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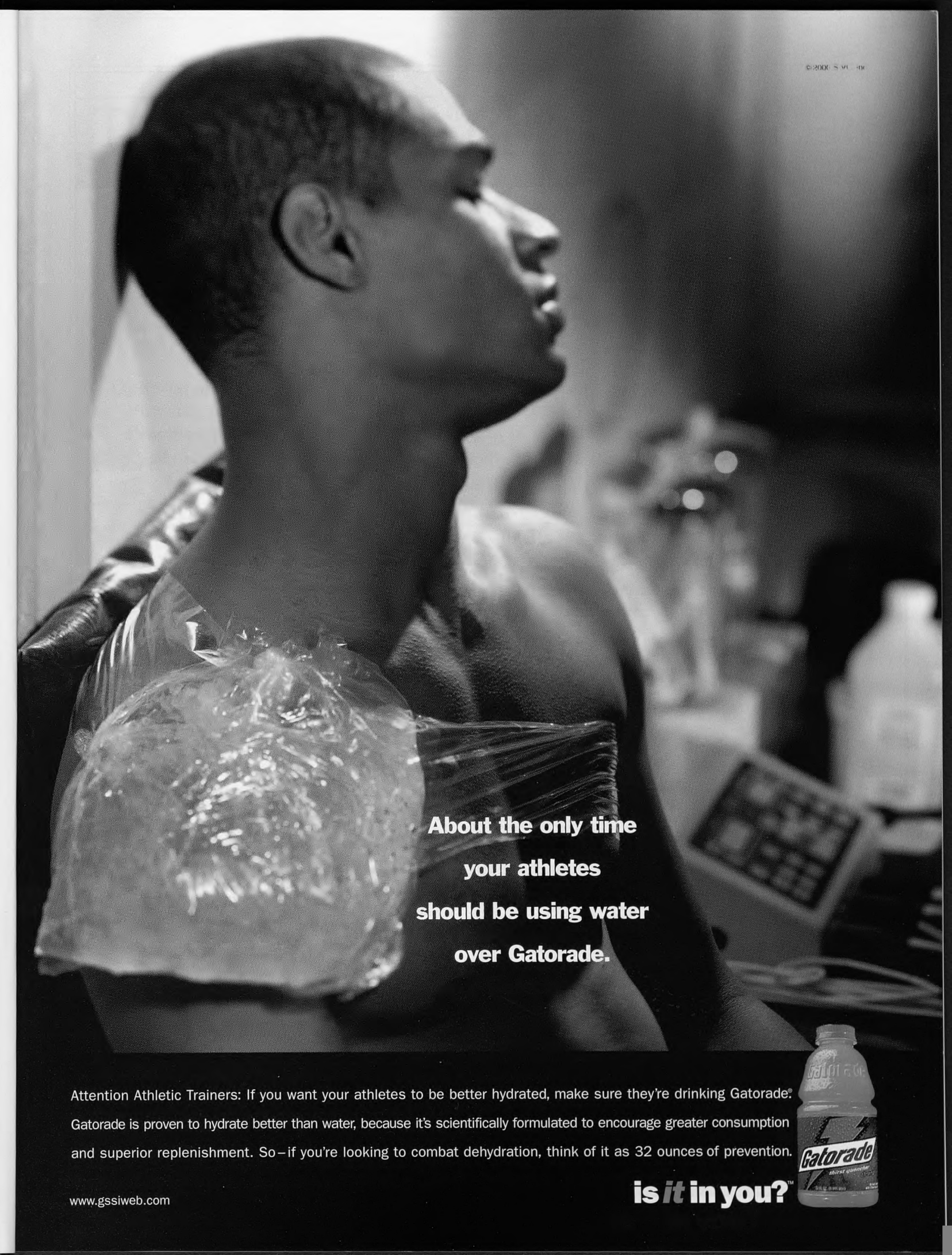
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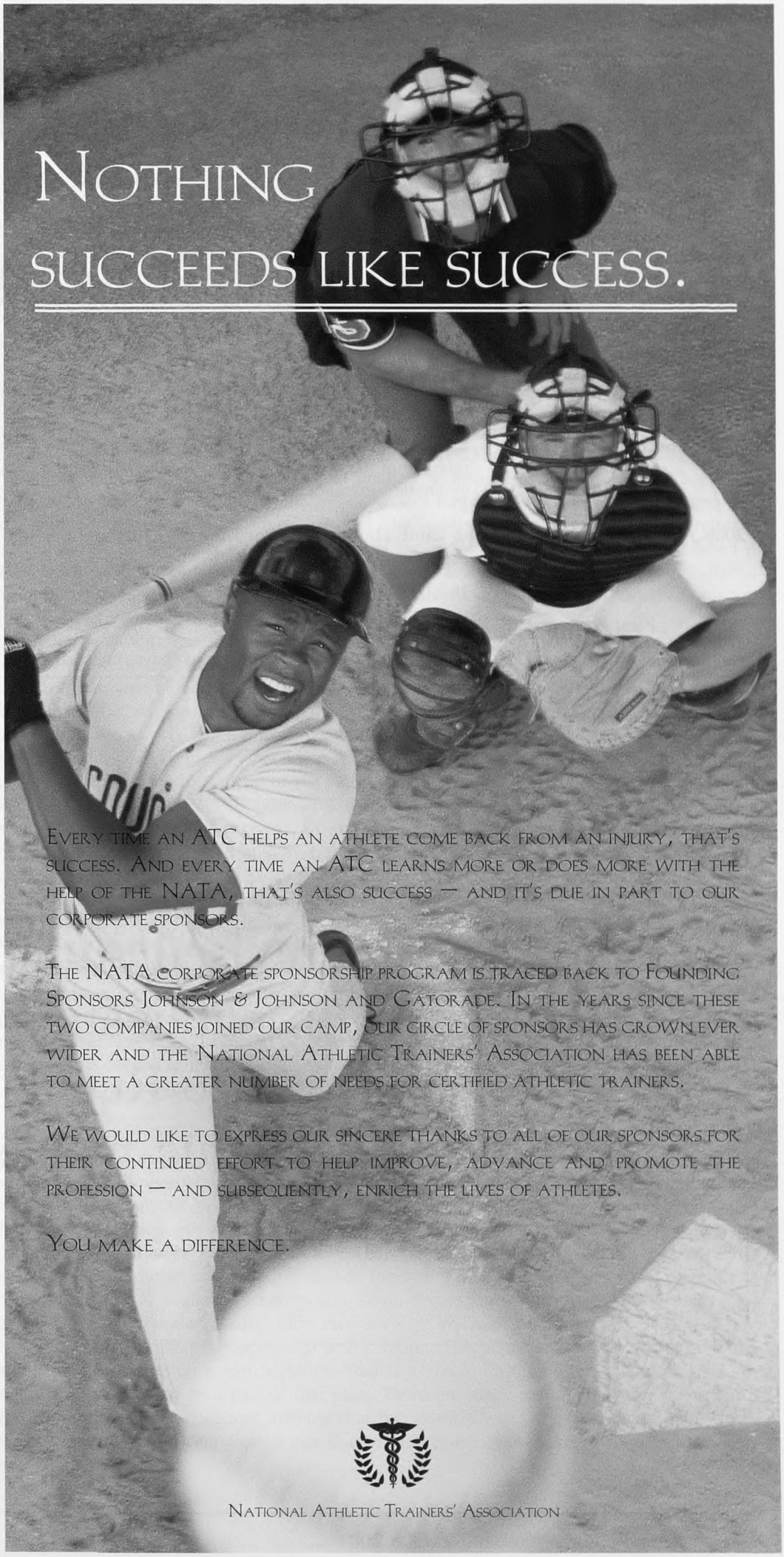

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2000 Outstanding Manuscript Awards

Congratulations to the winners and the runners-up of the 2000 Outstanding Manuscript Awards, as determined by the Editorial Board and the Associate Editors of the Journal of Athletic Training.

2000 *Journal of Athletic Training* Kenneth L. Knight Award for the Outstanding Research Manuscript

Winner: Garrett CL, Draper DO, Knight KL. Heat distribution in the lower leg from pulsed short-wave diathermy and ultrasound treatments. 1:50–55.

First Runner-Up: Riemann BL, Guskiewicz KM. Effects of mild head injury on postural stability as measured through clinical balance testing. 1:19–25.

Second Runner-Up: Shellock FG, Mullin M, Stone KR, Coleman M, Crues JV. Kinematic magnetic resonance imaging of the effect of bracing on patellar position: qualitative assessment using an extremity magnetic resonance system. 1:44–49.

2000 *Journal of Athletic Training* Clint Thompson Award for the Outstanding Non-Research Manuscript

Winner: Myers JB, Lephart SM. The role of the sensorimotor system in the athletic shoulder. 3:351–363.

First Runner-Up: Combs JA. It's not "just a finger." 2:168–178.

Second Runner-Up: Houglum JE. Asthma medications: basic pharmacology and use in the athlete. 2:179–187.

Please see the revised Authors' Guide for changes in publication policies regarding Case Report and Clinical Technique manuscripts.

Editorial

Are You Ready for Sudden Cardiac Death?

Francis Feld

Two phrases that are sure to strike terror into every athletic trainer's heart are sudden cardiac death and spinal cord injury. Even though the chances of either of these injuries happening are small, it is reasonable to assume that every athletic trainer will either care for an athlete who suffers one of these injuries or will know of a colleague whose athlete has suffered one of these events. For all involved, the event is catastrophic and will never leave them. After 23 years, I can still see the eyes of the 17-year-old football player who said "Fran, I can't move my legs." That image will never leave me.

This issue of the *Journal of Athletic Training* contains an article in which John W. and John A. McChesney describe important assessment tools for evaluation of the chest and abdomen and 2 articles that deal with sudden cardiac death. In athletic training, we emphasize injury prevention, and rightly so, but Michael Koester shows that we are not doing an adequate job when it comes to preventing sudden cardiac death. No matter how sophisticated we get with electrocardiograms and echocardiograms, the best screening tool is still a thorough history and physical examination by a qualified practitioner. Yet most schools are not using this more economical and more effective tool. We *must* do better.

The article by Glenn Terry and colleagues deals with resuscitation, a topic dear to my heart. Two issues come to mind here: should athletic trainers receive training in the use of automated external defibrillators (AEDs), and should institutions purchase AEDs? I suppose that 30-some years ago, there was a debate over whether athletic trainers should be certified in cardiopulmonary resuscitation (CPR), and the result is that we are all CPR certified. We need a card to take the certification test, and we need the card to maintain our certification. We receive continuing education units (CEUs) for it. We dread having to pound on poor Annie's chest every 2 years, but we do it. Well, guess what? The AED has become an integral part of CPR training. The American Heart Association (AHA) has recently revised many of the cardiac care standards for adults and children.[1] This revision requires all CPR instructors to become certified as AED providers and instructors. Every CPR course is to include a demonstration of the AED and how it fits into the chain of survival. In my opinion, AED training should be added to the educational competencies for student athletic trainers. Certified athletic trainers should train on the AED each year that they are not required to take a CPR class. That way, at least once a year, you practice your cardiac resuscitation skills. It uses 4 hours of your time at most. The Board of Certification should offer CEUs for AED training. Police officers, firefighters, and security guards have demonstrated they can use the AED effectively. Do we really want a mall guard to have a higher level of medical certification than a certified athletic trainer? We *must* do better.

The AHA is promoting public-access defibrillation in its newest guidelines.[1] We know that survivability depends upon early defibrillation. The goal is a call-to-shock time of less than 5 minutes, but emergency medical services (EMS) agencies cannot reliably and consistently meet this goal. Indeed, Cady and Lindberg[2] reported on EMS delivered by the largest 200 cities in the United States, and they found no universal method to measure or define response time. Public-access defibrillation is a way to meet this goal.[3] If you work in a sports medicine facility, don't think you are immune to this issue. The AHA has set a goal of collapse-to-shock time of less than 3 minutes for all areas of the hospital or ambulatory-care facility.[1] We *must* do better.

Sudden cardiac death in athletes is truly a rare event. Cardiac arrest in the general public is not. Athletic trainers have a greater chance of having to resuscitate a spectator, coach, or official than an athlete. We must be prepared. At a cost of US $3000 each, mandating the purchase of an AED by every institution would be difficult. Yet many schools are doing so, and they are buying more than one. Every school beyond the 50% mark that obtains an AED makes a stronger case that it is the standard of care. Athletic trainers should examine their own situations and decide what is best. It may be feasible to rely on campus security to have AEDs, but response time must be examined closely. Can they reliably and consistently get that AED to you in less than 5 minutes? Be reasonable in that assessment. Think outside the box. Work with your local EMS agency to obtain AED training. They can probably guide you to available grant money. Also, if campus security is buying 2 units, why not buy 2 more for the athletic department? Bulk purchases usually offer cost savings. We *must* do better.

Every institution should have an emergency action plan for injuries at every venue. The plan must be comprehensive and common knowledge. It should be practiced and revised on a regular basis—that is a given. Providing an AED at the side of every cardiac-arrest victim in less than 5 minutes should be a part of that plan. There are many ways this can happen, but it must happen. Having an AED readily available at every spectator event and at the victim's side in less than 5 minutes must be our goal. Although I can still see Jeff's eyes from 23 years ago, I can also see the eyes of several cardiac-arrest survivors who said thank you. We *will* do better.

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Editor's Note: Francis Feld, MS, MEd, CRNA, ATC, NREMT-P, is a certified, registered nurse anesthetist at the Veterans Administration Pittsburgh Health Center. He has been a certified athletic trainer at the high school, university, and professional football levels and is a paramedic with the UPMC St. Margaret Hospital Paramedic Response Team and Ross West View EMSA.

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Neuromuscular Evaluation of Trunk-Training Exercises

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Peter Konrad, PhD, Klaus Schmitz, PT, and Achim Denner, PhD, contributed to conception and design; acquisition and analysis and interpretation of the data; and drafting, critical revision, and final approval of the article.

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Objective: To evaluate the neuromuscular activation profiles of trunk muscles in commonly used gymnastic strength exercises with a polymyographic set-up and to describe the training effects of each exercise.

Design and Setting: Subjects performed 9 repetitions of each of 12 gymnastic exercises. Variations of 5 trunk flexions, 5 extensions, and 2 lateral-flexion movements were performed under standardized test conditions.

Subjects: Ten healthy subjects (men and women) who were familiar with the exercises participated in the study.

Measurements: We recorded surface electromyograms (EMGs) from the rectus abdominis, external oblique, rectus femoris, middle trapezius, erector spinae at T12 and L3, gluteus maximus, and semitendinosus and semimembranosus muscles. Recording of each repetition cycle was triggered by a flexible electronic goniometer attached to the trunk. The raw EMG signals were rectified, smoothed, amplitude normalized to maximal voluntary contraction (MVC), and averaged for the last 8 repetitions.

Results: Pure spine-flexion exercises, such as a curl-up, produced sufficient and isolated activation (greater than 50% MVC) of the abdominal muscles. When flexion of the spine was combined with hip flexion (sit-up), the peak activation was increased. Lateral-flexion tasks targeted primarily the external oblique muscle, which demonstrated high activity in side-lying flexion tasks. Back- and hip-extension exercises, such as bridging and diagonal hip and shoulder extension, produced only moderate mean activities (less than 35% MVC) in the trunk-extensor muscles. Trunk-extension exercises with combined hip extension increased the EMG activity to 50% MVC but only at the end of the extension.

Conclusions: Individual responses to each exercise varied markedly, which complicated the classification of exercise effects. However, within the limitations of the study, we found that the chosen abdominal exercises provided an effective training stimulus for the trunk-flexor muscles, whereas in the back- and hip-extension exercises, the neuromuscular activation tended to be too low or unspecific to qualify as muscle-specific training.

Key Words: electromyography, activation profiles, EMG normalization, EMG variability, movement standardization, back muscles, abdominal muscles, hip muscles, training effectiveness

A wide variety of different trunk exercises are currently used for training and conditioning purposes, both in athletic programs (eg, competitive sports and fitness) and in rehabilitation practice (eg, low back pain patients and back schools). The effectiveness of neuromuscular training is typically based on functional anatomical evaluations, empiric measurements, or subjective perception. Despite the large number of different exercise set-ups, scientific evaluation of their specific effects on the targeted musculature is lacking. The fundamental questions are (1) is the muscle active and (2) if yes, is its activation high enough and long enough to elicit a training response for strength or endurance improvement?

Surface electromyography (EMG) can be used as a quantitative method to detect the activation level and patterns of muscle groups in movement.[1] A review of the existing EMG literature related to this topic indicates that many study findings are limited by poor standardization, insufficient EMG processing, or missing statistical analyses. Typically, a qualitative approach of EMG calculation based on microvolts, ordinal scaling (more or less activity), or both was used.[2–4]

Recent investigators[5–10] have used state-of-the-art EMG methods incorporating fine-wire electrodes, amplitude-normalization techniques, and interfacing with other biomechanical sensors to evaluate the neuromuscular function of trunk and hip muscles in a wide variety of daily activities and training exercises. One drawback of most studies examining back and hip extension is the lack of control or detection of the hip-extensor muscles, such as the gluteus maximus and the hamstring muscles. We found no study detecting the dorsal and ventral "chain" of the main trunk and hip muscles within one measurement set-up.

The purpose of our study was to record both the dorsal and ventral superficial muscles simultaneously to demonstrate the activation and coactivation patterns of the main trunk and hip muscles. The EMG activation profiles for the main movements of the spine (extension, flexion, lateral flexion, and rotation) were determined in gymnastic exercises. Standardized methods and quantitative EMG analysis incorporating the latest amplifier technology were used to allow for comparisons among the exercises.

METHODS

We investigated 10 healthy subjects (3 women, 7 men; age, 27.8 ± 2.4 years; body weight, 75.8 ± 15.8 kg; height, 177.9

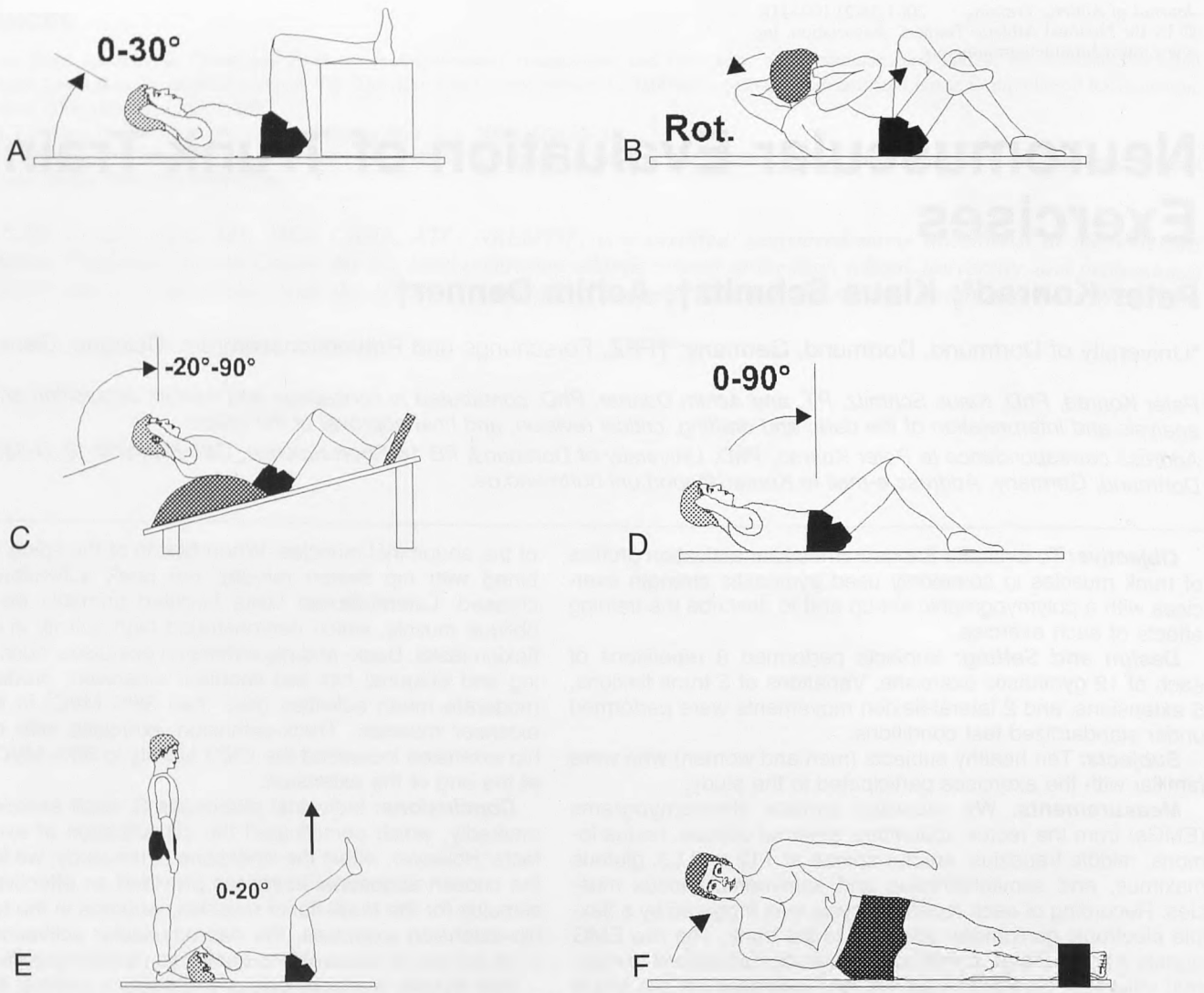

Figure 1. Gymnastic training exercises. A, Straight Curl-Up. Fingertips touch the temples, arms are in a fixed lateral position, the head and shoulders are lifted, and the feet are not fixed. B, Cross Curl-Up. As in A, but 1 leg is across the other, and the contralateral elbow is moved to the opposite knee. C, Curl Up, Hyperextended. Same arm position as A but inverse (-20°) starting position; trunk and hip flexion until the head and thorax are upright; no foot fixation. D, Sit-Up. Same arm position as A; trunk and hip flexion until the upper body is upright; no foot fixation. E, Vertical Hip Lift. Knees are flexed between 70° and 90°, arms are fixed, hip is lifted until lumbar spine is lifted from the ground. F, Lateral Flexion, Fixed Legs. Foot of upper leg is crossed over the lower leg and fixed; flexion until the upper body is lifted off the ground (30°). G, Lateral Hip Lift. Elbow support from a flexed position (30° from hip to ground), extension to the neutral position (0°). H, Diagonal Hip and Shoulder Extension. From a flexed position (elbow in contact with the contralateral knee), diagonal hip and shoulder extension to the horizontal position. I, Kneeling Back Extension. Same arm position as A, from a flexed position (chest-leg contact), isolated spine extension (head and thorax to 45°). J, Trunk Extension, Fixed Legs. Same arm position as A, fixed legs in prone position, from 90° hip flexion-extension to the horizontal (0°). K, Bridging. Supine position, trunk and arms resting on ground and knees bent (90°), hip extension to neutral position (0°). L, Hip Extension, Fixed Trunk. Fixed upper body in prone position, from 90° hip and knee flexion with extension of legs to the horizontal line (hip and knee, 0°).

± 10.4 cm). All subjects were familiar with strength training and gymnastic exercises, but none were specifically training at that time. Informed consent was obtained from each subject before participation in the study. Because the study was conducted at a sports institution rather than a medical facility, institutional review board approval was not required.

Trunk-training exercises were performed in randomized order: 12 gymnastic exercises (Figure 1), including 5 for trunk and hip flexion, 2 for trunk lateral flexion, and 5 for trunk and hip extension. After a standardized sequence of warm-up on a bicycle ergometer and stretching exercises, each subject performed 12 different static maximal voluntary contractions (MVCs), and each contraction (of 3 to 5 seconds' duration) was repeated 2 times (Figure 2). The rationale for these exercises is based on comprehensive pilot studies of the most effective task to produce maximum EMG activity. All subjects were familiar with the MVC tasks, especially with the machine exercises, on which they had trained extensively in the past. After the MVC set, the subjects performed 9 repetitions for each training exercise. The contraction duration was standardized by using an acoustic metronome at 30 beeps per minute. Between sets was a rest period of at least 5 minutes.

We recorded surface EMG signals from 8 muscles; the dorsal muscle extension chain was represented by 5 muscle groups and the ventral muscle chain by 3 muscle groups (Table 1).

