organizations, yet there is a substantial need for additional studies comparing baseline and immediate post-MHI evaluation. 19–22, 36 Baseline return-to-play criteria may be established using normal and MHI subjects, thus allowing clinicians the opportunity to review normative versus MHI data. The collection of future data involving collegiate athletes, high school athletes, and professional athletes may allow us to establish general normative baselines for each level of play and each individual sporting event. As stated previously, though, cognitive baselines often vary among individuals; thus, the establishment of performance trends and preseason baselines for individual athletes at risk of MHI is much more useful than overall group normative baseline scores. Ideally, all athletes would have a preseason baseline screening, but this will not always be the case. Therefore, the establishment of some normative data will at least provide comparison scores to make immediate, objective return-to-play decisions. Additionally, this study relied on practice conditions only. It may be helpful to investigate whether game conditions are significantly more distracting than practice conditions, with a resultant impact on cognitive testing performance.

CONCLUSIONS

Our most important finding was no apparent difference in cognitive testing performance in a controlled clinical environment versus performance in an uncontrolled sideline environment for 5 specific cognitive tests. The sole significant difference was that of the testing environment order learning effect for the Stroop Test. This finding may be beneficial for future MHI evaluation; we found no adverse effects for environmental differences on various cognitive testing abilities. Of more importance is the suggestion that clinicians can administer 5 specific cognitive tests in a setting that does not have to be highly controlled, with a note of caution about the variation in the learning effect for testing environment order for the Stroop Test. This finding will allow easier administration of cognitive tests during an acute, initial, uncontrolled sideline environment situation that does not have to be highly controlled, with a resultant impact on cognitive testing performance.

REFERENCES


Injuries and Illnesses in the National Basketball Association: A 10-Year Perspective

Chad Starkey, PhD, ATC
Northeastern University, Boston, MA

**Objective:** To present an overview of the medical conditions experienced by athletes competing in the National Basketball Association (NBA) from the 1988–1989 through the 1997–1998 seasons.

**Design and Setting:** Athletic trainers completed profiles that provided demographic information for each player. Injury reports indicated when and where the injury occurred, pathology, onset, activity, and the mechanism of injury. The amount of time lost, injured list status, hospitalization, and surgery were also reported. Reportable injuries were those that resulted in (1) physician referral, (2) a practice or game being missed, or (3) emergency care being rendered.

**Subjects:** A total of 1094 players appeared in the database 3843 times (mean, 3.3 ± 2.6 seasons). Mean player demographics were age 26.7 (±3.7) years, NBA playing experience 4.1 (±3.7) years, height 200.8 (±9.9) cm, and weight 100.2 (±13.5) kg. Players averaged 52 (±34.7) games and 1263.1 (±1073.8) minutes played.

**Measurements:** The frequency of injury, time lost, and game exposures were tabulated, and game-related injury rates were then calculated.

**Results:** Ankle sprains were the most frequently occurring orthopaedic injury (942, 9.4%), followed by patellofemoral inflammation (803, 8.1%), lumbar strains (491, 5.0%), and knee sprains (258, 2.3%). The greatest number of days missed were related to patellofemoral inflammation (7569, 11.5%), knee sprains (5712, 8.6%), ankle sprains (5122, 7.7%), and lumbar strains (3365, 5.1%).

**Conclusions:** Professional athletes in the NBA experience a rate of game-related injuries that is twice as high as their collegiate counterparts. Patellofemoral inflammation is a significant problem among NBA players.

**Key Words:** epidemiology, professional sports, ankle sprains, knee sprains

The nature of the game of basketball has changed dramatically over the years, evolving from a game of finesse to a collision sport to its current designation as a high-risk contact sport. The original concept for basketball was to avoid the fast pace of football, and it was based on the premise that “if the offense did not have the opportunity to run with the ball, there would be no necessity for tackling and we would thus eliminate roughness.”1 This premise was reinforced by a rule stating that any form of physical contact would result in the player’s being removed from the contest, without a substitute, until the next basket was scored, similar to the penalty box in ice hockey.

The contemporary game of basketball emphasizes the speed and power of its competitors. The strength and quickness necessary to control an opponent’s position, “muscle” a rebound, or “power” a shot are all prerequisites for a successful basketball career. Although the changes in the game of basketball can be seen at all levels, the changes are generally acknowledged to have had the greatest impact at the professional level. Athletes in the National Basketball Association, Inc (NBA) participate on a longer court, for a greater number of minutes per game, more times per week, for a longer season, and are older than their collegiate counterparts. Both the frequency and the intensity of the competition expose these athletes to potentially injurious forces across their 9-month season. My purpose is to present a normative overview of the injuries and illnesses seen by athletic trainers and team physicians in the NBA and to compare game-related injury rates with those seen in college basketball.

**METHODS**

**Subjects**

From 1988 through 1997, 86% of the 29 NBA teams complied with the study on an annual basis, with each team averaging 36.7 (±18.2) reports per year. (There were 25 NBA teams during the 1988–1989 seasons. The league added 2 expansion teams at the start of the 1989–1990 seasons and 2 additional teams at the start of the 1995–1996 season.) A total of 1094 individual players were included in this study, representing approximately 85% of the players who were on a regular-season NBA roster during this time (Table 1). Twenty-three players (2.1%) appeared in the database for each of the 10 seasons included in this study, and the average player was recorded in the database for 3.3 (±2.6) seasons, accounting for a total of 3843 player entries in the database. Of the 3843 player entries, 728 (18.9%) players were listed as centers, 1565 (40.7%) as forwards, and 1550 (40.3%) as guards, although the

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Table 1. Player Demographic Information

<table>
<thead>
<tr>
<th></th>
<th>Minimum</th>
<th>Maximum</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>18.0</td>
<td>43.0</td>
<td>26.7</td>
<td>3.7</td>
</tr>
<tr>
<td>Playing experience (years)</td>
<td>0.0</td>
<td>20.0</td>
<td>4.1</td>
<td>3.7</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>160.0</td>
<td>231.1</td>
<td>200.8</td>
<td>9.9</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>60.5</td>
<td>155.9</td>
<td>100.2</td>
<td>13.5</td>
</tr>
<tr>
<td>Games played</td>
<td>0.0</td>
<td>938.0</td>
<td>52.0</td>
<td>34.7</td>
</tr>
<tr>
<td>Minutes played</td>
<td>0.0</td>
<td>5895.0</td>
<td>1263.1</td>
<td>1073.8</td>
</tr>
</tbody>
</table>

contemporary game of professional basketball tends to blur these distinctions.

Procedures

The National Basketball [Athletic] Trainers’ Association (NBTA) maintains a database of all injuries and illnesses occurring to NBA players that (1) required physician referral or prescription medication, or both; (2) resulted in a practice or game being missed; or (3) caused emergency care to be rendered to the athlete. These records are based on a standard-ized league-wide injury-reporting instrument that is completed by the team’s athletic trainer and cosigned by the team physician. The primary information collected includes the player’s identification number, when and where the injury occurred, the specific pathology, and the onset, activity, and mechanism of the injury. Data regarding the number of practices and games missed, ankle support worn, injured reserve list status, hospitalization, surgery, and medication data were also collected.

A “Guide to Reporting” that describes the administrative aspects of this injury surveillance program, a definition of terms, and the reporting time table was distributed to each team’s head athletic trainer. Reports were submitted monthly from the start of preseason camp to the end of the postseason.

Before the start of preseason camp, each team returned a Player Profile detailing the position, height, weight, age, number of years of NBA playing experience, and permanent identification number for each member of the team. Updates to this document were made throughout the season as players were added to a team’s roster. At the end of the regular and postseasons, exposures for games and playing minutes for the 1988–1989 through the 1996–1997 seasons were acquired from the NBA’s official statistical publication, The NBA News (New York, NY). At the start of the 1997–1998 season, the NBA’s web site (http://www.nba.com) became the League’s official source of statistical information. At the end of each season, the Player Profile database was linked to the injury database to provide exposure and demographic information.

Because of the lack of a consistent and reliable method of calculating practice and preseason game exposures, I did not include incidence rates for participation in these activities. Frequency data for these injuries and illnesses were, however, still collected and tabulated.

Data Analysis

I analyzed data reported during the 1988–1989 through the 1997–1998 basketball seasons for league-sanctioned activities from approximately October 1 through June 15. Frequencies, means, standard deviations, and frequency rates were calculated using SPSS-PC, version 9.0 (SPSS, Inc, Chicago, IL). Incidence rates were calculated using Microsoft Excel 97 (Microsoft Corp, Redmond, WA).

Two methods were used to calculate game-related injury rate. To compare these data with research performed by the National Collegiate Athletic Association (NCAA) and other similar studies, injury rates were calculated per 1000 athlete exposures (AEs). This method describes the total number of athletes appearing in regular-season and postseason games whereby 1 athlete appearing in 1 game equals 1 AE (during a single game, the maximum AE would be 24 if all 12 players from each team participate in the contest). Incidence rates for AEs were calculated using the formula: number of injuries (group)/total game exposures (group) × 1000.

Although AE is a common method for determining injury rates, it lacks sensitivity because it does not take into account the length of time the player was actually participating. A player appearing in a game would record 1 AE, whether he played for 1 minute or 48 minutes. Each regulation game represents 480 total minutes of competition (10 players on the court for 48 minutes), and 10000 competition minutes (CMs) would then represent 10 players competing in 20.8 regulation games. Incidence rates for CMs were calculated using the formula: number of injuries (group)/total game minutes played (group) × 10000.

RESULTS

Return Rate and Player Demographics

Of the database’s 9991 total entries, 87 (0.9%) were omitted from the study for inaccuracy or not meeting the reporting criteria, thus yielding 9904 usable records. Of this number, 7449 (75.2%) were athletic-related and 2455 (24.8%) were non-athletic-related injuries or general medical conditions.

During the reporting period, 200012 AEs accumulated 4854051 total minutes of playing time. Forwards had the highest game-related injury rate (21.7/1000 AEs), followed by guards (21.3/1000 AEs) and centers (21.0/1000 AEs). Figure 1 presents the game-related injury rates based on the player’s age, years of NBA playing experience, height, and weight.

Injuries or illnesses were incurred by 961 (87.8%) individual athletes; 5188 (52.3%) time-lost injuries accounted for a total of 65956 days missed (32118 practices and 33838 games). Athletic-related injuries resulted in 61542 (93.3%) days missed, while non-athletic conditions caused 4414 (6.7%) days lost from practices and games. Also, 1121 (12.3%) incidences resulted in athletes being placed on the injured reserve list.

Of the 7449 athletic-related injuries, 3989 (56.3%) occurred at home, 2347 (31.5%) on the road, while 1113 (14.9%) were chronic or of insidious onset. Game competition accounted for 4277 (43.2%) injuries, with 2357 (55.1%) occurring at home and 1920 (44.9%) during road competition. Game-related injuries occurred at a rate of 21.4/1000 AEs. The rate of game-related injuries increased 12.4% during the 10-year period (Figure 2).

Injury Prevalence

The lower extremity was the most common site of injury and resulted in the greatest number of days being lost (Table
Figure 1. Game-related injury rates by player (A) age, (B) years of NBA playing experience, (C) height, and (D) weight based on 10000 competition minutes (1 regulation game = 480 competition minutes).

icantly correlated with the athlete’s age \( r = 0.986, P < .001 \) (Figure 3).

Ankle sprains were the most frequent orthopaedic condition reported (942, 9.4%), with most involving the lateral ligaments (874, 92.8%) (Table 5). Trauma to the deltoid ligament complex was reported in 60 (6.4%) instances, while the distal ankle syndesmosis was involved in 8 (0.8%) injuries. In all, ankle sprains were the most frequent game-related injury, occurring at a rate of 3.4/1000 AEs.

The patellofemoral complex was the second most frequent site of orthopaedic trauma and is ranked third among the number of days missed from competition. Ranking first in causing the greatest number of days lost, inflammation of the patellofemoral complex resulted in 7569 (11.5%) days missed. When considered as a single unit, the knee (tibiofemoral joint) and patellofemoral complex is the most prevalent site of orthopaedic trauma (1367, 13.8%), accounts for the greatest amount of time lost (17567 days, 26.6%), and follows only the ankle in the percentage of game-related injuries and incidence rate.

A total of 258 (2.6%) knee sprains occurred, with 8 instances involving multiple ligaments. The medial collateral ligament was the most frequently sprained ligament (126, 47.4%), followed by general capsular sprains (76, 28.6%), the anterior cruciate ligament (35, 13.2%), lateral collateral ligament (27, 10.2%), and
Table 2. Injury Frequency Classified by Body Area

<table>
<thead>
<tr>
<th>Body Area</th>
<th>Frequency (percentage)</th>
<th>Days Missed (percentage)</th>
<th>Frequency (percentage of area)</th>
<th>Percentage of All Game Injuries</th>
<th>Rate per 1000 CMs*</th>
<th>Rate per 1000 AEs†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head</td>
<td>846 (8.5%)</td>
<td>1312 (2.0%)</td>
<td>493 (58.3%)</td>
<td>11.5</td>
<td>1.0</td>
<td>2.5</td>
</tr>
<tr>
<td>Neck</td>
<td>145 (1.5%)</td>
<td>797 (1.2%)</td>
<td>87 (80.0%)</td>
<td>2.0</td>
<td>0.2</td>
<td>0.4</td>
</tr>
<tr>
<td>Upper extremity</td>
<td>1195 (12.1%)</td>
<td>8580 (13.0%)</td>
<td>818 (86.5%)</td>
<td>19.1</td>
<td>1.7</td>
<td>4.1</td>
</tr>
<tr>
<td>Torso</td>
<td>945 (9.5%)</td>
<td>8770 (13.3%)</td>
<td>412 (43.6%)</td>
<td>9.6</td>
<td>0.9</td>
<td>2.1</td>
</tr>
<tr>
<td>Genitals</td>
<td>49 (0.5%)</td>
<td>84 (0.1%)</td>
<td>4 (82.2%)</td>
<td>0.1</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Lower extremity</td>
<td>4993 (46.4%)</td>
<td>43785 (66.4%)</td>
<td>2428 (52.9%)</td>
<td>56.8</td>
<td>5.0</td>
<td>12.1</td>
</tr>
<tr>
<td>General medical</td>
<td>2131 (21.5%)</td>
<td>2628 (4.0%)</td>
<td>35 (1.5%)</td>
<td>0.8</td>
<td>0.1</td>
<td>0.2</td>
</tr>
<tr>
<td>Total</td>
<td>9904 (100.0%)</td>
<td>65956 (100.0%)</td>
<td>4277 (43.2%)</td>
<td>100.0</td>
<td>8.8</td>
<td>21.4</td>
</tr>
</tbody>
</table>

*CM, competition minute.
†AE, athlete exposure.

Table 3. Injury Frequency Classified by Structure*

<table>
<thead>
<tr>
<th>Structure</th>
<th>Frequency (percentage)</th>
<th>Days Missed (percentage)</th>
<th>Frequency (percentage of area)</th>
<th>Percentage of All Game Injuries</th>
<th>Rate per 1000 CMs*</th>
<th>Rate per 1000 AEs†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systemic</td>
<td>2144 (21.6%)</td>
<td>2817 (4.3%)</td>
<td>5 (0.2%)</td>
<td>0.1</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Ankle</td>
<td>1062 (10.7%)</td>
<td>7198 (10.9%)</td>
<td>724 (68.2%)</td>
<td>16.9</td>
<td>1.5</td>
<td>3.6</td>
</tr>
<tr>
<td>Patellofemoral</td>
<td>934 (9.4%)</td>
<td>8628 (13.1%)</td>
<td>236 (25.3%)</td>
<td>5.5</td>
<td>0.5</td>
<td>1.2</td>
</tr>
<tr>
<td>Lumbar spine</td>
<td>675 (6.8%)</td>
<td>7268 (11.0%)</td>
<td>271 (40.1%)</td>
<td>6.3</td>
<td>0.6</td>
<td>1.4</td>
</tr>
<tr>
<td>Femur</td>
<td>541 (5.5%)</td>
<td>3252 (4.9%)</td>
<td>337 (62.3%)</td>
<td>7.9</td>
<td>0.7</td>
<td>1.7</td>
</tr>
<tr>
<td>Tibia</td>
<td>471 (4.8%)</td>
<td>4822 (7.3%)</td>
<td>239 (50.7%)</td>
<td>5.6</td>
<td>0.5</td>
<td>1.2</td>
</tr>
<tr>
<td>Knee</td>
<td>433 (4.4%)</td>
<td>8939 (13.6%)</td>
<td>308 (71.1%)</td>
<td>7.2</td>
<td>0.6</td>
<td>1.5</td>
</tr>
<tr>
<td>Foot</td>
<td>414 (4.2%)</td>
<td>6056 (9.2%)</td>
<td>181 (43.7%)</td>
<td>4.2</td>
<td>0.4</td>
<td>0.9</td>
</tr>
<tr>
<td>Shoulder</td>
<td>296 (3.0%)</td>
<td>2386 (3.6%)</td>
<td>184 (62.2%)</td>
<td>4.3</td>
<td>0.4</td>
<td>0.9</td>
</tr>
<tr>
<td>Hip</td>
<td>234 (2.4%)</td>
<td>880 (1.3%)</td>
<td>164 (70.1%)</td>
<td>3.8</td>
<td>0.3</td>
<td>0.8</td>
</tr>
<tr>
<td>Eye</td>
<td>224 (2.3%)</td>
<td>575 (0.9%)</td>
<td>160 (71.4%)</td>
<td>3.7</td>
<td>0.3</td>
<td>0.8</td>
</tr>
<tr>
<td>Groin</td>
<td>220 (2.2%)</td>
<td>1444 (2.2%)</td>
<td>121 (55.0%)</td>
<td>2.8</td>
<td>0.2</td>
<td>0.6</td>
</tr>
<tr>
<td>Toes</td>
<td>202 (2.0%)</td>
<td>972 (1.5%)</td>
<td>80 (39.6%)</td>
<td>1.9</td>
<td>0.2</td>
<td>0.4</td>
</tr>
<tr>
<td>Wrist</td>
<td>187 (1.9%)</td>
<td>1798 (2.7%)</td>
<td>133 (71.1%)</td>
<td>3.1</td>
<td>0.3</td>
<td>0.7</td>
</tr>
<tr>
<td>Fingers</td>
<td>182 (1.8%)</td>
<td>731 (1.1%)</td>
<td>123 (67.6%)</td>
<td>2.9</td>
<td>0.3</td>
<td>0.6</td>
</tr>
<tr>
<td>Mouth</td>
<td>178 (1.8%)</td>
<td>80 (0.1%)</td>
<td>98 (55.1%)</td>
<td>2.3</td>
<td>0.2</td>
<td>0.5</td>
</tr>
<tr>
<td>Hand</td>
<td>175 (1.8%)</td>
<td>1470 (2.2%)</td>
<td>130 (74.3%)</td>
<td>3.0</td>
<td>0.3</td>
<td>0.7</td>
</tr>
<tr>
<td>Thumb</td>
<td>160 (1.6%)</td>
<td>1354 (2.1%)</td>
<td>116 (72.5%)</td>
<td>2.7</td>
<td>0.2</td>
<td>0.6</td>
</tr>
<tr>
<td>Nose</td>
<td>150 (1.5%)</td>
<td>285 (0.4%)</td>
<td>51 (34.0%)</td>
<td>1.2</td>
<td>0.1</td>
<td>0.3</td>
</tr>
<tr>
<td>Elbow</td>
<td>150 (1.5%)</td>
<td>386 (0.6%)</td>
<td>102 (68.0%)</td>
<td>2.4</td>
<td>0.2</td>
<td>0.5</td>
</tr>
<tr>
<td>Cervical spine</td>
<td>120 (1.3%)</td>
<td>781 (1.2%)</td>
<td>70 (58.3%)</td>
<td>1.7</td>
<td>0.1</td>
<td>0.4</td>
</tr>
<tr>
<td>Face</td>
<td>103 (1.0%)</td>
<td>77 (0.1%)</td>
<td>79 (76.7%)</td>
<td>1.8</td>
<td>0.2</td>
<td>0.4</td>
</tr>
<tr>
<td>Thorax</td>
<td>99 (1.0%)</td>
<td>445 (0.7%)</td>
<td>53 (53.5%)</td>
<td>1.2</td>
<td>0.1</td>
<td>0.3</td>
</tr>
<tr>
<td>Skull</td>
<td>85 (0.9%)</td>
<td>128 (0.2%)</td>
<td>49 (56.7%)</td>
<td>1.1</td>
<td>0.1</td>
<td>0.2</td>
</tr>
<tr>
<td>Fibula</td>
<td>77 (0.8%)</td>
<td>1401 (2.1%)</td>
<td>35 (45.5%)</td>
<td>0.8</td>
<td>0.1</td>
<td>0.2</td>
</tr>
<tr>
<td>Sacrum</td>
<td>60 (0.6%)</td>
<td>309 (0.5%)</td>
<td>33 (55.0%)</td>
<td>0.8</td>
<td>0.1</td>
<td>0.2</td>
</tr>
<tr>
<td>Jaw</td>
<td>58 (0.6%)</td>
<td>72 (0.1%)</td>
<td>45 (77.6%)</td>
<td>1.1</td>
<td>0.1</td>
<td>0.2</td>
</tr>
<tr>
<td>Abdomen</td>
<td>56 (0.6%)</td>
<td>589 (0.9%)</td>
<td>31 (55.4%)</td>
<td>0.7</td>
<td>0.1</td>
<td>0.2</td>
</tr>
<tr>
<td>Thoracic spine</td>
<td>50 (0.5%)</td>
<td>167 (0.3%)</td>
<td>24 (48.0%)</td>
<td>0.6</td>
<td>0.0</td>
<td>0.1</td>
</tr>
</tbody>
</table>

*Structures accounting for less than 0.5% of the total were excluded from this table.
†CM, competition minute.
‡AE, athlete exposure.

posterior cruciate ligament (2, 0.8%). In 9 (3.4%) instances, the ligament sprain occurred concurrently with a meniscal tear.

Trauma to the lumbar spine was the third most prevalent musculoskeletal injury. Lumbar strains and intervertebral disc ruptures (567, 5.7%) accounted for 6418 (9.7%) days missed.

Surgical intervention was required for 368 (3.7%) of the cases reported and accounted for 18761 (28.4%) days missed. The incidence rate for game-related injuries was 1.8/1000 AEs. The body areas most frequently requiring surgery were the knee (120, 32.6%); wrist, hand, and thumb (49, 13.3%); foot (29, 7.9%); and patella (26, 7.1%).

General Medical Conditions

More than one quarter of the conditions reported were not orthopaedic in nature; of these, systemic conditions were the most prevalent, representing 2144 (21.6%) of all cases. Upper respiratory infections were the most common conditions seen by the medical staff (1655, 16.7%), and gastrointestinal problems were ranked fifth in terms of frequently seen conditions (345, 3.5%) (Table 5). Local and systemic infections (94, 0.9%) and dermatologic conditions (86, 0.9%) were also frequently treated conditions.
Table 4. Injury Frequency by Type*

<table>
<thead>
<tr>
<th>Injury or Condition</th>
<th>All Injuries (n = 9904)</th>
<th>Game-Related Injuries (n = 4277)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Frequency (percentage)</td>
<td>Days Missed (percentage)</td>
</tr>
<tr>
<td>Sprain</td>
<td>2074 (20.9%)</td>
<td>17138 (26.0%)</td>
</tr>
<tr>
<td>Upper respiratory infection</td>
<td>1565 (16.7%)</td>
<td>1568 (2.4%)</td>
</tr>
<tr>
<td>Strain or spasm</td>
<td>1603 (16.2%)</td>
<td>12288 (18.6%)</td>
</tr>
<tr>
<td>Sprain</td>
<td>1516 (15.3%)</td>
<td>12948 (19.6%)</td>
</tr>
<tr>
<td>Contusion</td>
<td>1166 (11.8%)</td>
<td>2931 (4.4%)</td>
</tr>
<tr>
<td>Skin wound</td>
<td>360 (3.6%)</td>
<td>412 (0.6%)</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>348 (3.5%)</td>
<td>353 (0.5%)</td>
</tr>
<tr>
<td>Fracture</td>
<td>302 (3.0%)</td>
<td>8534 (12.9%)</td>
</tr>
<tr>
<td>Neurologic</td>
<td>178 (1.8%)</td>
<td>4315 (6.5%)</td>
</tr>
<tr>
<td>Other illness</td>
<td>134 (1.4%)</td>
<td>632 (1.0%)</td>
</tr>
<tr>
<td>Infection</td>
<td>94 (0.9%)</td>
<td>108 (0.2%)</td>
</tr>
<tr>
<td>Dermatologic</td>
<td>86 (0.9%)</td>
<td>111 (0.2%)</td>
</tr>
<tr>
<td>Dental</td>
<td>85 (0.9%)</td>
<td>65 (0.1%)</td>
</tr>
<tr>
<td>Eye</td>
<td>83 (0.8%)</td>
<td>447 (0.7%)</td>
</tr>
<tr>
<td>Meniscal tear</td>
<td>76 (0.7%)</td>
<td>3819 (5.8%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Injury and illness types accounting for less than 0.5% of the total were excluded from this table.

1CM, competition minute.

2AE, athlete exposure.

Figure 3. Rate of inflammatory conditions per player by age group (2-year increments).

DISCUSSION

The types of injuries experienced by professional basketball players reflect the physical demands of the game. As many as 4 nights per week, up to 38 weeks per year, athletes may log as players reflect the physical demands of the game. As many as 48 minutes, and travel the length of a 28.65-m (94-ft) court 200 to 300 times per game (the equivalent of 4.83 to 8.05 km), all while jumping, cutting, and contacting the opponent. These forces, combined with the stresses associated with traveling and time zone changes, all seemingly lead to the onset of acute and repetitive stress injuries.

The changing nature of professional basketball and its increased physical demands may be reflected by the 12.4% increase in game-related injuries during the period investigated. The frequency of inflammatory conditions and the high incidence rate of general medical conditions (eg, upper respiratory infections, influenza) may also serve as evidence that the stresses associated with game participation and travel take their toll on the athletes’ physical health.

While a number of researchers2–6 have examined specific injuries associated with participation in the NBA, this study provides perspective as to the scope, frequency, and relative magnitude of the broad range of conditions that occur to these athletes. Zagelbaum et al5 investigated eye injuries using data reported to the NBTA during the 1991 and 1992 seasons, finding that 5.4% of the injuries involved the eye and occurred at the rate of 1.4/1000 AEs. Both the magnitude and rate of eye injuries were determined to be somewhat less during the 10 seasons described in this paper, when eye injuries represented 3.0% of all athletic-related injuries with an incidence rate of 1.1/1000 AEs. Krinsky et al6 examined meniscal tears reported to the NBTA during the 1985 through 1990 seasons and reported that most (58%) involved the lateral meniscus. Similarly, I noted in this study that 59% of meniscal tears involved the lateral meniscus.

Empirically and anecdotally, ankle sprains are synonymous with the game of basketball. This study confirms that ankle sprains were the most common musculoskeletal injury suffered by professional basketball players, accounting for 9.4% of all injuries and illnesses and 7.7% of all time lost from practices and games. However, the injury rates for ankle sprains identified in this study of 3.4/1000 AEs and 1.4/10000 athlete-minutes are less than those reported for collegiate intramural7,8 and European professional basketball.9

Because of its insidious nature, patellofemoral inflammation may be the “silent endemic” among professional basketball players. This condition is the leading cause of players missing practices and games, but in multiple, small increments rather than the season-ending magnitude of other injuries in the database.

The NCAA maintains a longitudinal database of injuries occurring in 15 sports, including basketball. Similar to the NBTA, the NCAA’s reporting criteria were injuries that (a) occurred as a result of participation in an organized intercollegiate practice or game; (b) required medical attention by a team athletic trainer or physician; and (c) resulted in restriction of the student-athlete’s participation or performance for 1 or more days beyond the day of injury.10

A comparison of the rates and types of game-related injuries that occurred in the NBA and in NCAA Division I, II, and III...
Table 5. Specific Injury and Illness Rates*

<table>
<thead>
<tr>
<th>Injury/Illness</th>
<th>Frequency (percentage)</th>
<th>Days missed (percentage)</th>
<th>Percentage of All Game Injuries</th>
<th>Rate per 10000 CMs</th>
<th>Rate per 1000 AEs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper respiratory infection</td>
<td>1655 (16.7%)</td>
<td>1568 (2.4%)</td>
<td>16.1</td>
<td>1.4</td>
<td>3.4</td>
</tr>
<tr>
<td>Ankle sprain</td>
<td>942 (9.4%)</td>
<td>5122 (7.7%)</td>
<td>143 (17.9%)</td>
<td>3.3</td>
<td>0.3</td>
</tr>
<tr>
<td>Patellofemoral inflammation</td>
<td>803 (9.1%)</td>
<td>7569 (11.5%)</td>
<td>189 (38.5%)</td>
<td>4.4</td>
<td>0.4</td>
</tr>
<tr>
<td>Lumbar erector muscle group strain</td>
<td>491 (5.0%)</td>
<td>3365 (5.1%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gastrointestinal distress</td>
<td>345 (3.5%)</td>
<td>321 (0.5%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knee sprain</td>
<td>258 (2.6%)</td>
<td>5712 (8.6%)</td>
<td>195 (75.6%)</td>
<td>4.6</td>
<td>0.4</td>
</tr>
<tr>
<td>Hamstring strain</td>
<td>249 (2.5%)</td>
<td>1878 (2.8%)</td>
<td>139 (55.8%)</td>
<td>3.2</td>
<td>0.3</td>
</tr>
<tr>
<td>Head and face lacerations</td>
<td>248 (2.5%)</td>
<td>90 (0.1%)</td>
<td>197 (79.4%)</td>
<td>4.6</td>
<td>0.4</td>
</tr>
<tr>
<td>Foot inflammation</td>
<td>219 (2.2%)</td>
<td>2527 (3.8%)</td>
<td>71 (32.4%)</td>
<td>1.7</td>
<td>0.1</td>
</tr>
<tr>
<td>Adductor strain</td>
<td>210 (2.1%)</td>
<td>1352 (2.0%)</td>
<td>117 (55.7%)</td>
<td>2.7</td>
<td>0.2</td>
</tr>
<tr>
<td>Quadriceps contusion</td>
<td>208 (2.0%)</td>
<td>591 (0.8%)</td>
<td>157 (75.5%)</td>
<td>3.7</td>
<td>0.3</td>
</tr>
<tr>
<td>Knee contusion</td>
<td>179 (1.8%)</td>
<td>531 (0.8%)</td>
<td>139 (77.7%)</td>
<td>3.2</td>
<td>0.3</td>
</tr>
<tr>
<td>Triceps surae or Achilles tendon strain</td>
<td>149 (1.5%)</td>
<td>2518 (3.7%)</td>
<td>86 (57.1%)</td>
<td>2.0</td>
<td>0.2</td>
</tr>
<tr>
<td>Finger sprain or dislocation</td>
<td>133 (1.3%)</td>
<td>252 (0.4%)</td>
<td>94 (70.7%)</td>
<td>2.2</td>
<td>0.5</td>
</tr>
<tr>
<td>Lower leg contusion</td>
<td>132 (1.3%)</td>
<td>531 (0.8%)</td>
<td>100 (75.8%)</td>
<td>2.3</td>
<td>0.5</td>
</tr>
<tr>
<td>Hip contusion</td>
<td>127 (1.3%)</td>
<td>291 (0.4%)</td>
<td>109 (85.6%)</td>
<td>2.5</td>
<td>0.6</td>
</tr>
<tr>
<td>Wrist sprain</td>
<td>115 (1.1%)</td>
<td>852 (1.2%)</td>
<td>92 (80.0%)</td>
<td>2.2</td>
<td>0.5</td>
</tr>
<tr>
<td>Thumb sprain</td>
<td>112 (1.1%)</td>
<td>832 (1.2%)</td>
<td>83 (74.1%)</td>
<td>1.9</td>
<td>0.2</td>
</tr>
<tr>
<td>Triceps surae or Achilles tendon inflammation</td>
<td>109 (1.1%)</td>
<td>547 (0.8%)</td>
<td>34 (31.2%)</td>
<td>0.8</td>
<td>0.1</td>
</tr>
<tr>
<td>Foot sprain</td>
<td>102 (0.9%)</td>
<td>718 (1.1%)</td>
<td>65 (63.7%)</td>
<td>1.5</td>
<td>0.3</td>
</tr>
<tr>
<td>Ankle inflammation</td>
<td>86 (0.8%)</td>
<td>948 (1.4%)</td>
<td>20 (23.3%)</td>
<td>0.5</td>
<td>0.0</td>
</tr>
<tr>
<td>Lumbar intervertebral disc rupture or herniation</td>
<td>76 (0.8%)</td>
<td>3053 (4.6%)</td>
<td>16 (21.1%)</td>
<td>0.4</td>
<td>0.0</td>
</tr>
<tr>
<td>Meniscal tear*</td>
<td>76 (0.7%)</td>
<td>3819 (5.8%)</td>
<td>39 (51.3%)</td>
<td>0.9</td>
<td>0.2</td>
</tr>
<tr>
<td>Cervical muscle strain</td>
<td>73 (0.7%)</td>
<td>198 (0.3%)</td>
<td>43 (58.9%)</td>
<td>1.0</td>
<td>0.2</td>
</tr>
<tr>
<td>Rotator cuff inflammation</td>
<td>68 (0.7%)</td>
<td>350 (0.5%)</td>
<td>20 (29.4%)</td>
<td>0.5</td>
<td>0.0</td>
</tr>
<tr>
<td>Hand ligament sprain</td>
<td>66 (0.7%)</td>
<td>168 (0.3%)</td>
<td>48 (72.7%)</td>
<td>1.1</td>
<td>0.1</td>
</tr>
<tr>
<td>Lumbarosacral contusion</td>
<td>62 (0.6%)</td>
<td>93 (0.1%)</td>
<td>56 (90.3%)</td>
<td>1.3</td>
<td>0.3</td>
</tr>
<tr>
<td>Foot contusion</td>
<td>59 (0.6%)</td>
<td>305 (0.4%)</td>
<td>39 (66.1%)</td>
<td>0.9</td>
<td>0.1</td>
</tr>
<tr>
<td>Tarsal or metatarsal fracture</td>
<td>58 (0.6%)</td>
<td>2802 (4.2%)</td>
<td>25 (43.1%)</td>
<td>0.6</td>
<td>0.1</td>
</tr>
<tr>
<td>Quadriceps strain</td>
<td>58 (0.6%)</td>
<td>457 (0.7%)</td>
<td>29 (50.0%)</td>
<td>0.7</td>
<td>0.1</td>
</tr>
<tr>
<td>Tooth fracture</td>
<td>57 (0.6%)</td>
<td>28 (0.0%)</td>
<td>40 (70.2%)</td>
<td>0.9</td>
<td>0.2</td>
</tr>
<tr>
<td>Toe sprain</td>
<td>55 (0.6%)</td>
<td>208 (0.3%)</td>
<td>33 (60.0%)</td>
<td>0.8</td>
<td>0.1</td>
</tr>
<tr>
<td>Hip flexor strain</td>
<td>52 (0.5%)</td>
<td>427 (0.7%)</td>
<td>31 (69.6%)</td>
<td>0.7</td>
<td>0.1</td>
</tr>
<tr>
<td>Glenohumeral sprain</td>
<td>50 (0.5%)</td>
<td>1049 (1.6%)</td>
<td>43 (86.0%)</td>
<td>1.0</td>
<td>0.1</td>
</tr>
<tr>
<td>Nasal fracture</td>
<td>50 (0.5%)</td>
<td>165 (0.2%)</td>
<td>36 (72.0%)</td>
<td>0.8</td>
<td>0.1</td>
</tr>
<tr>
<td>Acromioclavicular joint sprain</td>
<td>47 (0.5%)</td>
<td>464 (0.7%)</td>
<td>31 (66.0%)</td>
<td>0.7</td>
<td>0.1</td>
</tr>
<tr>
<td>Elbow contusion</td>
<td>47 (0.5%)</td>
<td>62 (0.1%)</td>
<td>36 (76.6%)</td>
<td>0.8</td>
<td>0.1</td>
</tr>
</tbody>
</table>

*Injuries representing less than 0.5% of the total were excluded from this table.