Wet-gel, nondisposable, 1.2-cm electrodes (Type Blue Sensor P00S, Medicotest, Ølstykke, Denmark) were applied parallel to the muscle-fiber orientation, with an interelectrode distance of

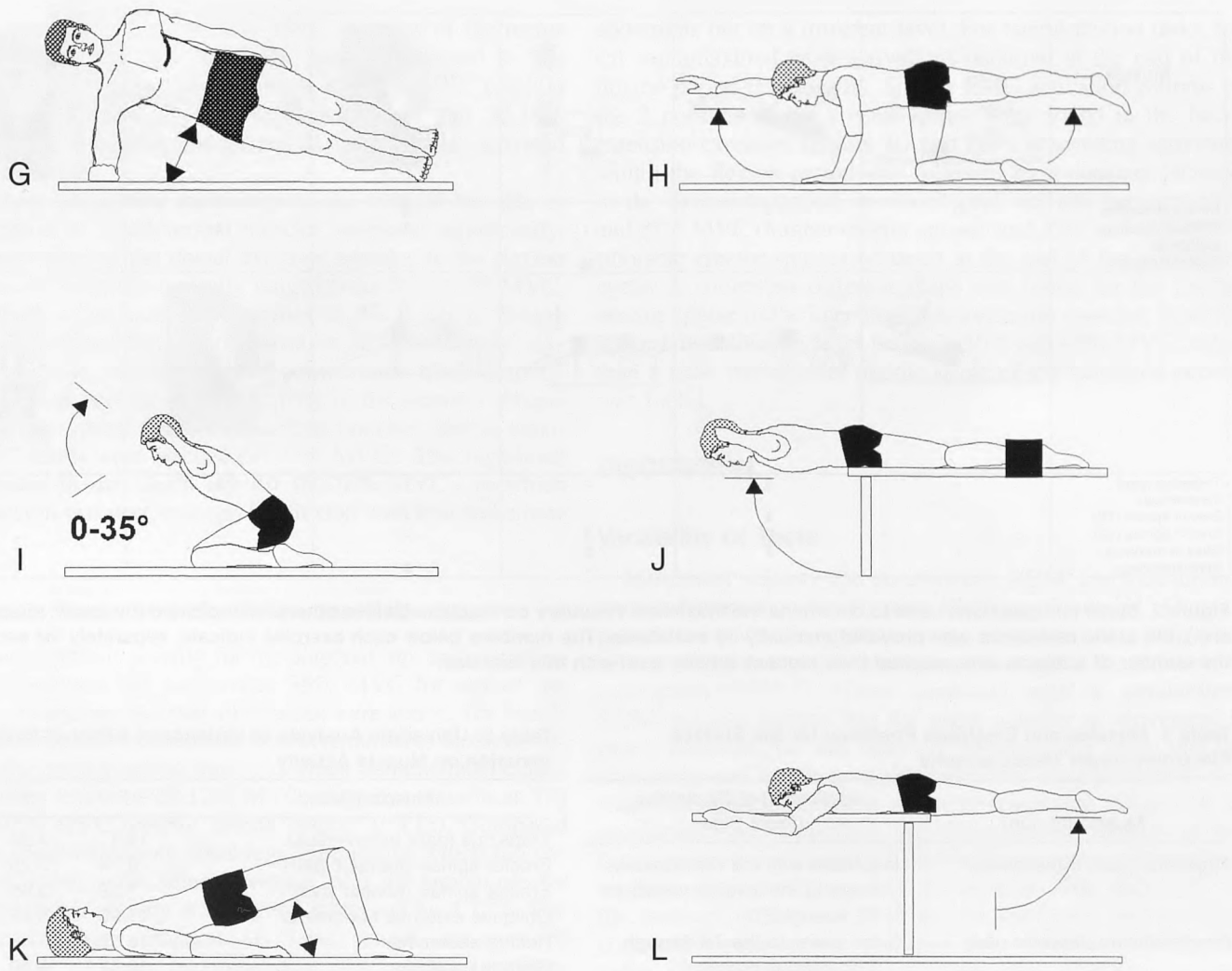

Figure 1. continued.

2 cm. We prepared the skin by using a special abrasive-conductive fluid that rubs and cleans the skin (Every, Neurodata, Vienna, Austria). Care was taken that interelectrode impedance was less than 10 kOhm. We tested the signal quality by visual inspection of the baseline while moving the cable and shaking the muscle. We performed spectral analysis on selected signals and analyzed the power spectrum to inspect the EMG quality and detect possible noise interference at 50 Hz.

The raw EMG data were measured at a bandwidth of 10 to 500 Hz, using a differential amplifier (MyoSystem 2008, Noraxon Inc, Scottsdale, AZ). According to the manufacturer's technical specifications, the common mode rejection ratio was greater than 110 dB, amplifier noise was less than 7 μVrms, and input impedance was equal to 10 mOhm. The signals were A/D converted with 1500 Hz and stored in a personal computer. The stored data first were full-wave rectified, then smoothed with a root mean square (150 milliseconds), and finally amplitude normalized to the highest activity level found in the set of MVC contractions (mean amplitude for 1 second). To define the start and end of each flexion-extension cycle for gymnastic exercises, a 2-dimensional goniometer (Penny & Giles Computer Products Ltd, Christchurch-Dorset, United Kingdom) was applied to the lateral trunk axis of the subjects. Because of possible starting effects, the first repetition of each set was excluded from the analysis; the remaining 8 repetitions were time normalized to 100 data points and expressed as an averaged repetition cycle ranging from 0% to 100%.[11] Finally, for each exercise, these ensemble averages were averaged again for all subjects included in the study.

The coefficient of variation (CV) was used to describe the variability of EMG data,[11] and the Pearson correlation coefficient was used to describe the similarity of EMG activation patterns. We used an analysis of variance for repeated measures to show the effect of different exercise tasks on the EMG amplitude within a muscle group and the post hoc multiple-comparison Newman-Keuls test to identify significant differences ($P < .05$) in mean values between tests.

RESULTS

Variability in Movement Execution

To standardize the tests, we asked all subjects to move with a cadence of 30 beeps per minute. The mean contraction duration for the first movement period (flexion, lateral flexion to the right side) of all 12 exercises was 1928 milliseconds, and the mean duration of the second movement period (extension, lateral flexion to the left) was 2031 milliseconds. A general trend indicated that the period to overcome load (concentric period) was performed faster than the backward movement (eccentric period). The mean duration difference between the periods was 254 milliseconds. The CV was calculated for each

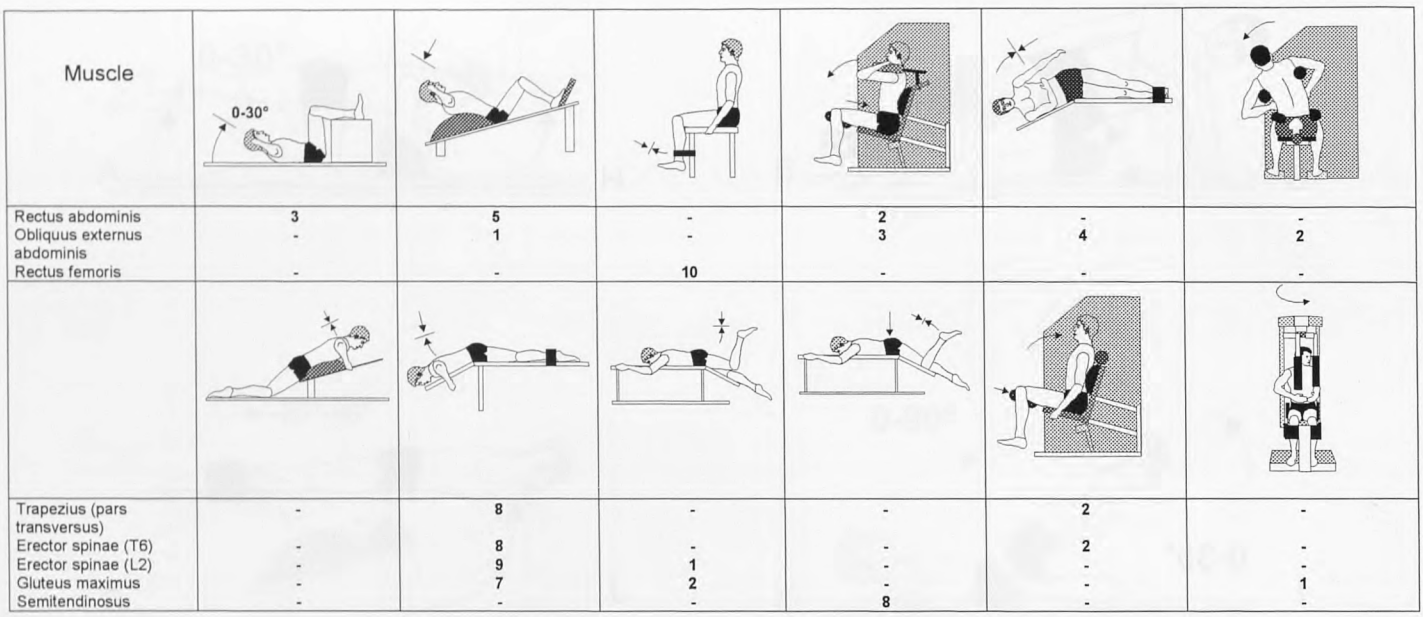

Figure 2. Static test positions used to determine the maximum voluntary contraction. Unless otherwise indicated (by arrow, straps, or lever arm), the static resistance was provided manually by assistants. The numbers below each exercise indicate, separately for each muscle, the number of subjects who reached their highest activity level with this exercise.

Table 1. Muscles and Electrode Positions for the Surface Electromyogram Measurements

Muscle (Group)	Application of Electrodes (Right Side)
Trapezius (pars transversus)	In parallel with the rhomboideus fibers at the level of vertebrae T3 through T6
Erector spinae (thoracic part)	3 cm lateral to the T8 through T12 spinous process
Erector spinae (lumbar part)	3 cm lateral to the L2 through L4 spinous processes
Gluteus maximus	At the center of the muscle belly
Semitendinosus and semimembranosus	At the center of the semitendinosus and semimembranosus group
Rectus abdominis	3 cm lateral to the umbilicus
Obliquus externus abdominis	At the level of the umbilicus, about 15 cm apart, 3 cm above the iliac crest
Rectus femoris	At the center

exercise to describe the variability of the contraction duration. For the concentric part of the movement, the mean CV for all exercises was 4.42%; for the eccentric period, 3.7%. The vertical and the lateral hip lift demonstrated values higher than 10% (maximum, 12.3%). The constancy of the range of motion (ROM) was calculated from the CV of the mean angle values obtained during each exercise. The average CV for the ROM was 10.68%.

Mean Activity Distribution Profiles

The analysis of variance revealed a significant effect for the exercise tasks for all muscles (Table 2). However, due to the EMG-specific variability (see the following sections), many differences among given tasks could not be confirmed by the post hoc multiple-comparison analysis. If not otherwise indicated in the text, the findings were not significant.

Table 2. Univariate Analysis of Variance of Effect of Exercise Variation on Muscle Activity

Muscle (Group)	F	*df*	*P*
Trapezius (pars transversus)	14.1	4.34	.0
Erector spinae (thoracic part)	8.74	3.25	.0
Erector spinae (lumbar part)	7.39	5.45	.0
Obliquus externus abdominis	11.96	5.45	.0
Rectus abdominis	16.58	4.45	.0
Gluteus maximus	16.42	6.50	.0
Semitendinosus and semimembranosus	16.51	6.56	.0
Rectus femoris	15.1	4.44	.0

Gymnastic Abdominal-Flexion Exercises

To investigate which muscle is involved in each exercise and what its activation level is for a given task, we calculated the mean EMG activity for each period within the averaged repetition cycle (Figure 3). Three exercises, the straight curl-up, the cross curl-up, and the vertical hip lift, were spine-flexion tasks without combined hip flexion. The distribution of EMG activity over the ventral muscles indicated remarkable isolation of the abdominal muscles. In the straight curl-up, rectus abdominis activity showed a middle exhaustion level (52.45% MVC) for the flexion period, which was slightly but not significantly increased when additional rotation was added in the cross curl-up exercise (55.96% MVC). External oblique muscle activity was increased from 28.71% to 36.2% MVC when the straight curl-up was varied to the cross curl-up. The next 2 exercises, the curl-up, hyperextended at prestretched start position, and the regular sit-up, added hip flexion to the spine flexion. For both abdominal muscles, flexion activity increased when the straight curl-up was changed to the curl-up, hyperextended; the increase of 17.78% MVC for the oblique muscle was significant, but the increase of 13.87% MVC for the rectus abdominis muscle was not significant. When the straight curl-up was varied to a sit-up, the flexion activity level of the rectus abdominis muscle was unchanged, but the oblique muscle showed a significant

increase from 28.71% to 56.51% MVC. Activity of the rectus femoris muscle, the only hip-flexor muscle evaluated in this study, increased significantly from less than 5% MVC (curl-up exercises) to 21.98% (curl-up, hyperextended) and 32.18% MVC (sit-up), indicating that the hip flexors were also activated in these exercises.

The most demanding exercise was the vertical hip lift, in which activation of all ventral muscles increased significantly. The coactivation of the dorsal extensor muscles in the flexion period of flexion tasks typically ranged from 2% to 7% MVC. The neuromuscular activation profiles of the 2 lateral flexion exercises illustrated their characterization as "whole-body" exercises: all trunk muscles showed considerable EMG activity. Most dominant was the flexion activity of the external oblique muscle in the lateral-flexion, fixed-legs exercise: almost-maximal EMG levels were reached (97.77% MVC). This high level was reduced in the lateral hip lift (80.78% MVC), in which lateral flexion was performed as hip flexion with foot and elbow support.

Gymnastic Back- and Hip-Extension Exercises

The mean muscle activity for the diagonal hip- and shoulder-extension exercise did not exceed 35% MVC for any of the muscles, despite the fact that all muscles were active. The kneeling back-extension exercise showed a remarkable isolation effect for the erector spinae muscles, which demonstrated extension activity levels of 68.12% MVC (trapezius muscle at T6) and 44.02% MVC (erector spinae muscle at T12). Compared with the trunk extension, fixed-legs exercise, in which hip extension is added to the spine extension, only the lumbar erector spinae extension activity was slightly increased by 4.77%. The hip extensors had comparatively lower mean activation of 32.44% MVC (gluteus maximus) and 24.6% MVC (semitendinosus and semimembranosus muscle) in the extension phase. In the bridging exercise, all dorsal extensor muscles were activated on a low level: the highest mean extension activity for this exercise was only 36.96% MVC for the lumbar erector spinae.

In the hip extension, fixed-trunk exercise, the lower body moves against the fixed upper body, opposite to the trunk extension with fixed legs. When these 2 exercises were compared, a similar activity distribution was found. However, the lumbar erector spinae EMG activation for extension was increased by 7.12% MVC, and trapezius and rhomboideus activity at level T6 was diminished by 15.5% MVC.

Averaged Activation Profiles

To demonstrate the development of EMG activity within a movement cycle, we analyzed the time- and amplitude-normalized activation profiles for selected target muscles (Figure 4). Clear differences in the cycle-specific activation can be seen for both abdominal muscles: the rectus abdominis had a single peak pattern in flexion exercises (eg, straight curl-up and vertical hip lift) and a biphasic pattern in combined spine- and hip-flexion movements (eg, curl-up, hyperextended, and sit-up). High activation peaks between 80% and 100% MVC were found at the beginning (curl-up, hyperextended, and sit-up) or at the end (straight curl-up, vertical hip lift) at the end of the flexion period.

As a general trend, the external oblique muscle showed a similar activation profile for flexion movements as the rectus abdominis but on a different level. For lateral-flexion tasks, local supramaximal peak activations occurred at the end of the flexion period (Figure 4B). Similar EMG activation patterns of the 2 portions of the erector spinae were found in the back-extension exercises (Figure 4C and D): a descending activation within the flexion period was followed by a constant increase in the extension period, in which peak activity between 65% and 87% MVC (lumbar erector spinae) and 35% and 65% MVC (thoracic erector spinae) occurred at the end of the movement cycle. A somewhat different shape was found for the lumbar erector spinae in the kneeling back-extension exercise, in which a constant activation level between 50% and 60% MVC, rather than a peak through the middle range of the extension period, was found.

DISCUSSION

Variability of Tests

Movement velocity and acceleration, ROM, and load are important factors that directly alter EMG amplitude.[12,13] One approach to standardizing the velocity of movement within a set of repetitions is to use a metronome to control the duration of contraction.[5–6,8,14–17] When combined with a standardized ROM, we can assume that the mean velocity of movement is nearly constant. Yet this does not automatically mean that the shortening and lengthening velocity of the muscle fibers is constant throughout the ROM and that temporary differences in acceleration are eliminated. We instructed our subjects to perform a smooth and controlled movement to minimize the effects of fluctuation in velocity and acceleration. The mean CV for the contraction duration, both for the concentric and eccentric contraction periods, was less than 5%, which is an acceptable value. Godfrey et al[2] investigated the effect of different movement velocities in sit-up exercises and observed higher amplitudes for faster movements. They suggested that the discrepancies in the literature were due to differences in the cadence of movements. In our study, movement velocity differed among exercises because of varied ROMs. In some exercises, such as the vertical hip lift or lateral flexion with fixed legs, the duration variability among subjects increased up to 12%, reflecting the difficulty of the motor task (complexity, load, or both). Interindividual differences in the combined hip joint and segmental spine movement could have been one reason for increased variability, even if the overall movement of the whole body had a standardized range. Because of the multisegmental character of spine and hip movements, there is remarkable individual freedom to solve the given motor task.[18]

EMG Variability and Normalization

Our subjects performed a set of static exercises according to the concept proposed by McGill et al[8–10,15] to achieve a valid MVC reference value. This approach is based on their observation that subjects do not all perform at maximum EMG activity in the same exercise but may show maximum EMG activity in other exercises.[10,15] Our findings confirmed this observation; for example, MVC activity for the external oblique muscle was found in 4 different MVC tasks (Figure 2). The most productive exercise for the dorsal extensor muscles was not a machine-based exercise (as expected) but the prone-lying extension of the whole body from a slight flexed-hip position. One explanation for this finding could be the combination of

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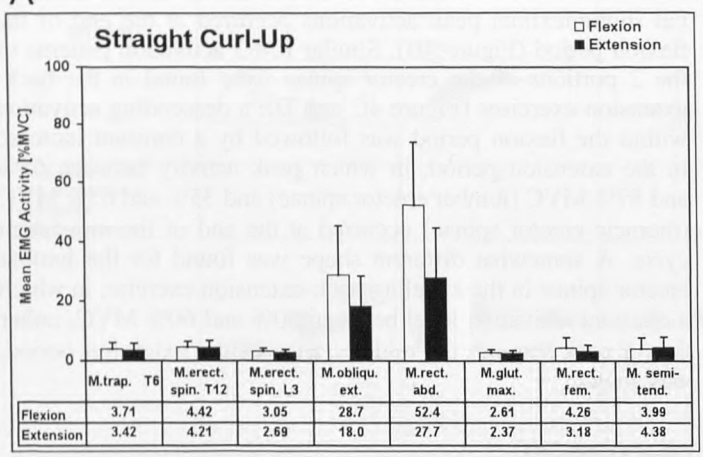

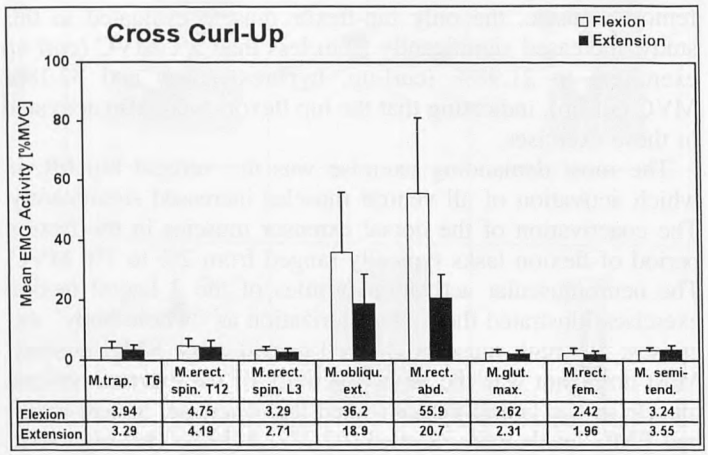

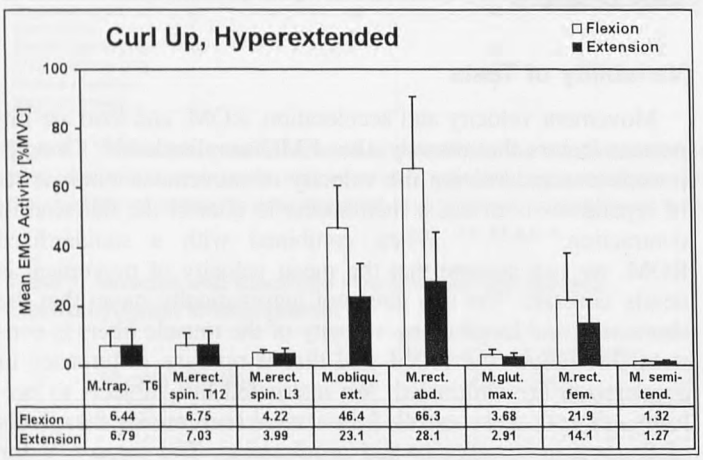

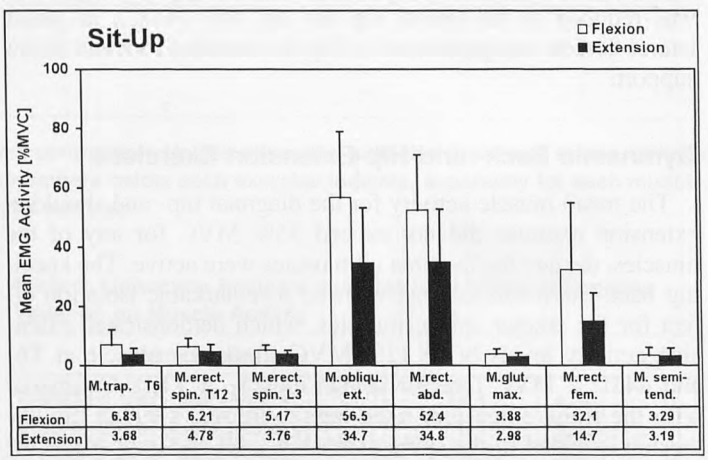

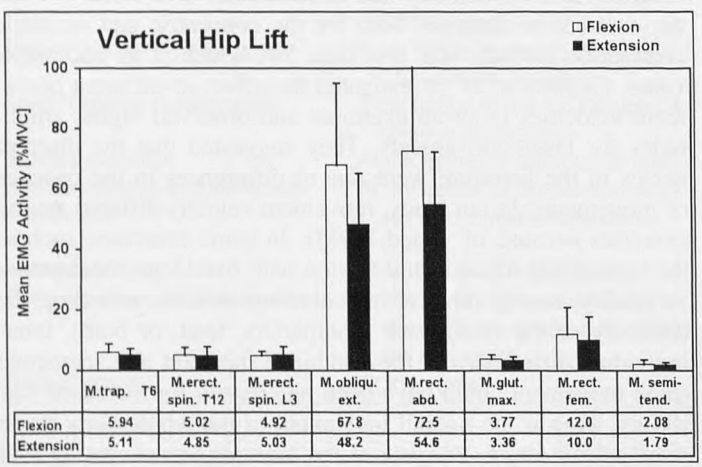

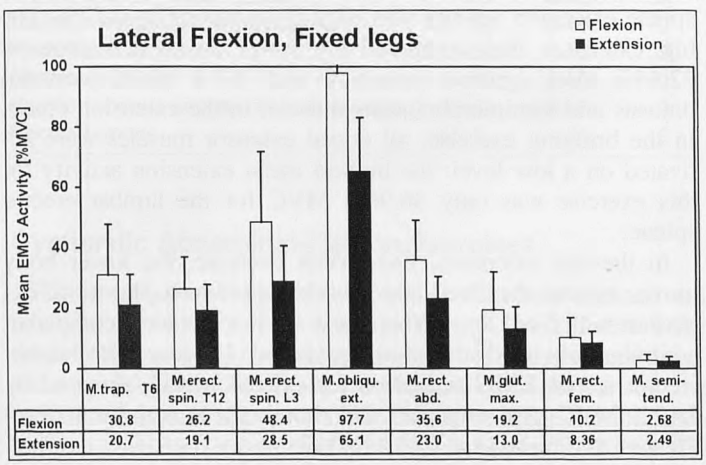

Figure 3. Mean activity distribution profiles. The mean electromyogram activity for the concentric and eccentric periods is calculated as the mean of 8 repetitions, which is averaged again for all subjects (n = 10). The thin lines indicate 1 SD. A, Profiles for exercises 1 through 6. B, Profiles for exercises 7 through 12.

the subject's stable position and the activation of the whole extensor chain, which facilitated the activity of all the synergistic muscles.

The activity distribution profiles (Figures 3A and B) can be used to estimate the effectiveness of an exercise set in terms of activating the main superficial trunk and hip muscles. However, the high SD ranges in all the EMG data reflect each individual's unique response to these exercises, despite the homogeneity of the subjects' skill levels and familiarity with the exercises. The mean CV of the average activation profiles of 3 selected target muscles ranged from 34.17% (external oblique in lateral-flexion tasks) to 41.18% (lumbar erector spinae in extension tasks) to 43.2% (rectus abdominis in flexion tasks). On average, this variability is comparable with or even lower than that found in other studies (eg, investigations of gait cycles).[11] As indicated by this variability, a general conclusion is that a training exercise does not automatically generate a certain stimulus or level of demand for the individual muscle.

Another feature of our study was the analysis of a typical training set for each exercise, including 8 repetitions. One expectation was that fatigue-induced changes of the EMG signal could occur within the sequence of repetitions (eg, increased EMG activity as a result of motor unit recruitment and increased firing frequency).[19,20] The statistical analysis of the mean EMG

B

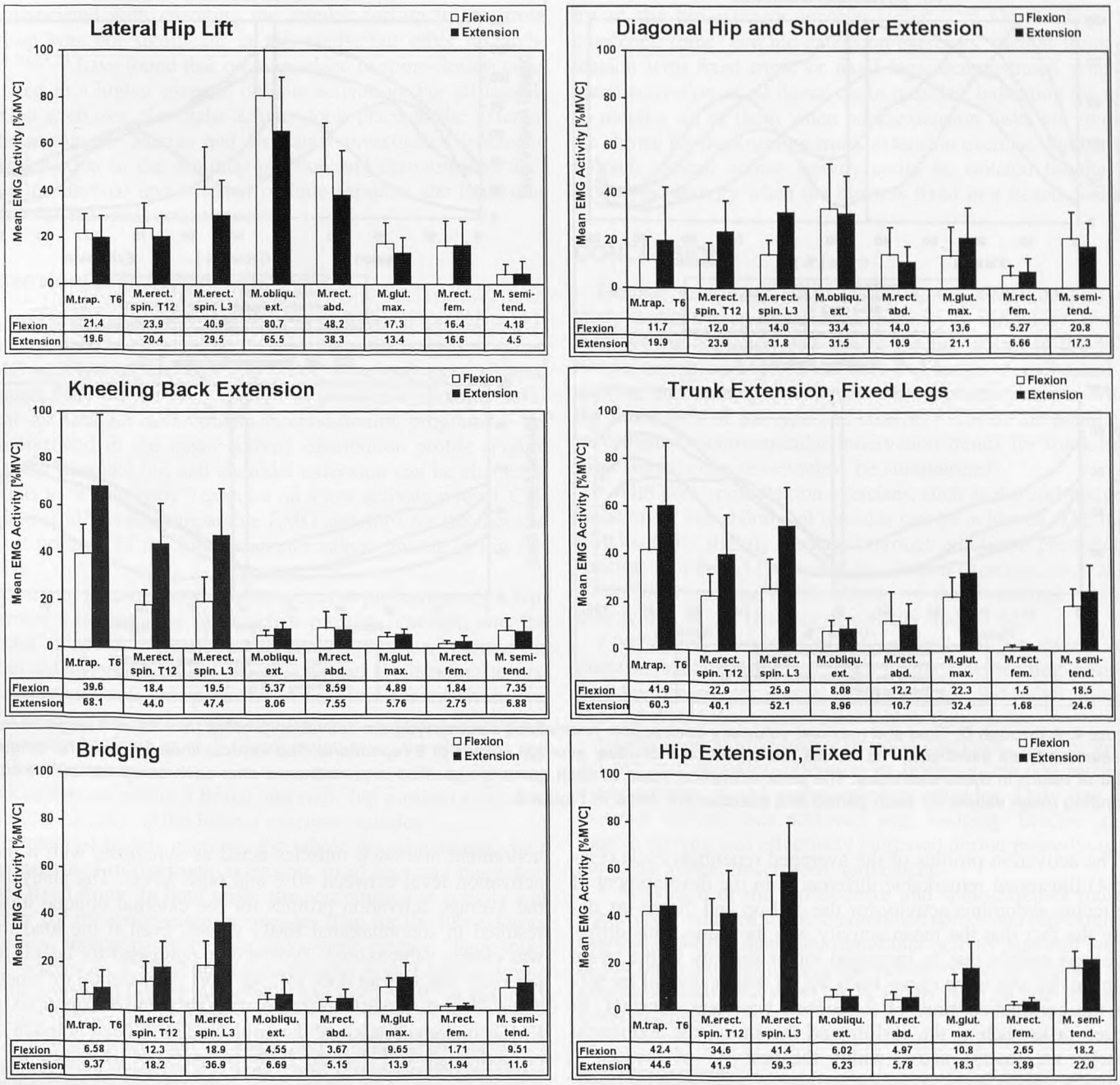

Figure 3. continued.

differences between the first and the last repetition revealed that only in 3 exercises did a significant increase in concentric EMG activity occur in the prime movers: the rectus abdominis in the curl-up, hyperextended, and sit-up and the external oblique in the lateral-flexion exercise with fixed legs. Probably as a result of the low initial activation level, typically not exceeding 50% MVC, no significant EMG increase was found for the other muscles and exercises. If training effectiveness is described in terms of strength development, the neuromuscular activation for these tasks may not be high enough, and increased load should be added to achieve an effective neuromuscular innervation higher than 50% MVC.[20,21] In practice, this would need to be determined individually, because contrary to the mean tendency, some individual subjects demonstrated steep increases, whereas others did not.

Flexion Exercises

A main finding was the isolation of the abdominal muscle activity in spine-flexion tasks without hip flexion (Figure 3A), which confirms earlier studies.[7,8] When hip flexion, such as in the sit-up, was added to spine flexion, the mean flexion activity for the rectus abdominis muscle was unchanged, but the external oblique and rectus femoris muscle activation was significantly higher. McGill et al,[22] comparing fine-wire and surface electrodes, found that with an error of about 12%, rectus femoris activity in common flexion tasks can be used to estimate the activity of the deep psoas muscle. Mean rectus femoris flexion activity in our data increased from 4.26% MVC (curl-up) to 32.18% MVC (sit-up), indicating clear involvement of the hip-flexor muscles. However, this finding demonstrated that the abdominal flexors remained the primary activated muscles.

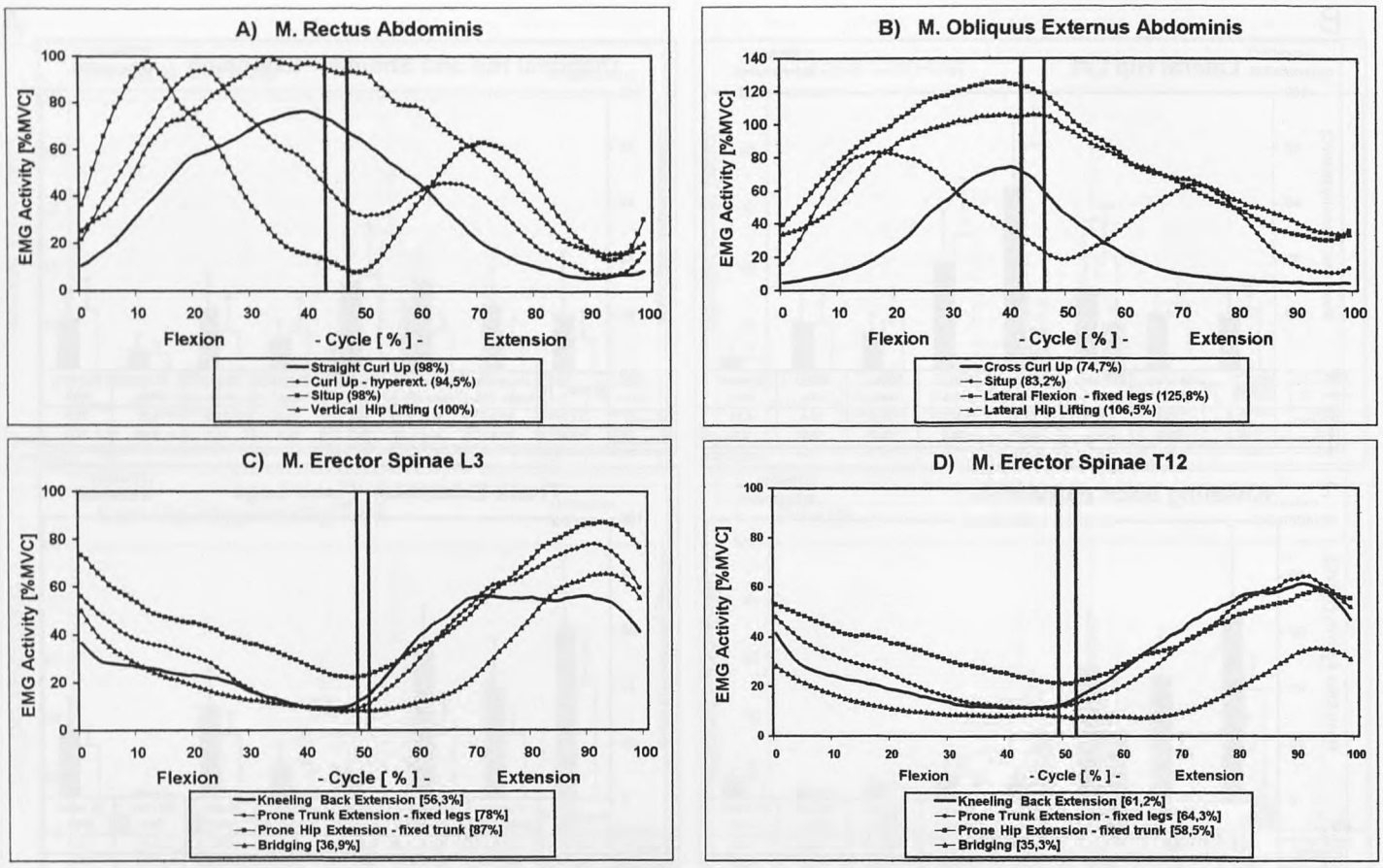

Figure 4. A through D, Time and maximal voluntary contraction–normalized electromyogram activation profiles (n = 10) of 4 target muscles in selected trunk exercises. The curves are based on individual average curves of 8 repetitions. The vertical lines indicate the range for point of return in these exercises. The peak activation value of each curve is specified in parentheses after each curve name. The corresponding mean values for each period and exercise are listed in Figure 3.

The activation profiles of the averaged repetition cycle (Figure 4) illustrated remarkable differences in the development of the rectus abdominis activity for the curl-up and the sit-up, despite the fact that the mean activity was the same. This difference was mainly due to increased initial velocity in the sit-up exercise, in which the upper body ROM was increased by 60°, but the contraction time of 2 seconds was kept constant. In flexion tasks such as the curl-up and the sit-up, the abdominal muscles are mainly active within the first 30° of flexion.[2–4] From the standpoint of muscular training effectiveness and peak activation, our data indicate that the sit-up is the more demanding exercise for both the rectus abdominis and the external oblique muscles due to the increased contraction velocity and the need to accelerate the upper body mass more quickly at the beginning of movement. As Axler and McGill[8] showed, this happens at the cost of higher compression forces acting at the lumbar vertebrae. Results from other studies[7,23,24] allow for the same conclusion of increased activation in the abdominal muscles in sit-ups, even if a direct comparison is limited due to different test standardization and EMG quantification. Similar to the study of Ekholm et al,[23] the mean EMG activity but not the peak activity for the (upper) rectus abdominis was significantly increased when the muscle was prestretched, as in the curl-up, hyperextended.

Lateral-Flexion Exercises

High neuromuscular demand on the external oblique was found in the 2 gymnastic lateral-flexion exercises. The other abdominal and back muscles acted as synergists with a mean activation level between 40% and 60% MVC. The analysis of the average activation profiles for the external oblique muscle resulted in supramaximal EMG values, even if the load itself was clearly submaximal. Typical data published for lateral-flexion tasks range from 40% to 75% MVC, depending on whether static[8,25] or dynamic[10] exercises were performed. Supramaximal EMGs under submaximal dynamic load have been noted in other studies as well, but the findings were not addressed by the authors.[8,14,18,26] The cause of this phenomenon remains unclear. A possible reason could be the incomplete excitation of the motoneurons within the static MVC test trial, despite the fact that similar exercise arrangements were used and the subjects were accustomed to performing at maximum effort. Another cause could be the changing electrode-to-muscle configuration and distance in dynamic vs static contractions, which is thought to influence the validity of static MVCs.[1,27–29] Changes in the recruitment scheme, increased cross-talk, metabolic changes occurring in repeated dynamic contractions, and signal-summation effects are other possible sources for supramaximal amplitudes. The estimation of the neuromuscular activation level is clearly limited by these uncertainties. However, because MVC amplitude normalization is mainly a rescaling function, the relative (muscle-specific) comparison of EMG activities among several tasks is not affected and should be the main focus of interpretation.

As compared with lateral-flexion tasks, a comparably low activation of the external oblique muscle was achieved with the

cross curl-up exercise (Figure 3A). The increase in mean activity associated with changing the regular curl-up to the cross curl-up was not significant in this study, but other researchers[8,10,23,24] have found that rotation added to spine-flexion tasks resulted in a higher external oblique activation. For all lateral-flexion exercises, the main activity took place in the external oblique muscle. Marras and Granata[25] investigated isokinetic lateral flexion in the standing position and demonstrated that, after the internal and external oblique muscles, the latissimus dorsi was the most active muscle.

Extension Exercises

Among the back-extension exercises, diagonal hip and shoulder extension and bridging showed comparatively low mean EMG activities of less than 35% MVC. The load and activation demand may not be high enough to produce a stimulus sufficient for strength development in conditioning programs.[21] As demonstrated in the mean activity distribution profile (Figure 3B), the diagonal hip and shoulder extension can be characterized as a "whole-body" exercise on a low activation level. Callaghan et al[9] found comparable EMG activities for the thoracic and 2 portions of the lumbar erector spinae muscle in this exercise.

Bridging is sometimes misinterpreted as predominantly a hip-extensor exercise. The most active muscle, however, was the erector spinae at the lumbar and thoracic portions, whereas the mean extension activity of the gluteus and semitendinosus and semimembranosus group was less than 14%. The relatively low activation of the gluteus was also found in a patient population studied by Liefring et al.[30] Good isolation of the thoracic and lumbar erector spinae muscles was achieved with the kneeling back extension, where a flexed and static hip position facilitated selective activity of the lumbar extensor muscles.

In contrast to this exercise, the whole dorsal extensor chain was activated in the trunk extension, fixed legs and hip extension, fixed trunk. In both tasks, the neuromuscular activity was higher for the spine-extensor muscles than for the hip-extensor muscles, indicating that these tasks were mainly "back-training" exercises. Callaghan et al[9] reported peak EMG activities of the erector spinae between 45% and 60% MVC in the trunk extension, fixed legs.

A direct comparison of these movements demonstrated a nonsignificant trend for the fixed-trunk version to enhance erector spinae muscle activity and diminish hip-extensor activity. As shown in Figure 4, peak erector spinae activity for combined spine- and hip-extension movement is located at the end of the extension period, when the body segments provide the highest lever arm. From the standpoint of training effectiveness, the last 25% of the extension cycle is the most productive part of movement. Callaghan et al[9] demonstrated that EMG activity is increased when true hyperextension of the spine and hip joint is performed. In 2 other studies,[6,30] near-maximal EMG activity was found in the lumbar erector spinae portion when unilateral and bilateral straight-leg lifting was performed to achieve prone hyperextension.

The increase in erector spinae activity with increasing extension is also reported for the combined back- and hip-extension task in the standing position.[28,31] In a comparison study between kneeling and standing back and hip extension, Gallagher[31] demonstrated that the angle-specific activation of the erector spinae muscle is strongly influenced by the hip and pelvis position and rotation. Many investigators have used seated or standing back- and hip-extension and lifting tasks without measuring the activity of the hip-extensor muscles.[17,26,28,31–34] Our findings for combined spine- and hip-extension exercises, such as trunk extension with fixed trunk or fixed legs, demonstrated synchronized activation of all dorsal chain muscles, indicating the need to monitor all of them when back-extension tasks are studied. As shown for the kneeling trunk-extension exercise, lumbar and thoracic erector spinae activity could be isolated for the hip extensors' activity when the hip was fixed in a flexed position.

CONCLUSIONS

Despite movement standardization, the EMG data in our study demonstrated remarkable intersubject variability. Even if some of this variation was due to the limitations of the MVC normalization concept, it outlines the range of individual responses and training effectiveness for a given exercise. Within the limitations of the type and size (n = 10) of the population investigated, neuromuscular innervation trends for trunk-training exercises can nevertheless be summarized.

- With pure spine-flexion exercises, such as the curl-up, good isolation of the abdominal muscles can be achieved. The mean EMG activity slightly increased through additional prestretch or rotation. Combined spine- and hip-flexion exercises, such as the sit-up, increased the EMG activity of the abdominal muscles, mainly due to the changes in velocity and ROM.
- Very high activation of the external oblique muscle took place in lateral-flexion exercises in the side-bending position. The lumbar erector spinae and the rectus abdominis muscles showed distinctive coactivation within these tasks.
- Double-supported back-extension exercises, such as diagonal hip and shoulder extension or bridging, elicited moderate mean activity in the dorsal-extensor muscles. Only low hip-extensor activity was achieved with bridging. Erector spinae muscle activity was effectively increased during prone-lying upper body versus lower body movements.
- Activation of the hip-extensor and spine-extensor muscles was closely coupled in these combined spine- and hip-extension exercises, but the main neuromuscular activity was still located in the back muscles. The spine extensors can be isolated with exercises based on a fixed hip in flexed position and segmental spine extension.

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Influence of Local Cooling on Proprioceptive Acuity in the Quadriceps Muscle

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Objective: To test the influence of cooling on proprioceptive acuity as reflected in the ability to discriminate weights.

Design and Setting: Participants were trained to perform a weight-discrimination task. Their ability to correctly report small increments in weight was compared before and after local cooling (a 20-minute application of a crushed-ice pack) of the quadriceps muscle group. Data were collected at a university research laboratory.

Subjects: Twenty young, physically active adults (undergraduate students; 14 men, 6 women; mean age, 22.1 ± 2.6 years).

Measurements: We calculated overall performance in the weight-discrimination task (percentage of discrimination correct) for each participant to estimate the differential threshold (ie, minimal increment in weight that yields a probability of 75% correct responses).

Results: Before local cooling, participants discriminated increments in the order of 4% to 10% from the standard weight (mean threshold, 0.17 ± 0.06 kg). After local cooling, the discriminative performance remained, on average, very similar to that seen before cooling (mean threshold, 0.17 ± 0.08 kg; paired *t* test: $t = 0.24$, $P = .81$). Only a small group of participants (n = 5) showed evidence of a decreased ability to discriminate weight after cooling.

Conclusions: The perception of force signals required for weight discrimination does not appear to be affected by local cooling of the quadriceps muscle group. This finding provides additional evidence for the relative safety of cold applications and their effect on proprioceptive perceptual abilities.

Key Words: cold therapy, weight perception, sensory discrimination

In the context of sports therapy, ice is frequently used to treat minor acute musculoskeletal injuries. Although ice is known to be effective in decreasing painful and tactile sensations, its effect on proprioceptive abilities has received comparatively little attention. La Riviere and Osternig[1] evaluated the effect of brief (5-minute) and prolonged (20-minute) cold-water immersion on ankle proprioception and concluded that cooling does not alter position sense. Thieme et al[2] reported similar findings for the knee joint after a 20-minute ice application. Thus, perception of static joint positions seems to be preserved after cooling. The ability to sense joint position, however, is only one of the perceptual attributes of the proprioceptive system, which also includes the ability to sense movement (amplitude and angular velocity) and to perceive force and weight.[3] The latter ability is particularly important both from historical and perceptual points of view. Weber in 1834 was the first to fully appreciate the importance of weight perception in relation to what he called the "muscular sense." Weber's seminal observation that weight discrimination was far more accurate when objects were actively lifted instead of being passively applied on skin provided the first evidence that signals from contracting muscles participate in weight perception.[4] At that time, however, it was not clear if the muscular sense, and therefore weight perception, was derived from signals of peripheral origin or from sensory signals arising centrally via corollaries of the descending motor commands producing the lifting action. More recent investigations have helped to clarify the issue and confirmed that sensory signals of both central and peripheral origins contribute to our ability to perceive force and weights.[4]

Given the acute sensitivity of the human proprioceptive system to changes in weight (typically, normal subjects can reliably discriminate a 5% to 10% change in weight with active lifting movements[5,6]), we thought that weight discrimination would be a good test to determine the influence of cooling on proprioceptive acuity. The purpose of our study, therefore, was to test whether the ability to perceive small differences in weight (ie, proprioceptive acuity) remained accurate after local cooling of the quadriceps muscle group.

METHODS

Participants

Twenty young, healthy adults (mean age, 22.1 ± 2.6 years) were recruited from among the population of undergraduate

Table 1. Participants' Characteristics

Sex	No.	Age, y	Physical Activity Level*		
			Low	Moderate	High
Women	6	21.5 ± 1.5	0	4	2
Men	14	22.3 ± 3.3	4	7	3

*As self-reported by subjects on a separate health questionnaire. Low indicates activity <2 times/week; moderate, activity 2 to 3 times/week; and high, activity ≥4 times/week.

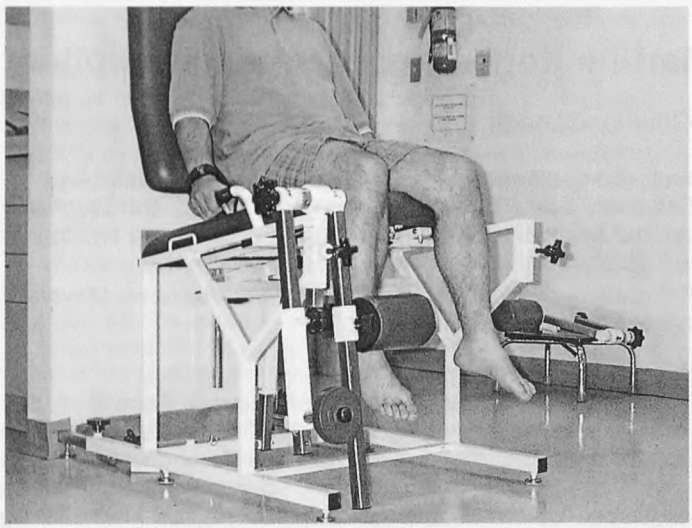

Figure 1. The participant's starting position on the exercise table with the 0.50-kg increment weight on the lever. Note that the standard weight corresponded to the weight of the lever unloaded (2.50 kg).

students at the Faculty of Health Sciences, University of Ottawa. All participants were physically active (Table 1), and some were even engaged in high-level activity (1 participant was a player on the Canadian women's Olympic hockey team). None reported a previous history of knee injuries or sensory dysfunction in the right lower extremity. The study's procedures were approved by the University Human Research Ethics Board, and subjects gave their informed consent.

Materials

The ability of participants to discriminate weights was tested on a conventional leg exercise table (Model 2400, Midland Co, Columbia, SC) equipped with a lever system that allowed for loading of free weights (Figure 1). The actual weight of the table's right lever system unloaded (2.50 kg measured separately on a precision numeric scale) was chosen as the standard. Pilot testing indicated that a standard weight of 2.50 kg was appropriate to prevent fatigue during testing because participants had to perform many repeated lifting movements. A set of metric metal weights (0.11, 0.28, 0.40, and 0.50 kg, Elgin Co, Elgin, IL) was used to gradually increase the mass of the standard weight. These comparison weights corresponded to increments of 4% (2.61 kg), 11% (2.78 kg), 16% (2.90 kg), and 20% (3 kg) from the standard. This set of comparison weights covered the range of human capacities for weight discrimination along a continuum from easy (16% and 20% changes) to increasingly difficult (4% and 10% changes) discrimination.

Discrimination Paradigm

Trial #	Order of presentation within a block (pseudo-random)	Alternatives (equally probable)	Subject's report on which was heavier: 1st or 2nd ?
1	(1) S_w→ (2) C_w	1st or 2nd	2nd
2	(1) C_w→ (2) S_w	1st or 2nd	1st
3	(1) C_w→ (2) S_w	1st or 2nd	1st
4	(1) S_w→ (2) C_w	1st or 2nd	2nd
↓	↓	↓	↓
14 trials	28 presentations	7 (1st) 7 (2nd)	Performance score: % correct

Figure 2. Protocol used for weight discrimination. S_w indicates standard weight (2.50 kg), which corresponded to the weight of the lever unloaded. C_w indicates comparison weights (0.50-, 0.40-, 0.28-, and 0.11-kg increments) that were added separately to the standard (lever) to increase its mass. Note that the comparison weight was always the same within a block of trials and that the blocks were presented in an overall sequence of increasing difficulty (ie, first block, 0.50 kg; second block, 0.40 kg; third block, 0.28 kg; and fourth block, 0.11 kg).

Weight-Discrimination Task

Precooling. As shown in Figure 1, the task was performed with the participant comfortably seated on the exercise table with the hips flexed to approximately 100° and the knee resting at 90°. The leg pad was adjusted in height approximately 5 cm above the medial malleolus of the right leg. Participants were then instructed regarding the task. They were told that it consisted of active-lifting movements in order to compare weights with the right leg. Participants were free to choose any movement ranges (from 90° to 180°), speeds, and modes of contraction (concentric or eccentric) they deemed appropriate to estimate the weights. To prevent any bias, participants were not informed that the standard weight corresponded to the weight of the lever unloaded. In addition, to avoid pressure cues, the lever was always pulled away from the participant's leg for weight loading and unloading. Participants were then blindfolded and provided with a series of practice trials, which consisted of 5 easy discriminations (0.50-kg increments) and 5 more difficult discriminations (0.28-kg increments). During practice trials, feedback was given on discrimination performance. Once participants felt comfortable with the task, the formal testing session began. Participants remained blindfolded and were fitted with earplugs to eliminate any auditory cues. The discrimination protocol is described in Figure 2. Briefly, it consisted of a 2-alternative, forced choice (2-AFC) procedure with each trial consisting of 2 weight presentations: the standard weight (2.50 kg) and a comparison weight (eg, a 0.50-kg increment). After receiving a tactile cue (a tap on the knee), participants lifted each weight successively and reported which in the sequence (the first or second weight) felt heavier. The order of presentation (standard versus comparison) was pseudorandom, and the 2 alternatives were equally probable within a block of 14 trials (Table 2). As usual in discrimination paradigms,[7,8] the comparison weights were presented in 4 successive blocks of increasing difficulty (ie, 0.50-kg increments

Table 2. Discrimination Paradigm Used for the Weight-Discrimination Task*

Trial No.	Order of Weight Presentation Within a Block†	Subject's Response: First or Second Heavier?
1	STD‡ → STD + COMP§	Second
2	STD + COMP → STD	First
3	STD + COMP → STD	First
4	STD → STD + COMP	Second

*Twenty-eight presentations in 14 trials. Performance score = No. correct/14 × 100.
†Order was pseudorandom within a block. The 2 alternatives (first or second) were equally probable.
‡STD indicates standard weight (2.50 kg), which corresponded to the weight of the lever unloaded.
§COMP indicates comparison weights (0.50, 0.40, 0.28, 0.11 kg) added to the STD (lever) to increase its mass. The comparison weights were always the same within a block of trials, and they were presented in 4 successive blocks of increasing difficulty (0.50 kg down to 0.11 kg).

down to 0.11-kg increments). Although no restriction was imposed on the lifting movement, most participants chose an up-and-down strategy to judge the applied weight, moving up the lever slowly near full extension (concentric mode), then returning slowly to the starting position (eccentric mode). No feedback was provided on the discrimination performance during or after formal testing. The whole precooling procedure was usually completed within 30 minutes, the task itself taking 10 to 15 minutes to complete.