1CM, competition minute.

1AE, athlete exposure.

Includes meniscal tears associated with ligament sprains: lateral meniscus, 59.2%; medial meniscus 40.8%.

Men’s basketball from 1988 through 1997 reveals some stark contrasts. The injury rate among NBA players (21.4/1000 AEs) was twice that experienced by collegiate players (10.9/1000 AEs). In collegiate basketball, the ankle (28.8%), knee (12.0%), and hip and pelvis (2.7%) were the most frequent sites of orthopaedic trauma. When only musculoskeletal damage (ie, eliminating general medical reports), the top 3 injury locations in professional basketball were the ankle (14.3%), patellofemoral complex (12.5%), and lumbar spine (9.1%). Similarly, the most common types of injuries experienced in college basketball were sprains (37.1%), strains (16.4%), and contusions (12.6%). In professional basketball, the most frequent types of injury were sprains (27.8%), strains (21.5%), and inflammatory conditions (20.4%).

The prevalence and types of general medical conditions suffered by NBA athletes dilute the perception that these individuals are otherwise healthy. Although the number of reports of general medical conditions may be inflated because conditions needing prescription medication require an injury report, many diseases would have once disqualified the athlete from competition. Athletes are competing in professional basketball with conditions such as asthma, diabetes, emotional and cognitive disorders, and cardiovascular and cardiopulmonary diseases. To appropriately recognize and refer athletes with these conditions to the appropriate medical professional, the athletic trainer’s scope of knowledge must extend beyond orthopaedic conditions. Athletic trainers must be familiar with the unique needs of these athletes and be prepared to respond in the event that athletic competition complicates the athlete’s condition.

CONCLUSIONS

Professional basketball players experience game-related injuries at a rate twice that of college basketball players. The
lower extremity is the most commonly injured body area, accounting for more than half of all orthopaedic injuries. Although ankle sprains are the most frequently occurring orthopaedic injury, the scope of conditions that NBA athletic trainers and team physicians must be familiar with is much broader. Patellofemoral inflammation accounted for the greatest number of practices and games missed, potentially reflecting the physical demands of competition in the NBA and the age of its participants. Extra precautions should be taken to reduce the onset of inflammatory conditions in athletes who are entering the league and those over age 30. Approximately one quarter of the conditions reported were nonorthopaedic, general medical conditions.

This paper serves as the foundation for further investigation into the nature and cause of injuries suffered by professional basketball players. Demographic factors, past injuries, and the concurrence of 1 condition to another, as well as the more detailed analysis of injuries suffered by body region, are logical extensions of this descriptive study.

ACKNOWLEDGMENTS

I thank the athletic trainers of the NBA, who, in addition to their other responsibilities, took the time to complete and return the instruments necessary to conduct this study. Joe O’Toole, ATC, former athletic trainer for the Atlanta Hawks, must be recognized for developing and implementing this study and seeing it grow from a simple idea to a league-wide concept. Also, gratitude is extended to Christopher D. Ingersoll, PhD, ATC, for his valuable assistance in the technical development of this paper.

REFERENCES

It’s Not “Just A Finger”

Jan A. Combs, MD, ATC, FACSM, FAAOS
Walter Reed Army Medical Center, Washington, DC, and The Curtis National Hand Center, Union Memorial Hospital, Baltimore, MD

Objective: To provide a source of information to the athletic trainer on injuries to the fingers; to detail the pertinent anatomy of the finger and explain the techniques of a proper examination; and to discuss treatment and possible outcomes.

Background: Injuries to the fingers are often dismissed as inconsequential and are usually only touched upon briefly in articles on hand injuries.

W e have all seen an athlete come off the field shaking a hand and, when questioned, reply “It’s nothing, it’s just a finger.” Unfortunately, we health care professionals often assume the athlete’s nonchalant attitude about a finger injury. However, seemingly innocuous finger injuries can cause significant, permanent disability for the player.

We use our fingers daily without thought as to how they function. The intricate working of the various structures in each finger is on the subconscious level, as it must be to allow the athlete to grasp the high bar, make the perfect volleyball set, or throw the 145-kph (90-mph) fastball while thinking of what the next move is going to be. Of course, as with any activity requiring such finely tuned actions, it only takes a small problem to cause the whole cascade to malfunction.

Recently, several journal issues have been devoted solely to hand and wrist injuries in sports,1-12 but rarely do these publications do more than just touch on injuries to the fingers. As exposed as the fingers are in most athletic activities, the athletic trainer must be well versed in the examination and treatment of these very important body parts.

ANATOMY

When practicing for any sport, everyone begins with the fundamentals. For the athletic trainer, the fundamental knowledge is anatomy.13-16 The structure of the human finger is extremely complex, but all athletic trainers should have a basic understanding of the major components. Once the basics have been mastered, the examination becomes much more manageable.

The support structure upon which the body is built is the skeleton. The 3 bones of the finger are uniquely shaped to impart stability to the interphalangeal joints (Figure 1). The head of the proximal phalanx has 2 concentric condyles, separated by the intercondylar notch. The base of the middle phalanx is flattened and broad. Its articular surface contains 2 concave fossae, separated by a median ridge. The condyles of the proximal phalanx articulate with these fossae, forming the proximal interphalangeal joint (PIPJ). This tongue-in-groove contour and the large congruent surface area help stabilize the joint by resisting lateral and rotatory stresses.

The primary stabilizers of the PIPJ are the pair of strong collateral ligaments and the volar plate.17 The ligament-box configuration produces a 3-dimensional structure that strongly resists displacement of the PIPJ (Figure 2). The lateral capsule consists mainly of the 2- to 3-mm-thick collateral ligaments, which are the primary restraints to radial and ulnar stress.

The fibrocartilaginous volar plate makes up the floor of the joint. This thick, tough structure allows the flexor tendons to glide past the joint without catching. During movement, the volar plate folds back into a sulcus on the proximal phalanx, permitting full finger flexion. The volar plate is the primary structure preventing PIPJ hyperextension.18

The osseous and ligamentous anatomy of the distal interphalangeal joint (DIPJ) is analogous to that of the PIPJ. The metacarpal-phalangeal joint (MCPJ) is also similar to the PIPJ, but the volar plate is quite different. At the MCPJ, the volar plate is constructed of criss-crossing fibers that collapse to become linear as the joint is flexed. So, instead of moving out of the way with flexion, the MCPJ volar plate actually becomes shorter. The functions of the volar plate are the same in all 3 joints.

Another restraining complex on the volar surface of the finger is the flexor tendon sheath.19 The flexor sheath encases the flexor tendons from the distal palm to the distal phalanx. The sheath is a fibroosseous tunnel, with predictable segmental thickenings, which form the strong annular pulleys. The cruciate pulleys are confluent with the thin synovial sections that interpose the annular pulleys. These sections collapse to allow the annular pulleys to approximate each other during finger flexion (Figure 3).

The purpose of the pulley system is to keep the flexor tendons close to the bone, thus optimizing the biomechanical functioning of the flexor tendons; the constraint of the pulley system governs the moment arm, excursion, and joint rotation produced by the flexor tendons. Loss of digital pulleys, in particular the second (A-2) or fourth (A-4) annular pulley, can...
significantly alter the normal integrated balance of the flexor and extensor tendon mechanisms.

The 2 flexor tendons are strong, oval, cord-like structures powered by extrinsic muscles: the flexor digitorum superficialis (FDS) and flexor digitorum profundus (FDP). The tendons pass through the carpal tunnel, across the palm, and into the flexor tendon sheath. The FDS is palmar to the FDP until they reach Camper’s chiasm, at the middle aspect of the proximal phalanx. Here the FDS splits, the resulting tails twist 180°, the FDP passes through the split, and the tails rejoin and insert onto the base of the middle phalanx. After the FDP passes through the split, it continues on to insert onto the base of the distal phalanx (Figure 4). The flexor tendons are responsible for bending the finger; the FDS acts at the PIPJ and the FDP at the DIPJ. These tendons can function independently, synergistically, or in coordinated fashion.20

The only connection the flexor tendons have to the tendon sheath is through the vinculae.21 These filmy, synovial-like tethers each contain a blood vessel, which contributes to the nourishment of the flexor tendons. There are 2 vinculae, a longus and a brevis, to each tendon. They are located on the dorsal surface of the tendon, between the tendon and the phalanx.

In contrast to the flexor tendons, the extensor tendons are thinner and less substantial, but significantly more complex22 (Figure 5). The extensor mechanism begins in the forearm, arising from the extensor digitorum communis, the extensor indicis proprius, and the extensor digiti minimi. The extensor tendons have independent function, owing to the fact that the extensor digitorum communis has 4 distinct muscle bellies, each with its own branch from the posterior interosseous nerve. The extensor tendons reach the hand by passing through 1 of the 6 dorsal wrist compartments. They then course, very superficially, over the dorsum of the hand into the extensor hood at the MCPJ level.

The extensor hood is a confluence of the extensor tendon, sagittal bands, and the conjoined tendons of the intrinsic muscles. The sagittal bands arise from the intermetacarpal ligaments and the MCPJ volar plate. The tendons from the lumbricals and the interossei join the extensor hood at the midportion of the proximal phalanx and continue distally to the DIPJ.

The extensor tendon trifurcates just proximal to the PIPJ, becoming the central slip and contributions to the lateral bands. The central slip inserts on the dorsal base of the middle phalanx. It is a very wide insertion, extending from 1 collateral ligament to the other, with the central portion being the thickest. The lateral bands travel on the sides of the PIPJ and proceed distally. They join at the DIPJ and insert on the dorsal base of the distal phalanx.

The complexity of the extensor mechanism becomes apparent when one realizes that it is responsible for extending 3 joints (and flexing 1 joint). This is very different from the flexor system, which has an independent flexor tendon for each joint, each with its own excursion length. The fixed tendon lengths of the extensor mechanism dictate that the position of any 1 joint exerts a particular effect on the others. This accounts for the complicated nature of normal finger extension and produces predictable, reciprocal joint deformities when damaged.20,22

The motor units of the fingers are innervated by the median, radial, and ulnar nerves. In very general terms, the flexors are innervated by the median nerve, the extensors by the radial nerve, and the intrinsics by the ulnar nerve. There are notable exceptions, which is another reason the hand examination is so challenging. The flexor digitorum profundus to the ring and small fingers is innervated by the ulnar nerve, rather than the
median, as the other flexors are. Although the intrinsics are, in large part, innervated by the ulnar nerve, the first and second lumbricals are innervated by the median nerve. All 3 nerves have both motor and sensory functions.

Sensation to the volar aspect of the fingers is supplied by the median and ulnar nerves. The thumb, index finger, middle finger, and the radial half of the ring finger are innervated by the median nerve. The ulnar nerve supplies both the volar and dorsal aspects of the small finger and the ulnar half of the ring finger. The dorsal aspects of the thumb, index finger, middle finger, and radial half of the ring finger are innervated by the radial nerve. The digital nerves are branches off the common digital nerves in the palm and travel volar to the midlateral line of the finger.

The digital arteries travel with the digital nerves in the neurovascular bundle. This close association often results in the nerve and the artery both being injured by a laceration. The digital arteries arise from the deep and superficial palmar arches. These arches are the terminal aspects of the radial and ulnar arteries.

The blood supplied to the finger by the digital arteries is returned to the central circulation by a venous plexus. Where the arterial system is uniform, the venous system has a random pattern, varying in number and course. The venous drainage is located primarily on the dorsal aspect of the finger, beginning about 4 mm proximal to the nail complex.

The nail complex, or perionychium, is a specialized skin structure. It provides protection to the tip of the finger (Figure 6) and is composed of the nail bed and the paronychium. The nail bed is all the soft tissue directly under the nail that is responsible for nail generation and migration. The hard nail plate is produced by the germinal matrix located within the floor of the nail fold. As it proceeds distally, it becomes exposed and is attached to the sterile matrix, a part of the nail bed. The nail bed is highly vascular and lies between the periosteum of the distal phalanx and the nail plate.

The skin located on the rest of the finger is of 2 types. The tough, thick skin on the volar aspect is referred to as glabrous skin. It has no hair follicles and very little pigmentation. It forms our personal identification, the fingerprint. It is held in place by septa, or vertical fascial bands, so that the skin does not slip subcutaneously during grasping and twisting actions. The skin on the dorsal aspect of the finger is the same as the skin that covers most of the body. It is thin, yet durable, and has the elasticity to accommodate even extreme positions during movement.

This brief review of the anatomy of the finger should provide a sufficient fund of knowledge to allow one to competently examine an acutely injured finger. If more information is desired, several excellent texts are available at varying levels of detail.

EXAMINATION

On the hectic sidelines, a quick, efficient primary examination is essential; performing it the same way every time will ensure that all areas are covered. A focused, detailed examination of the injured finger is guided by the findings on the primary examination.

The examination actually begins when you observe the injury occur on the playing field and you obtain the athlete’s history. The mechanism of injury is often one of your best sources of information. If a player catches a ball on the end of a finger, you might suspect a mallet finger or volar plate injury. Tacklers in rugby or football can avulse the FDP tendon, the so-called rugger jersey injury. Recognizing the mechanism of injury also helps to focus your examination without sacrificing thoroughness.

The next step is to observe the attitude, or general appearance, of the fingers. Look for such things as cuts, abrasions, bruising, swelling, and deformity. Note if the finger appears adequately perfused. Is it pink and warm or does it have a cool, dusky appearance? At rest, the fingers form a flexion cascade (Figure 7). The tips should point to the region of the scaphoid
A disrupted cascade with 1 finger obviously extended usually indicates a flexor tendon injury. When a deformity is present, be sure to note the direction and amount of the angulation, rotation, and displacement.

Palpate the finger to determine specific areas of point tenderness. Be careful to test each area separately. Do not apply pressure to both sides of the finger at the same time, which may confuse the picture. As you palpate, ask the athlete to tell you which area is the most tender, realizing that there may be more than 1 tender area. The eraser end of a pencil can be helpful in palpating small, discrete areas.

Assessing the range of motion in an acutely injured finger is often impossible. If attempted, it should only be done actively, within the limits of pain. If a fracture or dislocation is suspected, it is probably advisable to obtain x-ray films before testing the range of motion or stability of a joint.

Checking the functional continuity of the extensor tendons in an acute injury is not difficult. One must determine if the athlete can actively extend the finger. Note if full extension is achieved and the amount of force the athlete can produce. Conducting the intricate testing for an established boutonnière deformity or a chronic intrinsic-plus hand is not necessary in the acute setting.

Examination of the flexor tendons must be accomplished so that the actions of the tendons can be separated. Each tendon within each finger must be examined individually. To test the FDS, hold the other fingers in full extension and have the athlete flex the finger at the PIPJ (Figure 8). Holding the other fingers in extension keeps the common muscle belly of the FDP stretched to its maximum length. This blocks its function, so that it cannot aid in flexing the finger. To demonstrate this, flick the tip of the finger being tested. Note that the DIPJ is flail: the profundus cannot contract to help stabilize the joint.

The profundus tendon acts to flex the distal joint. To test it, hold the PIPJ in full extension, blocking the FDS from acting, and have the athlete attempt to bend the distal joint (Figure 9).

Stability of the interphalangeal joints to radial and ulnar stress is tested in the same way as the collateral ligaments of the knee. Apply stress in both extension and flexion to the opposite side of the joint to see if it will open, signifying damage to the ligament. Note whether or not there is a firm endpoint while stressing the ligament. If possible, have the athlete flex and extend the injured finger to check for dorsal and volar subluxation, a test of dynamic stability.

The neurovascular check of a finger on the sidelines is very cursory, at best, due to the nature of the setting. The vascular status of the finger should have been quickly assessed at the beginning of the examination, while noting the general appearance of the finger. Once in the training room, you can check the status of the nerves and vessels properly. Generally, if the finger is pink and warm, it is adequately perfused. To further evaluate the arteries requires a Doppler ultrasound. However, the digital nerves can be tested easily with an instrument found in most training rooms: a paper clip. Bend it so that the 2 ends of the wire are about 4 mm apart. Two-point discrimination is
tested on the pad of the finger (Figure 10). Test both sides on the volar aspect of the pad to check both digital nerves. Determine the athlete’s normal 2-point discrimination by testing the fingers on the uninjured hand. Normal static 2-point discrimination is approximately 4 to 5 mm. A 2-point discrimination of 6 to 10 mm is felt to be protective (ie, sensations such as hot and cold, sharp and dull, pain, rough texture, and extreme motion are detectable), but not normal, sensation. The finger is considered insensate if the 2-point discrimination is greater than 10 mm.

An interesting test of nerve continuity is the water immersion test. This is usually used in uncooperative persons, such as small children, unconscious patients, or those lacking verbal ability. The test is simple. Immerse the injured finger and at least 1 uninvolved finger in saline at room temperature. When the uninvolved finger wrinkles, look at the injured finger. If it is wrinkled, the nerves are intact. If it is still smooth on both sides, then both digital nerves have been disrupted. Moreover, by default, if only 1 side is smooth, that digital nerve is damaged.

With the above examination, you should be able to determine the nature of an acute injury nearly 100% of the time. Occasionally though, further information is needed. As mentioned earlier, any time you suspect a fracture or dislocation, an x-ray film should be obtained before stress testing or range of motion is performed. Use of a minifluoroscope for the initial radiographic examination is ideal. The x-ray images, both static and real-time, are immediately available. However, this device is usually only available to large university or professional programs. If the examination and x-ray films still do not elucidate the problem, the team orthopaedic surgeon or a hand surgeon should evaluate the athlete.

**INJURIES AND TREATMENT**

Finger injuries are very frequent in athletes, but they are usually minor and require minimal treatment. The difficulty comes in determining which injuries do require special attention. The following is a discussion of acute injuries that an athletic trainer may encounter. It does not include chronic or overuse problems.

![Figure 10. When testing the function of the digital nerves, the points of the paper clip should be aligned longitudinally on the side of the finger pad.](image1)

**Skin and Nail Injuries**

Athletes sustain minor cuts and abrasions often. These should be cleaned thoroughly to remove all dirt and foreign bodies from the wound. Soap and water are the best agents for cleaning wounds. Hydrogen peroxide is very helpful in removing dried-on blood around the wound. It is important to remove this blood so that small, but potentially significant, wounds are not missed. Once the wound is clean, a thin layer of a topical antibacterial ointment can be applied. However, use of the ointment should never take the place of thoroughly cleaning the wound with plain soap and water. A protective dressing should be used when the athlete is playing.

One wound that deserves special attention is the human bite. The human mouth has the highest concentration of bacteria anywhere on the body. The staphylococcus and streptococcus species, gram-positive cocci, are the most common. A bacterium of special interest in human bites is *Eikenella corrrodens*, a gram-negative rod. Any significant human bite (ie, a wound with a puncture component; a flap-type wound; a wound involving a joint, bone, tendon, nerve, or vessel; or any wound the athletic trainer is unsure about) should be evaluated by the team physician, as prophylactic antibiotics are generally needed in conjunction with proper wound care. When examining human bites, you must be sure that the wound does not violate the joint capsule. If it does, the athlete should be sent directly to a surgeon for a formal débridement of the wound as soon as feasible. Wounds of a minor nature, not requiring a physician’s attention, should be thoroughly cleaned and dressed as usual. Of note, human bites are never sutured or closed tightly, because of the high rate of infection. The wound is left open to heal by secondary intention.

Nail injuries usually occur from a crushing mechanism. If blood is visible under the nail (a subungual hematoma), the nail bed has been torn. Damage to the nail bed can sometimes cause future nail deformities, and the athlete should be advised of this possibility. If the nail plate is still firmly attached, the pressure from the bleeding can cause significant discomfort. Making holes in the nail plate can relieve the pressure (Figure 11). Several instruments can be used to make the holes: a hot paper clip, drill, needle, or electrocautery. Make the holes large (2 to 4 mm), so they will not clot off. Hydrogen peroxide is useful in keeping the holes free of dried blood. If the nail plate is partially or completely avulsed, the nail bed should be repaired. The laceration is repaired with very fine 6-0 ophthalmologic sutures. When the nail plate is present, it is cleaned, trimmed, and replaced over the wound to protect the repair and act as a stent to keep the nail fold open. Some surgeons believe that if a subungual hematoma covers 30% to
Ligament Injuries

The collateral ligaments of the proximal interphalangeal joint are usually injured by a bending or twisting mechanism. The joint will be swollen and point tender directly over the collateral ligament. If the joint opens when stress tested, the athlete has sustained a grade II or III ligament tear. Fortunately, due to the skeletal configuration of the interphalangeal joint, ligament injuries rarely lead to instability and almost never require surgical intervention. Treatment consists of buddy taping the injured finger and active range-of-motion exercises. The buddy taping should be worn full time for 10 to 14 days, or until full range of motion is achieved. Thereafter, the athlete need only buddy tape when playing for the remainder of the season. The most common long-term consequence of PIPJ injuries is decreased range of motion and stiffness. Injured PIPJs will remain swollen up to 6 or 8 months and sometimes permanently, which should be discussed with the athlete early in the treatment course.

Volar plate injuries are caused by hyperextension of the PIPJ. This injury is usually associated with a dorsal dislocation or subluxation of the middle phalanx. If the joint remains dislocated, the deformity can be quite impressive. However, it may not be readily apparent that a dislocation or subluxation has occurred if the athlete has already impulsively reduced it. The athlete will be exquisitely tender on the volar surface of the PIPJ, specifically at the base of the middle phalanx. If the athlete has delayed reporting the injury, ecchymosis in the PIPJ flexion crease and some diffuse swelling are usually evident. The volar plate can fail in 2 ways: the distal aspect can rupture or it can avulse its bony attachment from the base of the middle phalanx, the so-called “chip fracture.” Suspected volar plate injuries should be x-rayed to make sure the joint is congruent and to determine if a chip fracture exists, and, if so, how large the fragment is (Figure 12). A chip fragment is an insignificant piece of bone, usually just a tiny fleck. A large fragment involving a portion of the joint surface should probably be considered an intraarticular fracture, not a chip fracture. As long as the joint is congruent, treatment is the same, whether or not a chip fracture exists. A variation of dorsal block splinting is the usual regimen. The PIPJ is blocked 30° from full extension, but the athlete is allowed full active flexion. Over the next 3 to 4 weeks, extension is increased until full extension is achieved (Figure 13). The finger should be buddy taped for activities for the rest of the season.

Figure 12. Volar plate injury: the middle phalanx is subluxed dorsally.

If the PIPJ is unstable during active range of motion or the x-ray films show a persistent subluxation of the joint, more aggressive treatment is needed. The joint must be reduced and held in position, which may require surgical intervention, possibly in the form of a volar plate arthroplasty. Again, you must consider all the information obtained and maintain a high index of suspicion to avoid missing these injuries. If at any time you are unsure, refer the athlete for further evaluation.

Tendon Injuries

A mallet, or baseball, finger is an injury to the terminal slip of the extensor tendon. It is caused by sudden, forceful flexion of the distal phalanx, as when a player catches a ball on the tip of a finger (Figure 14). The injury can be of 3 types: stretch of the tendon, rupture of the tendon, or avulsion of the bony attachment from the distal phalanx. The athlete will be tender on the dorsal aspect of the DIPJ and unable to actively extend the distal phalanx. An x-ray film should be obtained to make sure the joint is congruent. It is rare that this injury cannot be treated with extension splinting. If volar subluxation of the distal phalanx persists, surgical intervention is usually necessary. Several commercial mallet finger splints are available, or you can make your own (Figure 15). The splint should hold the DIPJ in extension, but not hyperextension. When the DIPJ is in hyperextension, circulation to the skin covering the dorsal aspect of the DIPJ is compromised and skin slough may occur. The splint should avoid interfering with range of motion at the PIPJ. The athlete must wear the splint 24 hours per day for 6 weeks, then at night for another 2 to 4 weeks. The joint must be maintained in extension, even when the athlete is showering and changing the splint. If the DIPJ is flexed while out of the splint, the treatment time is reset, and the athlete has 6 weeks from that point. Unfortunately, it is sometimes difficult for the athlete (and
Mallet Finger

Stack Splint

Foam Aluminum Splint

Figure 15. Splint a mallet finger in extension, not hyperextension, and allow motion at the PIPJ.

When an injury to the central slip of the extensor tendon occurs and is untreated, the ensuing flexion deformity of the PIPJ is called a boutonnière deformity. However, common usage is to call the acute injury a boutonnière deformity as well, when a more proper term would be an impending boutonnière or a central slip injury (Figure 16). The usual mechanism of injury is a volar dislocation, a dorsal crush injury, or a laceration of the central slip. Unfortunately, the athlete often dismisses this injury as just a jammed finger. On acute presentation, if the finger is lacerated or still dislocated, it is an easy diagnosis. However, the only sign of injury may be exquisite point tenderness at the insertion of the central slip. The athlete will most likely not be able to extend the PIPJ. Occasionally, he or she can extend the joint using the lateral bands, even in the presence of a complete central slip disruption. With time, the joint will become swollen and will usually be held in a slightly flexed posture. An x-ray film should be obtained. If the x-ray film reveals a large articular fracture fragment, a subluxed joint, or significant comminution of the base of the middle phalanx, the athlete should be referred to a hand surgeon. For those injuries that can be treated nonoperatively, the treatment of choice is a static extension splint to hold the PIPJ in full extension. The DIPJ should be left free and active, and passive range of motion of the DIPJ should be encouraged (Figure 17). The motion at the DIPJ keeps the lateral bands from migrating volarly. The length of time the joint is splinted depends upon the severity of the injury. A complete rupture of the central slip treated nonoperatively is immobilized full time for 6 weeks, whereas a minor grade I strain (in which the only symptom is mild tenderness over the central slip) is splinted for 7 to 10 days. After the splint is discontinued, the finger is buddy taped 24 hours per day until full range of motion is regained. The finger should be buddy taped during play for the remainder of the season.

The rugger jersey injury is an injury to the terminal attachment of the FDP tendon. The mechanism of injury is a powerful extension force applied to the distal phalanx while it is attempting to flex forcefully. The FDP can also fail within the substance of the tendon or by avulsing its bony attachment at the base of the distal phalanx (Figure 18). The vinculae, if intact, keep the ruptured tendon from retracting into the palm. In addition, if the tendon avulses a large piece of bone, it may catch in the pulley system, also keeping the tendon from retracting further. It is important to keep these facts in mind when examining the athlete for a suspected rugger jersey injury. The end of the tendon may be caught in the A-4 pulley at the DIPJ or in the A-2 pulley at the PIPJ or it may have retracted all the way back into the palm. The athlete will usually report feeling a pop when the injury occurred. The normal cascade of the fingers will be disrupted, and the athlete will be unable to flex the DIPJ. A mass may be palpable where the end of the tendon has come to rest. If reporting of the injury has been delayed, ecchymosis will be visible on the volar aspect of the DIPJ and the finger will be swollen. Treatment of this injury is surgical; the tendon must be reattached. Surgery is followed by a complex rehabilitation program of dynamic splinting and therapist-supervised tendon-giding exercises. Rugger jersey injury is season ending.

Fractures

Fractures can occur by almost any mechanism, and it is the mechanism that determines the fracture pattern. A 3-point bend creates a butterfly fragment, a torsional stress causes a spiral fracture, and so on. If a finger is fractured, it will not necessarily be deformed. All fractures do cause tenderness, but that is where the similarities among fractures stop. Each one has its own personality and its own unique characteristics.

Figure 16. Avulsion of the central slip is 1 cause of a boutonnière deformity.

Figure 17. Boutonnière deformity is treated with an extension splint and motion of the DIPJ.
A crush injury to the tip of the finger causes 1 of the most common fractures in the finger, a distal tuft fracture. It can result from a finger being stepped on or caught between 2 football helmets. A nail bed injury can coexist. For the tuft fracture, there is no real treatment other than to protect the tip of the finger until the discomfort decreases. A prefabricated splint, such as one used for a mallet finger, or a foam aluminum splint can be used.

Midshaft fractures usually occur from a bending or twisting force. A transverse fracture results from a bending force and is inherently unstable. The resulting deformity is produced by the muscles pulling on the fracture fragments. It is often difficult to reduce and hold these fractures with a cast; therefore, surgical reduction and fixation may be needed.

A spiral fracture occurs when the finger is twisted. The deformity in this injury is much subtler than that of the transverse type. Sometimes the only sign, in addition to tenderness, is that the fingernails do not quite line up correctly. The injured finger is rotated out of the plane of the rest of the fingers. Rotational deformity is more of a problem than angulation. The body cannot remodel or change to correct rotation. Thus, it is very important to check the alignment of the fingernails and compare them with the other hand. A small rotation can cause significant functional impairment. Treatment is to reduce the fracture and hold it in position. If the fracture is nondisplaced, it can usually be treated with casting and close x-ray film follow-up. If it is displaced, surgical intervention is usually necessary.

Fractures that enter the joint are known as intraarticular fractures. By definition, mallet fingers and chip fractures are truly intraarticular fractures, but they are usually placed in separate categories from the condylar-type fractures. It is difficult to tell the difference between an intraarticular fracture and subluxation of the joint on examination without the use of x-ray films (Figure 19). Three views of the finger (not the hand), should be ordered in an x-ray film series. Sometimes, even with an examination and x-ray films, the fracture is still not apparent. In these cases, fluoroscopy, or real-time x-ray film, may be necessary. If the fracture is nondisplaced, it can be treated with immobilization and close x-ray film follow-up. However, any displacement of the fragment can result in a step-off of the joint surface, leading to significant arthritis. Surgery is needed to restore the joint surface.

One of the potentially most severe injuries to the finger is a pilon fracture of the base of the middle phalanx (Figure 20). This occurs from an axial load to an extended finger. Its presentation may not be significantly different from a volar plate or boutonniere injury. However, instead of being tender on only 1 side of the finger, the whole joint is tender. On x-ray film, the severe comminution and joint subluxation are apparent. The athlete must be seen by a hand surgeon for this injury. Many treatments have been developed for this problem, none of which offer an ideal solution. The athlete will be advised by the hand surgeon of the severity of the injury and of the possibility of never regaining full, complete range of motion compared with the uninjured side. However, with time and therapy, the athlete should be able to return to full activity. The occupational therapist will probably conduct the therapy in the immediate postinjury or postsurgery period. As an athletic trainer, you will be around the athlete most of the time thereafter and will have to provide support for the day-to-day problems of the injured athlete, not just of the injured finger.

Athletic trainers who work at the junior and senior high school levels should be aware of physeal fractures. The physis is the growth plate and the weak link in immature bone. When a young athlete sustains what appears to be a dislocation, an x-ray film must be obtained to determine if there is physeal involvement (Figure 21). If there is any question as to whether the physis is injured, the young athlete should be sent to an orthopaedic surgeon.

The previously described fractures are closed fractures: that is, the fracture is not exposed through an injury to the overlying tissues. Open fractures used to be called "compound fractures."
a term no longer in use. An injury in which bone is exposed is an open fracture and a surgical emergency, due to the high incidence of infection when the wound is not treated properly (Figure 22). The wound should be dressed, the hand splinted, and the athlete sent immediately (without waiting for the end of the game) to the hospital. It is important that the athlete not eat or drink anything on the way to the hospital, as this complicates the surgical process. Early surgical intervention for open fractures has greatly decreased the incidence of osteomyelitis, or infection of the bone, but has not totally eradicated it. Thus, as an athletic trainer, you must insist on transporting the athlete to the hospital immediately and not waiting until after the game.8,26,58

Neurovascular Injuries
Severe injuries to the nerves and arteries of the fingers are rare in sports. Athletes do not engage in activities employing objects with sharp edges that could cause lacerations to the neurovascular bundles. Thus, the most common cause of nerve and vessel injury is compression from swelling due to another injury. The numbness, which ensues from pressure on the nerve, will usually go away when the compression is alleviated. If sensation returns promptly, the nerve has sustained an injury called neurapraxia. Vascular occlusion can occur from extraneous swelling, but is very rare. For the finger to be at risk, both arteries must be occluded. A finger with both arteries blocked will feel cold and appear dusky blue, white, or pale. The athlete will complain of a vague, deep pain in the finger. This condition is a surgical emergency requiring, in essence, a fasciectomy to relieve the pressure and restore blood flow.

The second mechanism of injury to the nerves and vessels is a direct blow. Sensation distal to the injury will be diminished or absent. The nerve will then slowly recover sensation with time, from proximally to distally. The point at which the nerve is damaged, called a neuroma, can be determined by tapping along the course of the nerve. When the neuroma is reached, the athlete will feel an electric sensation to the tip of the finger. Recovery of the nerve can be followed by the same method. A direct blow to an artery will sometimes cause damage to the intimal lining of the vessel, resulting in disruption of the blood flow and development of a thrombosis. If only 1 vessel is involved and the finger is warm and pink, no treatment is necessary. If the finger is at risk (vascula rly compromised), immediate evaluation by a hand, vascular, or plastic surgeon is required.12,26

Devastating Injuries
A ring avulsion amputation is an injury that does not occur frequently. It results when the finger is caught in a net or other equipment.70 Due to the momentum and weight of the body, the soft tissues of the finger are literally ripped off their skeletal support, creating a degloving injury. Unfortunately, even with the microsurgical techniques available today, the amputation is rarely reimplantable. However, that is a decision for the hand surgeon to make. The athlete and the amputated part should be sent directly to a center that performs this type of surgery. The amputated finger should be wrapped in moist sterile gauze, placed in a sealed plastic bag, and the bag placed on ice water. Do not use dry ice or let the amputated part freeze. A properly handled amputated finger can survive up to 24 hours of cold ischemia. You should be
prepared for an injury such as this, but, I hope, will never have to actually make use of your preparation.26,70

There are a few injuries that can have devastating consequences, despite being treated promptly and properly. If osteomyelitis develops after an open fracture, occasionally the only means of eradicating the infection is to amputate the digit. A neurovascular injury that leaves the finger insensate poses a perplexing problem. Does one keep the digit for cosmetic reasons or perform a ray amputation to make a more functional hand? Fortunately, these decisions do not have to be made frequently.

**SUMMARY**

Injuries to the fingers occur frequently in sports. Most are minor in nature and need little in the way of treatment.1,4,27,71 However, severe injuries can often present with minimal, subtle signs and symptoms. Athletic trainers must be knowledgeable in the anatomy and biomechanics of the fingers and able to perform a competent examination of an injured digit. Missed or improperly treated finger injuries can lead to significant disability.72

Losing the use of just 1 finger can have a significant impact on an individual’s lifestyle. As health care providers, we must keep in mind that sports are not the only thing in a young person’s life. The athlete may play the piano or have plans to become a surgeon. There is life after sports, a fact we must always keep in mind when treating athletic injuries. Remember, it is not “just a finger”!

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**REFERENCES**

Asthma is a chronic inflammatory disorder of the airways that affects approximately 15 million people in the United States, with an increase in incidence of over 75% from 1982 to 1995. This inflammatory disease results in a hyperresponsiveness of the airways that can be initiated by exposure to various stimuli, including allergens, exercise, cold temperatures, aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs), viral infections, or other irritants. In addition, many asthmatic patients have nocturnal asthma, with symptoms that awaken them during the night or affect them upon awakening in the morning. Symptoms of asthma include episodic wheezing, chest tightness, shortness of breath, and cough. A discussion of the pathophysiology of asthma is available elsewhere.

Drug therapy plays an important role in the proper management of asthma. However, a study revealed that fewer than 20% of asthmatic patients received appropriate drug therapy. Inadequate patient education, poor patient compliance, and a communication gap between the patient and health care providers are major causes for inadequate therapeutic outcomes. Athlete trainers have the opportunity to influence these areas of deficiency and, thus, improve therapeutic outcomes. Consequently, it is important that athletic trainers have a basic understanding of the effects and appropriate use of the drugs available to treat athletes who have asthma, as well as knowledge of other related factors that may have an impact on the performance of these athletes.

GOALS OF THERAPY

The National Heart, Lung, and Blood Institute (NHLBI) established general goals of asthma therapy:

- Prevent chronic asthma symptoms and asthma exacerbations during the day and night.
- Maintain normal activity levels, including exercise and other physical activities.
- Have normal or near-normal lung function.
- Be satisfied with the asthma care received.
- Have no or minimal side effects while receiving optimal medications.

For the athlete, whether casual or competitive, maintenance of normal activity levels includes athletic performance, but a recent survey found that 48% of people with asthma say their asthma limits their ability to take part in sports or recreation. Ultimately, the athlete must determine an acceptable response from therapy, but to maximize the effectiveness of the therapy, the athletic trainer can help ensure that the athlete is compliant with the medication dosing schedules, is using inhaler devices appropriately, and is adequately monitoring the effectiveness of drug therapy. To achieve the best combination of medication and dosing, athletes not ideally managed may require a referral.
Another goal of therapy is to ensure that the athlete is not using any asthma medication banned by an agency regulating the athlete's participation. As discussed later, the US Olympic Committee, for example, only allows the use of some asthma medications by inhalation to minimize the systemic effects that may impart an advantage to the athlete (eg, central nervous system stimulation).

**INHALERS AND PULMONARY FUNCTION TESTS**

Inhalation of antiasthmatic medication provides a topical application of the drug in the airways and is the most effective route to administer the drugs of choice for quick relief of acute attacks (ie, β₂ agonists) and for long-term therapy (ie, corticosteroids). The most popular device used for inhalation of these drugs is the metered dose inhaler (MDI). These devices contain the drug in a pressurized container with a metering valve. When the device is pressed appropriately, it releases a propellant, which forces a metered amount of drug as an aerosol into the patient's mouth. The drug is either in solution or a suspended micronized powder. One disadvantage of the MDI is that the patient must be skilled in coordinating the activation of the MDI with inhalation to maximize delivery of drug to the lung. Even with good technique, only 10% to 20% of the drug reaches the lungs; most of the drug is deposited in the oropharynx. A spacer (or holding chamber) is a device that attaches to the MDI. The inhaler is discharged into the spacer, and the patient inhales from it. Some propellant evaporates as it travels through the spacer, resulting in smaller particles that have a better chance of reaching the lung. The spacer also reduces the speed of the drug so that some of the larger particles lose their momentum before reaching the oropharynx. When spacers are used with inhaled corticosteroids, the diminished amount of oropharyngeal deposition of the drug results in a lower incidence of hoarseness and fewer oral candidiasis infections (thrush). Rinsing the mouth with water and spitting after use of the inhaler also may reduce these localized side effects. The design of some spacers more effectively facilitates an increase in the number of particles reaching the lung. Use of a spacer also eliminates the need to precisely coordinate inhalation with activation of the MDI. Therefore, use of a spacer with the MDI decreases the precision necessary, improves inhaler technique, and increases the delivery of drug to the lungs in those patients who have difficulty achieving proper technique without the spacer.

Dry powders can be inhaled with the use of a dry powder inhaler (DPI). The powdered drug is placed in the DPI in a capsule or other package form, and the inhaler is used to break open the package. There is no propellant, but the process of inhalation through the inhaler causes the powder to reach the lung. This eliminates the need for hand-lung coordination but requires a higher inspiratory flow. DPIs tend to cause more drug to be deposited at the oropharynx and, thus, may necessitate patients rinsing their mouths, especially when using corticosteroids.

Nebulizers are used to deliver an aerosol of drug to the patient's lungs, but these are larger devices and are primarily used in hospitals and clinics and in homes for parents to administer drugs to young patients. Nebulizers use liquid drug and a stream of compressed air that forms the droplets of drug, which are inhaled during normal breathing by the patient. No hand-lung coordination is needed, and less drug is deposited in the mouth.

Another device that facilitates appropriate drug therapy is a peak flow meter (PFM), which measures the peak expiratory flow (PEF). The purpose of the PFM is to assess the patient's pulmonary function as a means of monitoring the effectiveness of the drug therapy. The handheld device can be used as a daily routine check of pulmonary function. The patient's personal best is established over 2 to 3 weeks of good asthma control. All other PFM tests are then compared with that value. Daily results can be used to evaluate the response to adjustments in therapy, and once as long-term, therapy or to signal that an acute attack is imminent even before symptoms arise. For example, if the PEF is 80% to 100% of the personal best, the existing treatment should be maintained. If the PEF is 50% to 80% of the personal best, the treatment should be adjusted, as previously determined by the physician. A medical alert is indicated if the PEF is less than 50%; a short-acting inhaled bronchodilator should be used and medical attention obtained immediately. The NHLBI recommends the use of the PFM for patients with moderate to severe persistent asthma or a history of severe exacerbations as a means to obtain optimal drug therapy outcomes.

Another pulmonary function test is the forced expiratory volume in 1 second (FEV₁). This can be measured using a forced expiratory spirometer, which allows a determination of the rate of forced expiratory volume after maximal inspiration. A comparison of FEV₁ with normal predicted values can be part of the criteria used to determine the severity of the asthma. Table 1 is the classification of the severity of asthma as defined by the NHLBI. Long-term therapy is modified based upon these classifications (Table 2). Patients at any level of severity may have acute exacerbations that range from mild to severe.

**CATEGORIES OF ASTHMA DRUGS**

Drugs used to treat asthma can be grouped into broad pharmacologic categories: bronchodilators and anti-inflammatory agents. Within these categories, subgroups exist based

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**Table 1. Classification of Asthma Severity: Clinical Features Before Treatment**

<table>
<thead>
<tr>
<th>Classification</th>
<th>Days with Symptoms</th>
<th>Nights with Symptoms</th>
<th>PEF or FEV₁,</th>
<th>PEF Variability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 4 (Severe persistent)</td>
<td>Continual</td>
<td>Frequent</td>
<td>≥60%</td>
<td>&gt;30%</td>
</tr>
<tr>
<td>Step 3 (Moderate persistent)</td>
<td>Daily</td>
<td>≥5/month</td>
<td>&gt;60% to &lt;80%</td>
<td>&gt;30%</td>
</tr>
<tr>
<td>Step 2 (Mild persistent)</td>
<td>3 to 6/week</td>
<td>3 to 4/month</td>
<td>≥80%</td>
<td>20 to 30%</td>
</tr>
<tr>
<td>Step 1 (Mild intermittent)</td>
<td>≤2/week</td>
<td>≤2/month</td>
<td>≥80%</td>
<td>&lt;20%</td>
</tr>
</tbody>
</table>

*Percentage of patient's best PEF or percentage of predicted values for FEV₁.*
Table 2. Stepwise Approach for Managing Asthma in Adults and Children >5 Years of Age

<table>
<thead>
<tr>
<th>Asthma Classification</th>
<th>Daily Medication(s) (preferred treatments are in bold)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 4 (Severe persistent)</td>
<td>• Anti-inflammatory: inhaled corticosteroid (high dose) and&lt;br&gt;• Long-acting bronchodilator: long-acting inhaled ( \beta_2 ) agonist, sustained-release theophylline, or long-acting ( \beta_2 ) agonist tablets and&lt;br&gt;• Corticosteroid tablets or syrup long term; make repeated attempts to reduce systemic corticosteroid and maintain control with high-dose inhaled corticosteroid</td>
</tr>
<tr>
<td>Step 3 (Moderate persistent)</td>
<td>• Anti-inflammatory: inhaled corticosteroid (medium dose) or&lt;br&gt;• Inhaled corticosteroid (low to medium dose) and add a long-acting bronchodilator, especially for nighttime symptoms; long-acting inhaled ( \beta_2 ) agonist, sustained-release theophylline, or long-acting ( \beta_2 )-agonist tablets&lt;br&gt;• If needed: Anti-inflammatory: inhaled corticosteroids (medium to high dose) and&lt;br&gt;• Long-acting bronchodilator, especially for nighttime symptoms; long-acting inhaled ( \beta_2 ) agonist, sustained-release theophylline, or long-acting ( \beta_2 )-agonist tablets</td>
</tr>
<tr>
<td>Step 2 (Mild persistent)</td>
<td>• Anti-inflammatory: either inhaled corticosteroid (low dose) or&lt;br&gt;• Cromolyn or nedocromil (children usually begin with either cromolyn or nedocromil)&lt;br&gt;• Sustained-release theophylline to serum concentration of 5 to 15 mcg/mL is an alternative, but not preferred, therapy. Zafirlukast or zileuton may also be considered for those &gt;12 years old, although their use in therapy is not fully established.</td>
</tr>
<tr>
<td>Step 1 (Mild intermittent)</td>
<td>• No daily medication needed.</td>
</tr>
</tbody>
</table>

* For quick relief for all patients, short-acting bronchodilator: inhaled \( \beta_2 \) agonist (2 to 4 puffs) as needed for symptoms. Intensity of treatment will depend on severity of exacerbation. Step down: Review treatment every 1 to 6 months. Gradually decrease treatment to the least medication necessary to maintain control. Step up: If control is not maintained, consider step up. Inadequate control is indicated by increased use of short-acting \( \beta_2 \) agonists. Review patient inhaler technique, compliance, and environmental avoidance of allergens or other precipitant factors. 

They are also the most effective medication to prevent an anticipated attack, such as immediately before exercise, without the onset of significant cardiac or other systemic effects. After inhalation of short-acting \( \beta_2 \) agonists, onset of action is usually within 5 minutes, and maximal bronchodilation occurs within about 15 minutes. Duration of action is 4 to 8 hours, depending upon the agent, although a shorter period of protection (2 to 4 hours) is experienced during exercise. Table 3 lists some short-acting \( \beta_2 \)-adrenergic agonists and a typical adult dose. When given at equipotent doses, all of the \( \beta_2 \) agonists will produce the same intensity of bronchodilation; their main difference is the duration of action. Some agents are effective orally, but the oral route delays onset of action and is less selective for bronchial muscle, thus increasing the incidence of side effects.

Salmeterol is a long-acting \( \beta_2 \) agonist available by inhalation, but its slower onset of action (up to 20 minutes) and time for maximal effect (1 to 4 hours) preclude its use as rescue therapy for treatment of acute attacks. Users of salmeterol must clearly understand that it is not effective for treatment of acute bronchospasms and that the dose should not exceed 2 puffs every 12 hours. Depending upon the duration of the athletic activity, some athletes may benefit from salmeterol, which has a longer duration of action (12 hours) as compared with albuterol (4 hours). Patients who suffer from nocturnal asthma may also benefit from the longer-acting \( \beta_2 \) agonist.

Regularly scheduled, daily use of a short-acting \( \beta_2 \) agonist is generally not recommended, since there is no apparent advantage over use on an as-needed basis. If the frequency of \( \beta_2 \)-agonist use increases or if use exceeds 1 canister of \( \beta_2 \) agonist (eg, 200 puffs of albuterol) per month to control exacerbations, asthma control is poor, and reevaluation of the anti-inflammatory therapy is necessary. Regular use of short-acting \( \beta_2 \) agonists has not demonstrated a clinically significant tolerance to the pulmonary effects, although daily use of salmeterol has resulted in a shorter duration of protection from exercise-induced bronchoconstriction (EIB). Regular use of a long-acting \( \beta_2 \) agonist is recommended for some patients with moderate to severe asthma.

upon the mechanism of action of the drug. According to the NHLBI recommendations, asthmatic patients with any level of persistent asthma require at least 1 drug from each pharmacologic category. From a therapy viewpoint, asthma drugs can be categorized differently: as drugs aimed at quick relief of acute asthma attacks or as drugs for long-term therapy to prevent recurrent attacks.

**BRONCHODILATORS**

**\( \beta_2 \) Agonists**

\( \beta_2 \) agonists are bronchodilators that act on the bronchial smooth muscle by combining with \( \beta_2 \)-adrenergic receptors to cause bronchial muscle relaxation and, thus, bronchodilation. Bronchodilation occurs regardless of the mechanism for the bronchial constriction, and \( \beta_2 \) agonists also provide protection against bronchial constriction; however, they do not significantly alter the progression of the inflammatory process. In other words, they dilate the bronchial muscle even though the inflammation is occurring.

Bronchial smooth muscle contains \( \beta_2 \)-adrenergic receptors, whereas the heart contains both \( \beta_1 \) receptors and \( \beta_2 \) receptors. Consequently, no therapeutic rationale exists for using nonselective \( \beta_2 \) agonists (ie, agents that are not selective for \( \beta_2 \) receptors relative to \( \beta_1 \) receptors); such agents (eg, isoproterenol, metaproterenol, epinephrine) have an increased incidence of sympathetic stimulatory effects, including increased heart rate and force of contraction. Since these cardiac effects could improve exercise performance, nonselective \( \beta \) agonists are not allowed in Olympic competition, and only selected \( \beta_2 \) agonists (albuterol, terbutaline, salmeterol) by inhalation are allowed upon written notification before competition. The \( \beta \)-agonist components of over-the-counter oral and inhalation asthma products are nonselective and, thus, a poor choice to treat asthma.

Inhaled \( \beta_2 \) agonists are the only agents providing an immediate response for acute asthma attacks (rescue therapy). They are also the most effective medication to prevent an anticipated attack, such as immediately before exercise, without the onset of significant cardiac or other systemic effects. After inhalation of short-acting \( \beta_2 \) agonists, onset of action is usually within 5 minutes, and maximal bronchodilation occurs within about 15 minutes. Duration of action is 4 to 8 hours, depending upon the agent, although a shorter period of protection (2 to 4 hours) is experienced during exercise. Table 3 lists some short-acting \( \beta_2 \)-adrenergic agonists and a typical adult dose. When given at equipotent doses, all of the \( \beta_2 \) agonists will produce the same intensity of bronchodilation; their main difference is the duration of action. Some agents are effective orally, but the oral route delays onset of action and is less selective for bronchial muscle, thus increasing the incidence of side effects.

Salmeterol is a long-acting \( \beta_2 \) agonist available by inhalation, but its slower onset of action (up to 20 minutes) and time for maximal effect (1 to 4 hours) preclude its use as rescue therapy for treatment of acute attacks. Users of salmeterol must clearly understand that it is not effective for treatment of acute bronchospasms and that the dose should not exceed 2 puffs every 12 hours. Depending upon the duration of the athletic activity, some athletes may benefit from salmeterol, which has a longer duration of action (12 hours) as compared with albuterol (4 hours). Patients who suffer from nocturnal asthma may also benefit from the longer-acting \( \beta_2 \) agonist.

Regularly scheduled, daily use of a short-acting \( \beta_2 \) agonist is generally not recommended, since there is no apparent advantage over use on an as-needed basis. If the frequency of \( \beta_2 \)-agonist use increases or if use exceeds 1 canister of \( \beta_2 \) agonist (eg, 200 puffs of albuterol) per month to control exacerbations, asthma control is poor, and reevaluation of the anti-inflammatory therapy is necessary. Regular use of short-acting \( \beta_2 \) agonists has not demonstrated a clinically significant tolerance to the pulmonary effects, although daily use of salmeterol has resulted in a shorter duration of protection from exercise-induced bronchoconstriction (EIB). Regular use of a long-acting \( \beta_2 \) agonist is recommended for some patients with moderate to severe asthma.
especially to control nighttime symptoms, in conjunction with corticosteroid therapy.

The most frequent side effects from inhalation of β₂ agonists are tachycardia and muscle tremor, although these are more pronounced with the nonselective agents and with oral use. During an acute asthma attack, the dose of the short-acting β₂ agonists can be increased severalfold to counter the bronchoconstriction without toxicity. However, if an athlete has escalating symptoms of asthma that are no longer being alleviated by the normal regimen of β₂ agonist, adjustment of the anti-inflammatortherapy may be required.

Albuterol, like many drugs, including all currently available selective β₂ agonists, is an equal mixture of R and S isomers. These isomers are mirror images of each other and interact differently at receptor sites. The R isomer, originally thought to be the only bioactive form, produces virtually all of the bronchodilation; the S isomer appears to contribute to some adverse effects (eg, nervousness and tremor) and has a longer duration because it is metabolized more slowly. Levalbuterol (Xopenex, Sepracor Inc, Marlborough, MA) is (R)-albuterol and is available for use with a nebulizer. Since levalbuterol contains only the active isomer, it provides comparable or better FEV₁ values than albuterol, with a lower incidence of side effects and at a smaller dose. R isomers of other β₂ agonists are also being investigated.29–31

Anticholinergics

Another group of bronchodilators is the anticholinergic agents. Rather than activating adrenergic receptors, these drugs inhibit the cholinergic receptors of the parasympathetic system, which, through the vagus nerve, maintain normal bronchial smooth muscle tone.4 The use of these agents has diminished over the years as the inhaled β₂ agonists have become available and because these agents cause significant anticholinergic side effects (eg, urinary retention, blurred vision, nasal congestion) and sedation through their central nervous system actions. Part of the bronchoconstriction caused by some asthma-inducing stimuli is mediated through the parasympathetic system, but the extent of this involvement varies significantly among patients. Anticholinergics are only effective in reducing bronchoconstriction mediated through this system.

Ipratropium bromide is a newer anticholinergic agent without sedative properties due to poor distribution into the central nervous system. It is available by inhalation for a more selective response. Onset of action is slower than that for the β₂ agonists, and peak bronchodilation occurs in 1 to 2 hours. The effectiveness of ipratropium varies considerably among asthmatic patients, although it does provide additional bronchodilation when combined with a short-acting β₂ agonist. It has limited effectiveness in preventing EIB.25–27,29

Methyloxanthines

Theophylline is a methylxanthine bronchodilator. Caffeine and theobromine are also members of this chemical group, but they are not used therapeutically to treat asthma since they have only a mild bronchodilating effect. The mechanism by which theophylline relaxes bronchial smooth muscle is not clearly understood, but the inhibition of phosphodiesterase and subsequent increase in intracellular cyclic adenosine monophosphate may be a contributor.4

Theophylline is used orally since it is not effective by inhalation, and it is available in immediate and sustained-release formulations. A major disadvantage of theophylline is that it has a narrow therapeutic window; that is, the blood level between too little and too much is narrow. Consequently, routine monitoring of theophylline blood levels is a standard procedure to prevent toxicity during long-term use. Potential side effects at the upper end of the normal therapeutic blood level include anorexia, nausea, vomiting, headache, and anxiety. As blood levels increase, seizures, arrhythmias, and death can occur. Complicating the problem is patient variability in the rate of excretion by the kidney and the potential for several other drugs (eg, erythromycin, cimetidine, zileuton) to alter the metabolism rate of theophylline by the liver, thus intensifying the need for blood-level monitoring.4,12,32

For most patients, theophylline is less effective than other bronchodilators. Nonetheless, long-term use at appropriate steady-state blood levels will maintain significant bronchodilation, and it is recommended as a second or third line of treatment for moderate to severe asthma in adults and children and to treat nocturnal asthma.3 For patients who do not respond to β₂-agonist therapy for prevention of EIB, theophylline offers an alternative. Long-term therapy is effective in preventing EIB, and, for some patients, protection from EIB is obtained from a single dose of theophylline before exercise.33

**ANTI-INFLAMMATORY AGENTS**

Since asthma is a chronic inflammatory disease, long-term use of anti-inflammatory agents is an important part of therapy.

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**Table 3. Characteristics of Some Drugs for Relief of Acute Symptoms**

<table>
<thead>
<tr>
<th>Generic Name</th>
<th>Trade Name</th>
<th>Category</th>
<th>Formulation; Typical Adult Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albuterol</td>
<td>Ventolin</td>
<td>β₂ agonist, SA*</td>
<td>MDI*: 2 puffs (90 mcg each) tid to qid prn*</td>
</tr>
<tr>
<td>Bitolterol</td>
<td>Tornalate</td>
<td>β₂ agonist, SA</td>
<td>DPI*: 1 to 2 capsules (200 mcg each) qid to q4h prn*</td>
</tr>
<tr>
<td>Ipratropium</td>
<td>Atrovent</td>
<td>Anticholinergic</td>
<td>MDI: 2 puffs (370 mcg each) tid to qid</td>
</tr>
<tr>
<td>Methylprednisolone</td>
<td>Medrol</td>
<td>Corticosteroid</td>
<td>MDI: 2–3 puffs (18 mcg each) qid</td>
</tr>
<tr>
<td>Pirbuterol</td>
<td>Maxair</td>
<td>β₂ agonist, SA</td>
<td>Oral: 40 to 60 mg/day for 3 to 10 days</td>
</tr>
<tr>
<td>Prednisolone</td>
<td>Prelone</td>
<td>Corticosteroid</td>
<td>MDI: 2 puffs (200 mcg each) tid to qid</td>
</tr>
<tr>
<td>Prednisone</td>
<td>Prednisone</td>
<td>Corticosteroid</td>
<td>Oral: 40 to 60 mg/day for 3 to 10 days</td>
</tr>
<tr>
<td>Terbutaline</td>
<td>Brethaire</td>
<td>β₂ agonist, SA</td>
<td>MDI: 2 puffs (200 mcg each) tid to qid prn</td>
</tr>
</tbody>
</table>

* SA, short acting.  
† MDI, metered dose inhaler.  
‡ prn, as needed for relief.  
§ DPI, dry powder inhaler.
to control inflammation and to prevent exacerbations. The cellular mechanism for airway inflammation includes the activation of phospholipase, an enzyme that releases arachidonic acid from membrane-bound phospholipid in the mast cell (Figure). Arachidonic acid is the precursor to prostaglandins and leukotrienes, which are mediators released by several cell types, including T lymphocytes, macrophages, and mast cells of the lung. These mediators contribute to bronchoconstriction, edema, and mucous production. These cells also release an array of other compounds (eg, histamine, platelet-activating factor, cytokines) that contribute to the inflammatory process, which includes increased vascular permeability, increased mucus secretion, and structural changes in the airways. The anti-inflammatory drugs affect 1 or more of the steps in the inflammatory process, thereby diminishing the destructive effects of chronic inflammation. Use of corticosteroids early in the disease can preserve lung function for a longer time compared with delayed use of these drugs.13

Anti-inflammatory agents can be grouped in the following categories: corticosteroids, mast cell-stabilizing agents, and antileukotrienes. Specific agents in all 3 groups are used as long-term therapy to prevent the onset of recurring asthma symptoms and to reduce the frequency of acute exacerbations, thus also reducing β2-agonist usage for quick relief. Poor asthma outcomes are often a result of underuse of these agents for long-term therapy.6

Corticosteroids

The corticosteroids have multiple mechanisms of action that contribute to their use in asthma. For example, they inhibit the production of prostaglandins and leukotrienes by inhibiting the action of phospholipase; inhibit cytokine gene transcription; and increase gene transcription of β receptors, which increases the responsiveness to β agonists.13,19 These drugs are effective in suppressing inflammation when used on a regular dosing schedule as long-term therapy.