Cooling. Participants were placed on a bed with the right knee in slight flexion and resting on a pillow. Before ice application, the right thigh-skin sensibility was briefly tested by checking response to pinprick. The ice was applied in the form of crushed ice in a moist towel over the anterior aspect of the thigh for 20 minutes to obtain effective cooling of the quadriceps muscle belly. This method of cooling has been shown to be effective in decreasing intramuscular temperature.[9,10] The ice application was adjusted for each participant so that it covered at least two thirds of the anterior thigh, excluding the most distal (patellar) and proximal (groin) areas. After the 20 minutes had elapsed, the ice pack was removed and the skin was reinspected. All participants reported diminished sensation and a decreased response to pinprick in the cooled area.

Postcooling. After the cooling procedure, participants were rapidly returned (usually within 2 minutes) to the exercise table to be tested again with the weight-discrimination task. As in the precooling procedure, testing took 10 to 15 minutes to complete.

Data Analysis

Proprioceptive acuity was determined by calculating for each participant the performance in the weight-discrimination task (ie, the number of correct discriminations for each comparison weight). A discrimination function was then constructed for each participant by plotting performance values (in percentages) against increments in weight. Although the discrimination function provides a description of the discriminative behavior, it is convenient to extract a single representative value for this capacity. For this purpose, we used the differential threshold, which, by convention in a 2-AFC procedure, corresponds to the minimal increment that yields a

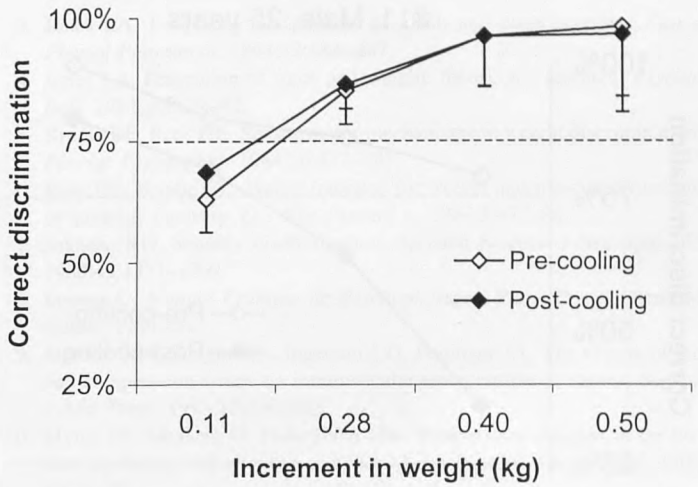

Figure 3. Performance in the weight-discrimination task before and after cooling. Each value represents the mean performance of all participants (n = 20) for each increment in weight with the associated SD in one direction. The dotted line indicates the 75% correct discrimination level, which corresponds by convention to the differential threshold.

probability of 75% correct responses (ie, midway between chance and perfection).[7,8] The differential threshold was computed by linear interpolation between the performance values on either side of the 0.75 value.[7] In some instances, performance was greater than or equal to the 75% level even with the lightest increment in weight (ie, 0.11 kg). In these cases, the threshold was set at 0.11 kg for statistical purposes. Precooling and postcooling differential thresholds were compared using a paired t test at the $P < 0.05$ level.

RESULTS

Figure 3 demonstrates the mean performance in the weight-discrimination task before and after local cooling. Before cooling, participants had no difficulty discriminating the 2 largest comparison weights (the 0.50- and 0.40-kg increments) from the standard. For the 2 other comparison weights, the discrimination performance decreased gradually and fell below the 75% level for the lightest increment (0.11 kg). Yet some participants (n = 6) were able to provide reliable reports of weight change (performance greater than or equal to the 75% level), even for the 0.11-kg increment. Overall, the differential threshold for weight discrimination before cooling represented, on average, a 6.8% increase from the standard weight (mean, 0.17 ± 0.06 kg). After cooling, the discriminative performance was, on average, very similar to that seen before cooling (Figure 3), and the differential threshold remained stable (mean, 0.17 ± 0.08 kg; $t = 0.24$; $P = 0.81$).

Although the ability to discriminate weights was unaffected by cooling in most participants, a small number of individuals (n = 5) experienced a decline in performance after cooling (ie, individual threshold values increased by a factor of 1.5 to 3.5 as compared with precooling values). Figure 4 displays the results for such an individual. Before cooling, this participant (a moderately active 25-year-old man) had no difficulty discriminating large and small increments in weight (threshold, 0.11 kg). However, after cooling, his discriminative ability greatly deteriorated, as evident in the major shift of the discrimination function (postcooling threshold, 0.40 kg). Such a decline in weight discrimination after cooling was not partic-

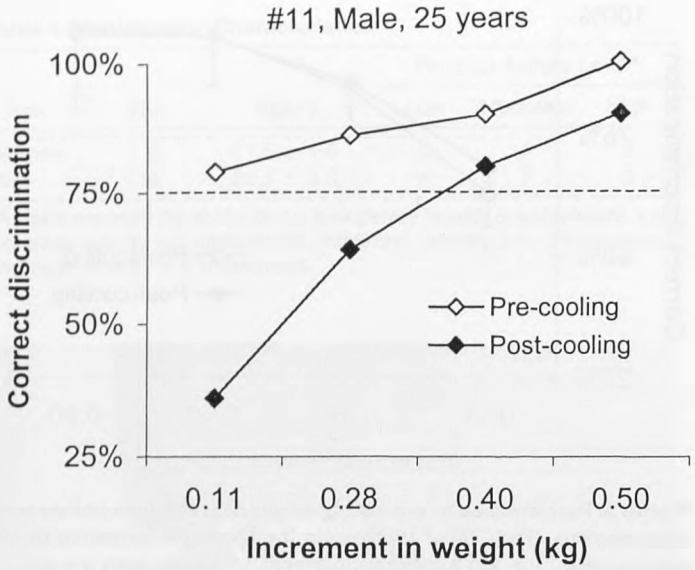

Figure 4. An individual whose discriminative performance was affected after cooling. The representation is similar to the representation in Figure 3.

ular to a sex (4 men, 1 woman) or to a level of physical activity (4 were moderately active, 1 was highly active).

DISCUSSION

Our study has shown that proprioceptive acuity in the quadriceps muscle, as reflected in the ability to perceive differences in weight, is preserved after a prolonged ice application. Before cooling, participants were able to reliably discriminate 4% to 10% increments in weight (threshold range, 0.11 to 0.26 kg), a range comparable to that reported in previous studies on weight discrimination.[5,6] After cooling, the perception of force signals generated during weight lifting remained accurate in most participants. In the following discussion, we will first address the issue of effectiveness of cooling methods as they pertain to proprioception. We will then discuss the possible sensory mechanisms underlying the current and previous observations that cooling has no effect on proprioceptive abilities.

The present finding that cooling did not affect the ability to discriminate weights is in agreement with the findings of previous studies that reported no change in the sense of position in the lower limb.[1,2] Thus, proprioceptive abilities appear to be relatively insensitive to the cooling effect. This finding raises the question of why proprioception is unaffected, whereas thresholds for cutaneous sensations rise sharply after local cooling.[11] One obvious possibility is that the common methods of cooling are simply not effective in decreasing the temperature in deep tissue, where important proprioceptors are located. Indeed, studies have shown that the degree of cooling achieved in deep tissues varies widely depending upon the method used (eg, ice packs versus gel packs or cold-water immersion).[12] We used a conventional method to cool the quadriceps muscle (crushed-ice pack for 20 minutes). Although we did not monitor tissue temperature, other authors have reported significant reductions in intramuscular temperature using similar applications.[9,10,13] Thus, we can reasonably assume that our cooling method was effective in decreasing muscle temperature. The degree of cooling achieved in each individual may have been different, however, due to uncontrolled factors such as thickness of adipose tissue. Nevertheless, all participants reported diminished sensation in the cooled area (response to pinprick) after the application and exhibited the usual objective signs of tissue cooling such as intense skin redness.

Another related issue pertains to the persistence of the cooling effect. As stated in the Methods section, the participants were tested within 2 minutes after application, and testing took 10 to 15 minutes to complete. Therefore, one may argue that by the time the testing procedure ended, the temperature had already returned to its preapplication levels. Although we cannot rule out this possibility, a recent study by Merrick et al[9] showed that an ice wrap applied over the anterior thigh for 30 minutes produced significant cooling in deep tissues (2 cm) that persisted up to 20 minutes after application. Thus, it is very unlikely that all of the cooling-induced effect had completely vanished by the time the postcooling testing procedure was administered.

Whatever the issues about the depth and duration of temperature changes, cooling is known to produce marked and persistent slowing of peripheral nerve conduction. For example, after cooling the calf muscles for 20 minutes, Halar et al[13] reported an average reduction of 7.4°C in skin temperature with corresponding drops in sural and tibial nerve conduction velocity of 11.2 and 6.4 $m \cdot s^{-1}$, respectively. The H-reflex latency, which reflects conduction in proprioceptive afferents from muscle spindles, increased, on average, by 5.3 milliseconds. Thus, peripheral signals of cutaneous and muscle origin were very likely (if not certainly) reduced after cooling. A reduction in skin afferents, although critical for tactile and pain sensations, is of less consequence for proprioceptive abilities. Indeed, skin mechanoreceptors contribute little to proprioceptive acuity, at least in the larger joints of the lower extremity.[14,15] Alternatively, any reduction in muscle afferents could be more detrimental for proprioceptive acuity because signals from muscle spindles appear to be critical to our ability to sense joint position and movement.[15–17] As previously noted, the ability to sense joint position is apparently unaffected after cooling. La Riviere and Osternig[1] suggested that inputs from joint receptors are probably able to compensate for cold-induced reductions in skin and muscle afferents. This proposal is unlikely, however, because joint receptors are only activated at the extremes of the joint range during passive movement,[14] a property that makes them poor candidates to signal joint position. Another possibility resides in the differential response of primary and secondary spindle endings to changes in muscle temperature.[18,19] Mense[18] studied the effect of temperature on cat muscle spindles and reported that primary endings were uniformly depressed in a cold muscle (approximately 29°C), whereas most secondary endings showed the reverse effect (ie, increased response to stretch). Whereas the reduced outflow from primary endings is consistent with reductions in tendon reflex amplitude reported in humans,[20] the increased outflow from secondary spindle endings may well explain the preserved ability to sense joint position after cooling, as previously noted. Indeed, secondary endings have been implicated in the perception of absolute joint angle because they are thought to function as length detectors.[15]

Another class of proprioceptive afferents that is particularly important with regard to the present findings is tendon organs. We deliberately restricted the cold application to the anterior thigh to reproduce situations often encountered in sports therapy (eg, treating a muscle bruise). The application, therefore,

did not cover the joint or the quadriceps tendon. The major role ascribed to tendon organs in the genesis of sensations of force and effort[4] may explain why weight discrimination was preserved in most participants, since tendon organs were, presumably, less affected by cooling. Evidence in animals, however, suggests that afferents from tendon organs are not markedly affected by changes in muscle temperature.[18] Yet, there remains the possibility that, even with a profound reduction in afferents from muscle spindles or tendon organs, weight discrimination can still be performed on the basis of corollary discharges associated with the active-lifting movements.[4,21] Psychophysical experiments have shown that when peripheral feedback is reduced or perturbed, individuals tend to rely more on sensory information generated centrally (via corollaries of descending commands) than on signals arising peripherally to make judgements about force and weight.[4,22] Thus, participants may have compensated for the reduction in peripheral signals by estimating the degree of efferent activity (via corollary discharges) necessary to lift the weight. Some participants did experience a decline in their ability to discriminate weight after cooling (Figure 4). The discrimination performance of those participants may have relied more on signals arising peripherally than on signals arising centrally, even if the participants were not necessarily aware of this effect. Interestingly, 2 of these participants made comments such as "my leg felt heavier" or "my quad was numb" after completing the postcooling testing, suggesting that they were more focused on sensations coming from their cooled muscle.

In conclusion, our study provides additional evidence that proprioceptive acuity in the quadriceps muscle group remains largely unaffected after prolonged ice application to the thigh. The fact that the sensory mechanisms underlying the perception of limb position[1,2] and sensations of force remained operational after cooling suggests that motor performance should not be affected. Indeed, there is evidence that cryotherapy has only minor consequences for motor performance.[23] Thus, a rapid return to play after ice therapy may not be necessarily detrimental for the athlete. Of course, other components of motor performance (eg, strength and flexibility) might be affected after ice therapy,[23,24] and health care professionals, therefore, must fully evaluate the conditions and circumstances before returning an athlete to action.

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The Effects of Low-Dose Creatine Supplementation Versus Creatine Loading in Collegiate Football Players

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Objective: To compare the effects of low doses of creatine and creatine loading on strength, urinary creatinine concentration, and percentage of body fat.

Design and Setting: Division IA collegiate football players took creatine monohydrate for 10 weeks during a sport-specific, periodized, off-season strength and conditioning program. One-repetition maximum (1-RM) squat, urinary creatinine concentrations, and percentage of body fat were analyzed.

Subjects: Twenty-five highly trained, Division IA collegiate football players with at least 1 year of college playing experience.

Measurements: We tested strength with a 1-RM squat exercise before, during, and after creatine supplementation. Percentage of body fat was measured by hydrostatic weighing before and after supplementation. Urinary creatinine concentration was measured via light spectrophotometer at 0, 1, 3, 7, 14, 21, 28, 35, 42, 48, 56, and 63 days. An analysis of variance with repeated measures was computed to compare means for all variables.

Results: Creatine supplementation had no significant group, time, or interaction effects on strength, urinary creatinine concentration, or percentage of body fat. However, significant time effects were found for 1-RM squat and fat-free mass in all groups.

Conclusions: Our data suggest that creatine monohydrate in any amount does not have any beneficial ergogenic effects in highly trained collegiate football players. However, a proper resistance training stimulus for 10 weeks can increase strength and fat-free mass in highly trained athletes.

Key Words: creatine phosphate, ergogenic aids, resistance training

Athletes continue to search for the most effective supplement to aid performance. Athletes use a wide variety of commercial supplements because of the belief that the supplements possess beneficial effects. Some examples include l-carnitine, ginseng, chromium, glutamine, amino acids, protein powders, and creatine monohydrate, taken alone or in combination. Some supplements may cause long-term or short-term harmful effects, a possibility that has stimulated concern among health care professionals, exercise physiologists, and coaches. Because we do not know conclusively which supplements are ergogenic or ergolytic, we need to further examine specific supplements and their effects on exercise.

Recently, creatine monohydrate has become the nutritional supplement of choice for athletes. This compound has accounted for most of the supplement sales during the past few years, and the market continues to grow as a result of endorsement by professional athletes. In addition, creatine does not appear on the banned list of substances of any sports federation.

Professional athletes have a tremendous influence on many other athletes at various levels of competition. Currently, college, high school, and recreational athletes are questioning the effects of creatine. Recent studies have supported creatine as an effective, harmless ergogenic aid with no short-term contraindications.[1–11] However, the effect of long-term use of creatine monohydrate on athletes is unclear; hence, the medical community has raised concerns, and possible health risks have been suggested.[12]

Creatine supplementation has resulted in positive physiologic effects on skeletal muscle creatine phosphate stores; short-duration, high-intensity anaerobic exercise; strength; and body composition in physically active subjects.[1–21] These subjects have used a loading protocol of 20 to 30 $g \cdot d^{-1}$ for up to 7 days, but most of these studies were not conducted in sport-specific settings.[1,2,13–18]

If creatine supplementation, specifically loading, causes a surplus of creatine in the muscle, then any excess can be converted and excreted in the form of creatinine.[13,19,20] Creatinine is produced from creatine as a byproduct of catabolism in skeletal muscle, and it appears in the urine. Therefore, creatine loading may not be beneficial to athletes if the muscle can only hold a predetermined concentration. Harris et al[22] showed that approximately 155 $mmol \cdot kg^{-1}$ of dry muscle mass may represent the upper storage limit of creatine when a subject ingests 5 g 4 to 6 times per day. Thus, creatine loading may

result in an excessive creatine concentration, which can be converted to creatinine and excreted. This finding suggests that creatine loading may not be necessary for those athletes (eg, football players) engaged in long-term supplementation.

Therefore, the purpose of our investigation was to compare the effects of low-dose creatine supplementation (3 $g \cdot d^{-1}$) to a loading protocol of 20 $g \cdot d^{-1}$ for 7 days and 5 $g \cdot d^{-1}$ thereafter for 10 weeks on 1-repetition maximum (1-RM) squat strength, creatinine excretion, and percentage of body fat in highly trained collegiate football players.

METHODS

Procedures

The subjects in this study were 25 male (age, 19 ± 1.02 years; height, 185.8 ± 5.27 cm; mass, 100.89 ± 21.79 kg), highly trained, NCAA Division IA collegiate football players with at least 1 year of college experience. All subjects were engaged in an off-season resistance training and conditioning program designed by the members of our strength and conditioning staff from the Department of Intercollegiate Athletics. Written consent was obtained from all subjects before testing and training, and the Institutional Review Board for the Protection of Human Subjects approved this project.

We selected subjects using the following criteria: (1) members of the institution's football team for at least 1 season; (2) weight training and conditioning limited to 4 specified sessions per week during the off-season program; (3) injury free; (4) not taking other substances that might aid athletic performance for 4 weeks before the study began and no other supplements for 10 weeks; and (5) maintenance of a normal diet while participating in the study.

We randomly placed 25 subjects into 1 of 3 groups: (1) creatine supplementation of 3 $g \cdot d^{-1}$; (2) creatine supplementation of 20 $g \cdot d^{-1}$ for 7 days, followed by creatine supplementation of 5 $g \cdot d^{-1}$ for the remainder of the study; or (3) a placebo group. The subjects were permitted to withdraw from the study at any time.

All subjects were familiarized with the scope of the study 1 week before its start. Since all subjects were team members for at least 1 year, they were extremely familiar with the training program, especially the squat exercise. Each subject took part in the preliminary testing 3 days before supplementation. The 1-RM squat, baseline urinary creatinine concentrations, and percentage of body fat (via hydrostatic weighing) were measured.

Creatine supplementation took place in a single-blind fashion, with each subject receiving a high or low dose of creatine or the placebo. Each creatine tablet (Createam Chewables, NutraSense Co, Shawnee Mission, KS) contained 1 g of creatine and 1.4 g of dextrose. The placebo tablet (Nutrasense) contained 2.4 g of dextrose. For the first week (loading phase), ingestion occurred 4 times per day. High-dose creatine (5 creatine tablets), low-dose creatine (3 creatine tablets and 2 placebo tablets), or placebo (5 placebo tablets) was taken when the subjects awoke, before and after the workout session, and in the evening before bed. For the subsequent 9 weeks, the high or low dose of creatine or the placebo dose was ingested once per day after workouts and at the same time on off days.

During the 10-week supplementation period, all subjects participated only in the University's off-season conditioning program, which consisted of periodized resistance and agility training. More specifically, the program consisted of 4 $h \cdot week^{-1}$ of heavy resistance training and 4 $h \cdot week^{-1}$ of conditioning. Weight training and conditioning were performed on Monday, Tuesday, Thursday, and Friday. All subjects completed the same number of morning workouts, sets, repetitions, and exercises at specified percentages of a 1-RM, regardless of position. The strength and conditioning coach led all workout sessions to ensure highly productive strength training and took a team approach in which each exercise is performed on the strength coach's cue. For example, all athletes started a lift on command. After each set was a specified rest interval, and then the next set would begin.

A 10-week, periodized resistance training program was designed. The primary exercises in the strength program were the front squat, back squat, hang clean, power clean, overhead press, bench press, single-arm dumbbell press, 1-arm rows, straight-leg dead lift, power shrugs, upright rows, chin-ups, dips, medicine ball plyometrics, and bumper-plate push-ups. The periodization protocol was a 5-week base hypertrophy phase (4 to 6 sets at 50% to 80% 1-RM), followed by a 2-week power phase (3 to 5 sets at 80% to 88%). After this 7-week period, a 2-week strength phase (1 to 3 sets at 90% to 95%) was implemented, followed by a 1-week peak strength phase (1 to 3 sets at 95% to 100%). The strength and conditioning staff, athletic trainers, and football coaches supervised all strength and conditioning sessions.

The 1-RM back-squat values were obtained before (week 0), during (week 5), and after supplementation (week 10). Urine samples were collected in sterile containers during the supplementation period on days 0, 1, 3, 7, 14, 21, 28, 35, 42, 49, 56, and 63, 4 hours after creatine tablet ingestion. During this 4-hour period, each subject was allocated 300 mL of water in a water bottle, the only liquid the athlete was allowed to consume until the urine sample was obtained. Each sample was analyzed using a spectrophotometric method.

Creatinine Measurement

A creatinine assay kit (Sigma Diagnostics, St. Louis, MO) was used to prepare the urine for analysis. The urine was diluted with distilled water in a standard test tube (0.5 mL of urine was added to 7 mL of distilled water). An alkaline picrate solution was prepared by mixing 5 parts of the creatinine color reagent (0.6% picric acid, sodium borate, and surfactant) to 1 part sodium hydroxide. Next, a pipette was used to place 300 μL of the diluted urine in a cuvette, where it was mixed with 3 mL of the alkaline picrate solution. This mixture was allowed to incubate for 10 minutes before the initial absorbance was read at 500 μm by the spectrophotometer. Finally, 100 μL of an acid reagent (mixture of sulfuric and acetic acid) was added to the assay and allowed to incubate for 5 minutes. Then the final absorbance was read at 500 μm by the spectrophotometer.

Back Squat

The back-squat exercise was performed on a standard lifting platform with an Olympic-size bar and rubber bumper plates. The parallel-squat position is defined as the subject's ability to lower a weight until the thigh is parallel to the floor and then return to the starting position. We instructed our subjects to ride a stationary bike for 5 minutes before performing the squat exercise. Each subject completed 2 repetitions at 60, 70,

Table 1. Group Means ± SDs for 1-Repetition Maximum (1-RM) Strength, Percentage of Body Fat, and Fat-Free Mass*

Variable	Low Dose (n = 8)	Loading (n = 8)	Placebo (n = 9)
1-RM strength, kg			
Before supplementation	169.6 ± 29.8	163.6 ± 22.2	169.1 ± 26.5
During supplementation	179.8 ± 26.5†	169.6 ± 16.1†	169.1 ± 19.1†
After supplementation	188.6 ± 37.3‡	172.4 ± 18.7‡	178.0 ± 22.4‡
Percentage of body fat			
Before supplementation	13.76 ± 5.57	14.85 ± 5.92	13.75 ± 5.85
After supplementation	13.37 ± 6.17	15.81 ± 5.28	14.53 ± 5.08
Fat-free mass, kg			
Before supplementation	81.67 ± 10.88	85.02 ± 8.97	81.53 ± 8.63
After supplementation	84.64 ± 9.79†	85.19 ± 8.60†	82.59 ± 7.29†

*Low dose indicates 3 g of creatine supplementation without loading phase. High dose indicates 5 g of creatine supplementation with a 20 g·d^{-1}, 7-day loading phase.
† Significance over time from before-supplementation values at α level $P < .05$.
‡ Significance over time from during-supplementation values at α level $P < .05$.

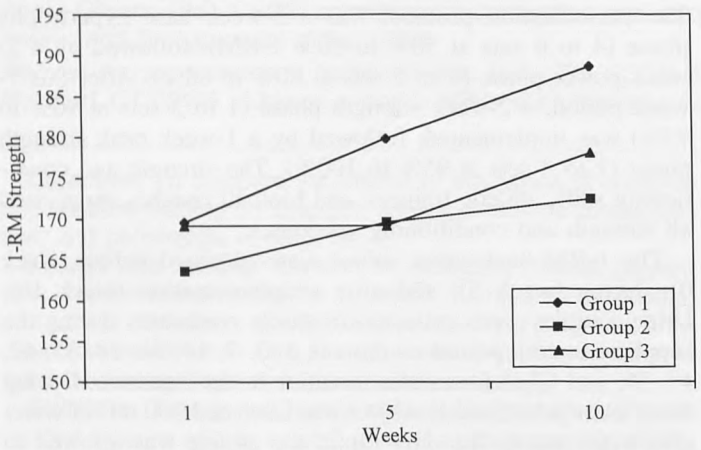

Figure 1. Changes in 1-repetition maximum for the squat exercise among groups throughout 10 weeks of creatine supplementation or placebo.

and 80% of a predicted 1-RM. After these 3 sets, subjects continued to complete 1 repetition, increasing the weight by 2.72-kg (5-lb) increments until either the parallel position could not be reached or the subject failed to return to the starting position.

Hydrostatic Weighing

Hydrostatic weighing was used to measure body density before (week 0) and after supplementation (week 10). Subjects reported to the underwater weighing tank in swimsuits. Body weight was first determined out of water by an electronic scale (model Toledo SL-39, Mettler Toledo Scales and Systems, Columbus, OH). Underwater weight was assessed by a force transducer (model 37–9.2, West Coast Research Co, Los Angeles, CA) connected by a transbridge (model TB-4, World Precision Instruments, Sarasota, FL), which was interfaced using Acknowledge software (version 3.0, BIOPAC System Inc, Goleta, CA) on a computer (Gateway 4DX2–66, Gateway, Inc, Sioux City, SD). Calibration was conducted daily by establishing linear interpolation from 2 known weights. Data points were recorded with data acquisition software, Acknowledge 3.0, from the force transducer. Subjects were submerged in warm water and asked to exhale a maximal amount of air while a signal from the force transducer produced a readable analog wave. The most stable waveform was selected, and the mean value was recorded. Subjects performed this procedure until at least 2 trials were within a 3% difference or a total of 10 trials was completed. If a 3% difference could not be obtained, mean values were calculated for the final underwater weight. Next, body density was calculated after weight was recorded in and out of water. The Siri equation was used to calculate percentage of body fat. Fat-free mass (FFM) was also calculated from the percentage of body fat.

Treatment of Data

Statistical analyses were performed using the Statistical Package for the Social Sciences for Windows software (version 8.0, SPSS, Inc, Chicago, IL). Our hypotheses were tested at an α level of .05. The 1-RM squat was measured before, during, and after supplementation. Urinary creatinine concentration was measured on days 0, 1, 3, 7, 14, 21, 28, 35, 42, 49, 56, and 63. Percentage of body fat was determined by hydrostatic weighing, and FFM was calculated before and after supplementation. All variables were analyzed between groups and over time by an analysis of variance with repeated measures. In addition, the Tukey pairwise comparison post hoc test for repeated measures was used for all data that showed significant differences.

RESULTS

Significant differences in absolute maximal strength were found over time with our periodized resistance training program ($P = .001$, $F_{1,22} = 13.52$); however, no group or interaction effects were noted ($P = .232$, $F_{2,22} = 1.50$) (Table 1). Each group showed significant increases: the low-dose group increased 19.03 kg, the loading group increased 8.80 kg, and the placebo group increased 8.83 kg (Figure 1). Using the post hoc pairwise comparison, we found that significant differences in absolute strength occurred before and during, during and after, and before and after supplementation.

When we analyzed urinary creatinine concentration output, we found a large amount of variation among subjects (Table 2). No statistically significant group, time, or interaction effects could be found ($P = .751$, $F_{22,242} = .778$). Initially, there was a nonsignificant decrease in urinary creatinine concentration at the beginning of supplementation for all groups (Figure 2). After initial ingestion, the low-dose and loading groups showed nonsignificant increases in creatinine concentration

Table 2. Urinary Creatinine Concentration (Mean ± SD)*

Day	Low Dose, μmol·L^{-1} (n = 8)	Loading, μmol·L^{-1} (n = 8)	Placebo, μmol·L^{-1} (n = 9)
0	1226.99 ± 284.65	884.00 ± 497.69	1179.26 ± 221.00
1	993.62 ± 622.34	704.55 ± 266.08	941.46 ± 393.38
3	1257.05 ± 436.70	1152.74 ± 631.18	1088.20 ± 371.28
7	1498.38 ± 293.49	1227.88 ± 680.68	1123.56 ± 568.41
14	1434.73 ± 586.98	1301.25 ± 792.06	1503.56 ± 656.81
21	990.96 ± 589.63	835.38 ± 544.54	1302.13 ± 746.98
28	1308.32 ± 696.59	1040.47 ± 536.59	1479.82 ± 406.64
35	1143.01 ± 577.25	1032.51 ± 633.83	1037.82 ± 360.67
42	1411.75 ± 701.01	1331.30 ± 574.60	1143.90 ± 525.98
49	1149.20 ± 477.36	1196.05 ± 500.34	1349.87 ± 699.24
56	962.68 ± 524.21	952.95 ± 623.22	1554.07 ± 762.01
63	1001.57 ± 226.30	1185.44 ± 794.72	1365.78 ± 594.05
Total mean concentration	1195.63 ± 187.55	1070.59 ± 195.36	1258.14 ± 198.90

*Low dose indicates 3 g of creatine supplementation without loading phase. High dose indicates 5 g of creatine supplementation with a 20 g·d^{-1}, 7-day loading phase.

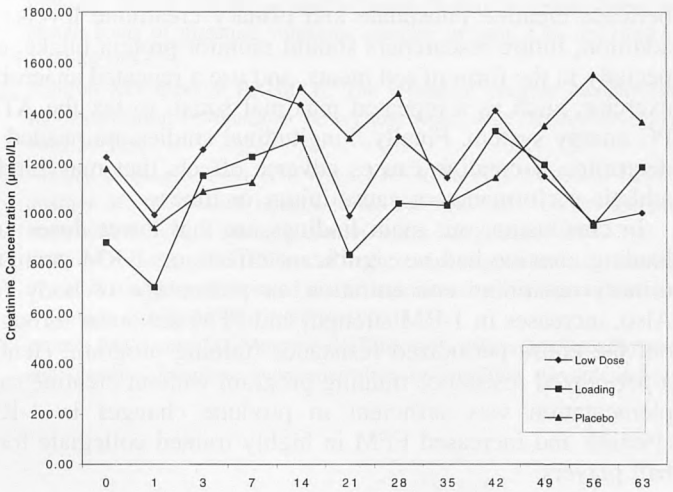

Figure 2. Group changes in urinary creatinine concentration throughout 10 weeks of creatine supplementation or placebo.

(0.034 μmol·L^{-1} and 0.057 μmol·L^{-1}) on day 3. The placebo's group urinary creatinine concentration actually decreased from week 0 (−1.66 mg·dL^{-1}) after day 3, but none of these values were statistically significant. At the end of the loading period, all groups continued to show similar urinary creatinine concentrations.

Percentage of body fat was analyzed by hydrostatic weighing. The group, time, and interaction effects showed no significant differences (P = .52, $F_{2,22}$ = .683) in hydrostatic weighing during 10 weeks of supplementation (Table 1). However, analysis of FFM showed significant differences over time (P = .037, $F_{1,22}$ = 4.93) but no group or interaction effects occurred (P = .211, $F_{2,22}$ = 1.67). The total change in FFM for the low-dose group was 2.46 kg, compared with 1.79 kg for the loading group and 1.16 kg for the placebo group.

DISCUSSION

Physiologically, creatine aids in recycling adenosine triphosphate (ATP) during short-term, high-intensity muscular work. Approximately 1 to 2 g of creatine is synthesized each day from the amino acids arginine, glycine, and methionine in the liver, kidneys, and pancreas.[14,23–25] Once synthesized, creatine must be transported by the blood to the tissues within the body for storage and utilization. Approximately 95% of creatine is found in skeletal muscle. Of this amount, 60% is in the form of creatine phosphate, with the remainder in the form of free creatine.[24] Some studies have indicated anabolic effects with short-term creatine loading; for example, 5 g 4 times a day may increase body mass, FFM, and strength.[16,17] Although these effects have been well documented in the laboratory, low doses and loading doses of creatine need to be compared.

Creatine loading has been found to increase creatine phosphate stores within skeletal muscle; thus, the possibility exists for enhancing performance, especially in short-term work bouts, and enhancing recovery between sessions.[2,6,25–30] Creatine loading is comparable with carbohydrate loading, in which the goal is to increase glycogen stores within the muscle during a week of high carbohydrate ingestion, However, it is possible that athletes are not just loading creatine for 1 week before competition but are ingesting high doses of creatine on a daily basis because of the proposed anabolic effects. Some research conducted on repeated anaerobic events has not shown performance enhancement,[7–9] although studies on strength training have shown significant increases in 1-RM strength.[8,16,19,31] We question whether or not creatine loading is necessary or even safe for athletes who are ingesting creatine habitually.

Our data suggest that creatine monohydrate use for 10 weeks did not produce any effects on 1-RM squat strength, urinary creatinine concentration, or body composition in collegiate football players. The 1-RM strength increased in all groups. Therefore, we believe that the periodized resistance training program was the cause of the strength increases. These results contradict the findings of other researchers of significant differences between creatine and placebo groups.[4,5,17] In addition, the changes occurred throughout the entire 10 weeks. We did observe that the low-dose group had the greatest change in 1-RM values, but it was not significantly different from the other 2 groups. In addition, all groups increased FFM throughout 10 weeks of training. This increase in FFM may have allowed subjects to perform at higher intensities, thus increasing overall strength because more actin and myosin were available for binding, which in turn may have allowed greater force production. This explanation is supported by other findings of similar increases in FFM and maximal 1-RM strength.[8,18] Furthermore, our subjects were highly trained athletes. We would expect this population to show minimal changes in strength compared with populations that are not involved in resistance training.

Creatinine formation is directly related to creatine phosphate concentration in skeletal muscle. Harris et al[22] showed that creatine saturation in muscle was associated with elevated levels of urinary creatinine. This result suggests that the muscle cannot phosphorylate excess free creatine; therefore, excessive supplementation yields elevated urinary creatinine. Other researchers have used muscle biopsy to show that creatine loading (20 g·d^{-1}) can increase creatine phosphate within muscle.[13,20,22] Moreover, a relationship exists between creatine loading and increased urinary creatinine concentrations.[19,20,22]

In our study, urinary creatinine concentrations were measured throughout 10 weeks, and large variations were seen

among subjects. These variations were also shown by Hultman et al,[20] who found in addition that creatinine degradation was directly proportional to muscle creatine phosphate content. We did not evaluate total creatine phosphate concentration within skeletal muscle but attempted to determine if creatine supplementation would increase levels of creatine phosphate within the muscle, as indicated by elevated urinary creatinine 4 hours after ingestion and exercise. We did not observe any group, time, or interaction effects. Some variation did occur during the loading phase, although the loading group did not show increased urinary creatinine concentrations compared with the low-dose and placebo groups. We expected that the loading group might have elevated urinary creatinine concentrations because this group was ingesting higher doses of creatine (5 g 4 times a day) compared with other groups. At the end of week 1, the low-dose and loading groups had similar creatinine concentrations (3.07 and 3.89 $mg \cdot dL^{-1}$) when compared with the placebo group. This result is consistent with the finding of Hultman et al[20] that lower doses of creatine produced similar effects to loading higher doses of creatine over time.

One possible explanation is that we did not collect 24-hour samples for analysis. It is possible that excess creatine was not excreted as creatinine within 4 hours of supplementation. It is also possible that creatine ingestion did not increase muscle creatine phosphate levels; we did not take biopsy specimens of the muscle and, therefore, were unable to accurately assess creatine phosphate levels. It is likely that our subjects were nonresponders, even though they had been free of creatine supplementation for 4 weeks before the study began. We did not monitor diet, and our subjects may have had previously elevated levels of creatine due to excessive protein ingestion. Finally, most of the creatine may have been converted to creatine phosphate and used during exercise. Our subjects were highly trained football players with large amounts of FFM, which may increase capacity for utilization of creatine phosphate. On the other hand, other tissues or organs (eg, liver or kidney) may be affected by elevated doses of creatine. Furthermore, hydration status may have an effect on creatinine concentration. We attempted to control hydration status by having the subjects drink only 300 mL of water during the 4 hours after creatine ingestion. We assumed that all subjects followed this procedure, but some subjects could have ingested more than 300 mL of fluid in the 4-hour window. In addition, it is very possible that the subjects' hydration status varied before workout sessions, which could have affected urinary creatinine concentration. These data suggest that creatine supplementation, with or without a loading phase, had no effect on urinary creatinine concentrations 4 hours after resistance training.

LIMITATIONS OF THE STUDY AND CONSIDERATIONS FOR FUTURE RESEARCH

We used a practical training design; therefore, our data differ slightly from other researchers' findings. We realize that our study does not support most of the published literature. Possible reasons that creatine supplementation did not cause significant effects are as follows. First, we did not take 24-hour urine samples to evaluate total urinary creatinine. Preexercise hydration status may have affected the urinary creatinine concentration 4 hours after exercise. Second, our creatinine data might have been more substantiated if we had taken muscle biopsy specimens to measure the current creatine levels of our subjects. Moreover, the possibilities of nonresponders and limited uptake in skeletal muscle are likely. Third, the subjects' diets were not monitored. Some of our subjects may have consumed more protein, which could have led to elevated creatine levels before supplementation. A high-protein diet may result in the ingestion of 3 or more $g \cdot d^{-1}$ of creatine, which could confound the effects of low-dose creatine supplementation. Fourth, the 1-RM squat exercise requires a single burst of energy that is fueled by the ATP-phosphocreatine (PC) energy system. Although significant strength gains did not occur among groups, the ingested creatine may still have had some effect in creatine phosphate resynthesis. To put further stress on the ATP-PC energy system, repeated anaerobic resistive exercises could be used to determine if creatine supplementation can affect creatine phosphate resynthesis.

Future studies should emphasize highly trained male and female athletes in controlled, sport-specific settings. Hydration status during exercise should be specifically addressed; 24-hour urine samples should be taken for more accurate measurement of creatine and creatinine levels; and muscle biopsy specimens should be taken to determine the actual relationship between creatine phosphate and urinary creatinine levels. In addition, future researchers should monitor protein intake, especially in the form of red meats, and use a repeated anaerobic exercise, such as a repeated maximal squat, to tax the ATP-PC energy system. Finally, longitudinal studies are needed to determine if creatine causes adverse effects that may hinder athletic performance or cause injury or illness.

In conclusion, our main findings are that lower doses and loading creatine had no significant effects on 1-RM strength, urinary creatinine concentration, or percentage of body fat. Also, increases in 1-RM strength and FFM did occur throughout the entire periodized resistance training program. Hence, a periodized resistance training program without creatine supplementation was sufficient to produce changes in 1-RM strength and increased FFM in highly trained collegiate football players.

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An Investigation of Undergraduate Athletic Training Students' Learning Styles and Program Admission Success

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Objective: The phrase *learning style* refers to the method one uses to obtain and use information to learn. Personal learning styles can be assessed by specifically designed inventories. We conducted this study to determine if undergraduate athletic training students possess a dominant learning style, according to the Kolb Learning Style Inventory IIA (KLSI IIA), the newest version of the Kolb Learning Style Inventory (KLSI), and whether this style is related to education program admission success.

Design and Setting: A 1 × 4 factorial design was used. The independent variable was learning style type with 4 levels (converger, diverger, assimilator, or accommodator). The dependent variable was successful versus unsuccessful admission into selected programs.

Subjects: Forty undergraduate students (21 men, 19 women) from 3 institutions (mean ± SD age, 20.7 ± 1.7 years; mean ± SD grade point average, 3.26 ± 0.43) participated in this study. No subjects had previously taken the KLSI IIA, and none had a diagnosed learning disability.

Measurements: The KLSI IIA was administered to the participants at their respective institutions. We used 2 separate χ^2 analyses to determine if the observed distribution of learning styles differed from the expected distribution. Additionally, a Mann-Whitney *U* test was performed to determine if the learning style distributions of those subjects who were successfully admitted to the selected programs differed from those who were not.

Results: No significant differences existed between the observed distribution and the expected distribution for those admitted and those not admitted ($\chi 2_3 = 3.8$, $P = .28$; and $\chi 2_3 = 3.1$, $P = .4$, respectively). Also, no significant differences existed between the learning style distributions of the groups when compared with each other (Mann-Whitney $U = 158$, $P = .5$).

Conclusions: Learning styles can be easily identified through the use of the KLSI IIA. We found no dominant learning style among undergraduate athletic training students and no particular learning style led to program admission.

Key Words: inventory, athletic training education program

People possess and use unique approaches to learn. These approaches are commonly referred to as learning styles. The method one specifically uses to obtain and then use information to learn is one's personal learning style.[1] Learning style inventories are commonly used to determine such styles.

The Kolb Learning Style Inventory (KLSI) is used extensively in learning style research.[1–17] The initial version of this questionnaire was created in 1976, and revisions were completed in 1985 and 1993.[3,6,10–12] This inventory is the most frequently used instrument for identifying learning styles.[18]

The KLSI classifies its respondents into 4 categories, which are representative of their dominant learning style.[3,6–9,12,13,17,19–22] These learning styles are termed *converger, diverger, assimilator,* and *accommodator*. The aforementioned styles are derived from the Kolb Experiential Learning Theory (KELT), and each style possesses distinct strengths and weaknesses with regard to learning experiences. Descriptions of each learning style are displayed in Table 1.

Learning style research in the field of athletic training has been limited, although its popularity has grown in recent years.[9,18,23] The paucity of research in this area prompts further investigation.

The effectiveness of athletic training education programs is critical to the profession. For athletic training students to eventually obtain employment, they must be skilled in a broad range of areas. Students use different methods to learn, and it is important for them and their instructors to recognize these styles. Instructors need to use the students' strengths and improve on their weaknesses to facilitate their total athletic training learning experience. The purpose of our study was to determine whether a specific learning style type among undergraduate athletic training students led to successful admission into athletic training education programs.

METHODS

A 1 × 4 factorial design was used in this study. The independent variable was the learning style type, as determined

Table 1. Learning Style Descriptions

Convergers	Divergers
Prefer: • Science-based fields[21,24] • To use hypothetical deductive reasoning[3,7,13,24] • To work with things rather than people[3,13,24] • To make their own decisions[6] • To problem solve in a practical manner[3,6] Described as being: • Technical[6] Tend to: • Be comfortable making decisions based on their understanding of a problem[7] • Do well on objective examinations[1] and conventional intelligence tests[21] • Have a narrow range of interests[13]	Prefer: • Arts and humanities[13,21] • Not to act until they have considered all options[7] • Open-ended questions[19] • To synthesize separate ideas into a whole[3] • To work with people over things[3,6,13,17,19,24] Described as being • Emotional[19,24] Tend to: • Be good at generating ideas[8,19] • Have active imaginations[3,8,13,24] • Have difficulty generalizing from one experience to another[7] • Have a broad range of interests[13,22,24]
Assimilators	**Accommodators**
Prefer: • Math and basic sciences[21] • Comparison and contrast–type questions[19] • Theoretical models and examples[3,8,13,17] • To work with abstract ideas and concepts rather than people[6,19,24] • To use inductive reasoning[3,7,13,17,24] Described as being: • Introverts[17] • Passive learners[17] Tend to: • Organize information[17] • Depend on others to give them facts[8] • Examine the soundness of theories and ideas[7,19,24] • Not be concerned with practical application of ideas or concepts[7]	Prefer: • Marketing and sales fields[21,24] • New experiences[3,8,13,24] • Action[6,7,13,24] and hands-on experiences[7] • Using trial-and-error methods and intuition to solve problems[3,6,13,17,24] • To have information given to them rather than collect it themselves[3] • To work with people over things[3] Described as being: • Pragmatic[6] • Active[3] Tend to: • Take risks[6,17,19,24] • Adapt well to situations[3,13,17,19] • Be good at carrying out plans made by others[3]

by KLSI version IIA (KLSI IIA), with 4 levels consisting of converger, diverger, assimilator, and accommodator. The dependent variable was successful versus unsuccessful admission into the professional component of the athletic training education programs.

Subjects

The subjects for this study were groups of undergraduate athletic training students enrolled in their last semester of preprofessional course work and observational clinical duties at 2 doctoral I institutions of higher learning and 1 junior college. Forty-seven volunteer subjects from the 3 institutions initially took part in the study.

The Commission on Accreditation of Allied Health Education Programs accredits the athletic training education programs at the 2 doctoral I institutions. The junior college does not offer an athletic training major. However, students are eligible to apply for selection to the athletic training education program at 1 of the 2 doctoral I institutions. The preprofessional classes completed at each institution are similar. Both programs require preprofessional students to take course work in emergency first aid, personal health, human anatomy, human physiology, athletic training practicum, and kinesiology. Clinical experiences in athletic training are also similar for these students. Observational hours are completed at each school to fulfill this requirement. The selection procedures at the universities also have some important similarities. Variables such as overall grade point average (GPA) and clinical evaluations of the students are used at each university. In addition, the acceptance-to-application ratio is comparable at both universities. The first university traditionally accepts 12 students per year of approximately 25, whereas the second university traditionally accepts 14 students per semester of approximately 30. These numbers vary because the number of applicants differs each selection cycle, but a 1:2 ratio is fairly consistent.

Despite the numerous similarities between the programs, some differences do exist. One university includes an interview and a faculty recommendation in its selection process, whereas the other university requires its applicants to take a proficiency examination consisting of written and oral/practical portions.

Some undergraduate students also choose not to pursue further athletic training education before the actual application and selection process. These students were not represented within this study, but it is important to remember that these students may self-select out of athletic training, and this decision may be related to their learning style.

No participants had been previously evaluated by the KLSI IIA, and none had a diagnosed learning disability. These criteria were determined through the use of an eligibility questionnaire. Before the study, the subjects were also requested to read and sign a GPA disclosure waiver form and an informed consent form that explained the purpose, risks, and benefits of the project. Approval was obtained from the Human Subjects' Research Committee.

Forty subjects were included in the final calculations for this study. Six participants completed the necessary paperwork required for the study and then withdrew their applications before selection procedures were complete. One participant was disqualified from the study because he had previously been evaluated using the KLSI IIA.

Instrumentation

The KLSI IIA was used to determine the learning styles of the subjects. The purpose of this inventory is to categorize respondents as convergers, divergers, assimilators, or accommodators based on their answers to a self-reported, 12-item questionnaire.[5,6,8–12,24] Each question begins with "When I learn. . . ," "I learn best when. . . ," "When I am learning. . . , I learn by. . . ," or "I learn best from. . . ," and 4 options for completing the sentence are supplied. The respondent ranks the 4 options, with 1 correlating with the respondent's least dominant learning style and 4 correlating with the respondent's most dominant learning style.[5–7,12,13,15,24] No ties should be made, and each question should contain 4 answers.[25] At this point, the respondent's duties were complete.

Hay/McBer, the distributor of the copyrighted inventory, supplied scoring directions for the questionnaire. Each answer

slot within the questionnaire corresponds with a step in the KELT. The steps are termed *concrete experience* (CE), *reflective observation* (RO), *abstract conceptualization* (AC), and *active experimentation* (AE).[3,4,6–8,10–12] Respondents placed the numbers 1, 2, 3, or 4 in the answer slots. Totals for CE, RO, AC, and AE were determined by adding the numbers in each answer slot by category. Next, the RO score was subtracted from the AE score, and the CE score was subtracted from the AC score.[26] These 2 remaining scores were then plotted on a grid consisting of 2 axes and 4 quadrants. The quadrants represent the 4 learning styles of converger, diverger, assimilator, and accommodator. The location of the scores plotted on the grid indicate the respondent's dominant learning style according to the KLSI IIA.[26]

Changes to the KLSI were completed in 1985, and the revised version was termed the *KLSI II*.[6] Further revisions of the KLSI II were recently completed to improve the instrument.[10,12,25] This newest version is referred to as the *KLSI IIA*.[9] Internal reliability of the inventory has been evaluated using the Cronbach α. The 4 basic scales (CE, RO, AC, and AE) and the 2 combination scores (AC-CE and AE-RO) of the KLSI IIA show very good internal reliability as measured by the Cronbach α: CE, .82; RO, .73; AC, .83; AE, .78; AC-CE, .88; and AE-RO, .81.[24,25] Internal consistency measurements of the KLSI II and KLSI IIA have also been completed using mean coefficient α values. These indexes were expected to decrease with the improvements made to the KLSI II. Response bias due to the consistent order of sentence endings in the KLSI I and KLSI II was reduced by scrambling the order of sentence endings of KLSI IIA. Based on these changes, the indexes were expected to decrease due to the anticipated elimination of response bias. Mean coefficient α values for the KLSI II ranged from .82 to .85, and mean coefficient α values for the KLSI IIA ranged from .52 to .78. Despite the decreases found, the values were still considered adequate in terms of internal consistency of the tool.[10] Additionally, test-retest reliability has been investigated. This measurement was expected to increase, and test-retest reliabilities for the 4 scales of the KLSI IIA across multiple administrations were very high in comparison with the KLSI II.[10] These values ranged from .92 to .99 for the KLSI IIA and .25 to .56 for the KLSI II.[10]

The validity of the instrument has not been as extensively investigated as its reliability. Construct validity of the KLSI and KLSI II has been examined, but the KLSI IIA has not been investigated with regard to validity.[11,13–16] Construct validity is determined by examining whether the inventory measures what it purports to measure as described in the KELT.[14] Factor analysis of the KLSI revealed poor word choices in descriptions pertaining to the CE category.[11] Also, results of the KLSI were not related to career choices or personality characteristics.[13,14] On the other hand, Merritt and Marshall's[15,16] comparisons of an alternate form of the inventory to the normative version revealed support for validity of the instrument based on the KELT. Despite these findings, the validity of the KLSI and KLSI II is questionable, but the inventory is considered reliable and is used a great deal in the determination and assessment of learning styles in many settings.[4,5,7,10,15,16,18,27]

Testing Procedures

Volunteer subjects reported to a specified classroom at their respective institutions on a predetermined testing day. Before testing began, the volunteers received a verbal explanation of the study, including a description of its purpose, risks, and benefits. Each subject then filled out an eligibility questionnaire and read and signed a disclosure waiver form allowing us to obtain the subject's current GPA from either the program curriculum director or the registrar's office of each institution. The volunteers also read and signed an informed consent form.