Inhaled corticosteroids are recommended as the first-line agents to control mild, moderate, and severe asthma, with dosage adjustment according to the severity of the disease.3 Benefit from inhalation corticosteroid therapy occurs over several weeks and may require 3 months for maximal effect.13 Consequently, inhalation corticosteroids are not beneficial on an as-needed basis to prevent EIB or to treat acute attacks (rescue therapy). As the cornerstone of long-term anti-inflammatory therapy, however, inhaled corticosteroids have been shown to reduce both symptoms and the number of acute exacerbations of chronic asthma, thus also reducing the reliance on β2 agonists. In addition, long-term use of inhaled corticosteroids is effective in managing nocturnal asthma and in providing protection against EIB symptoms.13,20,33,34

The potential for systemic side effects is minimized with long-term use of inhaled compared with oral corticosteroids and even more so with lower doses of inhaled corticosteroids.13,20,35 There is some concern that inhaled corticosteroids may reduce linear growth or delay growth in children, but generally the benefits of low-dose inhaled corticosteroids outweigh the risks.3 The most frequent side effects of inhaled corticosteroids are dysphonia (hoarseness), cough, and the potential for the development of oral fungal (candidiasis) infection.13 The incidence of these effects depends upon the total daily dose of corticosteroid in the oropharynx. Consequently, the incidence can be significantly reduced by the use of a spacer and by rinsing the mouth with water and spitting after every use; these procedures also reduce the amount of corticosteroid swallowed, which may otherwise contribute to systemic effects.13,14,19 The cough may be due to throat irritation from additives in the MDI and is less frequent with DPI.13 For some asthmatic patients, once-per-day, low-dose inhalation of corticosteroids may provide appropriate management of stable, mild-to-moderate asthma and may further diminish side effects.36

At equipotent doses, generally no corticosteroid appears to be significantly more efficacious than another.13,20 However, the 2 newest inhaled corticosteroids, budesonide and fluticasone, are potent and consequently require fewer puffs per day, thus providing an advantage for compliance in patients who require higher doses of inhaled corticosteroids. Budesonide and fluticasone also have the advantage of rapid metabolism by the liver compared with other inhaled corticosteroids, which reduces the potential for systemic side effects, especially in patients who require higher dosages. Additionally, fluticasone is poorly absorbed orally, thus further diminishing the potential for systemic side effects from drug swallowed after inhalation.13,14

Long-term use of oral corticosteroids is not recommended except for the control of severe asthma. Long-term oral use for treatment of severe persistent asthma presents the potential for an array of systemic side effects, such as fluid and electrolyte abnormalities, hyperglycemia, behavioral disturbances, osteoporosis, fat redistribution, cataracts, glaucoma, and growth suppression. Short-term use of oral or parenteral corticosteroids, however, is useful as a beneficial adjunct to β agonists for the treatment of acute exacerbations. The duration of systemic therapy for treatment of acute exacerbations depends
upon the patient’s response, but 3 to 10 days of therapy can reduce the effects of acute episodes. These short bursts of systemic corticosteroid therapy do not produce serious toxicities, although mood disturbances and loss of glucose control in diabetic patients may occur. Any adrenal suppression (ie, decrease in physiologic production of steroids) is readily reversible in a few days after therapy.4,20 Tapering of corticosteroid therapy is unnecessary in acute asthma therapy.20,37

Because systemic corticosteroids have an array of metabolic effects, some of which could provide an advantage in competitive sports, their use is carefully monitored. However, inhalation corticosteroids (eg, beclomethasone, budesonide, flunisolide, fluticasone, triamcinolone) are permissible in Olympic competition upon written notification before competition.31

Mast Cell-Stabilizing Agents

Two mast cell-stabilizing agents (also referred to as khellin derivatives) are currently available: cromolyn and nedocromil. These drugs are only effective by inhalation and are used primarily to prevent allergen-induced bronchospasm and EIB. They are most effective in maintaining protection when used 2 to 4 times daily, and although initial improvement is observed in 1 to 2 weeks, about 4 weeks of therapy are necessary to obtain maximal benefit.3,4 Since they do not produce bronchodilation, mast cell-stabilizing agents are not effective in alleviating acute asthmatic attacks.

Although the exact mechanism of action of the mast cell-stabilizing agents is unknown, they appear to stabilize the membrane to prevent the release of inflammatory mediators (eg, leukotrienes, prostaglandins, cytokines) from the cell. These mediators play an important role in the hyperreactivity response, especially as a result of exposure to allergens and exercise.4,12,20

At equipotent doses, cromolyn and nedocromil are equally effective. They are not as effective as inhaled corticosteroids for the prevention of asthma symptoms, but they have fewer potential side effects and virtually no systemic toxicity.3,4 These drugs are recommended for prevention of EIB and are frequently added if inhaled β2 agonist alone before exercise is insufficient to block the bronchospasms. They are also used for the treatment of mild persistent asthma, especially in children, since they lack the potential to delay growth.10,33 Reduced effectiveness with daily use has not been demonstrated. Some patients experience minor mouth and throat irritation, which can be alleviated by drinking water immediately after use. Additionally, some patients complain that nedocromil has such a bad taste that they discontinue the therapy.

Antileukotrienes

The antileukotrienes (also known as leukotriene modifiers) are the newest group of antiasthma drugs available. As shown in the Figure, these drugs either decrease the synthesis of cysteinyl leukotrienes by inhibiting 5-lipoxygenase (eg, zileuton) or by inhibiting the binding of leukotrienes at the receptor (eg, montelukast, zafirlukast).26,38,39 In either case, these agents diminish the effect of leukotrienes, which contribute to bronchoconstriction, edema, and mucous production. Antileukotrienes are effective for long-term control of mild to moderate asthma, especially in asthma induced by allergens, exercise, and aspirin.4,26,40 These drugs are only available for oral use and are not effective to treat acute asthma attacks.39,41

Use of these agents may allow for decreased dose of corticosteroids and decreased reliance on β-agonist inhalers.38 All 3 of the antileukotrienes are well tolerated orally. The most frequent side effects have been headaches and gastrointestinal disturbances. A potential problem associated with zileuton is an increase in blood alanine aminotransferase, an indicator of potential liver toxicity. Consequently, alanine aminotransferase should be checked monthly for the first 3 months and then every other month for the first year to monitor for hepatic toxicity.39 In addition, Churg-Strauss syndrome has been associated with the use of leukotriene receptor antagonists in a few patients. This syndrome is a vasculitis that primarily affects the respiratory tract during the early stages and can develop into a life-threatening systemic vasculitis.39,42-45 Most, but not all, of these reports have been in patients being withdrawn from corticosteroid therapy after being maintained on a leukotriene receptor antagonist. Therefore, there is some question as to whether undiagnosed Churg-Strauss syndrome existed before antileukotriene therapy and the symptoms, which include asthma, were masked by the corticosteroid therapy.

In addition to side effects, other differences exist among the antileukotrienes. The oral absorption of zafirlukast, the first of the leukotriene receptor antagonists approved for asthma therapy, is reduced significantly by food, and, thus, the medication should be taken either 1 hour before or 2 hours after meals. It has been approved for use in children as young as 7 years old. Zafirlukast acts as an inhibitor of P450 enzymes; these enzymes are important for the metabolism (inactivation) of many drugs. Consequently, the potential exists for zafirlukast to increase the blood level of other drugs as a result of the inhibitory action on the P450 enzymes. Warfarin is a drug that is metabolized by these same P450 enzymes, and, as a result, drug-dosing modifications may be necessary when patients are taking zafirlukast concurrently with warfarin or other drugs metabolized by selected P450 enzymes.32,39

Zileuton, the inhibitor of 5-lipoxygenase, is similar to zafirlukast in that it inhibits the ability of P450 enzymes to metabolize many drugs, such as warfarin, but also theophylline. Zileuton should not be used in children less than 12 years old. The absorption of zileuton from the gastrointestinal tract is not affected by food.26

Montelukast, like zafirlukast, inhibits the binding of leukotrienes to the receptor. Montelukast can be taken without regard to food, can be used to treat children at least 6 years old, and is available as a chewable tablet. Unlike zileuton and zafirlukast, montelukast does not inhibit the cytochrome P450 enzymes and, thus, does not have the same potential for drug interactions.26,38,46

Since antileukotrienes are relatively new agents for the treatment of asthma, their usefulness has not been clearly identified. Some patients respond well to therapy with these agents, whereas others do not. The reason for this difference is not certain, but the agents causing the asthma symptoms in some patients may not significantly affect the leukotriene pathway; other asthmatic patients may have a genetic disposition determining their response to these drugs.47-49 For example, asthma patients with diminished expression of the 5-lipoxygenase gene may not respond adequately to drug therapy targeting that pathway alone.47 Alternatively, some patients with aspirin-induced asthma (AIA) may have an enhanced expression of 5(S)-hydroxy-6(R)-S-glutathionyl-1,7,9-
THERAPY CONSIDERATIONS

General Principles

Several nondrug measures should always be a part of asthma therapy. Physical training, for example, has been shown to decrease the severity of asthmatic symptoms other than EIB and to reduce the number of emergency room visits.[50-52] Adequate education concerning the proper use of the MDI and the PFM can also reduce symptoms of asthma. A seemingly obvious nondrug measure is to avoid contact with stimuli that have the potential to initiate asthma symptoms. These causative agents include animal allergens from pets, indoor fungi, house dust mites, tobacco smoke, viral infections, outdoor allergens such as pollen, and occupational exposure to chemical irritants. Patients can be tested to assess their sensitivity to various allergens and irritants. Athletes who exercise outdoors and are hyperresponsive to outdoor allergens can reduce their exposure to these allergens by adjusting their exercise routine to an indoor, air-conditioned environment during peak pollen season or by avoiding outdoor exercise during midday, when allergen levels are typically higher.3

Along with animal and environmental stimuli of hyperresponsiveness in the asthmatic patient, from 3% to 39% of asthmatic patients have AIA. The incidence of AIA increases with age and with asthma severity.4,10,12,49 These asthmatics are also likely to be sensitive to other NSAIDs. Care should be taken when selecting over-the-counter analgesic products, since NSAIDs are components of many such products (Table 4).22 Aspirin desensitization is an option for people who require routine therapy with NSAIDs. The use of acetaminophen may be effective for treatment of minor pain for athletes with AIA. However, in 1 study,53 one third of asthmatics with AIA who were treated with 1000 to 1500 mg of acetaminophen experienced some degree of cross-reaction with acetaminophen. The bronchospastic reactions were dose dependent and milder than when induced by aspirin. Consequently, the authors indicate that the use of 650 mg or less of acetaminophen poses only a small risk of bronchospasm. In addition to being a major ingredient in many over-the-counter analgesics products, acetaminophen is also a component of many cold, cough, and allergy medications, although the amount of acetaminophen is usually not over 650 mg per dosage unit.22

When reviewing the athlete's asthma drug therapy, it is beneficial to group the drugs in therapeutic categories of agents either for quick relief (Table 3) or for long-term therapy to prevent recurring attacks (Table 5). As indicated in Table 2, the NHLBI guidelines10 recommend a stepwise course of long-term therapy based upon the severity of the asthma. The initial objective of this therapy is to gain control of the asthma symptoms by using sufficiently aggressive drug therapy. Subsequently, treatment should be reviewed and drug therapy gradually reduced to the least medication necessary to maintain control. For mild to moderate persistent asthma, use of a short-acting $\beta_2$ agonist on a daily basis or more than 3 to 4 times in 1 day indicates a need for a step up in therapy, assuming the patient is compliant with prescribed therapy, has

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Table 4. Examples of Over-the-Counter Products Containing Aspirin or Other NSAIDs22

<table>
<thead>
<tr>
<th>Brand-Name Product</th>
<th>Analgesic Component</th>
</tr>
</thead>
<tbody>
<tr>
<td>Actron</td>
<td>Ketoprofen</td>
</tr>
<tr>
<td>Advil Cold &amp; Sinus</td>
<td>Ibuprofen</td>
</tr>
<tr>
<td>Aleve</td>
<td>Naproxen</td>
</tr>
<tr>
<td>Alka-Seltzer Plus Sinus Medicine</td>
<td>Aspirin</td>
</tr>
<tr>
<td>Anacin</td>
<td>Aspirin</td>
</tr>
<tr>
<td>Arthritis Pain Formula</td>
<td>Aspirin</td>
</tr>
<tr>
<td>Ascriptin Regular Strength</td>
<td>Aspirin</td>
</tr>
<tr>
<td>BC Sinus-Cold</td>
<td>Aspirin</td>
</tr>
<tr>
<td>Buffaprin</td>
<td>Aspirin</td>
</tr>
<tr>
<td>Buffasal</td>
<td>Aspirin</td>
</tr>
<tr>
<td>Buffets II</td>
<td>Aspirin</td>
</tr>
<tr>
<td>Bufferol</td>
<td>Aspirin</td>
</tr>
<tr>
<td>Cope</td>
<td>Aspirin</td>
</tr>
<tr>
<td>Dristan Sinus</td>
<td>Ibuprofen</td>
</tr>
<tr>
<td>Ecotrin Regular Strength</td>
<td>Aspirin</td>
</tr>
<tr>
<td>Excedrin Extra Strength</td>
<td>Aspirin</td>
</tr>
<tr>
<td>Excedrin Migraine</td>
<td>Aspirin</td>
</tr>
<tr>
<td>Motrin IB</td>
<td>Ibuprofen</td>
</tr>
<tr>
<td>Nuprin</td>
<td>Ibuprofen</td>
</tr>
<tr>
<td>Orudis KT</td>
<td>Ketoprofen</td>
</tr>
<tr>
<td>Ultraprin</td>
<td>Ibuprofen</td>
</tr>
<tr>
<td>Valprin</td>
<td>Ibuprofen</td>
</tr>
<tr>
<td>Vanquish</td>
<td>Aspirin</td>
</tr>
</tbody>
</table>

* LA, long acting.

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Table 5. Characteristics of Some Drugs for Long-Term Asthma Control2,20,26

<table>
<thead>
<tr>
<th>Generic Name</th>
<th>Trade Name</th>
<th>Category</th>
<th>Formulation; Typical Adult Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albuterol</td>
<td>Proventil</td>
<td>$\beta_2$ agonist, LA*</td>
<td>Oral; extended release; 4 mg q12h</td>
</tr>
<tr>
<td>Beclomethasone</td>
<td>Beclovent</td>
<td>Corticosteroid</td>
<td>MDI; 6 to 10 puffs/day (84 mcg each)</td>
</tr>
<tr>
<td>Budesonide</td>
<td>Pulmicort Turbuhaler</td>
<td>Corticosteroid</td>
<td>DPI; 2 to 3 inhalations/day (200 mcg each)</td>
</tr>
<tr>
<td>Cromolyn</td>
<td>Intal</td>
<td>Mast cell stabilizer</td>
<td>MDI; 2 to 4 puffs (800 mcg each) tid to qid</td>
</tr>
<tr>
<td>Flunisolide</td>
<td>AeroBid</td>
<td>Corticosteroid</td>
<td>MDI; 4 to 8 puffs/day (250 mcg each)</td>
</tr>
<tr>
<td>Fluticasone</td>
<td>Flovent</td>
<td>Corticosteroid</td>
<td>MDI; 2 to 6 puffs/day (110 mcg each)</td>
</tr>
<tr>
<td>Fluticasone</td>
<td>Flovent Rotadisk</td>
<td>Corticosteroid</td>
<td>DPI; 3 to 6 inhalations/day (100 mcg each)</td>
</tr>
<tr>
<td>Montelukast</td>
<td>Singulair</td>
<td>Antileukotriene</td>
<td>Tablets; 10 mg qd</td>
</tr>
<tr>
<td>Nedocromil</td>
<td>Tilaide</td>
<td>Mast cell stabilizer</td>
<td>MDI; 2 to 4 puffs (1.75 mg each) bid to qid</td>
</tr>
<tr>
<td>Salmeterol</td>
<td>Serenvent</td>
<td>$\beta_2$ agonist, LA</td>
<td>MDI; 2 puffs (21 mcg each) q12h</td>
</tr>
<tr>
<td>Salmeterol</td>
<td>Serenvent Diskus</td>
<td>$\beta_3$ agonist, LA</td>
<td>DPI; 1 inhalation (50 mcg) every 12 hours</td>
</tr>
<tr>
<td>Theophylline</td>
<td>Theo-Dur</td>
<td>Methylxanthine</td>
<td>Oral; extended release; 300 mg bid</td>
</tr>
<tr>
<td>Trimcinolone</td>
<td>Azmacort</td>
<td>Corticosteroid</td>
<td>MDI; 10 to 20 puffs/day (100 mcg each)</td>
</tr>
<tr>
<td>Zafirlukast</td>
<td>Accolate</td>
<td>Antileukotriene</td>
<td>Tablets; 20 mg bid</td>
</tr>
<tr>
<td>Zileuton</td>
<td>Zyflo</td>
<td>Antileukotriene</td>
<td>Tablets; 600 mg qd</td>
</tr>
</tbody>
</table>

* LA, long acting.
appropriate inhaler technique, and is avoiding known precipitating factors. Inhaled β₂ agonists are the drugs of choice for quick relief of acute attacks, whereas inhaled corticosteroids are the drugs of choice for long-term control. The addition of another anti-inflammatory agent to inhalation corticosteroid therapy may allow for a reduction of the corticosteroid dose or may be necessary without reduction of the corticosteroid dose to achieve desired control.

**Exercise-Induced Bronchoconstriction (EIB)**

Since most asthmatic patients (70% to 90%) experience some degree of EIB, the treatment of athletes for EIB is of obvious importance to the athletic trainer. Of additional importance is that many athletes have no other history of asthma and are thus unaware that they have EIB. Symptoms of airway obstruction typically occur as a result of 5 to 8 minutes of strenuous exertion, but the time period of exercise-free symptoms can be extended somewhat with a warm-up period of 15 to 30 minutes of submaximal exercise. Maximal airway obstruction occurs 5 to 15 minutes after exercise cessation. Symptoms include coughing, wheezing, prolonged recovery times after exercise, and chest tightness. Airflow returns to baseline levels during the following 20 to 60 minutes. Some athletes experience a subsequent refractory period of 2 to 4 hours, in which exercise results in diminished bronchoconstriction, possibly due to depletion of mast cell mediators. A late asthmatic response occurs 3 to 9 hours after exercise causes additional bronchoconstriction in some athletes, but is typically less severe.

In athletes with EIB, inhalation of a β₂ agonist 5 to 15 minutes before exercise offers protection. If symptoms develop during exercise, puffs can be repeated. Increased use of a β₂ agonist by an athlete could indicate a need for additional anti-inflammatory therapy. Use of salmeterol, a long-acting inhaled β₂ agonist, provides protection against EIB for as long as 12 hours, although long-term daily use has been shown to diminish the duration of effect. Nonetheless, a long-acting inhaled β₂ agonist may be specifically advantageous for the athlete who is active for longer periods. The mast cell-stabilizing agents, cromolyn and nedocromil, have also demonstrated good effectiveness in protecting against EIB when administered before exercise. The duration of protection is dose dependent but similar to that of short-acting β₂ agonists (about 2 to 4 hours), and the low incidence of side effects make them appealing. In addition, cromolyn and nedocromil can alleviate the late asthmatic response, which can be used in combination with a β₂ agonist for enhanced protection if the agent alone is not sufficient. The antileukotrienes have also shown protective effects against EIB; they have the convenience of oral use and no tolerance to the protective effects with long-term use. Inhaled corticosteroid given immediately before exercise is of no benefit, but daily, long-term use may reduce the severity of EIB. Factors that reduce the effectiveness of all drugs used to treat EIB are increased intensity and duration of the exercise and exercise in cooler, drier air.

Inhaled corticosteroids and albuterol, terbutaline, and salmeterol by inhalation are allowed in Olympic competition upon advance written notification. Cromolyn, nedocromil, ipratropium, theophylline, and all the antileukotrienes are allowed by the US Olympic Committee without prior notification. The National Collegiate Athletic Association permits most asthma medications except for oral β₂ agonists.

**Considerations for the Athletic Trainer**

The athletic trainer has the opportunity to play a key role in ensuring that the asthmatic athlete achieves the desired therapeutic outcomes, including minimizing the effect of asthma on athletic performance. To accomplish this, the athletic trainer should consider the following:

- Ensure that asthmatic athletes are aware that asthma is a chronic inflammatory disease requiring compliance to the prescribed drug therapy to obtain maximal benefit (eg, not skipping doses or taking daily medication on an as-needed basis).
- Be certain that athletes are using the inhaler device(s) properly, including a spacer if good inhalation technique is not being achieved with the MDI.
- Remind athletes of the appropriate use of a prescribed β₂-agonist or mast cell-stabilizing agent (or both) before exercise for EIB and the value of 15 to 30 minutes of submaximal warm-up activity.
- Refer the athlete to a physician if the asthma is not being controlled at an acceptable level. Be especially alert to overuse of β₂-agonist inhalers, which may signal the need for adjustment in long-term anti-inflammatory therapy.
- Observe the routine use of the PFM in athletes with moderate to severe asthma, ensure they understand the importance of tracking their PEF, assist them in monitoring the results, and know the predetermined plan for making appropriate adjustments if PEF is below 80% of the athlete’s personal best.
- Be sure that athletes are aware of the stimuli that have the potential to initiate asthmatic symptoms and that cooler, drier air and more strenuous exercise increase the severity of EIB.
- Be alert to athletes not diagnosed with EIB but experiencing symptoms, who may require evaluation for diagnosis and treatment.
- Ensure that athletes with AIA understand that aspirin and other NSAIDs, which can also be found in over-the-counter combination products, must be avoided.
- Monitor each athlete’s drug therapy to ensure that the medications being used are not banned at the athlete’s level of competition.
- Reassure asthmatic athletes that proper therapy can allow them to compete at the level of nonasthmatic patients. In the 1996 Summer Olympic Games, the US Olympic athletes who had been told that they had asthma or had taken asthma medications fared as well as nonasthmatic athletes in winning team and individual medals.

**REFERENCES**

Accreditation and Continuous Quality Improvement in Athletic Training Education

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Objective: To apply the continuous quality improvement model commonly associated with the business sector to entry-level athletic training education program accreditation.

Data Sources: We applied athletic training educational program accreditation as a tool for ensuring quality in the entry-level athletic training education programs accredited by the Commission on the Accreditation of Allied Health Education Programs. Literature from the business, education, and athletic training fields is integrated to support this paradigm shift in athletic training education.

Data Synthesis: The advent of mandated entry-level athletic training educational program accreditation has forced institutions to evaluate their educational programs. Accreditation will promote continuous quality improvement in athletic training education through mechanisms such as control measures and process improvement.

Conclusions/Recommendations: Although accreditation of entry-level athletic training education programs has created some dissonance among athletic training professionals, it will strengthen the profession as a whole. Athletic training educators must capture the synergy generated from this change to ensure quality educational experiences for all our students as we move forward to secure a strong position in the allied health care market.

Key Words: process improvement, Commission on Accreditation of Allied Health Education Programs (CAAHEP), Joint Review Committee on Educational Programs in Athletic Training (JRC-AT), leadership

According to Rankin and Ingersoll,1 management theory is an area of development that has been ignored in athletic training programs. However, many principles that govern business also apply to athletic training education. As entry-level athletic training programs change to meet accreditation standards, the concept of quality has come to the forefront. Athletic training is not alone in the quest for quality. In the past decade, American higher education has been challenged with issues regarding the quality of education. The American Association for Higher Education has responded by forming an Academic Quality Consortium to apply Total Quality Management (TQM) philosophies of the business world in the academic setting.2 In the health care and academic fields, TQM is commonly referred to as Continuous Quality Improvement (CQI): “There has been a steady shift away from the notion of TQM in education to more benign language emphasizing continuous quality improvement.”2 Regardless of the label, this movement has certainly been met with both enthusiasm and skepticism in the academy. As institutions are faced with the internal and external challenges of the 21st century, quality issues will certainly take a commanding position in the increasingly competitive environment of higher education and allied health care.

Under the Commission on Accreditation of Allied Health Education Programs (CAAHEP), athletic training education programs have aligned themselves with practices commonly used in the business sector. According to Green,3 program and institutional accreditation involves an assessment of quality. Gelmon4 contended that accreditation also serves to promote continuous improvement. Accreditation further ensures that CQI is accomplished through control measures and process improvement. Seymour2 captured the essence of educational reform by stating, “Once you declare an unshakable commitment to quality, you begin to realize tremendous synergy from the alignment of the individual goals with the process and institutional goals.”

The athletic training profession will be faced with many challenges in the new millennium. Ray5 proposed that athletic trainers can benefit from the foundation in management theory and practices. Securing a strong position in the highly competitive allied health care market will be facilitated by standardizing the educational preparation of entry-level athletic trainers through accreditation. The combined work of the National Athletic Trainers’ Association Board of Certification (NATABOC), former Professional Education Committee, and Education Council have provided critical information to guide professional preparation for athletic trainers. As we meet this professional challenge, a proactive approach to CQI in athletic training education will keep us focused on the future. The purpose of our article is to present entry-level athletic training education accreditation as a vehicle for CQI for the athletic training profession.

CQI and Educational Accreditation

CQI emerged from the pioneering works of W. Edwards Deming,6 Joseph M. Juran,7 and Philip B. Crosby.8 These leaders identified 4 central themes regarding the CQI philosophy: 1) outputs must conform to requirements and meet customer expectations; 2) monitoring and evaluating are prospective as well as retrospective; 3) quality is a total organizational responsibility; and 4) quality improvement focuses on...
program standards that identify deficiencies through continuous improvement assessment and make modifications to improve the quality of the outcome. Institutional or program accreditation serves as a stimulus for change as internal and external pressures to provide quality education continue to increase. Athletic training is feeling the effects of these pressures.

Accreditation involves a process that serves to achieve several goals. Young defined accreditation as “a process that attempts to evaluate and encourage institutional quality,” and Seldon defined accreditation as “the process whereby an organization or an agency recognizes a college or university or program of study as having met certain predetermined qualifications or standards.” Accreditation is a means of standardizing athletic training educational programs that facilitates efforts to promote quality in the athletic training profession as a whole. The process-oriented approach of accreditation clearly blends with the basic tenets of CQI.

The educational preparation of allied health care providers is a critical issue in professional development. Accreditation ensures that standards have been achieved while fostering CQI: “Ongoing assessment of both students and institutions is proposed as the mechanism to attain and maintain quality.” Initial and periodic review of educational program objectives and outcomes are required for CAAHEP-accredited programs to facilitate CQI.

HISTORICAL PERSPECTIVES ON ATHLETIC TRAINING EDUCATION PROGRAMS

In the ever-changing field of health care, athletic trainers have chosen to compete with other allied health care professionals for a legitimate place as a provider for health care services. By expanding beyond the traditional athletic training settings, athletic trainers have broadened their role in the allied health care community. Until the recent ruling by the National Athletic Trainers’ Association (NATA) defining CAAHEP-accredited programs as the only route to certification by the year 2004, 2 predominant models of education were acceptable preparation for the NATABOC examination: the curriculum route and an internship route, both of which qualify individuals for eligibility via national certification. Because of the variation between the 2 models, athletic training education has fought an uphill battle due to the lack of standardization in the educational programs that produce certified athletic trainers. Although certification serves as a “process by which a nongovernmental agency or association grants recognition to someone who meets its qualifications,” the dual educational routes have resulted in a lack of credibility for athletic training professionals in the allied health care industry. In an effort to standardize athletic training education programs, CAAHEP has been appointed the task of granting accreditation to programs upon the recommendation of the Joint Review Committee on Educational Programs in Athletic Training (JRC-AT). Pete Carlson, former District 6 representative to the NATA Board of Directors, stated: “This landmark decision will affect education significantly into the millennium. It’s a major reform in how we prepare individuals to meet the demands of the profession, both entry-level and continuing education.”

Historically, the NATA granted program recognition through internal approval. As a subcommittee of the original NATA Committee on Gaining Recognition, the NATA Professional Education Committee evolved to grant approval to undergraduate athletic training curriculum programs. For approximately 3 decades, the Professional Education Committee continued to grant approval until the American Medical Association’s Commission on Allied Health Education and Accreditation (CAHEA) was selected as the accrediting body for athletic training education. The JRC-AT was established at this time to “develop standards and guidelines governing JRC-AT review and CAHEA accreditation of entry-level programs.” A new independent agency, CAAHEP, was later selected as the accrediting board for athletic training education in 1994 when CAHEA was disbanded.

In 1994, the NATA Board of Directors created an Education Task Force to specifically address the educational issues that would carry the NATA into the 21st century. This task force included members of the Board of Certification, athletic training educators, and athletic trainers from both internship and curriculum settings. This task force used the problem-solving model to formulate 18 initiatives for reform. In 1997, the task force had met its mission and was then discharged of its duties. Based on the recommendations of the task force, an Education Council was established. This 45-member Education Council was appointed by the Board of Directors to activate a quality control model focusing on the educational preparation of athletic trainers.

If quality is meeting the requirements set forth by the institution, then quality can only be measured if the requirements are known and defined. Accreditation serves this function to define the structure and expected outcomes of athletic training education programs. By using accreditation as the standard, Ray stated that the public and other members of the health care community will know that “when they work with an athletic trainer, they are always going to be working with a person of high quality and a person who has been educated to a very rigorous standard.”

ACCREDITATION PROCESSES AND IMPLICATIONS

Accreditation will require all athletic training education programs to follow the curriculum rather than the internship model to qualify the graduates of these programs to meet the eligibility criteria for the national certification examination. Traditionally, the internship models required extensive clinical hours (1500 minimum) and limited course work. Curriculum programs, on the other hand, offer expanded content areas and 800 minimum hours of clinical experience. One of the motivating factors in athletic training education reform stems from the difficulty in controlling “clinical experiences” and the acquired knowledge base from the internship experiences.

The disparity in educational preparation from dual certification routes will be eliminated as CAAHEP accreditation is required of entry-level programs in the near future. Standardization through accreditation has profound implications for athletic trainers in areas such as third-party reimbursement and licensure. The Education Task Force in the Recommendations to Reform Athletic Training Education introduced several major issues beyond entry-level educational accreditation that deal with CQI: quality education “across the life span,” continuing competence for practicing athletic trainers, and post-entry-level competencies. Further, the NATABOC will...
continue to require ongoing assessment of continuing education units for certified professionals in the field. Educational accreditation will serve as a tool to ensure quality in educational preparation that will affect athletic trainers far beyond the classroom.

"Accrediting agencies have the responsibility to hold programs accountable for clear statements of expectations for student learning, appropriate assessment programs to determine whether those expectations are being met, and a systematic effort to improve learning based on the results."17 Essentially, control measures of accrediting agencies through review processes are used to promote quality in a manner similar to that in which business applies these measures.

The actual accreditation process for educational programs in athletic training is a voluntary process that must be initiated by the chief academic officer at the institution. The process involves the completion of an in-depth self-study report, which is the crux of the peer-review system of accreditation. “This process critically examines the program in structure and substance, judges the program’s overall effectiveness relative to its mission, identifies specific strengths and deficiencies, and indicates a plan for necessary modifications and improvements.”18 Following completion of the self-study, an on-site visitation is arranged to validate the report and to assess the program’s compliance with the standards of the profession, the Standards and Guidelines.19 “These Standards are the minimum level of quality used to accredit programs that prepare individuals to enter Athletic Training.”19 Being under the jurisdiction of CAAHEP accreditation, the NATA has joined other prestigious organizations such as the American Academy of Family Physicians, the American Academy of Pediatrics, and the American Orthopaedic Society for Sports Medicine that have also adopted the Standards. These Standards, adopted in 1991, are the minimum requirements accredited programs must meet.19

Following the on-site visitation, a findings response is sent to the program director and department chair from the JRC-AT. In addition to other factors, this report indicates whether there were any violations of standards. Each program can provide a program response to be considered before the accreditation recommendation to the CAAHEP. Initial accreditation is granted for no more, and sometimes less, than 5 years. Continuing accreditation is granted for a maximum of 7 years, but it may be less. Once a program is accredited, program reviews and administrative functions prescribed by the JRC-AT must be carried out within specifications to maintain the accredited status.19

**CONTROL MEASURES**

The process model in Figure 1 depicting a business concept for control measures can be related directly to athletic training educational programs. The education of athletic trainers requires input resources (ie, faculty, facilities, and students), which then are integrated in a process (curriculum) resulting in outputs. The outputs are intended to be competent, fully prepared graduates who are ready to take the national certification examination and enter the work force. Each of these components are evaluated in the accreditation review process. Faculty roles, facilities, and student qualifications are assessed. Additionally, curriculum design, sequence, and delivery are elements for review in the self-study and on-site visitation. Lastly, graduate and employer satisfaction, as well as certification results, are considered critical outcomes in the educational process.

In education, CQI and control involve gathering information from the input, process, and output aspects of the institution to modify the inputs or processes to ensure quality educational practices (Figure 1). Control involves monitoring activities and comparing actual results with expected results. When appropriate, intervention occurs in the form of corrective action by changing inputs or processes. In accreditation, output control focuses on the quantity and quality achieved for objectives such as stakeholder satisfaction, graduates employed, and retention rates. Process control deals with the conversion of inputs to outputs. Rakich et al12 claimed that “standards and expectations are easier to develop for processes that deal with things, are consistent, and are simple to document and understand.” Accreditation ensures that the processes are consistent and clear. Last, input control involves using the best inputs before conversion to avoid output problems.12 Although all athletic training instructors and program directors are required to be credentialed by the NATA, educational programs such as Approved Clinical Instructor workshops are being developed by the Education Council to control the quality of inputs and, thus, the final outcomes.

Control measures are becoming more prominent in the educational setting. In education, the products are the new skills and knowledge acquired by the student. “For a person engaged in the acquisition of new skills and knowledge, the process of learning is very significant. Therefore, quality is sought in the service that is the provision of an environment that enables the new skills and knowledge to be acquired.”20 Quality must be defined and measured. By defining requirements, the standards to be achieved are set. Educational accreditation ensures that quality assurance is at the core of the educational process through initial accreditation as well as through re-accreditation reviews. Once accredited, a standard of quality is set that must be monitored and maintained.