Subjects then completed the KLSI IIA. We read the directions supplied with the inventory to the subjects and answered pertinent questions at this time. In addition, 2 further instructions were given. First, the subjects were told there were no right or wrong answers to the questions. Each of the learning styles described is considered valuable, and the true purpose of the inventory is to help assess personal skills related to learning. Second, to ensure correct scoring, the subjects were instructed to rank the 4 sentence endings for each question and to not create ties.

Once completed, the inventories were collected to be scored at a later time. The information from the questionnaires and inventories was entered into a data collection form to maintain anonymity of the subjects. Learning style type results for each subject were retained until admission procedures for the athletic training education programs were completed. Then the learning style distributions of the successful and unsuccessful candidates were examined and compared with expected distributions and each other.

Statistical Analysis

We used a χ^2 test to determine whether the observed learning style types of those subjects successfully admitted to the athletic training programs were similar to the expected distribution of learning style types. The expected distribution was determined to be 25% convergers, 25% divergers, 25% assimilators, and 25% accommodators. A χ^2 test was also used to determine if the observed learning style types of those subjects not admitted to the athletic training programs were similar to the expected distribution of learning style types. Once again, the expected distribution was set equally. Using a Mann-Whitney *U* test, the learning style type distributions of the unsuccessful candidates were also compared with those candidates who gained successful admission to the selected athletic training programs. The probability level was set at $P \leq .05$ for all tests.

RESULTS

We found no difference between the learning style distribution of the subjects who were successfully admitted to the selected athletic training programs and the expected distribution ($\chi^2_3 = 3.8$, $P = .3$) (Figure 1). There was no difference between the learning style distribution of the subjects who were not admitted to the selected athletic training programs and the expected distribution ($\chi^2_3 = 3.1$, $P = .4$) (Figure 2). Additionally, no differences were found between the learning style distributions of those subjects who were admitted to the selected athletic training programs and those who were not when compared with each other (Mann-Whitney $U = 158$, $P = .5$). The mean ± SD GPA for all subjects was 3.26 ± 0.43, and the mean ± SD GPAs for those subjects admitted to programs (n = 27) and those subjects not admitted to programs (n = 13) were 3.50 ± 0.31 and 2.82 ± 0.20, respectively.

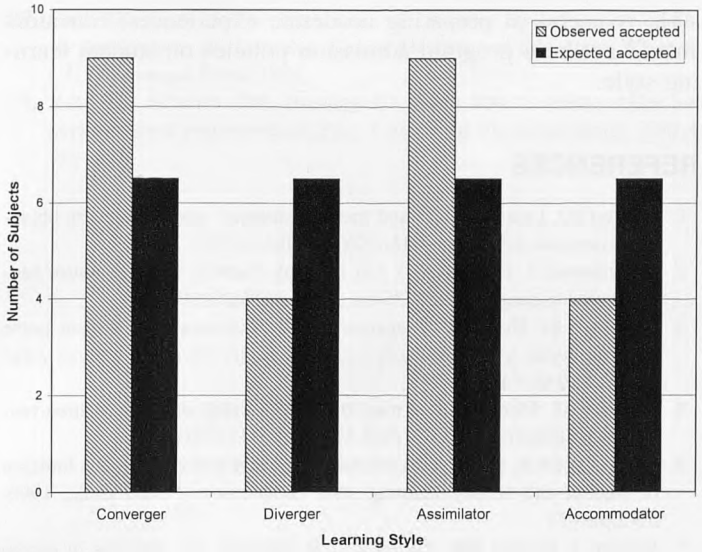

Figure 1. Observed and expected learning styles of subjects admitted to programs.

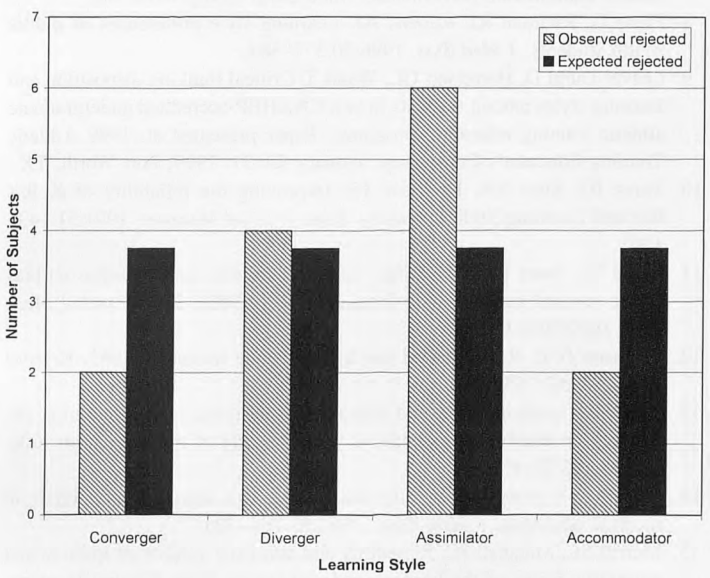

Figure 2. Observed and expected learning styles of subjects not admitted to programs.

DISCUSSION

Only one previous study, to our knowledge, has investigated the learning styles of undergraduate athletic training students. Recently, Leaver-Dunn et al[9] administered the KLSI IIA to 70 undergraduate athletic training students. They found that these students were mostly assimilators (38%), followed by accommodators (21%), convergers (21%), and divergers (20%). In comparison, the subjects involved in our study were mostly assimilators (37.5%), followed by convergers (27.5%), divergers (20%), and accommodators (15%). In both studies, most of the students were classified as assimilators, but the other categories were also represented. The results of our study revealed that no certain learning style among undergraduate athletic training students led to program admission.

The above-mentioned findings are unusual when compared with other fields of study. Research in other areas has shown dominant learning styles among nursing, physical therapy, medical, physician assistant, and medical technology students,[2,4,5,7,22,28] whereas the results of our study revealed that athletic training students had no dominant learning style. The results of other studies that have used the KLSI in some form are described in Table 2. Learning style researchers in medical technology, physical therapy, and physician assistant programs did not assess student learning styles using the KLSI.[22,28] However, dominant learning styles with characteristics similar to those described by Kolb were found. In medical technology and physical therapy, students (n = 100) preferred learning through concrete methods.[22] This is similar to the Kolb classifications of accommodator and diverger. Students (n = 42) in physician assistant programs preferred hands-on, step-by-step experiences.[28] This type of learning is consistent with the Kolb learning styles of assimilator and accommodator.

No previous researchers have investigated the relationship between learning styles and education program admission success. However, some have examined other measures of academic performance, such as overall GPA and examination scores.[1,6,8,23,29] The results of our study showed that no certain learning style was related to program admission, but learning style has affected other measures of academic performance in other fields. For example, nursing students categorized as assimilators have earned higher GPAs than their counterparts in other learning style classifications.[6] Also, medical students classified as convergers have performed better on objective examinations than divergers, assimilators, and accommodators.[1] In public health education, assimilators have scored better on written examinations in comparison with the 3 other learning style group members.[8] Lastly, in athletic training, academic variables rather than learning styles were considered predictors of success for students who took the National Athletic Trainers' Association Board of Certification examination.[23] This finding was verified again by Harrelson et al.[29] Therefore, it seems that, in terms of academic performance, learning styles of athletic training students have a limited ef-

Table 2. Learning Style Findings in Health Care–Related Fields

Field	Author	Sample Size	Learning Style	Percentage
Nursing	Jambunathan[2]	123	Assimilator	NA*
			Accommodator	
Nursing	Rakoczy and Money[4]	138	Assimilator	NA
Nursing	Joyce-Nagata[5]	334	Assimilator	42
			Accommodator	24
Medicine	Lynch et al[7]	227	Converger	45
			Assimilator	26
Athletic training	Leaver-Dunn et al[9]	70	Assimilator	38
Athletic training	Brower et al	40	Assimilator	37.5

*NA indicates not applicable since percentages were not supplied in all studies.

fect on academic success in comparison with students in other fields. Academic factors, on the other hand, seem to have a substantial effect on academic performance among undergraduate athletic training students, especially in terms of success on the certification examination.

Since academic variables seem to be important components in athletic training education, the mean GPAs of the subjects involved in this study were analyzed and comparisons were made by institution and admission status. The mean ± SD GPAs for subjects from each university were 3.22 ± 0.47, 3.11 ± 0.31, and 3.33 ± 0.41. The mean ± GPA for all subjects was 3.26 ± 0.43. Those subjects who gained successful admission to the selected programs possessed higher GPAs than those who did not. The mean ± GPA for those subjects admitted to programs was 3.50 ± 0.31, and for those not admitted to programs, 2.82 ± 0.20. In this study, GPA seemed to be related to admission success.

The subjects involved in this study consisted of 40 undergraduate athletic training students applying for admission to 2 selected athletic training programs. With regard to the large numbers of undergraduate curriculum athletic training programs, this subject pool was rather small. Our intent was to evaluate a significant number of athletic training students from similar programs to complete this study. Despite using a multisite approach, the total number of subjects was small. Also, these students were intended to represent typical undergraduate athletic training students, but this notion would be difficult to prove or disprove based on the small scope of this study. However, the results of this study have revealed a trend showing that these particular students do not have a dominant learning style. In addition, the learning styles of these individuals did not lead to successful admission into selected athletic training programs.

EDUCATIONAL APPLICATIONS

The findings of this study are helpful to both educators and students in demonstrating the importance of learning style identification. Most researchers agree that knowledge of one's personal learning style is advantageous.[6,17,19,28,30] The participants in this study were given the opportunity to request the results of their personal inventory. In addition, a brief explanation of the 4 learning styles, including strengths, weaknesses, and preferences of each style, was provided for the subjects. This explanation could prove helpful for some participants. Knowledge of one's learning style may be beneficial in that the participant will now be aware of his or her strengths and weaknesses in terms of learning experiences. Therefore, future learning may be enriched if the participants maintain their strengths and improve on their weaknesses.

Educators can also take part in this awareness of learning styles. By becoming knowledgeable about learning styles and assessing the learning styles of their students, educators can facilitate appropriate learning experiences based on these findings. Haislett et al[6] suggested providing a learning style assessment using an instrument such as the KLSI early in all students' academic experiences. In this way, students are sensitized to the fundamental strengths and weaknesses of their particular style, and they can use this information to improve their overall educational experiences.

Our study revealed no dominant learning style among undergraduate athletic training students admitted to athletic training education programs. Although knowledge about learning style is useful in preparing academic experiences, educators should not base program admission policies on student learning style.

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Predicting Performance on the National Athletic Trainers' Association Board of Certification Examination From Grade Point Average and Number of Clinical Hours

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Objective: To determine whether grade point average, hours of clinical education, or both are significant predictors of performance on the National Athletic Trainers' Association Board of Certification examination and whether curriculum and internship candidates' scores on the certification examination can be differentially predicted.

Design and Setting: Data collection forms and consent forms were mailed to the subjects to collect data for predictor variables. Subject scores on the certification examination were obtained from Columbia Assessment Services.

Subjects: A total of 270 first-time candidates for the April and June 1998 certification examinations.

Measurements: Grade point average, number of clinical hours completed, sex, route to certification eligibility (curriculum or internship), scores on each section of the certification examination, and pass/fail criteria for each section.

Results: We found no significant difference between the scores of men and women on any section of the examination. Scores for curriculum and internship candidates differed significantly on the written and practical sections of the examination but not on the simulation section. Grade point average was a significant predictor of scores on each section of the examination and the examination as a whole. Clinical hours completed did not add a significant increment for any section but did add a significant increment for the examination overall. Although no significant difference was noted between curriculum and internship candidates in predicting scores on sections of the examination, a significant difference by route was found in predicting whether candidates would pass the examination as a whole ($P = .047$). Proportion of variance accounted for was less than $R^2 = 0.0723$ for any section of the examination and $R^2 = 0.057$ for the examination as a whole.

Conclusions: Potential predictors of performance on the certification examination can be useful to athletic training educators in assisting students. These findings must be used cautiously because of the low proportion of explained variance. Low R^2 values suggest that the largest contributors to performance on the examination were not identified in this study. Although the results of this study support the decision to discontinue the internship route to certification, continued research focusing on identification and investigation of the constructs that contribute to examination success is needed.

Key Words: athletic training certification, certification examinations, credentialing examinations, athletic training education

The performance by route of curriculum and internship candidates on the National Athletic Trainers' Association Board of Certification (NATABOC) examination has been consistently reported in the literature. Candidates from the curriculum route to examination eligibility attain consistently higher scores on all 3 sections of the examination than internship-route candidates.[1–7]

This difference in the performance of curriculum and internship candidates on the certification examination raises questions for athletic training educators regarding the students' preparation for the examination. The ability to predict whether a student is likely to pass the certification examination by knowing academic performance as measured by grade point average (GPA) and the number of hours of clinical education completed is of interest to athletic training educators. To our knowledge, only one study[8] in athletic training investigated the ability to predict examination performance from performance in didactic learning and quantity of clinical education. This study only used subjects from a single institution. It is, therefore, important to expand the investigation to study candidates nationwide to determine whether the results are consistent.

The purposes of our study were to determine (1) whether a statistically significant relationship exists among GPA, quan-

tity of clinical education, and performance on any of the sections of the NATABOC examination; (2) whether GPA, the number of hours of clinical education completed, or both are significant predictors of performance of candidates on the NATABOC examination; and (3) whether the ability to predict performance on the examination for candidates who became eligible for the examination via the curriculum versus the internship route is statistically significant.

METHODS

Subjects

Data collection packets were mailed to 1360 first-time candidates for the April and June 1998 administrations of the NATABOC examination. The packets contained a data form and consent forms for study participation and the release of their examination results from Columbia Assessment Services.

Data and consent forms were received from 270 candidates: 117 men (43.33%) and 153 women (56.67%). Of these subjects, 143 (52.96%) were curriculum candidates and 127 (47.04%) were internship candidates. The study and consent forms were approved by the appropriate institutional review board for the protection of human subjects.

Data Collection Procedure

Each subject completed a data form that asked about the following variables: sex, overall GPA at the time of application for the certification examination, number of hours of clinical education completed at the time of application for the examination, and whether the candidate became eligible for the certification examination via the curriculum or internship route. The data forms were to be returned before the certification examination was administered.

Columbia Assessment Services provided quantitative scores on each section of the certification examination, including the written (written), practical (prac), and written simulation (simul) scores and pass/fail status on each section of the examination for each candidate in the study. Candidates who received a score of pass on all 3 sections of the examination also received a score of pass for the entire examination. Candidates not passing all 3 sections of the examination received a score of fail for the examination as a whole (pfall).

Statistical Analysis

We performed a 2-way analysis of variance to determine whether a significant difference existed between the means of the scores for the candidates by route to eligibility and by sex of the candidate. We also examined whether a significant interaction existed between the 2 variables. Because the variable for passing the examination as a whole (pfall) is only a dichotomous variable, we conducted a χ^2 analysis to determine whether curriculum and internship candidates passed the certification examination at significantly different rates.

Correlation coefficients were calculated to assess the degree of relationship among the variables in the study. Correlation coefficients among predictor variables were examined to assess the possibility of collinearity among the predictor variables.

We used multiple regression analysis to determine the ability to predict the outcome on each section of the examination and the examination as a whole from the predictor variables of GPA and clinical hours completed. Stepwise linear regression analysis was used to examine the ability to predict the quantitative score on each section of the certification examination from GPA and number of hours of clinical education completed. We examined the ability to predict whether the candidate received a score of pass or fail on the entire examination from GPA, number of clinical hours completed, or both using stepwise logistic regression analysis. This was done because the examination results are reported to the candidates as a dichotomous variable.

To determine whether the regression coefficients were significantly different among the groups in the regression analysis, the candidates were effect coded. We calculated an interaction vector consisting of the product of the value of the significant independent variables and the effect code. The variables GPA, clinical hours, effect code, and the interaction variable were entered sequentially into the appropriate equations. The significance of the regression coefficient for the interaction vector was used to determine whether a significant interaction existed between the independent variable(s) and route to examination eligibility. This test is equivalent to a test of the difference between the regression coefficients for the 2 groups.[9] Analyses were performed using the Statistical Package for the Social Sciences for Windows (version 6.0, SPSS Inc, Chicago, IL). For all analyses, the α level was set at $P < .05$.

Table 1. Descriptive Statistics by Route to Examination Eligibility

Variable	Mean (SD)
Grade point average	
Curriculum	3.375 (0.290)
Internship	3.306 (0.360)
Total	3.343 (0.326)
Hours	
Curriculum	1360.320 (366.020)
Internship	1767.940 (467.940)
Total	1551.953 (463.474)
Written score	
Curriculum	112.804 (10.204)
Internship	109.365 (11.165)
Total	111.137 (10.803)
Practical score	
Curriculum	37.091 (5.234)
Internship	35.500 (6.610)
Total	36.285 (5.881)
Simulation score	
Curriculum	533.042 (90.960)
Internship	531.865 (99.181)
Total	531.963 (94.943)

RESULTS

Descriptive statistics were calculated for each variable (Table 1).

The analysis of variance results indicated that the interaction between route to examination eligibility and sex of the candidate was not significant for all 3 sections of the examination ($P \geq .46$). The mean values for the written ($F_{3,266} = 7.05$, $P = .008$) and practical sections ($F_{3,266} = 6.09$, $P = .01$) of the examination showed a significant difference between curriculum and internship candidates. Neither the difference between the means of the scores on the written simulation section

Table 2. Correlation Coefficients Among Study Variables

	Grade Point Average	Hours	Sex	Written Score	Practical Score	Simulation Score	pfall*
Grade point average	1.000						
Hours	−0.072	1.000					
	(P = .24)						
Sex	0.097	−0.041	1.000				
	(P = .11)	(P = .50)					
Written score	0.404	0.005	−0.097	1.000			
	(P = .00)	(P = .93)	(P = .11)				
Practical score	0.266	0.049	−0.101	0.519	1.000		
	(P = .00)	(P = .43)	(P = .10)	(P = .00)			
Simulation score	0.267	0.090	0.101	0.532	0.364	1.000	
	(P = .00)	(P = .14)	(P = .10)	(P = .00)	(P = .00)		
pfall	0.203	0.109	−0.023)	0.553	0.516	0.597	1.000
	(P = .001)	(P = .07)	(P = .71)	(P = .00)	(P = .00)	(P = .00)	

*pfall reflects passing score on entire examination for candidates passing all 3 sections and failing score on entire examination for candidates not passing all 3 sections.

Table 3. Linear Regression Analysis

	Written	Oral Practical	Simulation
Grade point average			
b (SE)	13.370 (1.851)	4.794 (1.062)	77.585 (17.140)
t value	7.226	4.515	4.527
P value	0.000	0.000	0.000
R^2	0.163	0.071	0.071
Hours			
b	0.034	0.068	0.110
t value	0.614	1.155	1.870
P value	0.540	0.249	0.063
Incremental R^2	0.000	0.000	0.000

Table 4. Regression Coefficients for Comparison of Regressions

	Written	Practical	Simulated
b (SE)	2.357 (1.869)	1.676 (1.072)	18.420 (17.473)
P value	0.208	0.119	0.293

by route to eligibility ($F_{3,266}$ = .04, P = .85) nor the means of the scores of men and women on any section of the examination ($P > .05$) were statistically significant.

χ^2 analysis indicated that the difference in pass rate by route for the examination as a whole was not significant (P = .25). There was also no significant difference by sex of the candidate ($P > .05$).

The correlation coefficients among the independent variables of GPA, hours, and sex were not statistically significant ($P > .05$) (Table 2), but the correlation coefficients between GPA and the dependent variables written, prac, simul, and pfall were all statistically significant ($P < .05$). No significant correlation coefficients existed between the predictor variable clinical hours and any of the dependent variables, written, prac, simul, or pfall ($P \geq .07$). The correlations between sex and the variables written, prac, and simul were not significant ($P \geq .10$).

We used stepwise linear regression of the quantitative variables to determine if scores for each section of the examination could be predicted (Table 3). For each of the dependent variables (ie, written, prac, and simul), the regression coefficient for GPA was significant ($P \leq .05$). The regression coefficients for the quantitative dependent variables (ie, written, prac, and simul) were not significant ($P \geq .06$). Because there is no quantitative variable for passing the entire certification examination, we did not perform linear regression for this dependent variable.

To examine the ability to predict whether a candidate will pass the entire examination, pfall was regressed on GPA and clinical hours using logistic regression. The regression coefficient for GPA was significant (P = .001), and the coefficient for hours provided a significant incremental prediction of pfall (P = .04). The proportion of variance explained for the logistic regression model was 0.58 (R = 0.0762).

The regression coefficients for the interaction vectors between the effect code and each of the variables (ie, written, prac, and simul) were not significant ($P \geq .12$). These results indicated no significant difference between the regression coefficients for curriculum and internship candidates. We can infer from these results that no difference exists in the ability to predict examination scores for candidates who became eligible for the examination by different routes (Table 4).

The regression coefficient for the interaction vector among GPA, clinical hours, and effect code for the dependent variable pfall was significant ($\beta = 1.34 \times 10^{-5}$, P = .03). We can infer from these results a statistically significant difference in the ability to predict whether a curriculum or internship candidate will pass the entire examination from GPA and number of clinical hours completed.

DISCUSSION

Our examination of the data and the tests of differences in the means of examination scores suggests that curriculum candidates scored significantly higher than internship candidates on the written and practical sections of the examination, consistent with the previously reported literature.[1–7] Candidates from approved or accredited athletic training curriculums may have been better prepared for these sections of the certification examination than the internship candidates. Turocy et al[7] addressed potential reasons for the difference in scores, proposing that the more stringent didactic requirements of curriculum programs provide a wider knowledge base from which curriculum students can draw to enhance their clinical learning experiences.

The difference between the scores on the written simulation section of the examination for this sample was not statistically

significant, a result inconsistent with that reported in the literature. We have not found a specific reason for this departure from the pattern demonstrated in the past. However, potential explanations include bias in the sample due to subjects' self-selection in volunteering for participation in the study or the sample's inclusion of only 20% of the total pool of first-time candidates for the selected examination administration dates. The lack of significant difference in the simulation scores may also be explained by the type of questions used in this section of the examination and their relationship to the type of educational experience received by the candidates.

We found statistically significant positive correlations between GPA and scores on all 3 sections of the NATABOC examination and between GPA and whether a student passed the entire certification examination. These results imply that students who received higher grades were more likely to pass the certification examination than those who had a lower GPA.

We used the number of hours of clinical education as a predictor because currently no universally accepted measure of the quality of clinical education exists. Our analysis of the quantity of clinical education as measured by hours completed during the education of the candidates revealed no significant relationship between the number of hours accumulated and performance on any section of the examination. Draper[10] concluded that accumulating a high number of hours of clinical education does not necessarily result in passing the certification examination. Although he compared the number of clinical hours of internship and curriculum candidates in their preparation for the examination, he did not look at the combination of quantity of clinical education and academic performance as predictors of certification examination performance, as we did. We examined the ability to predict scores on the certification examination from both GPA and number of clinical hours completed.

The lack of a significant relationship between quantity of clinical education and performance on any section of the examination supports the current recommendations of the NATA Education Council to eliminate a count of the number of clinical hours as the sole criterion for examination eligibility based on the clinical portion of a candidate's preparation and replace it with a competency-based measure of student clinical education. This finding suggests that the nature of the students' clinical education differs among programs.

Changes in clinical education that move toward competency-based criteria are also supported by the lack of a significant correlation between GPA and number of hours of clinical education. The need to identify appropriate factors contributing to candidate performance on the certification examination is also supported. The lack of significant prediction of examination scores from the number of clinical hours completed and the low amount of the total variance accounted for by the data suggest factors contributing to examination performance that have not been identified.

Athletic training educators may find the ability to predict a candidate's performance on the certification examination useful in advising students during their professional preparation. An indication of the types of factors that are significant predictors of examination scores would assist educators in identifying students' areas of strength and weakness, allowing for early intervention. Therefore, a student with a low GPA could be directed to support services and resources that may improve the student's academic profile and, hence, the chances of success on the examination.

We regressed scores on the sections of the NATABOC examination on GPA and number of hours of clinical education completed by each subject. The regression coefficients of the curriculum and internship candidates on all sections of the examination were not significantly different from those of the internship candidates ($P > .05$). Lack of a significant difference in the regression coefficients implies no significant difference in the ability to predict performance on the individual sections of the examination, given the predictors of GPA and number of hours of clinical education completed.

The statistically significant difference in the regression coefficients of curriculum and internship candidates for the variable pfall implies a significant difference in the ability to predict whether a candidate will pass the entire certification examination based on the route to examination eligibility. On initial examination, the athletic training educator may be encouraged by these results, which may provide information to help predict a student's potential for success on the certification examination. Our examination of the proportion of variance accounted for in the regression analyses suggests that use of these results to predict a student's potential for success should be tempered. The proportion of variance accounted for in the prediction of the scores on the written ($R^2 = 0.16$), practical ($R^2 = 0.07$), and simulation ($R^2 = 0.07$) sections is very low. The proportion of variance accounted for in prediction of passing the entire examination ($R^2 = 0.057$) is also low. The low proportions of variance for the regression models imply that many other potential factors may contribute to a candidate's scores on the NATABOC examination.

The regression analyses in this study are consistent with those of studies in athletic training[7] and other allied health professions.[10–17] In general, GPA is a statistically significant predictor of performance on credentialing examinations. This may be due to the fact that most credentialing examinations are written, and they may tend to focus on material and testing methods that emphasize knowledge and understanding of the concepts normally developed in the classroom setting.

LIMITATIONS OF THE STUDY

Generalizability of our results may be influenced by the fact that subjects were self-selected for participation in the study by voluntarily responding to a request from the researchers. The results may also be affected by the return rate of 20%. The data should also be interpreted in light of the fact that this was a 1-time sampling of candidates from 2 examination administration dates in the same calendar year.

Using this study's findings to make decisions about how a student might score on the certification examination must be done cautiously because the proportion of variance accounted for was between 7.1% and 16.3% of the variance in the raw scores of the data and was 5.7% of the variance in the prediction of success on the examination as a whole. The relatively low proportion of variance accounted for also suggests that the factors accounting for success on the certification examination have not yet been identified. When evaluating the statistical significance of the correlation coefficients, we must also remember that the values fall in the low-to-moderate range.

CONCLUSIONS

Although potential predictors of examination performance can help athletic training educators advise their students, it

remains important to identify those factors that contribute significantly to that performance. No researchers have yet objectively identified the factors that contribute to student success on practical examinations. Research attempting to relate criterion-based performance to clinical education often uses subjective instructor assessments as a predictor. Additional research is required to identify factors that can be used to help predict candidates' performance on the certification examination. Issues relating to competency-based clinical education and specific didactic requirements for athletic training education need to be investigated further to determine those factors that more directly affect the candidates' outcome on the certification examination.

The development of a universal method to assess the content and quality of athletic training clinical education based on factors that contribute to examination scores would allow athletic training educators to assess students' professional preparation. Future research should focus on identification and investigation of the constructs that contribute to success on the NATABOC examination and appropriate methods for assessing student achievement within those constructs.

The evidence from this study against quantity of clinical education as a predictor of examination performance supports the efforts of the NATA Education Council to investigate competency-based clinical education. The process of revising the guidelines for athletic training education has already begun. Athletic training educators continue to strive for information to help them better prepare students for the certification examination. The identification of GPA as a significant predictor of performance on all sections of the examination is a first step toward identifying the factors that contribute to the success of candidates on the examination. More research is needed to identify content and psychometric factors that affect candidate performance on the examination.

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Preadmission Criteria as Predictors of Academic Success in Entry-Level Athletic Training and Other Allied Health Educational Programs

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Objective: To investigate preadmission criteria, Scholastic Aptitude Test (SAT) scores, and high school grade point average (HSGPA) and to determine the ability of those criteria to predict the college grade point average (CGPA) of graduates from programs in athletic training and 5 other allied health disciplines.

Design and Setting: Descriptive data, including age, sex, year of graduation, HSGPA, CGPA, and SAT scores (SAT mathematics [SATM], SAT verbal [SATV], and SAT total) were gathered from the files of graduates (1992 to 1997) of allied health education programs.

Subjects: The complete records of 373 graduates (244 women and 129 men) of 6 allied health education programs in athletic training, health management systems, occupational therapy, perfusion technology, physician assistant, and physical therapy were used in this study. Subjects with incomplete files were excluded from this study.

Measurements: We collected data from official college transcripts, official high school transcripts, and SAT scores reported to the university. Descriptive statistics, analysis of variance, Pearson correlation, and stepwise forward regression analyses were used to determine the ability of SATV score, SATM score, and HSGPA to predict CGPA.

Results: Both SATV score and HSGPA were found to predict 14% of the variance in student success (CGPA) in all allied health programs; however, only HSGPA was predictive of student success in athletic training ($P = .00$). Both SATV score and HSGPA were predictive of CGPA in both physical ($P = .02$ and .03, respectively) and occupational ($P = .02$ and .00, respectively) therapy graduates; however, they predicted only 12% and 21%, respectively, of the variance in CGPA. The SATM score was predictive of CGPA in both perfusion technology ($P = .05$) and physician assistant ($P = .00$) graduates, accounting for 7% and 18% of the variance in outcomes.

Conclusions: Overall, HSGPA and SATV score were predictive of student success (CGPA) in the allied health group as a whole and should continue to be used as part of the process of admission in higher education until such time that more predictive criteria are determined. The HSGPA was predictive of CGPA ($r^2 = 0.38$) in athletic training graduates, predicting more than one third of the professional program success. Athletic training educators should continue to use HSGPA as a predictor of success; however, they may wish to establish similar baseline comparisons between athletic training and other disciplines at their schools before determining the importance of standardized preadmission criteria for future students. This work should be considered a pilot study for the profession of athletic training and should be repeated at individual schools to determine the findings' applicability to other athletic training education programs.

Key Words: predictors of academic success, admission criteria, grade point average, Scholastic Aptitude Test

Education reform is one of the most important issues affecting the profession of athletic training today. The profession is responding to the 18 recommendations made by the National Athletic Trainers' Association (NATA) Education Task Force in 1997. Provision 12 of those recommendations encouraged "Athletic training education programs to consider aligning themselves in colleges of health-related professions."[1] If athletic training students are to be housed in schools with other allied health disciplines, they also may be required to adhere to and be evaluated by the same standards as are those other allied health professional students. Therefore, it is important to begin to prepare for this transition by comparing the academic preparation and outcomes of athletic training graduates with graduates of other allied health education programs. The purpose of our study was to compare the preadmission criteria of scores on the Scholastic Aptitude Test (SAT) and high school grade point average (HSGPA) among disciplines. We also examined the ability of those criteria to predict the professional grade point averages (GPAs) of the graduates from programs in athletic training, health management systems, occupational therapy, perfusion technology, physician assistant, and physical therapy. Professional

Table 1. Descriptive Data (Mean ± SD)

	Athletic Training	Health Management Systems	Occupational Therapy	Perfusion Technology	Physician Assistant	Physical Therapy
Age, y	23.22 ± 2.14	25.00 ± 3.61	24.83 ± 3.12	25.26 ± 4.21	27.55 ± 5.15	25.00 ± 3.18
High school grade point average	3.23 ± 0.47	3.22 ± 0.52	3.23 ± 0.52	3.29 ± 0.47	3.25 ± 0.59	3.89 ± 0.15
College grade point average	3.34 ± 0.28	3.19 ± 0.48	3.43 ± 0.27	3.38 ± 0.30	3.57 ± 0.33	3.58 ± 0.28
SAT mathematics score	504 ± 66.70	487 ± 103.16	498 ± 83.81	518 ± 79.58	515 ± 78.29	509 ± 80.54
SAT verbal score	445 ± 58.16	428 ± 60.54	437 ± 69.48	450 ± 69.20	451 ± 71.84	453 ± 76.79
SAT total score	934 ± 161.03	915 ± 140.35	934 ± 132.73	969 ± 119.61	966 ± 136.82	962 ± 138.28

Table 2. Stepwise Forward Regression

	Dependent Variable	Adjusted *R*	F	β
Athletic training	High school grade point average	0.384	29.62	.630
Perfusion technology	SAT mathematics score	0.072	4.159	.307
Occupational therapy	High school grade point average		19.799	.423
	High school grade point average and SAT verbal score	0.207	13.025	.342 and .229
Physical therapy	High school grade point average		13.973	.318
	High school grade point average and SAT verbal scores	0.123	9.786	.213 and .222
Physician assistant	SAT mathematics score	0.184	11.377	.449

program GPA refers to those courses required for successful completion of the accredited education and training as a health care professional in a specific discipline. In athletic training, this course work includes all courses required to comply with Commission on the Accreditation of Allied Health Educational Programs professional education standards.

METHODS

Sample

The subjects in this study were 373 graduates from 6 allied health education programs (1992 to 1997) from a private university in the northeast; 51 of those students were graduates of a Commission on the Accreditation of Allied Health Educational Programs–accredited athletic training education program. The other allied health disciplines we examined were health management systems, occupational therapy, perfusion technology, physician assistant, and physical therapy.

Measurements

Using the information found in the subjects' academic records, including official college transcripts, official high school transcripts, and SAT score reports, demographic information, HSGPA, SAT verbal (SATV) scores, SAT mathematics (SATM) scores, SAT total (SATT) scores, and college GPA (CGPA) data were gathered. We used descriptive statistics, analysis of variance, the Tukey post hoc test, the Pearson correlation, and stepwise forward regression models to analyze the data. The α levels were considered significant if $P \leq .05$. The dependent variable used to determine program success was CGPA, and the independent variables were HSGPA, SATV score, and SATM score. The investigators were blinded to the specific sources of the data. The SATT score was not used in the multiple regression analysis because it is the summation of the SATV and SATM scores and would create duplicate data. The institutional review board at the institution granted exempt status approval for this study.

RESULTS

All groups were first compared to determine the homogeneity of the sample (Pearson correlation). Only one significant difference was noted in the preadmission profiles (HSGPA; SATM, SATV, and SATT scores; and age) of the athletic training group compared with all other groups of allied health graduates. The mean age of the athletic training subjects was significantly different from the mean age for all other groups considered in this study ($F_{5,455} = 11.45$, $P = .00$). Overall, the ages of athletic training subjects ranged from 21 to 33 years, whereas the ages of all subjects ranged from 21 to 56 years. All other preadmission criteria were similar across all groups. The HSGPA ranged from 1.36 to 4.00 on a 4-point scale. The SATT scores ranged from 480 to 1300. The SATV scores ranged from 220 to 660, and the SATM scores ranged from a 260 to 710. The CGPA ranged from 2.00 to a 4.00 on a 4-point scale. All other descriptive statistical data are found in Table 1.

The stepwise forward regression analyses revealed that HSGPA and SATV score were predictive ($F_{2,370} = 31.32$, $P = .00$) of CGPA when the group was considered as a whole (n = 373). The independent variables predicted 14.5% of the variance in CGPA. The SATM score was not predictive as an independent variable. The HSGPA predicted 38% of the variance in CGPA of athletic training graduates ($P = .00$); however, SAT scores were not predictive (Table 2). The SATM score was the only significant predictor of CGPA for both perfusion technology ($P = .05$) and physician assistant ($P = .00$) graduates. The HSGPA and SATV score were predictive of CGPA in both occupational therapy ($P = .02$ and .00, respectively) and physical therapy ($P = .02$ and .03, respectively) groups. None of the dependent variables were predictive of CGPA for the health management systems group.

DISCUSSION

Selection criteria for entrance into allied health schools vary greatly among institutions; however, one standard remains consistent. Schools evaluate past academic performances as possible predictors of future performance and success in allied health education programs. Common criteria for evaluation of academic success (professional program GPA) include class rank, overall preadmission GPA, GPA in selected preadmission courses (eg, biology, chemistry), scores on standardized tests (eg, SAT, Graduate Record Examination), scores on specialized tests (eg, Allied Health Professions Aptitude Test), and quality of writing samples and personal interviews.

As more athletic training education programs consider the transition to schools of allied health, it is important to compare the academic preparation and outcomes of athletic training graduates with graduates of other allied health education programs. In this study, we investigated the effect of 2 traditional admission criteria, SAT scores and HSGPA, on the prediction of professional program performance as determined by CGPA. We found that, for students admitted at the freshman level into entry-level academic programs in the 6 allied health fields, SATV score and HSGPA predicted professional program GPA for the whole group ($n = 373$). However, these criteria predicted only 14% of the variances in CGPA. This finding demonstrates that other factors predicted the other 86% of the variance in CGPA. These findings also may have been influenced by the fact that 75% of the sample were graduates of the physical and occupational therapy programs, and HSGPA and SATV score were predictive of CGPA for both groups.

When analyzing the outcomes of this study further, we noted that HSGPA was the only predictor of CGPA in athletic training graduates. This is an important finding because HSGPA predicted 38% of the variance in CGPA in athletic training students. Since this independent variable predicted more than one third of the outcomes, with an associated high β weight, we advise that HSGPA continue to be used to predict success in future athletic training students.

Preadmission GPA is a common criterion measure for consideration of allied health program applicants.[2–7] Preadmission GPA may include HSGPA[8] or undergraduate,[4,6,9] freshman,[10] or preprofessional program GPA.[2,4,6–9] The findings from this study are similar to others in which preadmission GPA was a significant predictor of academic success in physical therapy[2,3,5,7] and pharmacy[6] professional programs. However, the variances in the ability to predict academic success noted in past studies ranged from 20% to 46%. The differences found in the Pearson correlation values may be explained by the rigors of different educational programs, professional discipline requirements, and the grading systems used by different instructors. Comparing GPA outcomes among different instructors, academic programs, or institutions is always suspect because of testing reliability errors. Similar to our findings, previous research provided support for the use of preadmission GPA as a predictor of success; however, evidence was insufficient to support preadmission GPA as the only criterion.

Preadmission GPA plays a confirming role when correlated with standardized test scores such as SAT, Graduate Record Examination, or American College Testing scores.[11] The results on these standardized tests are most commonly used to predict academic success as measured by final GPA[2–5] or board examination passing rates.[3,10] Standardized tests are purported to measure knowledge that has been acquired in and out of school. Scores of one portion of the SAT (SATV) and HSGPA were predictive of CGPA in both physical and occupational therapy graduates. These independent variables predicted 21% of the variance in CGPA in occupational therapy graduates and only 12% of the variance in physical therapy graduate outcomes. The SATM score was not found to be predictive of CGPA.

On the other hand, the SATM score predicted success in both the perfusion technology and physician assistant graduates. The SATM score predicted only 7% of the variance in CGPA in the perfusion technology group; this finding may be suspect because of the low power of the analysis ($F = 4.16$, $\beta = .31$). The SATM score predicted 18% of the variance in CGPA for physician assistant graduates. None of the independent variables predicted CGPA in health management system graduates; however, this finding may be a result of the small sample size of the group ($n = 22$).

Admission criteria (preadmission GPA and SAT scores) have been important considerations of candidates' qualifications for allied health education programs. These criteria are predictive of professional program success (CGPA); however, because of the great variance in their ability to predict professional outcomes, traditional selection criteria should not be the only factors used when considering students' applications. Other criteria, such as personal interviews, examination of student portfolios, student writing samples, and student motivation, should be considered. Personal interviews have been used for many years as part of the admission process into some athletic training education programs; however, little information is available on the effectiveness of such techniques. Further research should be conducted to determine the effectiveness of standard admission criteria, as well as the interview and other qualitative indexes, to assist in the prediction of success in athletic training and other allied health programs.

This study had several limitations, including sample size and sample selection. Differences in the number of subjects in each of the discipline-specific subgroups might have influenced the statistical data. The smaller groups may not have provided sufficient data to compare with the data collected from the larger groups. Also, the subjects used for this study, although from 6 different disciplines, were from the same institution. This restriction may bias the data and prevent generalization to a larger population. To confirm the findings from this study, a larger study of subjects from multiple institutions will help to control for institutional bias.

CONCLUSIONS

Overall, HSGPA and SATV score predicted academic success (CGPA) of the allied health group as a whole and should continue to be used as part of the process of admission in higher education until such time that more predictive criteria are determined. However, because these admissions criteria predicted only a small portion of the variance in graduate success, we suggest that other possible selection criteria continue to be investigated for their ability to predict success in athletic training and other allied health education programs.

The HSGPA was predictive of CGPA in athletic training graduates, predicting more than one third of the professional program success. Athletic training educators should continue to use HSGPA criteria as a predictor; however, they may wish to establish similar baseline comparisons between athletic training and other disciplines at their schools before determin-

ing the importance of standardized preadmission criteria for future students. This work should be considered a pilot study for the profession of athletic training and should be repeated at individual schools to determine the findings' applicability to other athletic training education programs.

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A Survey of Practice Patterns in Concussion Assessment and Management

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Objectives: To identify methods used by athletic trainers to assess concussions and the use of that information to assist in return-to-play decisions and to determine athletic trainers' familiarity with new standardized methods of concussion assessment.

Design and Setting: A 21-item questionnaire was distributed to attendees of a minicourse at the 1999 National Athletic Trainers' Association Annual Meeting and Clinical Symposia entitled "Use of Standardized Assessment of Concussion (SAC) in the Immediate Sideline Evaluation of Injured Athletes."

Subjects: A total of 339 valid surveys were returned by the attendees of the minicourse.

Measurements: We used frequency analysis and descriptive statistics.

Results: Clinical examination (33%) and a symptom checklist (15.3%) were the most common evaluative tools used to assess concussions. The Colorado Guidelines (28%) were used more than other concussion management guidelines. Athletic trainers (34%) and team physicians (40%) were primarily responsible for making decisions regarding return to play. A large number of respondents (83.5%) believed that the use of a standardized method of concussion assessment provided more information than routine clinical and physical examination alone.

Conclusions: Athletic trainers are using a variety of clinical tools to evaluate concussions in athletes. Clinical evaluation and collaboration with physicians still appear to be the primary methods used for return-to-play decisions. However, athletic trainers are beginning to use standardized methods of concussion to evaluate these injuries and to assist them in assessing the severity of injury and deciding when it is safe to return to play.

Key Words: mild brain injury, grading scales, head injury, evaluation

Although cerebral concussion has historically been an injury common to most contact and collision sports, only recently has the topic garnered increased interest from clinicians, researchers, the media, and sport-governing bodies. This shift is perhaps most clearly illustrated by the volume of research publications, continuing education workshops, and features by the print and electronic media on concussion in athletes relative to 10 years ago. The public health concern about concussion in sports has also resulted in more research grant funding available to study the assessment and management of concussion in sports. Currently, researchers in several major, large-scale studies are exploring these issues at the high school, collegiate, and professional levels, both in the United States and internationally.

Numerous individuals and organizations have published recommendations or guidelines for the management of concussion in sports.[1–3] Although investigators in several ongoing studies are attempting to identify factors that predict outcome and clarify the length of time necessary for adequate recovery after injury, current guidelines are based on consensus opinion of physicians and health care practitioners in the absence of empirical data. Some question exists about the level of consensus within the sports medicine community regarding certain aspects of different guidelines (eg, injury classification criteria, required time to withhold from competition after injury, etc). Past surveys[4] suggest that most athletic trainers do not adhere to any specific concussion classification system or return-to-play guidelines in the clinical decision-making process.

In 1994, the National Athletic Trainers' Association (NATA) Research and Education Foundation sponsored a summit on mild brain injury in sports. Since the summit, several advancements have been made in the assessment of concussion in athletes. Published studies[5,6] have highlighted the value of immediate, sideline, standardized mental status testing after concussion to clarify the acute neurocognitive effects of injury and to establish an index of severity for tracking recovery. Researchers[7–11] have also discovered the importance of assessing subtle deficits in balance and postural stability that may indicate concussion. The aim of standardized cognitive status and postural stability testing on the sideline immediately after injury is to reduce the amount of guesswork often encountered by sports medicine clinicians in assessing concussion during the acute stage. Studies[12–16] have also demonstrated the benefits of using more extensive neuropsychological

Table 1. Athletic Trainer Distribution by Setting and Experience

Clinical Setting	No. of Subjects	No. of Athletic Trainers	Average Experience Mean ± SD
Professional	18	16	10.9 ± 6.2
College	131	118	8.8 ± 7.0
High school	109	107	6.5 ± 4.9
Clinic	81	79	7.3 ± 6.2

Table 2. Number of Concussion Evaluations per Year by Setting

No. Evaluated	Professional (n = 18)	College (n = 126)	High School (n = 105)	Clinic (n = 78)	Total No. (%)
0–3	6	28	21	21	76 (23.2)
3–5	4	33	27	25	89 (27.2)
5–10	7	42	34	23	106 (32.4)
≥10	1	23	23	9	56 (17.1)

testing to clarify the persistent effects of concussion, track recovery, and make more informed decisions regarding return to play after injury. Aside from the obvious advantages of more accurate assessment methods, some researchers have expressed concerns about the potential misuse of these methods to return a player to competition earlier than what is deemed to be "safe" according to current practice standards.

The impact of recent research developments, continuing education offerings, media coverage, and various injury management policies on the attitudes and practice standards held by athletic trainers with respect to sports concussion is not clear. The purpose of our study was to clarify the methods used to assess concussions and the use of that information for making return-to-play decisions by athletic health care providers. This survey was also designed to determine athletic trainers' familiarity with and use of standardized methods of concussion assessment, including the Balance Error Scoring System (BESS), Standardized Assessment of Concussion (SAC), and neuropsychological testing.

METHODS

Instrument

A 21-item questionnaire was developed for the purpose of identifying athletic trainers' use of various concussion evaluation tools and to determine if patterns of use vary across practice settings. A panel of athletic trainers and neuropsychologists, considered experts in the current trends of concussion assessment and management, constructed the questionnaire and reviewed it for completeness. The questionnaire consisted of 5 demographic questions to ascertain level of education, certification status, employment position and setting, and sports for which the athletic trainer provided clinical coverage. Concussion-related questions addressed the number of concussions evaluated per year, methods used to assess and grade concussions, and how these methods affect return-to-play decision making by the sports medicine staff.

Subjects

The questionnaire was distributed to approximately 900 attendees of a minicourse at the 1999 NATA Annual Meeting and Clinical Symposia entitled "Use of the Standardized Assessment of Concussion (SAC) in the Immediate Sideline Evaluation of Injured Athletes." Attendees were given a questionnaire on arrival at the seminar and asked to complete the questionnaire before the lecture. A total of 403 completed surveys were voluntarily returned at the end of the session. For ease of comparison, completed surveys were then grouped according to setting of primary clinical responsibility. Surveys on which respondents failed to select a single answer to the question concerning setting of clinical responsibility were considered invalid, resulting in a final sample of 339 subjects. Table 1 describes this sample, grouped by clinical setting according to certification status and years of experience. Variations on sample size throughout the study occurred because not all respondents answered all questions on the survey. We used basic descriptive statistics to describe the population and clinical practice parameters. Analysis of variance was used to determine significant differences between number of concussions evaluated per year by clinical setting.

RESULTS

Table 2 presents a frequency distribution of the average number of concussions per year evaluated by athletic trainers in their respective employment settings. The mean (±SD) number of concussions evaluated per year was 7.04 (±5.0). Analysis of variance revealed no significant differences in the number of concussions evaluated per year by clinical setting ($F_{3,323} = 1.374$, $P = .25$).

More respondents reported using the Colorado Guidelines (28%) than the American Academy of Neurology, Cantu, or other specific set of injury management guidelines (Table 3). More than 18% of the total respondents reported currently not using any type of concussion grading scale or return-to-play guidelines, whereas only a small percentage (2.6%) reported using more than a single set of guidelines. Further analysis by sport revealed that a relatively high percentage of athletic trainers did not report using grading scales in contact sports

Table 3. Primary Grading Systems Used for Concussion by Setting*

Grading Scale	Professional (n = 16)	College (n = 122)	High School (n = 94)	Clinic (n = 73)	Total
American Academy of Neurology	2 (12.5)	16 (13)	12 (13)	10 (14)	40 (13.1)
Cantu	3 (19)	17 (14)	20 (21)	19 (26)	59 (19.3)
Colorado	4 (25)	43 (35)	23 (24)	17 (23)	87 (28.0)
Other	2 (12.5)	23 (19)	22 (23)	8 (11)	55 (18.0)
Multiple	0 (0)	4 (3)	4 (4)	0 (0)	8 (2.6)
None	5 (31)	19 (16)	13 (14)	19 (26)	56 (18.4)

*Data are number (percentage).

Table 4. Evaluative Tools Used in the Assessment of Concussion

Evaluative Tool	Professional (n = 18)	College (n = 131)	High School (n = 109)	Clinic (n = 80)	Total No. (%)
Clinical examination	9	103	71	55	238 (33.0)
Balance Error Scoring System	2	11	15	8	36 (5.0)
Standardized Assessment of Concussion	2	29	24	21	76 (10.6)
Symptom checklist	13	94	91	57	255 (35.7)
Neuropsychological testing	6	38	41	24	109 (15.3)

Table 5. Principal Evaluative Tools Used in the Return-to-Play Decision After Concussion

Evaluative Tool	Professional (n = 15)	College (n = 126)	High School (n = 98)	Clinic (n = 73)	Total No. (%)
Physician recommendation	4	39	33	13	89 (28.5)
Clinical examination	3	28	22	22	75 (24.0)
Return-to-play guidelines	1	24	20	13	58 (18.6)
Symptom checklist	2	19	14	15	50 (16.0)
Standardized Assessment of Concussion	0	4	4	3	11 (3.5)
Player self-report	2	4	0	2	8 (2.6)
Neuropsychological testing	1	2	2	1	6 (1.9)
Other	2	6	3	4	15 (4.8)

such as football (n = 44, or 16.4%), ice hockey (n = 10, or 17.2%), wrestling (n = 25, or 14.3%), lacrosse (n = 42, or 16.3%), and men's soccer (n = 39, or 17.0%).