Johnson and Dumas21 presented an interesting model for quality control that parallels the manufacturing approach in business but integrates specific nuances for nonmanufacturing environments such as education. Their approach is basically a 4-step process. The first step is to empower the people in the organization to obtain their commitment. This involves an explicit understanding of the outcomes and the processes used. Educational reform has empowered athletic trainers via the efforts of the Education Task Force and the Education Council to commit to a single route to certification through program accreditation. Second, this model advocates the process of remediation for commitment failures such as employee training.

![Figure 1. From JS Rakich, BB Longest, and K Darr, Managing Health Service Organizations. Baltimore: Health Professions Press; 1992, 444. Copyright 1992 by JS Rakich, BB Longest, and K Darr. Used by permission.](image-url)
and managerial styles, which are sources of empowerment for the personnel. Next, data are gathered regarding the failures. Lastly, outcomes are measured, maintained, and charted as the organization seeks to continually improve through problem solving. This process allows institutions to evaluate the educational program and provides guidelines for improving the program to meet the standards of quality expected by the accrediting agency. This model can further be applied to athletic training education, which seeks to improve the quality of its entry-level educational programs through process improvement.

**PROCESS IMPROVEMENT**

The goal of process improvement is to do things right the first time. Ray defined a process as a “collection of incremental and mutually dependent steps designed to direct the most important tasks of an organization.” Although the processes may vary from program to program, process improvement should be an ongoing endeavor involving all aspects of the organization and program. On an organizational level, process improvement can be directly linked to CAAHEP accreditation in athletic training. Griffith et al viewed process improvement “as the continuous endeavor to learn about the cause-and-effect mechanisms to change the process to reduce variation and complexity and improve customer satisfaction.” The FOCUS-PDCA methodology of process improvement is frequently used by noneducational organizations (Figure 2). The process of accreditation for athletic training education programs reflects application of this model’s methodology of process improvement. The individual institution must find a process to improve (educational programs for athletic trainers) and then organize a team that knows the process (JRC-AT and Education Council). The next step is to clarify the current knowledge of the process and to understand the causes of process variation (curriculum versus internship routes for certification). At this point, the process improvement task is selected (accreditation). The next phase presented in the model involves 4 steps. The first is planning improvement and data collection (self-study process). Second, the doing occurs with additional data collection and analysis. The third step involves checking data for process improvement and improved customer outcomes (accredited program statistics on the NATABOC examination and graduate placement). Finally, acting to continue the improvement (renewal of accreditation and continuing education revisions) is required.

This FOCUS-PDCA model, borrowed from other sectors, can be applied to athletic training education programs at the national and program levels.

**MANAGERIAL ROLES**

Although CQI is traditionally a business concept, the role of the academic manager has been found to closely parallel that of the business manager. Both in education and in business, the qualifications of a manager are typically quite variable; however, in both settings, managers adopt various roles in the discharge of their responsibilities. Meh contended that “there is similarity between administrative work of the department chair/program director and that of the corporate executive.” Rankin and Ingersoll discussed managerial roles with specific relevance to athletic training administrators such as human relations managers, leaders, and total quality managers. When CQI is the desired administrative result, academic administrators can successfully use common management theories, practices, and roles.

Several dominant classifications of managerial roles are presented in the Table. Mintzberg’s classification is based on the manager’s power and authority associated with a position. These roles are classified as Interpersonal, Informational, and Decisional. Meh applied Mintzberg’s managerial roles to the chief academic officer (CAO) role in an educational institution. He noted that the interpersonal role essentially involved the development of relationships with the constituencies of the institution. The informational role of the CAO

![FOCUS-PDCA](image-url)
translated into the receipt and processing of information, since the CAO serves as the "nerve center" for academic affairs. Lastly, the decisional role revolved around the CAO's access to the information that facilitated strategy-making processes to promote the mission of the institution. Mech also applied Alexander's typology of managerial roles to the academic setting. He classified these roles into strategic, operational, and interface. The strategic role "links the organization with its environment, determines basic goals and policies, identifies growth opportunities, and allocates resources." The operational role involves the daily operations of the institution and focuses primarily on the directing functions of the manager. Last, the interface roles are anchored in the formation of internal and external relationships. The athletic training education program director also fulfills these roles in the operations of the academic program both in the clinical and academic settings.

Stark and Lattuca classified managers' roles in a different fashion, focusing primarily on curriculum and dividing the managerial roles into 4 distinct categories. The human relations roles include those of the mentor and the facilitator. Athletic training educators must facilitate relationships involving students, coaches, colleagues, and administrators. The internal process roles are those associated with a higher degree of control, including monitor and coordinator. Monitoring student progression academically and clinically and coordinating student experiences lead to CQI in the educational program. Rational goal roles include the producer and the director, who are more externally focused but still highly controlled. Producing educationally appropriate opportunities and orchestrating the students, clinical instructors, and academic program requires significant insight from the program director. Lastly, open systems roles are those of the innovator and the broker. Innovative teaching techniques, technologic advances, and dwindling educational resources require much innovation and brokering on the part of the program director. As presented in the Table, Stark and Lattuca's roles are related to those of Mintzberg and Alexander in defining the importance of managerial roles in both the business and educational settings.

LEADERSHIP FOR SUCCESS

The roles of the athletic training program director are multifaceted. A strong understanding of management and leadership theory will help guide the director's behaviors. "Personal leadership by CEOs and clinical leaders will start and sustain the process of developing tomorrow's lean and agile health care system." Academic leaders play both a transactional and a transformational leadership function in the institution. As transactional leaders, educators are involved in the "simple exchange" of information and skills. On a daily basis, faculty and administrators interact with colleagues and students regarding the role of the course content in promoting the educational development of the students. They interact with others to ensure quality educational practices. However, the role of the faculty, particularly department chairs and program directors, includes transformational leadership aspects as well. As educators and administrators, transformational leaders use "change and conflict" to perpetuate quality standards in the professional development of students and colleagues. They must have a vision and ensure that the vision is consistent with the goals and mission of the institution. Cornesky believed that the vision of educators must "grab attention and encourage others to make a commitment for institutional achievement." It is the vision that stimulates strategic planning, which serves to shape and clarify the organization's profile.

Seymour recommended that "you lead between paradigms. That suggests 2 things: a clear vision of what the new paradigm looks like and a willingness to stay on the course as the culture is transformed." The accreditation process represents CQI and is part of the vision from the leaders in the field of athletic training. It will require transactional leadership as individual programs undergo significant change. More importantly, it will require transformational leadership at the institutional and national levels to ensure that a legitimate place in the workforce for athletic trainers is secured. With accreditation, "the initial investment in time alone is substantial, but it is necessary to ensure that each step toward improvement is carefully analyzed to maximize the potential for success." Effective leadership is critical to both this process and to CQI.

CONCLUSIONS

Change is a powerful force that is most often met with resistance. The strong legacy of higher education institutions further complicates the change process. An institution's culture is its mainstay in a constantly changing environment. However, to improve the quality of American higher education, change is necessary. CQI in athletic training programs requires a quality improvement initiative such as accreditation. Standards for professional education programs are set relative to inputs used, the process itself (ie, curriculum), and the quality of the outcomes. Academic leaders will be forced to exercise "increased leadership and implement the managerial techniques required to solve higher education's challenges." Although ambiguous in definition, quality is a critical issue for education in America. As society demands increased accountability in education, perhaps the principles of CQI should be considered when formulating strategic plans to ensure the viability and success of the institution. Accreditation is 1 step in ensuring quality; however, it must be an ongoing process. Chickering and Potter stated that "our customer is not only the individual student; we have a contract with the collective social enterprise." Higher education must rise to meet the demands of our changing society.

As we enter the 21st century, the trend of increased control over professional health care education programs is evolving. The role of the academic manager in attaining accreditation status, as well as the continued role of program administration after accreditation, has definite implications on the functioning and success of athletic training education programs. Managed care has created a highly competitive environment, and athletic training programs undergo significant change. More importantly, it will require transformational leadership at the institutional and national levels to ensure that a legitimate place in the workforce for athletic trainers is secured. With accreditation, "the initial investment in time alone is substantial, but it is necessary to ensure that each step toward improvement is carefully analyzed to maximize the potential for success.” Effective leadership is critical to both this process and to CQI.
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The Effect of Spirituality on Health and Healing: A Critical Review for Athletic Trainers

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Objective: To provide a comprehensive overview of the published literature regarding the effect of spirituality on health and healing.

Data Sources: I searched MEDLINE from 1976 to 1999 using the terms “spirituality,” “religion,” “faith,” “healing,” and “health.”

Data Synthesis: Strong scientific evidence suggests that individuals who regularly participate in spiritual worship services or related activities and who feel strongly that spirituality or the presence of a higher being or power are sources of strength and comfort to them are healthier and possess greater healing capabilities. Numerous research investigations have reported positive correlations between spirituality and decreased rates of stroke, cancer, cardiovascular disease, hypertension, drug abuse, suicide, and general mortality. It has been suggested that faith is beneficial for health and healing because it helps people avoid unhealthy behaviors such as smoking and excessive drinking. However, studies designed to statistically control for such factors also report positive associations between spirituality and health in individuals with unhealthy behaviors.

Conclusions/Recommendations: The impact of spirituality on health and healing is a topic that has been virtually ignored in the disciplines of athletic training and sports medicine. Because of their lack of exposure to this topic, most athletic trainers are unaware of the many positive associations that exist between spirituality and health and healing. The available literature base regarding this topic is quite large; its findings need to be explored and integrated into our profession.

Key Words: faith, health status, commitment

Clinical and scientific communities are continually trying to improve the quality of care offered to individuals seeking medical attention. These efforts may include improving existing and developing new pharmacologic interventions, medical equipment, and therapeutic and surgical interventions. However, with all of the sophisticated technology advances in medicine today, a relatively unknown factor has been shown to have a profound impact on health and healing: the relative strength of an individual’s faith or spirituality.

Spirituality is defined as having to do with the spirit or the soul, as distinguished from the body, and is often thought of as the better or higher part of the mind. Religion is defined as any specific system of belief, worship, conduct, etc., often involving a code of ethics and a philosophy. The terms “religiousness” and “spirituality” are often used interchangeably, and there is no solid consensus about the boundaries between them. I will use these terms interchangeably throughout this review. Ultimately, the meanings of religiousness and spirituality for a particular individual are idiosyncratic, reflecting numerous variables including cultural, theologic, developmental, and even biological factors.

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GENERAL HEALTH

A large number of investigators have evaluated the link between spiritual commitment and general health. Levin and Schiller reviewed nearly 250 research studies and examined the relationship between spiritual commitment and stroke, cancer, cardiovascular disease, colitis, hypertension, enteritis, general health status, and mortality. They reported consistently positive correlations between spiritual commitment and these health variables.

The relationship between blood pressure and spiritual commitment has been studied extensively. In 1 study, men who believed that spiritual commitment was important to them and who regularly attended spiritual services had significantly lower diastolic blood pressures than men who were not spiritually committed. This study also analyzed smoking behavior among participants and found that smokers who regularly attended spiritual services and who reported strong personal spiritual convictions were 7 times less likely to have abnormal diastolic blood pressures. Some have suggested that religious proscriptions against alcohol, drugs, and tobacco are largely responsible for lower disease rates in spiritually committed individuals. However, strong spiritual commitment seemed to have a protective health effect even in the presence of unhealthy behaviors, such as smoking. A review of nearly 20 published studies examining the relationship of spiritual commitment to blood pressure concluded that strong spiritual commitment was consistently associated with lower blood pressures.
Researchers have also shown that general health status is positively affected by strong spiritual commitment. Ferraro and Albrecht-Jensen\textsuperscript{7} reported that frequency of prayer and spiritual service attendance were significantly related to health status independent of age. This study also provided evidence that the impact of spiritual commitment on health paralleled the effect of an individual's level of education, which has long been shown to be a critical factor in health. The authors controlled statistically for social class (ie, affluence) and social club membership (ie, Rotary, Lions Club) and reported that spiritual commitment remained a strong predictor of general health status. Levin and Schiller\textsuperscript{3} also reported superior health status in individuals who attended spiritual services on a regular (weekly) basis.

A variety of investigators have reported that strong spiritual commitment is positively associated with lower rates of atherosclerotic disease in men and women,\textsuperscript{8} better health and longer life span,\textsuperscript{9} decreased rates of suicide,\textsuperscript{8} fewer suicidal thoughts,\textsuperscript{10} decreased rates of alcohol and drug abuse,\textsuperscript{11,12} and greater levels of happiness and life satisfaction.\textsuperscript{13} A strong body of evidence appears to reveal a positive relationship between spiritual commitment and health status. Keep in mind that these studies do not establish a direct cause-and-effect relationship; however, they provide evidence for significant associations between strength of spiritual commitment and good health. These associations appear to be so consistent that infrequent spiritual service attendance has been suggested as a definite health risk factor.\textsuperscript{14}

HEALING AND RECOVERY

In addition to general health status, numerous scientific studies have examined the effect of faith and spiritual commitment on healing and recovery. Harris et al\textsuperscript{15} explored the role of spiritual commitment in heart-transplant recipients' long-term health and well being. Heart-transplant recipients who regularly attended spiritual services and who reported having strong spiritual beliefs complied better with their rehabilitation protocols, reported higher emotional well-being indexes, and had superior physical functioning capabilities. The authors stated that it was wise for health care professionals to encourage spiritual commitment and spiritual participation to their patients. An additional study\textsuperscript{16} conducted on cardiac surgery patients explored the relationship between spiritual commitment and surgery survival rates. Patients described themselves as being 1) deeply spiritual, 2) involved in organized groups, such as a senior center, or 3) uninvolved. At the 6-month follow-up point, 14% of the patients who considered themselves uninvolved had died. 4% of the patients who stated that they were involved in organized groups had died, and of the patients who considered themselves deeply spiritual, none had died. The authors asserted that the strength of a person's faith was the strongest predictor of who survived cardiac surgery. Those patients who reported deriving at least some strength from their spiritual commitment were 3 times more likely to survive surgery. Women who suffered hip fractures were studied for depression and functional capacity.\textsuperscript{17} Patients who reported that a higher being or power was a source of strength to them and who frequently attended spiritual services fared better on a functional outcome measure (distance walked at discharge) and were less depressed during their medical care.

Research studies examining more subjective measures have been conducted on patient populations as well. Advanced breast cancer patients who reported having a sense of self-transcendence had less distress dealing with their illness and reported greater emotional well-being.\textsuperscript{18} Self-transcendence is described as a profound and potentially transformative experience that can have a vast array of manifestations.\textsuperscript{2} Ovarian cancer patients reported that they depend greatly on their spirituality to help them cope with their disease\textsuperscript{19}; the authors advised that it may be wise to add a chaplain to the treatment team of cancer patients, if the patient so wishes, since spirituality is such a strong coping mechanism for individuals fighting this disease. In a patient population of hospitalized, medically ill men, religious commitment and religious coping helped patients recover from depression more quickly.\textsuperscript{9} Finally, patients with increased commitment to their spirituality reported their health as better than the health of other patients.\textsuperscript{20}

One of the most commonly referenced studies was done by Byrd\textsuperscript{21} who examined the therapeutic effects of intercessory prayer in a coronary care population. Byrd randomly assigned 393 patients to receive either daily prayer or no prayer. The prayers were offered by a group of nondenominational, committed Christians. The Christians offered prayers for a rapid recovery, for prevention of complications and death, and for any other areas they believed would be beneficial to the patients. The study used a double-blind, randomized design. Physicians, nurses, and patients did not know who was or was not being prayed for; only the individuals offering the prayers knew the names of the recipients of the prayers. Pretesting revealed no significant differences between the 2 groups in cardiac or noncardiac diagnoses. However, at discharge, the patients who received the intercessory prayer differed significantly on 6 variables: they required significantly less intubation and ventilation assistance, fewer antibiotics, and fewer diuretics and had significantly fewer cardiopulmonary arrests, fewer episodes of congestive heart failure, and fewer cases of pneumonia. Less research has been conducted on prayer and its effect on health, yet approximately 90% of women and 85% of men pray, and approximately 80% pray on a weekly basis.\textsuperscript{22} Research on healing and recovery closely parallels the research reported for general health. Once again, evidence shows that the stronger one's spiritual commitment is, the more likely fast and effective healing and recovery will occur.

ASSESSMENT OF FAITH

Approaching the topic of a patient's faith can be an extremely delicate situation for an athletic trainer or any health care provider. One of the reasons this task may be extremely challenging is that health care providers usually do not receive adequate training in their various educational programs with regard to assessing their patients' spiritual beliefs. In fact, only about 5% of physicians receive regular training on spiritual topics during their educational tenures.\textsuperscript{23} I found no studies that examined the addressing or teaching of spiritual issues in athletic training or sports medicine educational programs.

Patients may be uncomfortable or even offended if asked about their spiritual beliefs and the relative strength of such beliefs. In the past, a person's faith was a topic that was rarely discussed or brought up in clinical or sports medicine settings. Reiser and Rosen\textsuperscript{24} stated that family physicians are adept at assessing and treating disease but are usually ignorant of the spiritual component of illness. These authors strongly criti-
cized contemporary medicine, saying the spiritual concerns of patients have been abysmally neglected.

One way to approach the topic of spirituality with patients is by asking them a simple, nonthreatening question regarding their spiritual beliefs. This simple introductory question should give the care provider insight into whether the patient would be interested in pursuing the subject of spirituality and spiritual commitment. Waldfogel2 offered a number of sample questions and statements that could be used to assess a patient’s interest in the topic of spirituality:

- Do you belong to a religious or spiritual community?
- How important is your religious and spiritual identification?
- What aspects of your religion or spirituality would you like me to be aware of?
- What does your belief in God mean to you? Has it changed during your illness?
- Tell me of your belief in God or a higher power.
- Tell me of your religious and spiritual practices, such as prayer or meditation.

These questions could certainly be modified to fit the care giver’s and the patient’s comfort levels.

Research to assess spiritual health and well being is in its infancy.26 However, a number of tools are available that attempt to quantify or assess a person’s level of spiritual commitment. One of the simplest is Kasl’s9 3-item religious index:

1. How often do you attend regular religious services during the year?
2. Aside from your frequency of attendance, how religious do you consider yourself to be?
3. How much is religion (and/or God) a source of strength and comfort to you?

The answers to these questions range from never to more than once a week, very religious to against religion, and not very much to a great deal, respectively. Kuhn’s27 Spiritual Inventory is a 25-item questionnaire also used to assess patients’ spiritual health. Among the items evaluated by Kuhn’s Inventory are hope, faith, love, prayer, worship, ability to laugh and celebrate, and the attachment of meaning and purpose to life. A 12-item scale developed by Strayhorn et al28 considers a number of devotional factors in its assessment of spiritual commitment, including frequency of prayer, reading the Bible, and service to one’s spiritual community. Finally, one of the more popular assessment tools contains 20 items that examine both existential and religious issues to give more of an encompassing measure of spiritual health.29

The question of whether patients desire their spiritual beliefs to be addressed by their care providers has been studied. Surprisingly, 75% of patients surveyed wished to have their spiritual commitment addressed by their health care provider,30 and nearly 50% of the respondents wanted to pray with their physicians. Patients in this study also reported that their faith was rarely discussed during medical visits. The fact that so many patients want their spiritual commitment addressed is less surprising when we consider that 95% of Americans express belief in God.31 Additionally, Bergin and Jenson12 studied marriage and family therapists, social workers, psychiatrists, and psychologists and reported that more than 75% of these professionals stated that they try hard to live their lives according to their spiritual beliefs; nearly 50% stated that their whole approach to life is based on their spirituality. Whether the findings of Bergin and Jenson’s12 study can be inferred or generalized to the general population, however, is unclear.

Approaching the topic of spiritual beliefs and commitment with patients or clients appears to be potentially less taboo and threatening than medical care providers may have traditionally thought.

INTEGRATION INTO CLINICAL PRACTICE

As athletic trainers become more aware of the positive associations among health, healing, and spirituality, they can begin to make a conscious effort to incorporate this dimension into their health care paradigm if they so choose. The athletic trainer’s comfort level, as well as the comfort level of the patient, must be given serious consideration before spiritual issues are explored. As I outlined previously, a simple, nonthreatening question can inform the care giver if the patient is receptive to a conversation about spirituality. Because this topic is relatively new to our profession, no standards or guidelines exist to assist athletic trainers in the practical application in various clinical settings. If patients have a desire to explore spiritual issues, athletic trainers may engage in a spiritual conversation with the patient, recommend readings from the religious or spiritual literature, encourage the patient to attend spiritual worship services or activities, and possibly even engage in prayer sessions with the patient. It is strongly recommended that athletic trainers take a nondenominational, nonthreatening approach when integrating religious and spiritual components into their practice to avoid pushing personal beliefs and convictions onto their patients. As career opportunities for athletic trainers continue to expand (eg, sports medicine clinics; high school, collegiate, and professional sporting teams; corporate health care centers; and health and fitness settings), the opportunity to share this information with interested patients and clients will certainly be available.

CONCLUSIONS

Spirituality and health is a topic that has seemingly received little attention in our profession. Study after study has suggested that a relationship does exist between the strength of one’s spirituality and one’s overall health. In a comprehensive review of more than 300 published studies in the medical field, Levin33 concluded that “although most of these studies are correlational, use inadequate measures of religious commitment such as religious denomination or other single-item measures, and seldom assess the intensity of religious commitment, they generally have suggested a beneficial, or salutary, effect for religious or spiritual involvement on physical and mental health status.” The question of how spirituality exerts this protective effect over health has not yet been answered. Waldfogel2 stated, “many benefits of spirituality that are felt to occur secondary to spiritual practices per se directly affect physiologic processes via mechanisms that are incompletely understood.”

Health care providers need not embrace or share their patients’ spiritual convictions,34 but a patient’s spiritual beliefs must always be highly respected and never addressed in a cynical or demeaning manner. It is also inappropriate for health care providers to impose or push personal spiritual convictions or agendas onto their patients.

Much of the research on this topic has been performed with elderly individuals and severely diseased (ie, cardiac surgery) patients and has been conducted in conjunction with physicians. Future research must address this topic in younger and...
more athletic populations and could potentially be performed in conjunction with athletic trainers in clinical as well as sports medicine settings. The goal of athletic trainers should be to provide the highest quality, comprehensive care possible to the athletes and patients who are under our supervision. Learning more about the effects of spirituality on health and healing and possibly incorporating these principles into our prevention and treatment philosophies may be one way to enhance the care we give to our clients and to continue to advance our profession.

REFERENCES

An Acute, Traumatic Supraspinatus Lesion in an Intercollegiate Football Player: A Case Report

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Objective: To describe the evaluation, management, and rehabilitation of an acute, supraspinatus tendon injury in an intercollegiate football player.

Background: While attempting to block a defender, a 19-year-old collegiate football player slipped on the artificial turf and landed on his right elbow, causing an injury to his right shoulder. The athlete was initially seen by the head athletic trainer and then referred to the team physician for further evaluation.

Differential Diagnosis: acromioclavicular joint sprain, brachial plexopathy, subacromial impingement syndrome, supraspinatus lesion.

Treatment: The athlete was managed surgically with an open acromioplasty and a 3-bone tunnel repair of the supraspinatus tendon. After surgery, the athlete underwent a 4-month rehabilitation protocol in preparation for return to competition.

Uniqueness: This case involved a teenage athlete rather than the older individuals who normally sustain rotator cuff lesions. Also, the mechanism was a compressive force on the supraspinatus tendon rather than the tensile force common to rotator cuff lesions.

Conclusions: By presenting this case report, we hope to give sports medicine clinicians a better understanding of rotator cuff injuries and how to successfully manage and rehabilitate supraspinatus lesions.

Key Words: acromioplasty, rotator cuff repair, shoulder injury

Rotator cuff lesions often occur chronically in an older population. Classically, Neer3 described rotator cuff injury as having a progressive onset. In overhead athletes younger than 25 years of age, edema and hemorrhage initially accumulate within the subacromial space. As rotator cuff injury progresses, fibrosis of the subacromial structures and tendinitis result, generally in overhead athletes from 25 to 40 years of age. Finally, partial- and full-thickness tears occur in overhead athletes over the age of 40. While the stages described above are termed stage I and stage II rotator cuff impingement, respectively, rotator cuff tears are categorized as stage III rotator cuff injury and often result from abnormalities within the subacromial space. Over an extended period of time, these abnormalities cause chronic microtrauma to the soft tissue structures (rotator cuff), leading to tissue failure. Rotator cuff injuries in younger athletes most often occur due to repetitive impingement within the subacromial space and usually are secondary to eccentric tendon failure, secondary impingement from glenohumeral instability, or posterior glenoid impingement.

The case we present is unique for 2 reasons: the age of the athlete and the mechanism of injury. The supraspinatus tendon tear sustained by the teenage athlete resulted from 1 traumatic, compressive mechanism rather than the repetitive microtrauma or tensile mechanisms often implicated in rotator cuff lesions. Our purpose is to describe the evaluation, management, and rehabilitation of an acute, traumatic rotator cuff injury in a teenage intercollegiate football player. We hope to provide sports medicine clinicians with a better understanding of rotator cuff injuries and how to successfully manage and rehabilitate supraspinatus tendon tears.

HISTORY

A 19-year-old intercollegiate football player participated in a home contest on artificial turf. He was a sophomore offensive guard with no previous history of shoulder injury or pain. During 1 play, the athlete was caught between 2 linemen. As the opposing defensive players tried to elude him, the athlete slipped and fell onto the turf, with his right elbow making the initial contact. The direct contact of his elbow on the turf caused an axial load along the long axis of the humerus, driving the humeral head into the undersurface of the acromion. The athlete got up slowly and made his way to the sidelines without assistance.
EVALUATION

Both the head athletic trainer and the team physician performed the initial evaluation on the sidelines. The injury first presented as a suspected brachial plexopathy (stinger) in his right shoulder. He complained of a numbing sensation radiating from his shoulder to his arm and pain over the right acromioclavicular joint. Special tests, including the acromioclavicular distraction-compression test, the Yergason test for biceps involvement, the empty-can test for supraspinatus involvement, the Neer impingement test, the O'Brien test for a superior labral anterior posterior lesion, and all instability assessments were normal. Manual muscle tests of all pertinent musculature about the shoulder were considered equal bilaterally. In light of the athlete’s discomfort, the team physician and head athletic trainer withheld the athlete from competition for the remainder of the game and treated him with cryotherapy. The team physician prescribed a standard set of radiographs for follow-up evaluation. Reevaluation 5 days after the injury revealed negative radiographs and minimal pain and tenderness over the acromioclavicular joint. All clinical tests were negative, and manual muscle tests were equal bilaterally. The athlete returned to competition for the last game of the season with a diagnosis of mild acromioclavicular joint sprain.

One month after the injury, the athlete still complained of pain on top of his right shoulder. The head athletic trainer sent the athlete to the team physician for further evaluation. The team physician elicited a positive impingement sign in 90° to 120° of elevation and a mildly positive Hawkins impingement sign. A magnetic resonance image arthrogram with gadolinium injection to evaluate rotator cuff integrity revealed a high-grade, partial-thickness tear of the supraspinatus tendon without gross retraction (Figure 1). The athlete then left school for the holidays, and all records were transferred to a second orthopaedic surgeon in the athlete’s hometown.

The evaluation by the second orthopaedic surgeon confirmed a partial-thickness tear of the supraspinatus tendon. In addition to the standard radiographs prescribed by the team physician, an additional supraspinatus outlet view revealed a possible type II-III acromion and a thickened lateral acromion (Figure 2). Once a rotator cuff lesion is suspected, the supraspinatus outlet radiograph is warranted to assess acromion morphology. Clinical evaluation revealed a positive impingement sign, mild weakness with supraspinatus testing, excellent active external rotation and internal rotation strength, no acromioclavicular joint tenderness, and no instability (including a negative sulcus sign and apprehension test).

SURGICAL MANAGEMENT

The athlete wanted to continue to participate in intercollegiate football, and so he elected surgical management of the supraspinatus lesion. Under anesthesia, the athlete exhibited no signs of instability. Arthroscopic evaluation revealed a normal biceps tendon and biceps anchor on the superior rim of the glenoid. The articular surfaces of both the humerus and glenoid were normal. No glenohumeral ligamentous pathology was evident. A 3-cm supraspinatus lesion was evident at the insertion on the greater tuberosity. Immediately after arthroscopic evaluation, the orthopaedic surgeon released the deltoide from the acromion for open exploration, which revealed a type II acromion and a fairly thickened subacromial space, particularly laterally, in the area of the tear. No full-thickness tear was revealed. Elevation of the supraspinatus revealed a 60% to 70% undersurface partial-thickness tear that was consistent with lateral acromion impaction on the greater tuberosity and a partial avulsion (approximately one half) of the supraspinatus tendon.

The athlete was managed surgically with an open rotator cuff repair. Because of the increased slope of the lateral acromion, an acromioplasty was performed, and then the supraspinatus lesion was repaired using a 3-bone-tunnel tech-

Figure 1. Magnetic resonance imaging arthrogram revealing a high-grade, partial-thickness tear of the supraspinatus tendon without gross retraction.

Figure 2. Supraspinatus outlet view radiograph revealing a type II-III acromion arthrology and thickened lateral acromion.
nique. Three transhumeral bone tunnels were drilled through the lateral portion of the humeral head, allowing fixation (via sutures) of the supraspinatus tendon to the greater tuberosity.

REHABILITATION

The rotator cuff repair rehabilitation protocol was developed through a collaborative effort of the head athletic trainer, the team physician, and the orthopaedic surgeon who performed the surgery. The program lasted 16 weeks and contained 7 phases; the athlete progressed from phase to phase only after evaluation by the team physician (Table). The head athletic trainer and team physician adapted the protocol to reflect the athlete’s progress. We emphasize that the protocol outlined is only a framework for rehabilitation after open rotator cuff repair. Each athlete must be closely supervised throughout the protocol so as to avoid disrupting the healing process.

Phase I (Weeks 0 to 4)

Rehabilitation began the day after surgery with pain-free, passive range-of-motion (PROM) exercises for shoulder flexion, abduction, and internal rotation. The athlete’s family was instructed in the PROM exercises and advised to perform the exercises on the athlete several times daily. The surgeon mandated that the athlete wear a pillow sling 24 hours a day, with the shoulder supported in abduction to facilitate circulation and decrease pain. Cryotherapy was prescribed 3 to 5 times daily for pain management. Shoulder extension was contraindicated for 4 weeks to protect the anterior deltoid and supraspinatus repair from tensile load.

The athlete reported to the athletic training room 1 week after surgery. The athlete continued to wear the sling 24 hours a day and perform the PROM exercises with the assistance of the athletic training staff. For weeks 2 through 4, the rehabilitation protocol included the pain-free PROM exercises as described above (avoiding shoulder extension) and supine passive external rotation exercises (elbow supported by the table and flexed to 90°) with the aid of a bar. The athletic training staff instructed the athlete to perform soft tissue massage once the incision was sufficiently healed. Active range of motion (AROM) of the elbow, wrist, and hand was initiated with the arm supported by the table. The athlete reported to the athletic training room twice a day for weeks 2 and 3 and once a day for weeks 4 and 5. All rehabilitation exercises were followed by cryotherapy. The athlete also exercised and iced at home according to the protocol.

Phase II (Weeks 4 to 6)

The goals of this phase of the protocol were full PROM by 6 weeks and weaning the athlete from the sling, first at night and then totally by 6 weeks. During this phase, the athletic trainer initiated gentle manual resistance for scapular protraction-retraction and elevation-depression and gentle, submaximal glenohumeral isometrics in all planes. By 6 weeks, the athlete no longer relied on the sling and had achieved a passive range of 170° of forward flexion, 170° of abduction, 90° of external rotation, and 55° of internal rotation. The athlete still complained of anterior shoulder pain, typically at end ranges of motion with internal rotation.

Phase III (Weeks 6 to 8)

The purpose of this phase was to begin active assisted range-of-motion (AAROM) exercises in the pain-free range. The AAROM exercises included self-assisted forward elevation with a bar, progressing from supine to standing. The athletic trainer started gentle open kinetic chain rhythmic stabilization to facilitate neuromuscular cocontraction of the glenohumeral stabilizers and scapular stabilization. The rhythmic stabilization technique began with the athlete supine and required him to maintain his arm in the resting position while the athletic trainer applied various directions of force to the upper arm. The rhythmic stabilization progression included placing the upper extremity in a position of vulnerability (abduction with external rotation) with the athlete in a standing position with his eyes shut. The athlete was able to tolerate wall climbs in forward flexion and abduction with no pain through the range of motion. He began horizontal wall walking and internal rotation stretching behind his back with the aid of a towel. Goniometric evaluation of AROM revealed limited shoulder flexion and abduction (approximately 10°), 65° of internal rotation at 90° of abduction with anterior shoulder pain at end range, and 93° of external rotation at 90° of abduction. The athlete also began general cardiovascular training on an elliptical trainer. At this point, he performed AAROM exercises once daily in the training room and once a day at home.
Phase IV (Weeks 8 to 10)

During this phase, the athlete began pain-free AROM exercises on rotator cuff exercises (without resistance). These exercises included standing forward elevation to 90° and side-lying internal and external rotation exercises. He progressed to prone horizontal abduction (thumbs up) at 100° and at 90° of abduction, prone external rotation in a 90°–90° position, and prone extension. In addition, the athletic trainer initiated scapulothoracic strengthening exercises, including supine ceiling punches and seated rows. The athlete progressed to prone horizontal abduction (thumbs up) at 150° of abduction for the last 20° of available range only. He performed gentle closed kinetic chain balance and stabilization progressions. These exercises included leaning on both hands at shoulder level on a wall and weight shifting from left to right. The athlete progressed to shoulder-level weight shifts on a table, requiring additional support of body weight. He had now achieved active flexion and abduction within 5° of normal limits, external rotation within normal limits, and 73° of internal rotation. Before exercise, the athletic trainer administered moist heat and ultrasound to assist with flexibility. The athlete continued cryotherapy for 20 minutes after each exercise session.

Phase V (Weeks 10 to 12)

The goal for this phase was AROM within functional limits by 12 weeks. The athlete progressed to self-stretching exercises, including doorframe hangs for forward flexion and corner stretches for horizontal abduction and external rotation. The protocol called for upper extremity endurance training on an upper body ergometer. Since the athletic training facility had no such equipment, the athlete performed this portion of the protocol in the swimming pool or warm whirlpool with his shoulders submerged. He used barbells in the swimming pool or exercised in the jet flow of the whirlpool to provide resistance. In this phase, the athlete jumped rope (3 sets of 5-minute intervals) for cardiovascular exercise.

Phase VI (Weeks 12 to 14)

In this phase, the athlete began isotonic and isokinetic progressive resistance exercises prone and standing. Hand weights were used initially, focusing on rotator cuff and scapulothoracic strengthening. Isokinetic internal and external rotation exercises were started at 0° of abduction in a standing position, progressing to a position of vulnerability at 90° of abduction in a supine position. The closed kinetic chain exercises described in Phase IV progressed to wide-base seated press-ups, wide-base step-ups on a box, tripod exercises on a wobble board, and wall push-ups with a plus. Manual resistive exercises using proprioceptive neuromuscular facilitation diagonal pattern techniques were instituted.

Phase VII (Weeks 14 to 16)

The goal of the last phase of this protocol was to achieve equal bilateral strength by 16 weeks with emphasis on frequency, intensity, and duration of training. Progressive resistive exercises were stressed. The athletic trainer initiated weight-room exercises, including military press, bench press, latissimus dorsi pull-downs, seated rows, elbow extensions for the triceps, biceps curls, and side-lying humeral rotation exercises with dumbbells. The athlete began proprioceptive neuromuscular facilitation diagonal pattern exercises with surgical tubing and a low-level plyometric progression (2-handed ball tosses and dribbling). Sport-specific exercises included activities on a blocking sled and resistance training on upper extremity power trainer (The Attacker, Powernetics, Riverside, TX).

At 16 weeks, the team physician cleared the athlete for athletic activities, with clearance for full-contact competition following at 6 months. The 5-month follow-up visit with the orthopaedic surgeon who performed the surgery yielded normal active and passive range of motion in all shoulder movements, as well as normal strength assessment. Isokinetic evaluation revealed symmetric bilateral torque measures in all planes of motion. The athlete is currently competing without difficulty.

DISCUSSION

This case is unique in that it involved a teenage athlete with an acute, traumatic lesion of the supraspinatus tendon. Rotator cuff lesions in the younger population are rare but often occur chronically in the older population secondary to impingement syndrome. Over time, bony alterations (acromion thickening and bone spurs) develop within the subacromial space, leading to partial- and eventually full-thickness tears of the rotator cuff.

Neer stated that the typical age of these individuals with stage III impingement was greater than 40 years old. When traumatic rotator cuff lesions are seen in a younger population, they are still believed to result from years of repetitive microtrauma rather than an isolated acute episode. Craven described this phenomenon as “athletically accelerated aging” of the shoulder. With the increase in youth leagues, longer seasons, and weight training that places excessive stress on the shoulder, younger overhead athletes are exposing their shoulders to repetitive trauma that, in a sense, ages the shoulder.

This increased stress causes tissue alteration, including decreased tensile strength at the musculotendinous junction, leading to increased risk of a traumatic rotator cuff lesion. In addition, Neer reported that acute trauma rarely seems to be the principal cause of a tear but only enlarges an already existing lesion.

The mechanism by which the injury occurred in this patient is unique: an axial load along the long axis humerus, driving the humeral head into the undersurface of the acromion. Often rotator cuff tears result from an eccentric tensile load placed on the tissue, rather than a compressive force. Glousman reported that partial tears on the undersurface of the rotator cuff usually occur from traction loads. This athlete sustained a 60% to 70% undersurface supraspinatus lesion, but from a compressive mechanism rather than traction.

Evaluation of the athlete yielded interesting and conflicting results. Manual muscle testing of the supraspinatus during the on-field evaluation and by the physician in the office revealed no decrease in strength. Manual muscle testing of the supraspinatus was performed by resisted elevation of the humerus in the scapular plane with the thumbs pointed down. In light of the fact that the athlete was diagnosed with a 60% to 70% lesion, it is surprising that a strength deficit was not found. The athlete was an offense lineman with a mesomorphic build and a high level of muscular strength. We can only speculate that the reason for the lack of observable decrease in strength was muscle substitution as a compensatory mechanism. Kendall et
al12 described an alternative method for testing the supraspinatus muscle. The patient sits or stands with the arm at the side, head and neck extended and laterally flexed to the ipsilateral side, and the head rotated toward the contralateral side.12 This position shortens the upper trapezius to minimize its ability to compensate for a weak supraspinatus muscle. Manual pressure resisting shoulder abduction is applied by the clinician. This test might have better isolated the supraspinatus in our athlete and revealed a strength deficit.

The athlete opted for surgical intervention. The acromioplasty is the initial stage in any rotator cuff repair.13 Orthopaedists perform acromioplasty by removing approximately 2 cm of bone from the anterior-inferior portion of the acromion.2,3,13,14 The undersurface of the resected acromion is then smoothed with a rasp or burr to flatten any abnormalities, allowing sufficient space and uninterrupted gliding of the rotator cuff within the subacromial space.14 In conjunction with the acromioplasty, the athlete was treated with a 3-bone-tunnel suture technique to repair the supraspinatus lesion. Three transhumeral bone tunnels were drilled from the metaphysis-diaphysis junction to the greater tuberosity. Transhumeral sutures were then passed through both the bone tunnels and the rotator cuff, where the sutures were tied, securing the supraspinatus to the humeral head in a bone trough.13,15

After surgical intervention, the athlete participated in a 4-month, 7-phase rehabilitation program. The initial goals of the rehabilitation program were to decrease pain through modality use; to protect the surgical repair with a sling; and to restore range of motion, first passively and progressing to full AROM. Once sufficient range of motion was achieved, rotator cuff resistance training was initiated.1,16,17 The rotator cuff both centralizes and approximates the humeral head within the glenoid.18,19 Restoring sufficient rotator cuff strength is vital if the athlete plans to return to athletic activity. Sharkey and Marder17 reported that the rotator cuff musculature opposes superior translation of the humeral head during abduction. If the rotator cuff fails to perform this humeral depression function (as in this athlete), the supraspinatus can become impinged, placing the repaired supraspinatus tendon in a compromising position. In phase IV, the athlete performed prone rotator cuff strengthening exercises such as those described by Blackburn et al.20 Initially, the only resistance provided was arm weight against gravity, progressing to dumbbells. Blackburn et al.20 found increased electromyographic rotator cuff activity with the prone exercises, suggesting their suitability in training the rotator cuff musculature. In phase VI, the athlete performed isokinetic humeral rotation exercises. At first, the athlete performed the exercises at 0° of abduction, advancing to a position of vulnerability at 90° of abduction.21

In addition to rotator cuff strengthening, the athlete performed a full scapular stabilization program. The scapular stabilizers play a vital role in the rehabilitation of rotator cuff repair. Motion of the scapulothoracic articulation is essential for fluent, coordinated movement of the shoulder.21 The scapular stabilizers provide a firm base of support for gleno-humeral movements and simultaneously rotate the scapula as the humeral head moves within the glenoid fossa (scapulo-humeral rhythm). Proper scapulohumeral rhythm maintains the humeral head in optimal alignment within the glenoid, allowing for the proper length-tension relationship among the rotator cuff muscles, glenoid, and humeral head.22 Asynchronous scapulohumeral rhythm by the scapular stabilizers disrupts this glenohumeral-scapulothoracic alignment and coordinated movement by the shoulder.21 With this humeral head alignment disrupted, the likelihood of the greater tuberosity's impinging the subacromial structures (supraspinatus tendon, subacromial bursa, and bicepital tendon) increases.23 This increased risk of impingement could be especially problematic in an individual recovering from a supraspinatus repair. By initiating isometric scapular stabilization exercises early in the program, we hoped to limit the risk of scapular inhibition that often accompanies shoulder pain.23 Naturally, the progression from isometric exercises was to isotonic scapular exercises, such as the ceiling punches and seated rows in phase IV, to the resistive scapular strengthening exercises performed in phases V through VII.

In addition to the traditional rehabilitation exercises described above, functional rehabilitation was performed throughout the protocol. This rehabilitation program focuses on both restoration of neuromuscular activation patterns and sport-specific activities.24 Neuromuscular restoration exercises are designed to reestablish the coordinated muscle actions vital to functional stability. In phase III, the subject performed rhythmic stabilization exercises. The goal of these exercises was to enhance the recruitment of the force couples present at the glenohumeral joint, vital for dynamic glenohumeral stability, as well as to stimulate scapular stabilization.25 In phase IV, the athlete performed closed kinetic balance tasks on a stable surface, progressing to tripod exercises on an unstable wobble board (phase VI). The latter exercise promotes joint compression and cocontraction of the force couples presented at the shoulder, increasing dynamic stability.26–28

Along with neuromuscular activation pattern restoration, sport-specific exercises are an important aspect of functional rehabilitation, with the goal of minimizing the stress placed on the shoulder joint with return to activity.20 It is important to mimic athletic activity with the exercises.21 In phase VI, the athlete performed proprioceptive neuromuscular facilitation exercises with both manual resistance and surgical tubing. In addition to strengthening the shoulder musculature, the D2 proprioceptive neuromuscular facilitation pattern mimics the functional plane of throwing and restores neuromuscular mechanisms important for coordinated movement. Stretch-shortening drills (plyometrics) were used in phase VI to mimic athletic activity. Similar to most athletic movement patterns, stretch-shortening exercises involve a short prestretch followed by a quick powerful concentric muscle contraction. Plyometrics impart eccentric loads common to most upper extremity athletic activity. Finally simple, high-velocity hand shivers on a blocking bag simulate the type of athletic activity performed by an offensive lineman.

### SUMMARY

Our purpose was to present the case of an intercollegiate football player who sustained an acute, traumatic supraspinatus lesion while playing football. The case is unique in that the supraspinatus lesion occurred during 1 traumatic episode, rather than as the result of repeated microtrauma, and that a younger individual sustained the injury. By describing the surgical technique and outlining the rehabilitation program, we hope to provide sports medicine clinicians with the framework to successfully manage and rehabilitate individuals who sustain acute, traumatic supraspinatus lesions.
REFERENCES
A Classification System for the Assessment of Lumbar Pain in Athletes

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Objective: To present a lumbar pain classification system and its rationale as an alternative to pathology-based diagnosis for athletes.

Background: Lumbar pain is a common problem in both athletes and in the general population. Although common, the etiology of lumbar pain cannot be identified in 80% to 90% of patients. Lumbar injuries are often evaluated using a pathology-based model. This approach attempts to pinpoint the cause of the pain, making lumbar pain evaluation difficult.

Description: We developed a 4-level classification system that groups athletes based on signs and symptoms. It does not necessarily attempt to determine the cause of pain. We use this classification system for referral, participation, and rehabilitation decisions.

Clinical Technique: This assessment technique eliminates much of the ambiguity associated with lumbar pain. Athletic trainers can use this technique on all athletes presenting with lumbar symptoms.

Key Words: low back, back pain, evaluation, spine, athletic injuries

Lumbar pain is a common complaint, both in the overall population and among athletes. It is the leading cause for visits to orthopaedists and neurosurgeons and the second leading cause of visits to family physicians among the general population.1 In athletes, reported incidence rates vary between 7% and 27%.2-6

Although lumbar pain is common, identifying the anatomical source of back pain is extremely difficult.1,7-12 Nearly all the significant lumbar spine anatomy can cause local and radiating pain. The diagnostic terms are numerous,2,12,13 vague, and often confusing.14 Intertester variability in common evaluation techniques is high, and diagnoses are often based on personal biases.11,15-18 The relationship between imaging studies and symptoms is also unreliable.19-21 Adding to the difficulty, the etiology of lumbar pain varies widely and is often unknown.9,22-24 Many health care professionals consider the low back evaluation frustrating and their findings inconsistent.17,19,25-28

The relationship between evaluation and rehabilitation of low back pain is unique. Successful rehabilitation depends more on symptoms and function than on determining a clear anatomical cause.14,25,29,30 Therefore, we take an approach to the evaluation of lumbar pain that is based on signs and symptoms, which the recent literature supports.1,8,10-13,29,31

We developed a general 4-level classification system that does not necessarily identify an anatomical cause. One level describes “red flag” lumbar pain, and the other 3 relate to the severity of the athlete’s signs, symptoms, and decreased function. We use the classification system for referral, participation, and rehabilitation decisions.

PAIN SOURCES

Few clinical evaluation techniques clearly distinguish 1 cause of lumbar pain from another. Following are the significant anatomical and physiologic lumbar pain sources.

Facet Joints

The facet joints play important mechanical and functional roles in the lumbar spine. The inferior and superior articulating processes of adjacent vertebrae form the joint (Figure 1). In a standing posture, the facets transmit only between 10% and 20% of compressive loads.14,27 Extension of the lumbar spine further loads the facets and unloads the intervertebral discs.14,27 The alignment of the lumbar joints makes them most resistant to rotation and least resistant to flexion and extension. As the primary resisters to lumbar rotation, the facets may be injured with rotation.14 Decreased disc space increases the load on the facets and is probably related to degeneration of the facet joints.14,27

The exact role of the facets as a source of lumbar pain is unclear. The facet joints are capable of causing both local lumbar pain and pain radiating as far as the foot.14,32,33 However, pain sites vary widely among individuals with facet injuries.27,35 Unfortunately, no reliable clinical method exists to determine injury to the facets. Imaging studies are also unreliable. Patients with images showing degenerated facets may be asymptomatic, and those with normal-appearing joints may have pain.3,14

Intervertebral Discs

Each disc has 3 main components. The nucleus pulposus is an incompressible gel located at the disc’s center. The annulus fibrosus is multilayered with woven fibers and surrounds the pulposus. The third component is the cartilaginous endplates.
Figure 1. Posterior view of L4–L5.

Figure 2. Superior view of L4.

Figure 3. Lateral view of L4–L5.

that anchor the disc to each vertebral body. These structures are very synergistic, and abnormal function of 1 affects the others.

The lumbar discs distribute force across the spine, absorb force directly, and maintain space between the vertebrae, allowing normal function. The disc withstands between 80% and 90% of compressive loads while the individual is standing.14 Flexion increases the load on the discs and unloads the facet joints. The components of the disc tolerate compression well but are vulnerable to rotation with a flexion component.34 Excessive torsion usually results in failure of the nucleus or endplate.

After age 12, the disc is avascular.14 It depends upon the annulus fibrosus and endplates for passive nutrition. Therefore, injury to these structures can result in disc degeneration from interruption of nutrition. Disc degeneration can also result from the normal aging process. Degeneration leads to decreased tensile strength, decreased disc height, and increased stress on the facets.34

It appears that only the annulus fibrosus is innervated with nociceptors3,14,36; thus, injury to this structure can result in local pain. Clinically, it is very difficult to differentiate annular injuries from other lumbar sprains and strains.8,14 A weakened annulus can, in turn, lead to escape of the nucleus pulposus.3 The disc’s role as a secondary cause of radiating pain via the displaced nucleus is recognized in the literature.

Disc herniation can result in local back pain, radiating pain, or neurologic deficits, or a combination of these. Neurologic deficits include altered sensation, muscle weakness, and decreased reflexes. Ninety percent of disc herniations occur at the L4–L5 or L5–S1 junctions.14 Only 1% of disc herniations affect individuals between the ages of 10 and 20 years.34 Classically, the disc herniation’s mechanical pressure on the nerve root is considered the cause of radiating pain and neurologic symptoms.33 However, not all mechanical pressure caused by disc herniation is symptomatic.14,38,39 Mechanical pressure alone does not seem to explain all radiating pain symptoms.

Spinal Nerve Root and Dorsal Root Ganglion

The spinal nerve roots exit the spinal canal through the intervertebral foramen and have an important role in lumbar pain (Figures 2 and 3). Both disc herniations and spinal stenosis can cause compression of the nerve root. Compression of a normal root causes neurologic change, but does not cause pain.36,38 Some degree of inflammation and irritation must preexist for pain to occur, and as a consequence, normally pain-free lumbar activity can result in radiating pain.36

The dorsal root ganglion (Figures 2 and 3) is a lateral prominence of the spinal nerve root after it exits the vertebral foramen. Disc herniation is a likely cause of mechanical compression to the dorsal root ganglion. However, the ganglion’s reaction to mechanical compression differs from that of the spinal nerve root. It appears that mechanical compression alone of a normal dorsal root ganglion may cause radiating pain and neurologic symptoms.14,37 It is also unique because it releases painful stimuli long after the mechanical pressure has been relieved.36

Inflammation

The role of inflammation as a significant cause of low back pain is becoming better understood. An abnormal chemical environment may be most responsible for nerve root pain.14,37–39 Chemicals from the herniated pulposus3,14,36,38,39 or from by-products of local inflammation may inflame and
The sacroiliac joint is an accepted, although controversial, cause of low back pain. While the evaluation of the sacroiliac joint is beyond the scope of this article, it should be considered when evaluating low back pain.

CLASSIFICATION VERSUS DIAGNOSIS

Injury diagnosis relies upon a pathology-based model. The assessment is directed at identifying an anatomical abnormality or injury. The model assumes that the anatomical abnormality relates to the athlete’s signs and symptoms and is the pain source. Treatment and rehabilitation are based upon identifying the injury, correcting it, and thereby eliminating the signs and symptoms.

While this methodology works well with other anatomical sites, it does not work well with lumbar pain. The precise diagnosis of lumbar pain is unidentifiable in approximately 80% to 90% of patients. Numerous factors contribute to this difficulty. Mechanisms of injury vary so widely that they often produce little useful diagnostic information. Palpation is unreliable with lumbar injuries. An exact diagnosis cannot be given to most patients due to a loose association among symptoms, physical examination, and anatomical findings. Imaging studies do not add much credence to the diagnostic effort. Magnetic resonance imaging, computed tomography, and radiographs all have high false-positive findings in asymptomatic individuals. Nonselective use of these studies can mislead diagnostic efforts because abnormal imaging findings may be coincidental and not the actual cause of pain.

Examiners’ personal beliefs are probably the foundation of lumbar pain diagnoses. According to Bigos and Davis, the most diagnostic terms are synonymous with saying “I don’t know.” Evaluation and diagnostic methods were observed to vary significantly with profession, area of the country, and even within individual states. Professions differ in their beliefs of the most common anatomical cause of lumbar pain. The health care professional the athlete sees is often more of a determinant in diagnosis than his or her signs and symptoms. In reality, diagnostic efforts are often disappointing, and few lumbar diagnoses hold up to the rigors of science.

A classification system is an alternative to the diagnosis model. This system categorizes injuries by groups of signs and symptoms and does not rely on identifying the anatomical cause of pain. Decisions are based on the theory that specific treatments have a higher likelihood of succeeding in patients with specific clusters of signs and symptoms. Recently, classification systems have gained popularity as a viable alternative to the diagnosis of low back pain, although they are not directed specifically at the athlete.

Preliminary data suggest that patients treated with a classification approach have better results than patients treated without regard to classification.

A lumbar classification system is also a useful foundation for rehabilitation decisions. Successful rehabilitation is not dependent upon identifying an anatomical cause of pain. Each plan must be individualized, and a specific diagnosis often has no impact on initial rehabilitation choices. The patient’s pain-free functional ability primarily drives rehabilitation decisions. Exercises and activities that reduce symptoms are emphasized and those that exacerbate symptoms are avoided.

If the cause of most low back pain cannot be identified, then it is reasonable to use a classification system based on pain. We developed a lumbar injury classification system for athletes based primarily on the judgment approach. The decision basis for this approach is traditional custom, conventional wisdom, and personal experience. We believe the system resolves many of the difficulties associated with a pathology-directed evaluation. The classification system consists of 4 levels (Table 1). We use the system for referral decisions and determining participation status and as an initial guide for the rehabilitation plan.

CLASSIFICATION SYSTEM

Level 1: Lumbar Pain Only

Most athletes that we encounter fall into the level 1 classification, which consists of local lumbar pain only. Mechanism of injury does not affect our classification system; we have found it highly variable and often unknown, providing us with very little useful information. We do, however, use mechanism of injury as a tool to guide the athlete’s rehabilitation.

<table>
<thead>
<tr>
<th>Level</th>
<th>Description</th>
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<tbody>
<tr>
<td>1</td>
<td>Local lumbar pain only</td>
</tr>
<tr>
<td>2</td>
<td>Radiating pain</td>
</tr>
<tr>
<td>3</td>
<td>Neurologic deficits</td>
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<td>4</td>
<td>Serious conditions</td>
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Table 1. Classifications for Lumbar Injuries
History. We believe a thorough history is crucial for every athlete with lumbar pain. It consists of questions about the onset and duration of back pain, along with previous episodes of back pain. We obtain perceived pain levels from the athlete, both at rest and with activity. The level 1 athlete has a negative history for radiating pain and neurologic deficit, and thus, need not undergo a full neurologic evaluation.\textsuperscript{11} We discuss additional history questions in levels 2, 3, and 4, all of which are negative for the level 1 athlete.

Observation. We evaluate the athlete’s standing posture. This allows us to check postural alignment, willingness to weight bear evenly, and gait. It also gives us an opportunity to observe any asymmetries or contusions. We look for muscular definition along the entire back and abdomen and inspect for apparent differences in hip height.

Palpation. Palpation of the lumbar spine, whether for symmetry,\textsuperscript{14} segmental movement patterns,\textsuperscript{15} or muscle spasm,\textsuperscript{1} is of questionable value. Many clinicians promote palpation of the vertebrae to identify asymmetries or irregularities.\textsuperscript{15} However, not only are variations normal between individual vertebrae, but normal variations also exist between 2 sides of the same vertebra.\textsuperscript{12} Asymmetry is the rule, rather than the exception in the lumbar spine.\textsuperscript{14} In our experience, the level 1 athlete often reports point tenderness directly above and lateral to the posterior superior iliac spine. Overall, palpation is not integral to our classifications.

Active Range of Motion. Since the association between low back pain and spinal flexibility is not clinically significant,\textsuperscript{47} we do not take active range-of-motion measurements. We grossly assess active range of motion of the trunk for flexion, extension, side bending, and rotation and use it to direct our treatment approach. For instance, an athlete who demonstrates limitations or discomfort, or both, with lumbar flexion is more likely to benefit from a treatment program based on extension exercises.\textsuperscript{19,45} Additionally, we assess the effect of repetitive movements on the athlete’s pain level and location.

Decision and Action. The athlete with level 1 lumbar pain generally has full relief within a few weeks on conservative treatment. The athlete begins rehabilitation immediately and may remain in competition, although activity level may be altered based on the athlete’s tolerance. Occasionally, we completely remove the athlete from participation due to the pain level. If there is no improvement in symptoms after 4 to 6 weeks of treatment, we refer the athlete to our orthopaedist.

Spondylolysis. Spondylolysis is a defect of the pars interarticularis, often due to a stress fracture. It is a precursor to spondylolisthesis,\textsuperscript{48} which is a forward slippage of 1 vertebra on another. Spondylolysis is far more common in adolescent athletes.\textsuperscript{40,47,49,50} The occurrence rate in the overall population is 5\%,\textsuperscript{7,49} while it may be as high as 47\% in adolescent athletes with lumbar pain.\textsuperscript{40} Sports that involve repetitive hyperextension increase the risk for this injury.\textsuperscript{34,40,47,49–53}

It seems that spondylolysis is an exception in lumbar diagnosis. It can be accurately suspected with a clinical examination\textsuperscript{3,40,54} and identified on radiographs.\textsuperscript{3,40,44,49,51,54} When this diagnosis is suspected, the injury warrants a different decision and action. Therefore, we have made it a subgroup of level 1.

Signs and symptoms of spondylolysis are shown in Table 2. The spondylolysis can be unilateral or bilateral,\textsuperscript{2,54} which is reflected in the lumbar pain. Severe spondylolisthesis can produce radiating pain.\textsuperscript{1} We use both the single-leg\textsuperscript{52,54} (Figure 4) and the standing extension\textsuperscript{54} stress tests. While several authors\textsuperscript{40,52,54} cite hamstring spasm as a sign of spondylolysis, we have found it very subjective and of limited value.

Decision and Action. Any athlete who is less than 20 years of age, participates in a repetitive hyperextension sport, and has a positive extension test warrants referral to rule out spondylolysis. We may allow this athlete to participate based upon the severity of symptoms until a diagnosis is made.

Level 2: Radiating Pain

The hallmark of the level 2 classification is radiating lower extremity pain. Only 2\% to 3\% of individuals with low back pain complain of radiating pain.\textsuperscript{1} The level 2 athlete may have

![Figure 4. A positive single-leg extension test increases pain on the supported side in the lumbar region and indicates unilateral spondylolysis.](image-url)
any of the signs and symptoms of level 1 or no lumbar pain at all.

History. In the literature, the terms “sciatica,” “referred pain,” and “radiating pain” are used extensively and often interchangeably, which can be confusing and frustrating. Therefore, we refer to all of these terms collectively as “radiating pain” and define it as pain that occurs at a distance from the source. As described earlier, various structures of the lumbar spine have the potential to elicit radiating pain.

It is important to inquire about the exact location of the athlete’s symptoms. Normally, the athlete complains of pain in the buttock, posterior thigh, and posterior calf. Radiating pain along the anterior thigh may be related to the upper lumbar region. Anterior thigh pain is much less common and should alert the athletic trainer to consider a more serious condition, such as underlying hip pathology, diabetic neuropathy, kidney stones, or a retroperitoneal tumor. The athlete in the level 2 classification has a negative history for both altered sensation and lower extremity weakness.

Observation. The athlete occasionally presents with a list to 1 side. This is often a protective mechanism to relieve tension on the nerve roots or other inflamed structures. The athlete’s lower extremities will not demonstrate atrophy.

Neurologic Assessment. Even without indication of neurologic involvement in the history or observation, we conduct a neurologic assessment. However, no deficits are noted in the level 2 athlete. We describe our neurologic assessment further in the level 3 classification.

Special Tests. The 2 special tests we use are the straight-leg raise and the slump test, both of which may be positive. Although positive results on these tests alone do not warrant that the athlete be referred, they give us better insight into the severity of the condition. We consider a test positive if it reproduces the athlete's symptoms. It is our opinion that back pain alone does not constitute a positive test.

The straight-leg raise is more reliable in individuals under 30 years of age. If reproduction of the athlete’s symptoms occurs between 35° and 70° of leg elevation, we consider the test positive (Figure 5). The test places the greatest tension on the nerve roots in this range. Reproduction of the athlete’s symptoms beyond 70° is not a positive test.

The slump test maximally stresses the dural sheath and the nerve roots in the sitting position (Figure 6). Although the straight-leg raise test also stresses the neural structures, it only uses an inferior force. The slump test exerts both superior and inferior forces on the dura mater and places a tensile stress on the lumbar nerve roots. In addition to using the slump test as an evaluation tool, we also use the test to measure progress in the athlete who presented with radiating pain or neurologic deficits.

Decision and Action. Participation status for the level 2 athlete varies from full to none. We base this participation decision on the athlete’s functional ability, pain tolerance, and symptom severity. As in the level 1 athlete, we implement a rehabilitation program. We allow for a maximum of 2 weeks of intervention without improvement before we refer the athlete to our team physician.

Level 3: Neurologic Deficits

The level 3 classification comprises athletes with neurologic deficits. We define neurologic deficits as altered sensation, muscle weakness, atrophy, and reflex changes. The level 3 athlete may have any combination of these signs and symptoms. Additionally, the athlete may or may not present with the signs and symptoms discussed in levels 1 and 2.
Figure 6. A positive slump test reproduces radiating or neurologic symptoms in the extremity. The test may also be used to gauge progress during rehabilitation.

**History.** It is important to question the athlete about any altered sensation, including numbness, tingling, or pins and needles. We also ask about lower extremity strength. Has the athlete noticed weakness in a specific muscle or difficulty with a particular skill? A positive history for altered sensation or weakness suggests the presence of a neurologic deficit and, therefore, a level 3 classification.

**Observation.** As in levels 1 and 2 patients, the athletic trainer should complete a postural assessment. The level 3 athlete may also present with a list to 1 side. We examine the athlete’s lower extremities, looking for any observable atrophy. Circumferential measurements are among the most objective parts of a low back examination, and we take them if muscle wasting is suspected.

**Neurologic Assessment.** Neurologic compromise can result in muscle weakness, altered sensation, and reflex changes. The lower lumbar spine is responsible for 98% of nerve root compression. We reflect this in our suggested neurologic assessment in Table 3. Muscle weakness almost always occurs unilaterally; bilateral weakness indicates a much more serious condition and is covered in level 4. Sensory involvement due to nerve root compression is most evident in the distal extremities. Our sensory evaluation reflects this.

Testing of the deep tendon reflexes can be diminished or absent in the event of neurologic impairment.

**Special Tests.** We use the same stress tests as in level 2. Once again, both tests may be positive.

**Decision and Action.** The level 3 athlete is removed from participation and referred for further evaluation. We do, however, begin a rehabilitation program as soon as possible.

**Level 4: Red Flags**

Our primary objective in assessing lumbar pain is to rule out systemic disease and surgical emergencies. This can be effectively accomplished with an appropriate history. We ask every athlete with lumbar pain the “red flag” questions. Positive answers increase the suspicion for a serious condition and place the athlete in level 4, where the athlete remains until such a condition is ruled out.

**Cancer.** Although cancer is the underlying cause of low back pain in less than 1% of the population, it is the most common systemic disease that affects the spine. Cancer-related back pain is usually due to spinal metastases, most often from breast, lung, and prostate cancer. The historical features associated with cancer-related low back pain are shown in Table 4. We feel that unexplained weight loss of at least 10% of body weight is a positive finding, although the literature does not mention a threshold. Previous history of cancer has such a high specificity that any athlete who presents with low back pain and a history of cancer is referred immediately for further diagnostic testing.

**Spinal Infections.** Spinal infections can include, but are not limited to, vertebral osteomyelitis, diskitis, and epidural abscess and are the causes of low back pain in 0.01% of all patients. Spinal infections are usually blood borne from other

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**Table 3. Lower Lumbar Neurologic Tests**

<table>
<thead>
<tr>
<th>Tests</th>
<th>Vertebral Level</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>L3</td>
</tr>
<tr>
<td>Motor</td>
<td>Hip flexion</td>
</tr>
<tr>
<td></td>
<td>Knee extension</td>
</tr>
<tr>
<td>Sensory</td>
<td>Medial knee</td>
</tr>
<tr>
<td>Reflex</td>
<td>Patellar</td>
</tr>
</tbody>
</table>

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Journal of Athletic Training 209
In addition to lumbar pain, an athlete with cauda equina involvement usually complains of pain bilaterally in the buttocks, posterior thighs, and calves. Severe, progressive sensory deficits are often present over the buttocks, posterior-superior thighs, and perineal regions,12 commonly referred to as “saddle anesthesia.”7,11,12

Decision and Action. Cauda equina syndrome is the only true surgical emergency involving the lumbar spine.7,19,45,58 If cauda equina syndrome is suspected, then emergency department referral is necessary.

CONCLUSIONS

Our classification system groups athletes by signs and symptoms to make initial participation, rehabilitation, and referral decisions. Our system is also suggestive of a hierarchy of concern for the athletic trainer. Lowest on this hierarchy is level 1, once spondylolysis is eliminated. Participation is based on the athlete’s pain and function, rehabilitation is begun, and referral can wait 4 to 6 weeks. An athlete in level 2, with radiating pain, should increase the athletic trainer’s concern. It is important to remember, however, that minor trauma and inflammation can result in radiating pain. As in level 1, rehabilitation is implemented, and participation is based on pain and tolerance. Referral can wait up to 2 weeks for a level 2 patient.

Levels 3 and 4 should cause the athletic trainer the most concern. An athlete with neurologic deficits, level 3, warrants removal from participation and referral in a timely manner. A level 4 classification represents back pain due to a systemic condition or cauda equina syndrome. The athletic trainer must take a thorough history to ensure that an athlete with a potentially serious condition is identified. Athletes suspected of a serious condition should be immediately referred for follow-up, but these athletes may participate based on tolerance and begin rehabilitation until diagnosed by a physician. The most urgent problem regarding lumbar pain is cauda equina syndrome. This level 4 classification should be ruled out in any athlete presenting with neurologic deficits.

ACKNOWLEDGMENTS

We thank Dr. Harvey Kesselman and The Richard Stockton College for their support of this project and for funding the cost of our illustrations. We also extend our sincere appreciation to Tracie Aretz, MSMI, for her priority production and reproduction of our artwork.

REFERENCES

National Athletic Trainers’ Association Position Statement: Fluid Replacement for Athletes

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Objective: To present recommendations to optimize the fluid-replacement practices of athletes.

Background: Dehydration can compromise athletic performance and increase the risk of exertional heat injury. Athletes do not voluntarily drink sufficient water to prevent dehydration during physical activity. Drinking behavior can be modified by education, increasing accessibility, and optimizing palatability. However, excessive overdrinking should be avoided because it can also compromise physical performance and health. We provide practical recommendations regarding fluid replacement for athletes.

Recommendations:

Educate athletes regarding the risks of dehydration and overhydration on health and physical performance. Work with individual athletes to develop fluid-replacement practices that optimize hydration status before, during, and after competition.

Key Words: athletic performance, dehydration, heat illness, hydration protocol, hydration status, oral rehydration solution, rehydration

During exercise, evaporation is usually the primary mechanism of heat dissipation. The evaporation of sweat from the skin’s surface assists the body in regulating core temperature. If the body cannot adequately evaporate sweat from the skin’s surface, core temperature rises rapidly. A side effect of sweating is the loss of valuable fluids from the finite reservoir within the body, the rate being related to exercise intensity, individual differences, environmental conditions, acclimatization state, clothing, and baseline hydration status. Athletes whose sweat loss exceeds fluid intake become dehydrated during activity. Therefore, a person with a high sweat rate who undertakes intense exercise in a hot, humid environment can rapidly become dehydrated. Dehydration of 1% to 2% of body weight begins to compromise physiologic function and negatively influence performance. Dehydration of greater than 3% of body weight further disturbs physiologic function and increases an athlete’s risk of developing an exertional heat illness (ie, heat cramps, heat exhaustion, or heat stroke). This level of dehydration is common in sports; it can be elicited in just an hour of exercise or even more rapidly if the athlete enters the exercise session dehydrated. The onset of significant dehydration is preventable, or at least modifiable, when hydration protocols are followed to assure all athletes the most productive and the safest athletic experience.

The purpose of this position stand is to 1) provide useful recommendations to optimize fluid replacement for athletes, 2) emphasize the physiologic, medical, and performance considerations associated with dehydration, and 3) identify factors that influence optimal rehydration during and after athletic participation.

RECOMMENDATIONS

The National Athletic Trainers’ Association (NATA) recommends the following practices regarding fluid replacement for athletic participation:

1. Establish a hydration protocol for athletes, including a rehydration strategy that considers the athlete’s sweat rate, sport dynamics (eg, rest breaks, fluid access), environmental factors, acclimatization state, exercise duration, exercise intensity, and individual preferences (see Table 1 for examples of potential outcomes).
2. A proper hydration protocol considers each sport’s unique features. If rehydration opportunities are frequent (eg, baseball, football, track and field), the athlete can consume smaller volumes at a convenient pace based on sweat rate and environmental conditions. If rehydration must occur at specific times (eg, soccer, lacrosse, distance running), the athlete must consume fluids to maximize hydration within the sport’s confines and rules.

3. Fluid-replacement beverages should be easily accessible in individual fluid containers and flavored to the athlete’s preference. Individual containers permit easier monitoring of fluid intake. Clear water bottles marked in 100-mL (3.4-fl oz) increments provide visual reminders to athletes to drink beyond thirst satiation or the typical few gulps. Carrying water bottles or other hydration systems, when practical, during exercise encourages greater fluid volume ingestion.

4. Athletes should begin all exercise sessions well hydrated. Hydration status can be approximated by athletes and athletic trainers in several ways (Table 2). Assuming proper hydration, pre-exercise body weight should be relatively consistent across exercise sessions. Determine the percentage difference between the current body weight and the hydrated baseline body weight. Remember that body weight is dynamic. Frequent exercise sessions can induce nonfluid-related weight loss influenced by timing of meals and defecation, time of day, and calories expended in exercise. The simplest method is comparison of urine color (from a sample in a container) with a urine color chart (Figure). Measuring urine specific gravity (USG) with a refractometer (available for less than $150) is less subjective than comparing urine color and also simple to use. Urine volume is another indicator of hydration status but inconvenient to collect and measure. For color analysis or specific gravity, use midstream urine collection for consistency and accuracy. Remember that body weight changes during exercise give the best indication of hydration status. Because of urine and body weight dynamics, measure urine before exercise and check body weight (percentage of body weight change) before, during, and after exercise sessions to estimate fluid balance.

5. To ensure proper pre-exercise hydration, the athlete should consume approximately 500 to 600 mL (17 to 20 fl oz) of water or a sports drink 2 to 3 hours before exercise and 200 to 300 mL (7 to 10 fl oz) of water or a sports drink 10 to 20 minutes before exercise.

6. Fluid replacement should approximate sweat and urine losses and at least maintain hydration at less than 2% body weight reduction. This generally requires 200 to 300 mL (7 to 10 fl oz) every 10 to 20 minutes. Specific individual recommendations are calculated based on sweat rates, sport dynamics, and individual tolerance. Maintaining hydration status in athletes with high sweat rates, in sports with limited fluid access, and during high-intensity exercise can be difficult, and special efforts should be made to minimize dehydration. Dangerous hyperhydration is also a risk if athletes drink based on published recommendations and not according to individual needs.

7. Postexercise hydration should aim to correct any fluid loss accumulated during the practice or event. Ideally completed within 2 hours, rehydration should contain water to restore hydration status, carbohydrates to replenish glycogen stores, and electrolytes to speed rehydration. The primary goal is the immediate return of physiologic function (especially if an exercise bout will follow). When rehydration must be rapid, the athlete should compensate for obligatory urine losses incurred during the rehydration process and drink about 25% to 50% more than sweat losses to assure optimal hydration 4 to 6 hours after the event.

8. Fluid temperature influences the amount consumed. While individual differences exist, a cool beverage of 10°C to 15°C (50° to 59°F) is recommended.

9. The Wet Bulb Globe Temperature (WBGT) should be ascertained in hot environments. Very high relative humidity limits evaporative cooling; the air is nearly saturated with water vapor, and evaporation is minimized. Thus, dehydration associated with high sweat losses can induce a rapid core temperature increase due to the inability to dissipate heat. Measuring core temperature rectally allows the athlete’s thermal status to be accurately determined. See the NATA position statement on heat illnesses for expanded information on this topic.

10. In many situations, athletes benefit from including carbohydrates (CHOs) in their rehydration protocols. Consuming CHOs during the pre-exercise hydration session (2 to 3 hours pre-exercise), as in item 5, along with a normal daily diet increases glycogen stores. If exercise is intense, then consuming CHOs about 30 minutes pre-exercise may also be beneficial. Include CHOs in the rehydration beverage during exercise if the session lasts longer than 45 to 50 minutes or is intense. An ingestion rate of about 1 g/min (0.04 oz/min) maintains optimal carbohydrate metabolism: for example, 1 L of a 6% CHO drink per hour of exercise. CHO concentrations greater than 8% increase the rate of CHO delivery to the body but compromise the rate of fluid emptying from the stomach and absorbed from the intestine. Fruit juices, CHO gels, sodas, and some sports drinks have CHO concentrations greater than 8% and are not recommended during an exercise session as the sole beverage. Athletes should consume CHOs at least 30 minutes before the normal onset of fatigue and earlier if the environmental conditions are unusually extreme, although this may not apply for very intense short-term exercise, which may require earlier intake of CHOs. Most CHO forms (ie, glucose, sucrose, glucose polymers) are suitable, and the absorption rate is maximized when multiple forms are consumed simultaneously. Substances to be limited include fructose (which may cause gastrointestinal distress); those to be avoided include caffeine, alcohol (which may increase urine output and reduce fluid retention), and carbonated beverages (which may reduce voluntary fluid intake due to stomach fullness).

11. Those supervising athletes should be able to recognize the basic signs and symptoms of dehydration: thirst, irritability, and general discomfort, followed by headache, weakness, dizziness, cramps, chills, vomiting, nausea, head or neck heat sensations, and decreased performance. Early diagnosis of dehydration decreases the occurrence and severity of heat illness. A conscious, cognizant, dehydrated athlete without gastrointestinal distress can aggressively rehydrate orally, while one with mental compromise from dehydration or gastrointestinal distress should be transported to a medical facility for intravenous rehydration. For a complete description of heat illnesses and issues
related to hyperthermia, see the NATA position statement on heat illnesses.

12. Inclusion of sodium chloride in fluid-replacement beverages should be considered under the following conditions: inadequate access to meals or meals not eaten; physical activity exceeding 4 hours in duration; or during the initial days of hot weather. Under these conditions, adding modest amounts of salt (0.3 to 0.7 g/L) can offset salt loss in sweat and minimize medical events associated with electrolyte imbalances (eg, muscle cramps, hypernatremia). Adding a modest amount of salt (0.3 to 0.7 g/L) to all hydration beverages would be acceptable to stimulate thirst, increase voluntary fluid intake, and decrease the risk of hypernatremia and should cause no harm.

13. Calculate each athlete's sweat rate (sweating rate = pre-exercise body weight – postexercise body weight + fluid intake – urine volume/exercise time in hours) for a representative range of environmental conditions, practices, and competitions (Table 3). This time-consuming task can be made easier by weighing a large number of athletes before an intense 1-hour practice session and then reweighing them at the end of the 1-hour practice. Sweat rate can now be easily calculated (do not allow rehydration or urination during this 1 hour when sweat rate is being determined to make the task even easier). This calculation is the most fundamental consideration when establishing a rehydration protocol. Average sweat rates from the scientific literature or other athletes can vary from 0.5 L/h to more than 2.5 L/h (0.50 to 2.50 kg/h) and are not ideal to use.

14. Heat acclimatization induces physiologic changes that may alter individual fluid-replacement considerations. First, sweat rate generally increases after 10 to 14 days of heat exposure, requiring a greater fluid intake for a similar bout of exercise. An athlete's sweat rate should be reassessed after acclimatization. Second, moving from a cool environment to a warm environment increases the overall sweat rate for a bout of exercise. The athlete's hydration status must be closely monitored for the first week of exercise in a warm environment. Third, increased sodium intake may be warranted during the first 3 to 5 days of heat exposure, since the increased thermal strain and associated increased sweat rate increase the sodium lost in sweat. Adequate sodium intake optimizes fluid palatability and absorption during the first few days and may decrease exercise-associated muscle cramping. After 5 to 10 days, the sodium concentration of sweat decreases, and normal sodium intake suffices.

15. All sports requiring weight classes (ie, wrestling, judo, rowing) should mandate a check of hydration status before weigh-in to ensure the athlete is not dehydrated. A USG less than or equal to 1.020 or urine color less than or equal to 4 should be the upper range of acceptable on weigh-in. Any procedures used to induce dramatic dehydration (eg, diuretics, rubber suits, exercising in a sauna) are strictly prohibited.

16. Hyperhydration by ingesting a pre-exercise glycerol and water beverage has equivocal support from well-controlled studies. At this time, evidence is insufficient to endorse the practice of hyperhydration via glycerol. Also, a risk of side effects such as headaches and gastrointestinal distress exists when glycerol is consumed.

17. Consider modifications when working with prepubescent and adolescent athletes who exercise intensely in the heat and may not fully comprehend the medical and performance consequences of dehydration. Focus special attention on schedules and event modification to minimize environmental stress and maximize time for fluid replacement. Make available the most palatable beverage possible. Educate parents and coaches about rehydration and the signs of dehydration. Monitor and remove a child from activity promptly if signs or symptoms of dehydration occur.

18. Large-scale event management (eg, tournaments, camps) requires advance planning. Ample fluid and cups should be conveniently available. With successive practice sessions during a day or over multiple days (as in most summer sport camps), check hydration status daily before allowing continued participation. Be aware of unhealthy behaviors, such as eating disorders and dehydration in weight-class sports. Use extra caution with novice and unconditioned athletes, and remember, many athletes are not supervised on a daily basis. If the WBGT dictates, modify events (change game times or cancel) or change game dynamics (insert nonroutine water breaks, shorten game times). Recruit help from fellow athletic trainers in local schools, student athletic trainers, and athletes from other sports to ensure that hydration is maintained at all venues (ie, along a road race course, on different fields during a tournament). Be sure all assistants can communicate with the supervising athletic trainer at a central location. For successive-day events, provide educational materials on rehydration principles to inform athletes and parents of this critical component of athletic performance.

19. Implementing a hydration protocol for athletes will only succeed if athletes, coaches, athletic trainers, and team physicians realize the importance of maintaining proper hydration status and the steps required to accomplish this goal. Here are the most critical components of hydration education:

- Educate athletes on the effects of dehydration on physical performance.
- Inform athletes on how to monitor hydration status.
- Convince athletes to participate in their own hydration protocols based on sweat rate, drinking preferences, and personal responses to different fluid quantities.
- Encourage coaches to mandate rehydration during practices and competitions, just as they require other drills and conditioning activities.
- Have a scale accessible to assist athletes in monitoring weight before, during, and after activity.
- Provide the optimal oral rehydration solution (water, CHO, electrolytes) before, during, and after exercise.
- Implement the hydration protocol during all practices and games, and adapt it as needed.
- Finally, encourage event scheduling and rule modifications to minimize the risks associated with exercise in the heat.

**BACKGROUND AND LITERATURE REVIEW**

**Dehydration and Exercise**

**Physiologic Implications.** All physiologic systems in the human body are influenced by dehydration. The degree of
evaporation (ie, high humidity, dehydration) will have pro-
during exercise in the heat. Therefore, any factor that limits
the rate of water loss via sweating, progressive dehydration
hot, dry conditions, evaporation may account for as much as
from evaporation is the predominant heat-dissipating mecha­
nism for the exercising athlete. In warm, humid conditions,
98% of cooling.5 If sufficient fluids are not consumed to offset
contribution of each method depends on the ambient temper­
ature, relative humidity, and exercise intensity. As ambient
rise, conduction and convection decrease mark­
ly, and radiation becomes nearly insignificant.4'5 Heat loss
from evaporation is the predominant heat-dissipating mech­
anism for the exercising athlete. In warm, humid conditions,
may account for more than 80% of heat loss. In hot,
ditions, evaporation may account for as much as
98% of cooling.2 If sufficient fluids are not consumed to offset
the rate of water loss via sweating, progressive dehydration
will occur. The sweating response is critical to body cooling
during exercise in the heat. Therefore, any factor that limits
(ev, high humidity, dehydration) will have pro-
found effects on physiologic function and athletic perfor­
mance.
Water is the major component of the human body, accounting
for approximately 73% of lean body mass.6 Body water is
distributed within and between cells and in the plasma. At rest,
approximately 30% to 35% of total body mass is intracellular
fluid, 20% to 25% is interstitial fluid, and 5% is plasma.5'6
Water movement between compartments occurs due to hydro­
static pressure and osmotic-oncotic gradients.5,6 Because sweat
is hypotonic relative to body water, the elevation of extracel­

tular tonicity results in water movement from intracellular to
extracellular spaces.5-9 As a consequence, all water com­
partments contribute to water deficit with dehydration.6,10 Most of
the resultant water deficits associated with dehydration, how­
ever, come from muscle and skin.11 The resulting hypovole­
ic-hyperosmolality condition is thought to precipitate many of
the physiologic consequences associated with dehydration.12

A major consequence of dehydration is an increase in core
temperature during physical activity, with core temperature
rising an additional 0.15 to 0.20°C for every 1% of body
weight lost (due to sweating) during the activity.13,14 The
added thermal strain occurs due to both impaired skin blood
flow and altered sweating responses.15-21 which is best illus­
trated by the delayed onset of skin vasodilation and sweating
when a dehydrated person begins to exercise.6 These thermo­
regulatory changes may negate the physiologic advantages
resulting from increased fitness21,22 and heat acclimatiza­
tion.21,23 Additionally, heat tolerance is reduced and exercise
time to exhaustion occurs at lower core temperatures with
hypohydration.24

accompanied by the increase in thermal strain is greater
vascular strain, as characterized by decreased stroke
volume, increased heart rate, increased systemic vascular
resistance, and possibly lower cardiac output and mean arterial
pressure.25-31 Similar to body temperature changes, the mag­
nitude of cardiovascular changes is proportional to the water

<table>
<thead>
<tr>
<th>Parameter to Consider</th>
<th>Example A: College Soccer, Katie (60 kg)*</th>
<th>Example B: High School Basketball, Mike (80 kg)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) WBGT</td>
<td>28.3°C (83°F)</td>
<td>21.1°C (70°F)</td>
</tr>
<tr>
<td>2) Sweat rate†</td>
<td>1.7 L/h</td>
<td>1.2 L/h</td>
</tr>
<tr>
<td>3) Acclimatized</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>4) Length of activity</td>
<td>2 45-minute halves</td>
<td>4 10-minute quarters</td>
</tr>
<tr>
<td>5) Intensity</td>
<td>Game situation (maximal)</td>
<td>Game situation (maximal)</td>
</tr>
<tr>
<td>6) Properly prehydrated</td>
<td>No (began −2% body weight)</td>
<td>Yes</td>
</tr>
<tr>
<td>7) Individual container</td>
<td>Yes</td>
<td>(just cups)</td>
</tr>
<tr>
<td>8) Type of beverage</td>
<td>5% to 7% CHO solution</td>
<td>5% to 7% CHO solution</td>
</tr>
<tr>
<td>9) Assess hydration status</td>
<td>At halftime (with scale)</td>
<td>No</td>
</tr>
<tr>
<td>10) Available breaks</td>
<td>Halftime</td>
<td>Quarters, half timeouts</td>
</tr>
<tr>
<td>11) Amount given</td>
<td>Maximal comfortable predetermined amount</td>
<td>200 mL at quarter breaks</td>
</tr>
<tr>
<td></td>
<td>given at half time (about 700 to 1000 L)</td>
<td>400 mL at half time</td>
</tr>
<tr>
<td></td>
<td></td>
<td>100 mL at 1 timeout/half</td>
</tr>
<tr>
<td>12) End hydration status</td>
<td>−4.8% body weight</td>
<td>Normal hydration</td>
</tr>
<tr>
<td>13) Hydrated body weight</td>
<td>60 kg</td>
<td>80 kg</td>
</tr>
<tr>
<td></td>
<td>Pre-exercise body weight</td>
<td>80 kg</td>
</tr>
<tr>
<td></td>
<td>Halftime body weight</td>
<td>No measure</td>
</tr>
<tr>
<td></td>
<td>Postexercise body weight</td>
<td>80.1 kg</td>
</tr>
</tbody>
</table>

*Assumptions: Both are starters and play a full game.† Sweat rate determined under similar parameters described in example (ie, acclimatization state, WBGT, intensity, etc) under normal game conditions (ie, no injury timeouts, overtime, etc).

Note: Keep results on record for future reference.

Table 2. Indexes of Hydration Status

<table>
<thead>
<tr>
<th>Condition</th>
<th>% Body Weight Change*</th>
<th>Urine Color</th>
<th>USG†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Well hydrated</td>
<td>[pre-exercise body weight - postexercise body weight] / pre-exercise body weight x 100</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Minimal dehydration</td>
<td>−1 to −3</td>
<td>3 or 4</td>
<td>1.010-1.020</td>
</tr>
<tr>
<td>Significant dehydration</td>
<td>−3 to −5</td>
<td>5 or 6</td>
<td>1.021-1.030</td>
</tr>
<tr>
<td>Serious dehydration</td>
<td>&gt;5</td>
<td>&gt;6</td>
<td>&gt;1.030</td>
</tr>
</tbody>
</table>

†USG, urine specific gravity.

See Figure for urine color chart and references. Please note that obtaining a urine sample may not be possible if the athlete is seriously dehydrated. These are physiologically independent entities, and the numbers provided are only general guidelines.

dehydration dictates the extent of systemic compromise. Isol­
ating the physiologic changes that contribute to decrements in
performance is difficult, as any change in 1 system (ie, cardio­
vascular) influences the performance of other systems
(ie, thermoregulatory, muscular).5

The body attempts to balance endogenous heat production
and exogenous heat accumulation by heat dissipation via
conduction, convection, evaporation, and radiation.4 The relative
contribution of each method depends on the ambient temper­
ature, relative humidity, and exercise intensity. As ambient
temperature rises, conduction and convection decrease mark­
edly, and radiation becomes nearly insignificant.4,2 Heat loss
from evaporation is the predominant heat-dissipating mech­
anism for the exercising athlete. In warm, humid conditions,
evaporation may account for more than 80% of heat loss. In hot,
dry conditions, evaporation may account for as much as
98% of cooling.2 If sufficient fluids are not consumed to offset
the rate of water loss via sweating, progressive dehydration
will occur. The sweating response is critical to body cooling
during exercise in the heat. Therefore, any factor that limits
evaporation (ie, high humidity, dehydration) will have pro-


deficit. For example, heart rate rises an additional 3 to 5 beats per minute for every 1% of body weight loss.14 The stroke-volume reduction seen with dehydration appears to be due to reduced central venous pressure, resulting from reduced blood volume and the additional hyperthermia imposed by dehydration.28-32

Both hypovolemia7,17,35,36 and hypertonicity7,35,37-39 have been suggested as mechanisms for the altered thermoregulatory and cardiovascular responses during dehydration. Manipulation of each factor independently has resulted in decreased blood flow to the skin and sweating responses.28,34 Some authors7,35 have argued that hypovolemia is primarily responsible for the thermoregulatory changes by reducing cardiac preload and may alter the feedback to the hypothalamus via the atrial pressure receptors (baroreceptors). The hypothalamic thermoregulatory centers may induce a decrease in the blood volume perfusing the skin in order to reestablish a normal cardiac preload. Some studies30,31 have provided support for this hypothesis, but it is clearly not the only variable influencing thermoregulation during hypohydration. Two hypotheses explain the role of hyperosmolarity on the thermoregulatory system. Peripheral regulation may occur via the strong osmotic pressure influence of the interstitium, limiting the available fluid sources for the eccrine sweat glands.42 However, while this peripheral influence is likely, it seems more feasible that central brain regulation plays the largest role.7 The neurons surrounding the thermoregulatory control centers in the hypothalamus are sensitive to osmolality.43,44 Changes in the plasma osmolality of the blood perfusing the hypothalamus affect body water regulation and the desire for fluid consumption.28,32,45 It is likely that both hypovolemia and hypertonicity contribute to body fluid regulation.

Potential changes at the level of the muscle tissue include a possible increased rate of glycogen degradation,18,46,47 elevated muscle temperature,48 and increased lactate levels.49 These changes may be caused by a decrease in blood perfusion of the muscle tissue during the recovery between contractions.50

The psychological changes associated with exercise in a dehydrated state should not be overlooked. Dehydration increases the rating of perceived exertion and impairs mental functioning.14,51 Dehydration also decreases the motivation to exercise and decreases the time to exhaustion, even in instances when strength is not compromised.52-54 These are important factors when considering the motivation required by high-level athletes to maintain maximal performance.

Performance Implications. Studies investigating the role of dehydration on muscle strength have generally shown decrements in performance at 5% or more dehydration.15,33,55-58 The greater the degree of dehydration, the more negative the impact on physiologic systems and overall athletic performance.

Most studies30,55,59-62 that address the influence of dehydration on muscle endurance show that dehydration of 3% to 4% elicits a performance decrement, but in 1 study,33 this finding was not supported. Interestingly, hypohydrated wrestlers who were working at maximal or near-maximal muscle activity for more than 30 seconds had a decrease in performance.53 The environmental conditions may also play an important role in muscle endurance.33,48

The research concerning maximal aerobic power and the physical work capacity for extended exercise is relatively consistent. Maximal aerobic power usually decreases with more than 3% hypohydration.6 The heat, aerobic power decrements are exaggerated.53 Even at 1% to 2% hypohydration in a cool environment,34 loss of aerobic power is demonstrated. Two important studies have noted a decrease in physical work capacity with less than 2% dehydration during intense exercise in the heat.66,67 When the percentage of dehydration increased, physical work capacity decreased by as much as 35% to 48%.68 Physical work capacity often decreased even when maximal aerobic power did not change.46,64,65 Hypohydration of 2.5% of body weight results in significant performance decrements while exercising in the heat, regardless of fitness or heat acclimation status, although enhanced fitness and acclimation can lessen the effects of dehydration.69 Partial rehydration will enhance performance during an ensuing exercise session in the heat, which is important when faced with the reality of sports situations.49,70 The performance decrements noted with low to moderate levels of hypohydration may be due to an increased perception of fatigue.50

Rehydration and Exercise

Factors Influencing Rehydration. The degree of environmental stress is determined by temperature, humidity, wind speed, and radiant energy load, which induce physiologic changes that affect the rehydration process.71-73 Fluid intake

Table 3. Sample Sweat Rate Calculation*

<table>
<thead>
<tr>
<th>Name</th>
<th>Date</th>
<th>Before Exercise</th>
<th>After Exercise</th>
<th>ΔBW (C-D)</th>
<th>Drink Volume</th>
<th>Urine Volume†</th>
<th>Sweat Loss (E+F–G)</th>
<th>Exercise Time</th>
<th>Sweat Rate (H/I)</th>
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</thead>
<tbody>
<tr>
<td>Kelly K</td>
<td>9/15</td>
<td>61.7 kg</td>
<td>60.3 kg</td>
<td>1400 g</td>
<td>420 mL</td>
<td>90 mL</td>
<td>1730 mL</td>
<td>90 min</td>
<td>19 mL/min</td>
</tr>
</tbody>
</table>

†Weight of urine should be subtracted if urine was excreted prior to postrace body weight.
‡In the example, Kelly K. should drink about 1 L (32 oz) of fluid during each hour of activity to remain well hydrated.
increases substantially when ambient temperature rises above 25°C; the rehydration stimulus can also be psychological. An athlete exercising in the heat will voluntarily ingest more fluid if it is chilled. Individual differences in learned behavior also play a role in the rehydration process. An athlete who knows that rehydrating enhances subsequent performance is more apt to consume fluid before significant dehydration occurs, so appropriate education of athletes is essential.

The physical characteristics of the rehydration beverage can dramatically influence fluid replacement. Salinity, color, sweetness, temperature, flavor, carbonation, and viscosity all affect how much an athlete drinks. Since most fluid consumed by athletes is with meals, the presence of ample fluid during meals and adequate amount of time to eat are critical to rehydration. When access to meals is limited, a CHO-electrolyte beverage will help maintain CHO and electrolyte intake along with hydration status.

Other factors that contribute to fluid replacement include the individual’s mood (calmness is associated with enhanced rehydration) and the degree of concentration required by the task. For example, industrial laborers need frequent breaks to rehydrate because they must remain focused on a specific task. This need for concentration may explain why many elite mountain bikers use a convenient back-mounted hydration system instead of the typical rack-mounted water bottle. The back-mounted water reservoir may allow the cyclist to enhance rehydration while remaining focused on terrain, speed, gears, braking, and exertion. Accessibility to a fluid and ease of drinking may explain why athletes consume more fluid while cycling compared with running in a simulated duathlon.

Hydration before Exercise. An athlete should begin exercising well hydrated. Many athletes who perform repeated bouts of exercise on the same day or on consecutive days can become chronically dehydrated. When a hypohydrated athlete begins to exercise, physiologic mechanisms are compromised and the extent of the dysfunction is related to the degree of thermal stress experienced by the athlete. Athletes may require substantial assistance in obtaining fluids as evidenced by the phenomena of voluntary (when individuals drink insufficient quantities to replace fluid losses) and involuntary dehydration.

Athletes should ingest 500 mL of fluid 2 hours before the event (which allows ample time to urinate excess fluid) to ensure proper hydration and physiologic function at the onset of exercise. Mandatory pre-exercise hydration is physiologically advantageous and more effective than hydration dictated by often insufficient personal preference. Ingesting a nutritionally balanced diet and fluids during the 24 hours before an exercise session is also crucial. Increasing CHO intake before endurance activity may be beneficial for performance and may even enhance performance for activities as short as 10 minutes, but it may have a limited effect on resistance exercise.

There has been recent interest in potential benefits of purposefully overhydrating before exercise to postpone the onset of water deficit. While an enhanced hydration state is often reported with glycerol use, this does not always translate into a performance improvement. A recent study found increased exercise time and plasma volume during exercise to exhaustion in the heat when subjects were rehydrated with water and glycerol before exercise as compared with rehydration using an equal volume of water without glycerol. However, another study found no benefits of glycerol ingestion when the ensuing exercise took place in a thermoneutral environment. Hyperhydrating before exercise, even without glycerol, may enhance thermoregulatory function and limit the performance decrements normally noted with dehydration while exercising in the heat (WBGT > 25°C). A key point is that the benefits associated with glycerol use seem to be negated when proper hydration status is maintained during exercise. However, many athletes are unable to maintain hydration, so hyperhydration may be beneficial in extreme conditions when fluid intake cannot match sweat loss.

Rehydration during Exercise. Proper hydration during exercise will influence cardiovascular function, thermoregulatory function, muscle functioning, fluid volume status, and exercise performance. This topic has been extensively reviewed through the years, but some recent compilations are especially notable. Proper hydration during exercise enhances heat dissipation (increased skin blood flow and sweating rate), limits plasma hypertonicity, and helps sustain cardiac output. The enhanced evaporative cooling that can occur (due to increased skin blood flow and maintained perfusion of working muscles) is the result of sustained cardiac filling pressure. Rehydration during exercise conserves the centrally circulating fluid volume and allows maximal physiologic responses to intense exercise in the heat.

Two important purposes of rehydration are to decrease the rate of hyperthermia and to maintain athletic performance. A classic study showed that changes in rectal temperature during exercise depended on the degree of fluid intake. When water intake equaled sweat loss, rise in core temperature was slowest when compared with ad libidum water and no-water groups. This benefit of rehydration on thermoregulatory function is likely due to increased blood circulation and heat dissipation (increased skin blood flow and sweating rate), limits plasma hypertonicity, and helps sustain cardiac output. The enhanced evaporative cooling that can occur (due to increased skin blood flow and maintained perfusion of working muscles) is the result of sustained cardiac filling pressure. Rehydration during exercise conserves the centrally circulating fluid volume and allows maximal physiologic responses to intense exercise in the heat.

Athletes generally do not rehydrate to pre-exercise levels during exercise due to personal choice, fluid availability, the circumstances of competition, or a combination of these factors. Athletes should aim to drink quantities equal to sweat and urine losses, and while they rarely meet this goal, athletes can readily handle these large volumes (> 1 L/h). Additionally, athletes may not need to exactly match fluid intake with sweat loss to maintain water balance given the small contribution of water from metabolic processes. Appealing to individual taste preferences may encourage athletes to drink more fluids. In addition, including CHOs and electrolytes (especially sodium and potassium) in the rehydration drink can maintain blood glucose, CHO oxidation, and electrolyte balance and can maintain performance.

if the exercise session exceeds about 50 minutes in duration.\textsuperscript{79,118,130,134–152} Also, recent evidence\textsuperscript{153,154} indicates that athletes performing extremely intense intermittent activity with total exercise times of less than 50 minutes may benefit from ingestion of CHOs in the rehydration beverage.

Rates of gastric emptying and intestinal absorption should also be considered.\textsuperscript{118,155–160} Fluid volume,\textsuperscript{161} fluid calorie content, fluid osmolality, exercise intensity,\textsuperscript{162} environmental stress,\textsuperscript{162} and fluid temperature\textsuperscript{107} are some of the most important factors.\textsuperscript{28} in determining the rates of gastric emptying and small intestine absorption (the small intestine is the primary site of fluid absorption). The single most important variable may be the volume of fluid in the stomach.\textsuperscript{163,164} Maintaining 400 to 600 mL of fluid in the stomach (or the maximum tolerated) will optimize gastric emptying.\textsuperscript{79} If CHOs are included in the fluid, the concentration should be 4\% to 8\%. Concentrations higher than 8\% slow the rate of fluid absorption.\textsuperscript{165,166} Intense exercise (>80\% of VO\textsubscript{2} max) may also decrease the rate of gastric emptying.\textsuperscript{155} Frequent ingestion (every 15 to 20 minutes) of a moderate fluid volume (200 mL) may be ideal, but is not feasible in sports with extended periods between breaks. The rates of gastric emptying and intestinal absorption likely influence the speed of movement of the ingested fluids into the plasma volume.\textsuperscript{167} Since the gastric emptying and intestinal absorption rates are not compromised with the addition of a 6\% carbohydrate solution as compared with water, fluid replacement and energy replenishment are equally achievable.\textsuperscript{116,167–171} The rate of gastric emptying is slowed\textsuperscript{163,172} by significant dehydration (>4\%), which complicates rehydration and may increase gastrointestinal discomfort.\textsuperscript{163,172} Regardless, rehydration will still benefit the athlete’s hydration status.\textsuperscript{172}

Rehydration during exercise is also influenced by the state of acclimatization of the athlete. Heat acclimatization is achieved after 5 to 10 days of training in a hot environment and will increase sweat rate, decrease electrolyte losses in the sweat, and allow athletes to better tolerate exercise in the heat.\textsuperscript{173,174} Heat acclimatization modestly increases rehydration needs due to greater sweating. Fortunately, an athlete who is heat acclimatized has fewer deficits associated with dehydration\textsuperscript{172} and tends to be a “better” voluntary drinker (ingests fluid earlier and more often).\textsuperscript{1,34}

An athlete who exercises for more than 4 hours and hydrates excessively (well beyond sweat loss) only with water or low-solute beverages may be susceptible to a relatively rare condition known as symptomatic hyponatremia (also known as water intoxication).\textsuperscript{76,108,176,177} Ultimately, the body cannot excrete the consumed fluid rapidly enough to prevent intracellular swelling, which is sufficient to produce neuropsychological manifestations. Patients present with serum sodium levels below 130 to 135 mmol/L, and the sequence of hyponatremia can result in death if not treated.\textsuperscript{177} The condition can most likely be avoided if sodium is consumed with the rehydration beverage and if fluid intake does not exceed sweat losses.\textsuperscript{76,79,108}

Every athlete will benefit from attempting to match intake with sweating rate and urine losses. Individual differences exist for gastric emptying and availability of fluids during particular sports. Rehydration procedures should be tested in practice and individually modified to maximize performance in competition.\textsuperscript{97,108,116,156}

**Rehydration after Exercise.** Replenishing fluid volume\textsuperscript{176,179} and glycogen stores is critical in the recovery of many body processes, including the cardiovascular, thermoregulatory, and metabolic activities.\textsuperscript{71,97,178,180,181}

Based on volume and osmolality, the best fluid to drink after exercise to replace the fluids that are lost via sweating may not be water.\textsuperscript{71,182–184} Consuming water alone decreases osmolality, which limits the drive to drink and slightly increases urine output. Including sodium in the rehydration beverage (or diet) allows fluid volume to be better conserved and increases the drive to drink.\textsuperscript{71,125,178,184–186} Including CHOs in the rehydration solution may improve the rate of intestinal absorption of sodium and water\textsuperscript{118,178} and replenishes glycogen stores.\textsuperscript{118,187,188} Replenishing glycogen stores can enhance performance in subsequent exercise sessions\textsuperscript{189,190} and may enhance immune function.\textsuperscript{191} While a normal diet commonly restores proper electrolyte concentrations,\textsuperscript{192} many athletes are forced to rehydrate between exercise sessions in the absence of meals.\textsuperscript{178} In addition, some athletes’ meals are eaten as long as 6 hours after an exercise session, which may compromise electrolyte availability during rehydration after intense exercise in hot conditions.

While replenishing fluid to equal sweating losses is often recommended, this formula does not replace urine losses. Ingestion equal to 150\% of weight loss resulted in optimal rehydration 6 hours after exercise.\textsuperscript{185}

**Assessment of Hydration Status.** Body weight changes, urine color, subjective feelings, and thirst, among other indicators, offer cues to the need for rehydration.\textsuperscript{192} When preparing for an event, an athlete should know the sweat rate, assess current hydration status, and develop a rehydration plan. Determinations of sweat rate can be made.\textsuperscript{16,134} Hydration status can be assessed by measuring body weight before and after exercise sessions; monitoring urine color, USG, or urine volume; or using a combination of these factors.\textsuperscript{194,195} A urine color chart is included in this manuscript (Figure).\textsuperscript{196} The general indexes of hydration status are provided in Table 3. A refractometer offers a precise reading of USG and can be used as a general indicator of hydration state. A reading of less than 1.010 reflects a well-hydrated condition, while a reading of more than 1.020 reflects dehydration.\textsuperscript{134} Urine osmolality and urine conductivity may also be useful tools in assessing hydration status.\textsuperscript{197}

The hydration plan should take into account the length of the event, the individual’s sweat rate, exercise intensity, the temperature and humidity, and the availability of fluids (is fluid constantly available, as in cycling, or is it consumed in a large bolus during a break?). Habits of the coach or athlete, or both, may need to be altered in order to maximize the hydration process. Any plan for rehydrating during competition should be instituted and perfected during practice sessions; it should also be individually implemented, given the large variation among people in what constitutes a “comfortable” amount of rehydration.\textsuperscript{198,199} A sample hydration protocol for preparing an elite athlete for an event has been documented.\textsuperscript{200}

**Composition of Rehydration Fluid.** During exercise, the body uses 30 to 60 g of CHOs per hour that need to be replaced to maintain CHO oxidation and delay the onset of glycogen depletion fatigue.\textsuperscript{201–205} Thus, including 60 g of CHOs in 1 L of fluid will not hinder fluid absorption and provides an adequate supply of CHOs during or while recovering from an exercise bout. The CHO concentration in the ideal fluid-replacement solution should be in the range of 4\% to 8\% (g/100 mL).\textsuperscript{117} The simple sugars, glucose or sucrose in simple or polymer form, are the best additives to the replacement fluid.
fluid. Absorption is maximized if multiple forms of CHO are ingested simultaneously (ie, fluid is absorbed more quickly from the intestine if both glucose and fructose are present than if only glucose is present). The amount of fructose in the beverage should be limited to about 2% to 3% (2 to 3 g/100 mL of the beverage), since larger quantities may play a role in decreasing rates of absorption and oxidation and causing gastrointestinal distress. Ultimately, CHO composition depends on the relative need to replace fluids or CHO. During events, when a high rate of fluid intake is necessary to sustain hydration, the CHO composition should be kept low (eg, <7%) to optimize gastric emptying and fluid absorption. During conditions when high rates of fluid replacement are not as necessary (ie, during recovery from an exercise session, mild environmental conditions, etc), the carbohydrate concentration can be increased to optimize CHO delivery with minimal risk of jeopardizing the hydration status.

Small quantities of sodium may enhance palatability and retention, stimulate thirst, and prevent hyponatremia in a susceptible individual. Sodium concentration should be approximately 0.3 to 0.7 g/L. Other valuable sources of practical information concerning the composition of rehydration beverages and rehydration in general are available.

Recognizing Dehydration in Athletes. The early signs and symptoms of dehydration include thirst and general discomfort and complaints. These are followed by flushed skin, weariness, cramps, and apathy. At greater water deficits, dizziness, headache, vomiting, nausea, heat sensations on the head or neck, chills, decreased performance, and dyspnea may be present. The degree of dehydration, the mental status, and the general medical condition of the athlete will dictate the mode, amount, type, and rate of rehydration. Identifying the early signs of dehydration can limit the onset or degree of an exertional heat illnesses. A comprehensive review of the prevention, identification, and treatment of the exertional heat illness can be found in the position stands by the NATA and the American College of Sports Medicine.

Event Management. Some events are conducted under environmental conditions that are extreme and force the athlete to reduce intensity or risk a heat illness. These hazardous heat stresses can be avoided by scheduling athletic events during the coolest part of the day or a cooler time of the year. The reality of sport administration is that many events take place regardless of the environmental conditions. Individuals supervision an event in a hot humid environment must ensure that athletes have ample access to fluids, are encouraged to match fluid intakes with sweat losses, and are monitored for dehydration and exertional heat illness. Whenever possible, minimize the exercise intensity of athletes in the extreme heat, since this is the largest contributor to dehydration and heat illness. When successive exercise sessions occur on the same day or on ensuing days, hydration status, sleep, meals, and other factors that maximize performance and enhance safety should be maintained. Given the variety of events an athletic trainer may supervise, we cannot formulate an event management recommendation for all sports. However, the general concepts are interchangeable across sports and venues. For example, game modifications such as decreasing the length of play or inserting nontraditional water breaks (especially in youth sports and practice situations) will reduce the rate of heat illness. Closely monitoring environmental conditions via the WBGT or the heat index will allow an informed approach to hydration and sweat modification. Athletes who are educated on how to prevent and recognize dehydration are empowered to participate actively in implementing their own hydration protocols, thereby enhancing both performance and safety. The person responsible for the medical supervision of an event should have a detailed plan to address facilities, equipment, supplies, staffing, communication systems, education, and implementation of event policy.

References


Acknowledgments

This position statement was reviewed for the NATA by the Pronouncements Committee and reviewers Kristine L. Clark, PhD, RD, David Lamb, PhD, and Jack Ransone, PhD, ATC.
220 Volume 35 • Number 2 • June 2000


47. Caldwell JE, Ahonen E, Nousiainen U. Differential effects of sauna-


74. Welch BE, Buskirk ER, Jampietro PF. Relation of climate and temperature to food and water intake. *Metabolism.* 1958;7:141.


The Urine Color Chart shown here will assess your hydration status (level of dehydration) in extreme environments. To use this chart, match the color of your urine sample to a color on the chart. If the urine sample matches #1, #2, or #3 on the chart, you are well hydrated. If your urine color is #7 or darker, you are dehydrated and should consume fluids.

The scientific validation of this color chart may be found in the International Journal of Sport Nutrition, Volume 4, 1994, pages 265-279; and Volume 8, 1998, pages 345-355. Adapted by permission from Larry Armstrong, 2000, Performing In Extreme Environments, (Champaign, IL: Human Kinetics).
### Books

<table>
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<tr>
<th>Title</th>
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<th>Edition</th>
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<td>Weight Training: Steps to Success, 2nd edition</td>
<td>Thomas R. Baechle and Barney R. Groves</td>
<td>Human Kinetics, Champaign, IL</td>
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<td>1998</td>
<td>0-88011-718-4</td>
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<td>Stress Fractures:</td>
<td>Peter Brukner, Kim Bennell, and Gordon Matheson</td>
<td>Blackwell Science, Victoria, Australia</td>
<td></td>
<td>1999</td>
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<td>Sports and Children</td>
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<td>Counseling in Sports Medicine</td>
<td>Richard Ray and Diane M. Wiese-Bjornstal, Editors</td>
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### The Clinical Pharmacology of Sport and Exercise:

**Proceedings of the Esteve Foundation Symposium VII**

- Thomas Reilly and Michael Orme, Editors

### Exercise and Circulation in Health and Disease

- Bengt Saltin, MD, PhD, Robert Boushel, DSc, Niels Secher, MD, PhD, and Jere Mitchell, MD, Editors

### CD-ROM

**Upper Extremity Injury Evaluation: An Interactive Approach**

- Denise Wiksten and Brian Barry

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**Weight Training: Steps To Success**

Thomas R. Baechle and Barney R. Groves

Human Kinetics, Champaign, IL

2nd edition

1998

181 pages

ISBN: 0-88011-718-4

Price: $15.95

*Weight Training: Steps To Success,* 2nd edition, is another contribution to the strength and conditioning literature. This text brings together a wealth of strength training knowledge in a comprehensive and easy-to-understand format, which translates into the skills necessary for the development of successful weight-training programs. Excellent step-by-step instructions are provided via explanations, practice procedures, drills, and self-assessment tools. This book is part of the “Steps-to-Success Activity Series” and is broken down accordingly.

The initial steps concentrate on basic strength training principles, including the body’s physiologic response to strength training, dietary and body weight considerations, and an introduction to weight-lifting equipment. The intermediate steps focus on basic weight-lifting techniques and core exercises used in a well-balanced basic program. The final steps present more advanced exercises, followed by instruction in the techniques necessary to implement and adjust weight-training programs and parameters. The major points for each of the steps are well reinforced with tables and illustrations, including a weight-training workout chart specific to parts of the body, days of the week, and sets and repetitions.

This book would be especially helpful for entry-level athletic training, physical education, physical therapy, and exercise physiology students who are interested in learning how to establish and adjust weight-training programs. Sound advice is offered regarding rest periods and techniques to avoid injuries, load determination coefficients to establish appropriate starting weights, load adjustment charts, and a formula for predicting 1-repetition maximum values. The authors do an excellent job of addressing and correcting many of the myths and misconceptions about strength training. The authors also list many common technique errors and the appropriate corrections. Further, references are presented throughout the text, which is extremely helpful for persons wanting to further pursue knowledge in a specific area.

Overall, this text provides thorough instruction in weight-training principles, describes weight-training exercises as part of a well-balanced weight-training program, and discusses the tools necessary to devise and adjust weight-training programs. While this text is not designed with the advanced strength and conditioning specialist in mind, it is very appropriate for anyone interested in learning the fundamental principles of weight training. At $15.95, the book is well worth the price.

Jeff Erickson, MS, PT, ATC, CSCS

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**Stress Fractures**

Peter Brukner, Kim Bennell, and Gordon Matheson

Blackwell Science, Victoria, Australia

1999

190 pages


Price: $59.00

In *Stress Fractures,* the authors have compiled the most comprehensive review of stress fractures available. The first 5 chapters describe the pathophysiology, epidemiology, risk factors, diagnosis, and treatment of stress fractures. The specific types of stress fractures are described by region in the last 5 chapters.

The first chapter gives a brief description of the physiology of normal and pathologic bone remodeling. There are clear definitions of stress and strain and a description of the microdamage that usually precedes a stress fracture. Of the first 5 chapters, this chapter goes into the least detailed description of the literature. For example, there is little mention of the role of the periosteum as a temporary support to remodeling bone (Engh CA, Robinson RA, Milgram J). Stress fractures in
The next chapter on epidemiology not only describes the literature relating to injury rates, characteristics, and morbidity, it summarizes the literature into easy-to-read tables throughout the chapter. In addition, the authors provide an explanation of the different types of epidemiologic studies with an understandable flow chart, so that a reader less familiar with this type of research can begin to draw reasonable conclusions regarding the studies presented. In chapter 3, the authors used their collective research experience to detail the possible roles of bone density, geometry, alignment, hormones, and nutrition as risk factors for stress fractures. At the end of this chapter, the authors make a point of mentioning the benefits of "cyclic training" to prevent stress fractures. However, they did not use the opportunity to justify this concept with the data available from military studies mentioned previously in the chapter (Scully TJ, Besterman G. Stress fracture: a preventable training injury. Mil Med. 1982;147: 285–287).

Chapter 4 explains the current literature on stress fracture diagnosis. It provides a clear description of the clinical presentation of stress fractures and suggests an appropriate use of radiologic techniques depending on the location, presentation, and clinical findings. The authors describe a 2-phase management strategy for the treatment of stress fractures in chapter 5. This strategy is similar to those commonly referred to in the literature over the past 2 decades, with inclusions of new evidence that suggests the benefits of air splints and discussion of conservative management of stress fractures at "high-risk" sites. There is no mention, however, of the psychological aspects of care that are often an important part of stress fracture rehabilitation.

In chapters 6 through 10, the authors use a complete review of the literature to describe the different presentations, incidence, diagnosis, and management strategies for stress fractures of the upper extremity, trunk, pelvis and thigh, lower leg, and foot and ankle. In many instances, case series or case reports are described and compared, with particular emphasis on the more common stress fractures of the femoral neck and tibia. The authors are to be commended on their suggestion that the term "shin splint" is confusing and should not be used to describe tibial pain. A similar case could be made for the many terms that are used to describe the presentation of stress fractures on radiologic tests, such as "stress reaction," "bone strain," and "abnormal remodeling."

Overall, Stress Fractures would be an excellent supplement to a course on orthopaedic sports injuries or an equally valuable clinical resource. The material contains the most comprehensive review available of the current literature on stress fractures and is presented at the graduate level. The tables and figures provide the reader with an easy-to-read overview of the studies described and synthesize the concepts proposed in the text. The organization and complete inclusion of the literature make it a valuable reference for the educator and clinician. **William Romani, PhD, PT, MHA, ATC** University of Maryland, Baltimore

**Sports and Children**

Editors: Kai-Ming Chan and Lyle J. Micheli

Williams & Wilkins Asia Pacific, Hong Kong

1998

293 pages


Price: $35.00

Originally transcribed and compiled from material presented by the World Health Organization and International Federation of Sports Medicine (WHO-FIMS), this text offers an excellent overview of research and clinical experience from experts on sports and children throughout the world. The softbound text is organized into 5 sections, with appendices that include FIMS statements on exercise and physical activity for children, from the WHO-FIMS Consensus Symposium on Organized Sport held in Hong Kong in January 1997.

This book would be appropriate for the primary care physician interested in the field of sports medicine for children or as an adjunct for the master’s-level student in athletic training. Knowledge of human growth and development is needed to fully appreciate the material in this book. Physicians, exercise physiologists, physical therapists, athletic trainers, youth sports coaches, and all health care professionals who treat injured child athletes will find this resource comprehensive.

The editors organized this text in a logical manner, answering relevant questions regarding the participation of children in various sports and physical activities. Overall, the authors did an excellent job of presenting their specialty areas, and all the information is appropriately referenced.

Section 1, "Sports and Health in Children," includes a review of child performance capacity, physical activity, skeletal health, physical fitness, and intellect. This section is thorough, with references that are complete and up-to-date. Section 2, "Sports and Children: Global Experience," features excellent presentations from Europe, Asia, and North America on the benefits of exercise for children. The references in this section are in-depth and current. The strongest part of the textbook is Section 3, "Sports Training in Children." This section establishes baselines and guidelines on such important topics as "Growth and Maturation" and "Sports Injuries in Children." Excellent references offer an opportunity for further study. Section 4, "Sports for Specific Child Groups," provides well-documented, organized, and current presentations on such topics as chronic illness, disabilities, and sex differences. The second strongest section of the text is Section 5, "Position Statement and Reference Article," statements 17–20. This section alone would be reason enough to purchase the book. It presents an overview of the Symposium, with policy statements by some
of the world’s foremost authorities on the subject of children in sports.

The information in this text comes from authors who are presenting their primary research emphases, providing a great deal of valuable youth sports information in 1 publication. If all the recommendations and appropriate allied health interventions in the text could be implemented, the safety of sports and physical activity for children would be markedly enhanced.

This book contains youth sports position statements and growth and development charts. Excellent graphs and black-and-white photographs are found throughout the text. In my estimation, the only shortcoming of the book is that large data tables were compressed to fit the 5- × 8-inch format, making them difficult to read.

This book provides valuable information previously unavailable to the health care professional who oversees strengthening and conditioning training for children’s sports. I recommend that it be used as a resource in athletic training curriculums, in health facilities, in rehabilitation clinics, and anywhere information is dispensed to parents in clinical settings.

Gerald W. Bell, EdD, ATC/L, PT
University of Illinois at Urbana-Champaign
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Counseling in Sports Medicine
Editors: Richard Ray and Diane M. Wiese-Bjornstal
Human Kinetics, Champaign, IL
1999
361 pages
ISBN: 0-88011-527-0
Price: $42.00

Counseling in Sports Medicine would be an excellent addition to the library of any sports medicine professional. It should be considered for use in advanced athletic training courses and seminars. Counseling in Sports Medicine should be dedicated to stimulating progress in the athletic milieu.

The Clinical Pharmacology of Sport and Exercise is the published version of the 7th symposium sponsored by the Esteve Foundation, an organization dedicated to stimulating progress in pharmacotherapy. The 7th symposium, an international multidisciplinary meeting held in Spain in October 1996, brought together well-respected authorities from the fields of pharmacology, exercise science, nutrition, environmental health, and anatomy. They presented the scientific evidence concerning links between drugs and exercise and stimulated discussion on these topics. The primary focus of the symposium and the book, however, is the use (and misuse) of ergogenic drugs and nutrients in the competitive athletic milieu.

The book is organized into the same 5 categories featured at the symposium. Within each part are 5 articles, typically written by a single invited contributor. Following each article is the transcript of a roundtable discussion. Each article is either a review or a presentation of the speaker’s research project, or a combination of the 2.

The book begins by describing the broader aspects of the use and misuse of drugs (ergogenic and recreational) in sports. The extent of the problem, as well as laboratory testing and research procedures, is generally presented by drug category (anabolic steroids, stimulants, beta blockers, narcotics, etc). The limitations of laboratory analyses and research designs are also discussed, particularly as they (in addition to athletes’ inaccurate self-reporting) contribute to our inability to truly understand the extent of drug misuse in sports. The last article in this part discusses the triad of overtraining, immunosuppression, and exercise-induced muscle damage and includes a brief mention of the effects of anti-inflammatory drugs and nutritional status.

The second part of the book is a presentation of the pharmacology, physiology, and health-related aspects of beta blockers, free radicals, nicotine, and estrogen. The more common ergogenic aids used for short-term performance (anabolic-androgenic steroids, alkalinizers, and creatine) and long-term performance (blood doping, branched-chain amino acids, growth hormone) are presented next. Included in the short-term performance section are chapters devoted to the physiology
of muscle fatigue and the pharmacology of bodybuilding. Endurance performance is highlighted in the chapters on nutrient (rather than drug) ergogenic aids, with chapters on dietary factors and use of beverages.

The final part of the text addresses the psychomotor aspects of performance enhancement. The pharmacologic and physiologic effects of methylxanthines, tranquilizers, and alcohol are presented. A single chapter is devoted to the pharmacology of the traveling athlete, and another chapter discusses the psychological strategies currently used in the "real world" of athletics.

This book would best serve as a reference volume. It could also serve as a supplementary text for a graduate-level seminar or a continuing education course, although the cost is certainly a deterrent. The material goes well beyond the basics for each topic. Everything from molecular physiology to sport psychology is included, as are discussions of research design and testing limitations. Most readers will appreciate the updated review of familiar material, but they will also appreciate the new knowledge contained in this book. A few content areas lack complete reviews because of the advancements made since the symposium was held. These include the presentations on creatine, growth hormone, fatigue, and the immunologic aspects of overtraining. Also, I felt that 1 chapter demonstrated somewhat of a professional bias, reflected by the author's statement that the field of sport psychology lacks a theoretic basis.

Most remarkable about this book is the wide range of material and the clarity and brevity of the writing. A few passages, however, suffer from English translation problems. It is not hard to imagine the need for a separate symposium on each of the topics presented; in fact, several seminars, workshops, and conferences have already been presented on anabolic-androgenic steroids and creatine supplementation. Thus, the price of this book could be compared with the price of admission to several symposiums, which may be the only way to justify the high cost. From a cost perspective, it may be best used as a library reference volume.

Titles of cited references are not included in the bibliography, although the references are well worth the effort of reading. The roundtable discussions put the finishing touches on the content of the individual articles. Not only are the questions and answers relevant to the real world of performance enhancement, but they should stimulate discussion in their own right. Indeed, the reader will appreciate the questions posed by the many multidisciplinary speakers.

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Exercise and Circulation in Health and Disease
Editors: Bengt Saltin, MD, PhD; Robert Boushel, DSc; Niels Secher, MD, PhD; Jere Mitchell, MD
Human Kinetics, Champaign, IL 1999
360 pages
ISBN: 0-88011-632-3
Price: $59.00

Exercise and Circulation in Health and Disease represents a compilation of integrated topics in cardiovascular regulatory physiology from more than 40 authors. The concept of the text was developed after a 1995 scientific meeting in Copenhagen to address the state of knowledge of circulatory control during exercise. The editors, Drs. Bengt Saltin, Robert Boushel, Niels Secher, and Jere Mitchell, recruited an exceptional roster of contributors internationally recognized for their respective work in cardiovascular control mechanisms.

The result is a well-organized text separated into 4 logical subsections. Part I provides a particularly effective in-depth discussion of cardiovascular control mechanisms during exercise, including regulation of blood pressure, circulation, and respiration. Part II addresses regional blood flow and oxygen delivery during exercise, including the impact on each of the major organs, and coronary and cerebrovascular blood flow. It devotes 3 superb chapters to muscle blood flow, its regulation, microvascular control, and integration with cardiac output. A separate, albeit brief, chapter concisely addresses oxygen transport in blood and to mitochondria. All chapters are supplemented by relevant data-based illustrations and appropriate schematics, radionuclide scans, and histology slides, in black and white, but clearly presented. Part III examines the effects of environmental factors, such as hypoxia, hypobaric pressure, microgravity, and temperature, on cardiovascular regulation. Although not a logical fit with this section of the text, 2 important areas, cardiovascular regulation with endurance training and prevention of the effects of aging, are addressed. These are best described as concise current reviews and are well referenced. The final section examines cardiovascular regulation in disease, with individual chapters devoted to heart failure, muscle disease, peripheral arterial disease, hypertension, and orthostatic stress and autonomic dysfunction. These areas, as in section III, fairly concise reviews, well referenced and easily read.

It should be emphasized that this text is heavily weighted toward the basic sciences and an understanding of current knowledge regarding the mechanisms of cardiovascular regulation during exercise in the presence of different physical, environmental, and biological challenges. It is not intended for the clinician seeking an overview of these mechanisms, which are intricate by their very nature. The text appears best suited as a primary source for graduate students at the doctoral or postdoctoral level engaged in courses involving cardiovascular regulation and circulatory physiology. For those in practice, the text would find a comfortable place on the shelf of basic and applied physiologists and those research clinicians with a serious interest in understanding cardiovascular control. The text would have minimal direct value in clinical practice, as it does not include recommendations or implications for clinical practice, appropriately so, given the focused intent of the text. The price is reasonable at $59.00 at the time of this review and as such may be a good addition to the clinician's library. Without question, the text represents the most comprehensive compilation of data on the underlying control of the cardiovascular system and the influence of disease and other physical and environmental stimuli.

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Upper Extremity Injury Evaluation: An Interactive Approach
Denise Wiksten and Brian Barry
Slack, Inc, Thorofare, NJ
2000
Almost every health care provider is faced with the challenge of understanding the special tests used to evaluate upper extremity pathology. A unique and excellent method of presenting selected tests is provided in the Wiksten and Barry CD-ROM, Upper Extremity Injury Evaluation: An Interactive Approach. The CD-ROM can be viewed using Windows 95/98/NT or the Macintosh platform.

The CD-ROM details selected techniques of physical examination of the cervical spine, brachial plexus, shoulder, elbow, wrist, and hand. Various learning styles are accommodated in verbal descriptions, video demonstrations, and text. Short video segments are viewed on QuickTime (provided on the CD-ROM) and can be viewed selectively, a much more efficient means of review than a full-length videotape.

This CD-ROM would be an important adjunct to the student's textbook, due to the detailed presentation of the patient position, examiner position, examination procedure, and positive and negative test results. Additional links for students to follow will aid in reviewing some of the pertinent anatomy.

Some of the more outstanding features of the CD-ROM include
- Navigation through the special tests. A student or clinician wishing to review the specifics of a test could do so in a very efficient manner. Launching the program and selecting the area of the upper extremity in question brings the viewer to a screen listing the specific tests for that area. Clicking on the specific test desired will bring the viewer to the first screen of the test description. At the test description screen, the viewer can review each step of the test procedure or click forward to the specific information needed. This ease in navigation is virtually unavailable using a videotape.
- Dynamic presentation of the testing procedure. Rather than reading about the positioning of the patient and the examiner, the viewer can see the positioning and hear a verbal description of the procedure.
- Test significance. Both positive and negative test results are demonstrated and explained. This is often an area overlooked in conventional textbooks.

Compared with the other available methods of providing such information, this format offers not only the dynamic movement involved in the special tests, but also the unique ability to view specific tests and aspects of tests without having to view the entire CD-ROM.

Improvements to this CD-ROM might include increasing the number of tests presented. This CD-ROM is not exhaustive in the tests used to fully evaluate the upper extremity. It serves its purpose well by reviewing the main tests employed in evaluation of the various body areas, although additional information regarding manual muscle testing and goniometric evaluation would provide a more complete review. Also, improving the anatomy sections would be helpful. While the physical tests are well done, the anatomical renderings are less than satisfactory. Even with a firm understanding of the structures involved, the viewer may be confused by the anatomy illustrations, both in some of the specific tests as well as the anatomical review provided via the "anatomy" selection button.

In summary, this CD-ROM provides an excellent review of the major evaluation techniques used in assessing upper extremity pathology. Every health care student should have this CD-ROM to assist in learning the examination procedures, and every clinic should keep the CD-ROM on hand for staff reference. With our changing technology, this is certainly the start of many more such references to come.

Sue Hillman, ATC, PT
Arizona School of Health Sciences
Phoenix, AZ
SUBMISSION POLICIES

1. Submit 1 original and 5 copies of the entire manuscript (including figures and tables) to: Journal of Athletic Training. Submissions, Houghton Sports Medicine Foundation, Inc., 6262 Veterans Parkway, PO Box 9517, Columbus, GA 31908. The term figure refers to items that are not editable, either halftones (photographs) or line art (charts, graphs, tracings, schematic drawings), or combinations of the two. A table is an editable item that is not a figure.

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   a. Title
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15. Title pages 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 study, the technique 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.

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   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-constructed illustrations.

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230 Volume 35 • Number 2 • June 2000
Authors' Guide

20. Communications articles, including official Position Statements and Policy Statements from the NATA Pronouncements Committee; technical notes on such topics as research design and statistics; and articles on other professional issues of interest to the readership are solicited by the Journal. An author who has a suggestion for such a paper is advised to contact the Editorial Office for instructions.

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22. References should be numbered consecutively, using superscripted Arabic numerals, in the order in which they are cited in the text. References should be used liberally. It is unethical to present others' ideas as your own. Also, use references so that readers who desire further information on the topic can benefit from your scholarship.

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