Clinical examination (n = 238, or 33%) and a symptom checklist (n = 255, or 15.3%) were the most common evaluative tools used to assess concussion (Table 4). A significant number of respondents also reported using various standardized assessment methods, including SAC (10.6%), BESS (5%), and neuropsychological testing (15.3%). Twenty-five percent of the athletic trainers surveyed had access to a neuropsychologist for consultation after concussion, and, of those who had access, 40% routinely referred their athletes to these professionals for further evaluation. A large number of respondents (86.2%) believed that athletic trainers should be trained to administer neuropsychological tests to assess concussion.

Athletic trainers (34%) and team physicians (40%) were primarily responsible for clinical decision making regarding return to play after concussion. In contrast, very few respondents indicated that decisions are primarily made by outside physicians (12%), coaches (2%), players (<1%), or parents (<1%). Respondents reported relying most frequently on physician recommendations (27.6%), clinical examination findings (23.3%), return-to-play guidelines (18%), and symptom checklists (15%) when making decisions about return to play after injury (Table 5). Fewer than 5% of the respondents reported using any single form of standardized assessment as the stand-alone method relied on most in making decisions regarding return to play after concussion.

A large number of respondents (84%) believed that standardized methods of concussion assessment (SMCA) provide more information than a routine clinical or physical examination alone. Forty-seven percent of the sample believed that this information would likely result in a player's being withheld longer from competition after injury, whereas only 21% thought that a player might return to play sooner based on this information. Most respondents (97%) indicated that they would not return an athlete to play when results from an SMCA were normal, but the clinical examination results were abnormal. Nearly all respondents (99%) said they would not return an athlete still experiencing postconcussion symptoms (Table 6). Eighty-six percent of respondents indicated that they would not allow the athlete to return to play when the clinical examination results were normal, but the results of standardized assessment measures were abnormal. Twenty-four percent of the athletic trainers thought that the information from standardized methods of concussion assessment could potentially be misused to allow a player to return to competition earlier than what would be deemed "safe" after concussion.

Table 6. Clinical Assessment and Standardized Methods of Concussion Assessment (SMCA) in Return-to-Play Decisions

Clinical Findings	Return-to-Play Decision: Yes	Return-to-Play Decision: No
The clinical examination results are abnormal, but the SMCA results are normal.	11	316
The clinical examination results are normal, but the SMCA results are abnormal.	4	326
The athlete demonstrates postconcussion symptoms, but the SMCA results are normal.	44	282

Of the small percentage (17%) of athletic trainers who currently use the SMCA in the initial assessment of concussion, most (86%) believed the instrument provides a more accurate assessment than routine clinical examination alone. Sixty-three percent of this subsample indicated that they were more likely to withhold a player from return to competition as a function of using the SMCA information, whereas 30% said the results would have no effect on their return-to-play decision.

DISCUSSION

This survey was designed to clarify the current trends in clinical practice by athletic trainers in the assessment and management of sport-related concussion. The collective responses represent the opinions and practice patterns reported by a relatively large sample of certified athletic trainers and other

health care providers responsible for coverage in a variety of male and female sports. These data certainly substantiate the notion that athletic trainers routinely encounter concussion in the care of their athletes during a sport season. On average, our sample reported being responsible for evaluating and managing more than 7 concussions per year across a wide range of sports with and without recognized risk of head injury.

Athletic trainers and team physicians were most often directly responsible for decisions regarding an athlete's readiness to return to play after concussion. Coaches, players, and parents are seldom primarily responsible for return-to-play decisions. Although routine clinical examination and input from an attending physician remain the primary sources of information used to make decisions about return to play after concussion, the specific criteria on which decisions are based remain less clear. More clinicians appear to be using a multidimensional approach to concussion assessment and management, which incorporates clinical examination findings, concussion grading scale criteria, symptom checklist information, return-to-play guidelines, standardized tests, and neuroimaging studies.

It can safely be said that more athletic trainers are looking to standardized cognitive assessment, postural stability testing, and formal neuropsychological testing to guide their clinical decision making compared with several years ago. Our results indicate that a small but increasing number of athletic trainers currently use SMCA and that most (86%) believe these measures significantly improve the accuracy of their evaluation. A promising finding is that athletic trainers are receptive to the use of standardized assessment methods, despite also reporting that results from these measures may actually cause a player to be withheld longer from competition after concussion than if they were not used. Although athletic trainers are beginning to recognize the value of standardized methods of assessment, it is vital that they understand the importance of obtaining baseline measurements with which postinjury comparisons can be made. Although time consuming, the process can be made more manageable when it is incorporated into preseason physical examinations and screenings.

We found no current consensus for any single preferred concussion grading scale and set of return-to-play guidelines among athletic trainers. More respondents reported using the Colorado Guidelines than the Academy of Neurology guidelines, the Cantu guidelines, or any other system. Nearly 20% of respondents indicated that they are not currently using any specific concussion grading scale or return-to-play guidelines. Most agree that it is not critical that all clinicians adhere to the same specific set of classification criteria but rather that each sports medicine staff have a systematic means of concussion assessment and management to avoid making "on-the-fly" decisions that could result in undue risks. The current survey results are favorable in the sense that more athletic trainers appear to be using some form of systematic injury management recommendations than was the case several years ago. Clinicians should recognize that most concussion grading scales depend on amnesia and loss of consciousness, yet a very small percentage of concussions result in either of these 2 symptoms.[4] Some of the grading scales have been criticized for placing too much emphasis on loss of consciousness while neglecting other symptoms. Future research is necessary to provide clinicians with an empirical basis on which to make the classification of injury severity and decisions regarding an athlete's readiness to safely return to competition after concussion. Thus, it is important to use a combination of overall symptom severity, a standardized assessment method, and an acceptable concussion grading scale when classifying injury.

A common theme in the recent literature on sports concussion is that any abnormalities reported or exhibited by the player after a suspected concussion require close attention by the responsible health care provider. All concussions are not created equal, and these injuries may manifest differently in individuals. Our results illustrate that athletic trainers recognize the importance of weighing all clinical information when assessing the injured athlete after concussion. Most would not return a player to competition if any findings from routine clinical examination, standardized assessment methods, or symptom screening were considered abnormal. The current data suggest that athletic trainers consider results from standardized sideline assessment methods (eg, SAC, BESS) and neuropsychological testing to be valuable but continue to place more emphasis on their own clinical examination and the symptoms reported by the player.

Our findings also suggest an increased frequency of collaboration between certified athletic trainers and neuropsychologists in the assessment of sport-related concussion. One in every 4 athletic trainers surveyed has access to a neuropsychologist for consultation, and 40% of those with access routinely refer injured players for testing and consultation after injury. Although athletic trainers incorporate neuropsychological test findings into their return-to-play decision making, seldom are these results the primary criteria considered.

Most respondents believe that standardized methods provide more information clinically than routine examination alone, yet a quarter of respondents expressed concern about the potential misuse of information from standardized assessment methods, including the danger of returning a player to competition too soon. A high percentage of athletic trainers already using these methods reported an improvement in the accuracy of their sideline assessment and indicated that they are now more likely to withhold a player even longer based on the information yielded by these measures.

Several issues related to neuropsychological testing in concussion assessment require attention in the near future, including the use of lengthy test batteries that are not practical in the sports medicine setting, the interest by athletic trainers in being trained to administer neuropsychological tests to assess concussion, and the acquisition of parental consent to perform neuropsychological assessments of high school athletes. Professionals from the fields of neuropsychology and athletic training need to jointly address these issues to develop solutions that meet the clinical demands of the sports medicine community and uphold the professional testing standards required of neuropsychologists.

In summary, recent research publications, continuing education efforts, and increased attention to sports-related concussion appear to have had a small but significant impact on the clinical practice of athletic trainers in the assessment and management of concussion. Clinical judgment and collaboration with an attending physician continue to drive clinical decision making on return to play after injury, but the inclusion of standardized assessment methods appears to be making increasing contributions to these decisions. Also, no consensus yet exists on specific guidelines for injury classification and return-to-play recommendations. Some time is likely to pass before clinical research findings are galvanized into standards of clinical practice and embraced by the larger sports medicine

community. Further research and education are necessary to demonstrate the potential value of innovative methods in improving the accuracy of concussion assessment, tracking post-injury recovery, and making more informed decisions regarding safe return to play.

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The Effect of Life Events on Incidence of Injury in High School Football Players

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Objective: To investigate the potential relationship between life events and injury among high school football players.

Design and Setting: This was a prospective cohort study. We collected athlete-exposure and injury data through 2 consecutive seasons at 13 high schools. Injury data consisted of injury status, frequency, severity, practice versus game, time of season, and total time lost from football participation. A life event survey was administered at the end of each season to obtain total, negative, and positive life event scores.

Subjects: A total of 331 varsity high school football players.

Measurements: Data collected included athlete exposures, injury reports, and life event surveys. Statistical analysis was completed using the Wilcoxon rank sum W test, the Pearson χ^2 test using an iterative proportional fitting procedure, and contingency χ^2 test.

Results: Total and negative life change measures significantly affected injury status and frequency of injury ($P < .05$). However, they did not reflect differences in severity of injury or time lost due to injury, and the positive life change measure was not associated with significant differences for any injury factor ($P > .05$). Preseason injury rates were significantly higher than season and playoff injury rates ($P < .05$).

Conclusions: High school football players who experience high degrees of total and negative change were at greater risk of becoming injured and of sustaining multiple injuries. Playing situation and time of season also affected risk of injury.

Key Words: stress, sports psychology, epidemiology

Stress has been defined as any disruption, change, or adjustment in a person's mental, emotional, or physical well-being caused by an external stimulus, either physical or psychological.[1,2] It has long been recognized that the stress produced by life events has a bearing on emotional health in people of all age groups.[1] However, prolonged stress or stressful events have recently been observed to have an effect on physical health as well.[1,3,4]

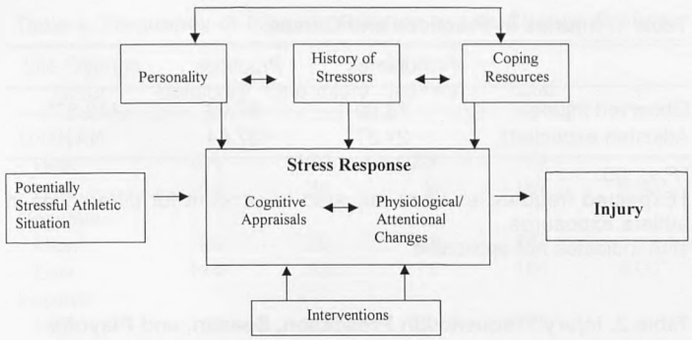
In 1988, Andersen and Williams[5] proposed a theoretical model that explained the relationship between stressful events and occurrence of injury. Previous researchers had not considered the complex relationship between potentially stressful athletic situations and resulting injury. This model was designed to illustrate this relationship. Williams and Andersen updated the model in 1998 to more clearly illustrate the relationships among the contributing factors (Figure).[6]

At the core of the model is the stress response to potentially stressful situations.[5,6] This response is similar to that described in previous models[1,2] but is divided into 2 aspects. The first aspect is the cognitive appraisal of the situation: the demands, resources, and consequences of the situation. The second aspect of the stress response is the physiologic and attentional responses: increased general muscle tension, narrowing of the visual field, and increased distractibility.

Prolonged stress can lead to chronic muscle tension as a form of direct defense.[1,5] This is a result of the constant tensing of the muscles in the attempt to guard the body from the stressor. The increased muscle tension can lead to tension headaches, migraine headaches, and backaches.[1,2] Generalized peripheral muscle tension may leave an athlete more susceptible to muscle strains. Williams et al[7] reported that individuals with high life stress experienced greater peripheral narrowing (awareness of activities surrounding an athlete) and trait anxiety when placed in a stressful, dual-task laboratory situation. This increased peripheral narrowing is potentially hazardous when an athlete is placed in a high-stress, high-risk activity such as football. Lysens et al[8] argued that stress related to life change may lead to a potentially dangerous blocking of adaptive responses in risky situations.

The Andersen and Williams[5,6] model differs in its description of the stress-injury relationship because of its consideration of individual factors, such as personality traits, history of stressors, and coping resources, which may alter or affect the stress response. This model indicates that individual factors will determine the severity of the stress response to a particular situation,[5,6] which may then increase an athlete's risk of being injured.[9]

The relationship between life events and incidence of injury



A model of stress and athletic injury. Reprinted by permission.[6]

has been studied extensively in collegiate football players.[10–13] However, to our knowledge, only Coddington and Troxell[14] have examined the relationship between life events and high school football. Their study was limited by 3 factors. The first factor was the size of the sample (n = 114). The second problem occurred in the reporting of injuries. The coaches were responsible for injury reporting, as opposed to qualified personnel such as certified athletic trainers (ATCs) or physicians. The third factor was that the researchers used a general life event questionnaire rather than a questionnaire designed for an athletic population. The authors stated in the study that its results should only be considered as a pilot study.[14] However, it appears that no researcher has attempted to follow up this pilot study.

The primary purpose of our study was to assess whether the stress-injury relationship identified in collegiate football players also existed in the high school setting. A secondary goal of the study was to further reveal factors predisposing high school football players to injury.

METHODS

Experimental Design

We collected data prospectively during the 1995 and 1996 football seasons. At the end of the season, each athlete completed a modified Life Events Survey for Collegiate Athletes (LESCA).[15] The independent variable for this study was the score on the modified LESCA. The modified LESCA was analyzed according to the number of events the athlete reported having occurred during the previous year and the effect the events had on the athlete. Totals were calculated for the cumulative effect of negative events, positive events, and all events. The dependent variables were the injury data points.

Subjects

Subjects were members of the varsity football teams from 13 high schools in 4 counties in Florida. These schools were chosen due to the availability of ATCs to aid in injury data collection. The schools ranged in size classification from 2A to 6A. Class 1A schools were not included because they do not sponsor football programs. The University of Florida Institutional Review Board approved this study. The study was explained to all eligible participants, and informed consent was obtained from those athletes willing to participate. Parental signatures authorizing participation were required for minor athletes. A total of 331 athletes in grades 8 through 12 chose to participate.

Instruments

The life events questionnaire used in this study was a modified version of the LESCA,[15] which was initially designed by Petrie in 1992. Sixty-nine questions were chosen from an initial master list of 109 events based on frequency of agreement among expert raters. The interrater agreement was 0.81 for those events considered most relevant to collegiate athletes.[15] Test-retest correlations were significant ($P < .001$) at 1 week and 8 weeks for all 3 life stress scores, indicating reliability of the measure.[15]

The LESCA was modified, with permission from the author, to make it appropriate for use with a high school athletic population. The scale was reviewed by ATCs working with high school athletes and by high school athletes who would not be a part of the study. Further refinements were made based on their input. After the modifications, 3 faculty members at the University of Florida reviewed the scale and determined it to have face validity.

The modified LESCA consisted of 65 general and athletic specific life events. The instructions call for the athlete to check each event experienced during the last year and indicate the overall effect of the event on his life. Effect is rated on a scale from −4, indicating an extremely negative effect, to +4, indicating an extremely positive effect. The modified LESCA was analyzed according to the number of events that occurred to an athlete and the effect the events had on the athlete. The survey was scored according to the instructions for the original survey.[15] Negative and positive life event scores were calculated by summing the effect scores of those events rated undesirable (negative) and desirable (positive). Adding the absolute values of the negative and positive scores determined total life event scores.

Procedures

At the beginning of preseason practices, a roster was made and each athlete was assigned a subject number, which was known only to the ATC at each site. Each school was assigned a code number. The combination of the school code and the subject number formed the subject identification number. The subject identification number was the only form of identification on the participation logs, life event survey, and injury questionnaire.

Throughout the season, the ATC at each site tracked athlete exposures (AEs) and injury data. Athlete exposure was defined as any situation, either game or practice, in which the athlete was at risk for injury.[16] Athlete exposure logs were completed each day, indicating the level of participation for each athlete. These levels included full participation, present but not participating, and not present.

We defined injury as any condition treated by the ATC that required modification of participation for the remaining time in the same practice or game or that resulted in the athlete's inability to participate in football for at least 1 day after injury.[9,13] All head injuries and dental injuries were reported, regardless of their effect on participation. Severity of injury was defined by 2 methods. The first definition was based on time lost,[17] and the second used anatomical structure damage.[18] A follow-up injury questionnaire was completed for all

incidents fitting this definition of injury. These questionnaires were completed only for injuries that were directly attributable to football participation.

At the end of each season, participants were asked to complete the modified LESCA. Standardized instructions were read to the participating athletes by the ATC at the site. When the athlete completed the scale, he placed it directly into an envelope, which maintained confidentiality. Only the researcher viewed the results of the surveys.

Statistical Analysis

The independent variable for this study was the score on the modified LESCA. Three levels of this variable were analyzed: overall negative, positive, and total life change scores. The third level (total score) is a combination of levels 1 and 2. The dependent variable was injury status. The number of levels varied with the injury factor being analyzed. Levels included injured or uninjured, frequency of injury, severity of injury, and total time lost from participation due to injury.

Injury rates were calculated by dividing the number of injuries by the number of exposures and multiplying by 1000. These rates were calculated for the total sample and for the entire season. We used the Pearson χ^2 test for comparison of injuries that occurred in practices and games and time of injury within the season. To adjust for differences in AEs, expected frequencies were generated through an iterative, proportional-fitting procedure.[19] Expected frequencies were adjusted because of the difference in the number of practice and game exposures and the different numbers of AEs experienced by each athlete.

We used descriptive and inferential statistics to assess injury frequencies and severities. Descriptive analyses included injury history, injury severity, mechanism of injury, time lost due to injury, and playing situation (game versus practice). Comparative analyses were conducted between athletes with high and low degrees of negative, positive, and total life change. Athletes were placed in high and low life change groups according to the reported score in relation to the median score.

The Wilcoxon rank sum W test was used to identify possible differences in the life event scores for injured and uninjured athletes. This test ranks the individual survey scores and then looks at differences in relation to the mean ranks rather than the scores themselves. We chose this test to eliminate the effect of outlying scores. Athletes were then divided into high and low life change groups based on their life change score in relation to the median score. The median was also chosen to eliminate the effect of outlying scores. These subgroups were analyzed by a contingency χ^2 test[20] to determine differences for injury factors. These factors included injury status, frequency of injury, severity of injury, and total time lost during the season due to injury. All hypotheses were tested in the null form. Data were analyzed with the level of significance set at $P < .05$ using the Statistical Package for the Social Sciences for Windows (version 6.1, Chicago, IL).

RESULTS

A total of 165 injuries were sustained by 121 (36.6%) of the 331 subjects who experienced 21 054 AEs throughout 2 seasons. The total injury rate for this study during the 1995 and 1996 seasons was 7.84 injuries per 1000 AEs. Of the 165 injuries reported during the study, 87 occurred in practice and 78 occurred during games. Practices had an injury rate of 4.95 injuries per 1000 AEs, whereas the game rate was more than 4 times higher, at 22.34 injuries per 1000 AEs. The χ^2 analysis revealed a significant difference between the observed and expected injury frequencies ($P < .05$, Table 1). This difference appears attributable to the increased game injury rate when adjusted for AEs. Although accounting for only 16% of all AEs, games were responsible for 47.3% of all injuries.

Table 1. Injuries in Practices and Games

	Games	Practices	χ^2
Observed injuries	78.00	87.00	112.37*
Adjusted expected†	27.37	137.64	NA‡

*$P < .05$.
†Expected frequencies were adjusted to account for differences in athlete exposures.
‡NA indicates not applicable.

Table 2. Injury Frequency in Preseason, Season, and Playoffs

	Preseason	Season	Playoffs	χ^2
Observed injuries	50.00	112.00	3.00	6.01*
Adjusted expected†	39.26	118.18	7.55	NA‡

*$P < .05$.
†Expected frequencies were adjusted to account for differences in athlete exposures.
‡NA indicates not applicable.

Table 3. Life Change Groups for Injured and Uninjured Athletes

Life Change Groups	Injured	Uninjured	Total	χ^2
Total				
High	73	101	174	4.61*
Low	48	109	157	
Negative				
High	71	96	167	5.16*
Low	50	114	164	
Positive				
High	68	107	175	0.84
Low	53	103	156	

*$P < .05$.

We analyzed time of injury during the season to determine if injury rates increased toward the end of the season. We speculated that the increased stress of late-season games might lead to higher injury rates. However, we found no significant differences between incidence of injury during the first and second halves of the season ($\chi^2 = 3.76$, $P > .05$). Analysis comparing preseason, season, and playoff injury rates revealed significant differences ($P < .05$, Table 2), apparently attributable to the preseason injury data. The preseason injury rate was 9.98 injuries per 1000 AEs, compared with the season and playoff rates, which were 7.43 and 3.1, respectively.

Of the 121 injured athletes, 89 (73.6%) sustained only 1 injury, 23 (19.0%) sustained 2 injuries, and 9 (7.4%) were injured 3 or more times during their season. The total and negative life event scores affected the athlete's injury status (injured or uninjured) and injury frequency. The Wilcoxon rank sum W and contingency χ^2 tests each indicated that those athletes with higher degrees of total and negative life change were more likely to become injured ($P < .05$, Table 3). Ath-

Table 4. Frequency of Injury in Relation to Life Change Groups

Life Change Group	Uninjured	One Injury	Multiple Injuries	Total	χ^2
Total					
High	101	50	23	174	
Low	109	39	9	157	6.93*
Negative					
High	96	50	21	167	
Low	114	39	11	164	6.00*
Positive					
High	107	48	20	175	
Low	103	41	12	156	1.54

*$P < .05$.

letes in the high life change groups appeared more likely to sustain multiple injuries ($P < .05$, Table 4). Similar results were not observed for the positive life event data.

For the purposes of this study, we defined injury severity in 2 ways. The first definition was based on anatomical structural damage.[18] The second definition used time lost from participation due to injury.[17] Analysis did not reveal significant differences in severity of injury between the low and high life event score groups for total, negative, or positive life change ($P > .05$).

We hypothesized that the life event score would significantly affect an athlete's chance of becoming injured and his chance of sustaining multiple injuries. If this were true, we expected that injured athletes in the high life change groups would miss more time during a season because of injury than athletes in the low life change groups. Athletes were placed in 3 groups based on total time lost: athletes missing 1 to 5 days, 6 to 10 days, or more than 10 days of participation. These groups provided sufficient data points in each cell. Comparison of the high and low life change groups for total, negative, and positive life change revealed no significant differences in total time lost ($P > .05$).

DISCUSSION

Each year, more than 3 million high school students actively participate in boys' sports programs.[21] This number does not include students participating in city, county, or other recreational leagues. In 1994, 13 877 schools had competitive football teams, accounting for 955 247 participants.[21] Football participants accounted for almost one third of all boys' sports participants. In 1994, Florida alone reported 374 schools with football teams and 29 911 participants. Similarly, football accounted for almost one third of the 102 775 boys' sports participants for the year.[22] Despite extensive research on college athletes, relatively little literature is available with respect to this large population of high school football players.

The significance of this study lies in the identification of a stress-injury relationship in the high school football player. This finding is an important step that must be taken in the process of assessing variables that may be moderated to prevent injury, but it does not constitute the entire story.

The stress-injury model has 3 components: the stressor, the stress response, and the injury.[5,6] The key to prevention is modifying 1 of the components of the model. In this type of prevention, it is almost impossible to prevent the stressor from occurring, and it is not feasible to remove the athlete from practice or games because of fear of injury. Therefore, the research and focus must be on the stress response. Once the stress-injury relationship was identified in college-aged subjects, researchers continued studies to identify means of altering the stress response, such as improving coping skills, increasing social support, and moderating personality traits. Injury prevention methods can be developed for these issues in the stress-injury response.

Current Research

The current game-practice data support previous football epidemiologic research. DeLee and Farney,[23] Prager et al,[24] Halpern et al,[25] and Zemper[26] all reported that athletes were at greater risk of injury during games than practices; however, previous authors of studies of life events and incidence of injury have not reported data regarding time of injury. This difference may be related to the fact that games are a more stressful exposure situation than are practices. The stress of game situations, in addition to preexisting stress levels, may make athletes more susceptible to injury. Although full-contact practices are common in football, the intensity of the contact often does not equal that in games. If the athlete is not focused on the task at hand, he may be more likely to become injured in a game, which is even less controlled than a full-contact practice. Finally, the stress response of peripheral narrowing could be particularly dangerous in game situations. The narrowing of the field of vision could leave the athlete at greater risk of being injured because he is not as aware of the situation surrounding him.

We did not find significant differences in injury frequency and rate when comparing the first and second halves of the season. However, when comparing preseason, season, and playoff data, a significant difference was noted, most likely related to the preseason injury rate and the week 2 data. In this study, week 2 had both the highest frequency of injury (n = 25) and the highest injury rate (16.19 per 1000 AEs). The reason for this was probably more related to conditioning than to stress. The high schools participating in this study limit the first 3 days of week 1 to no pads and no contact. Therefore, only the last 2 days of week 1 place the athlete in a full-contact, high-risk situation. During week 2, athletes are practicing in full pads and frequently practice 2 or 3 times a day. Many athletes do not participate in summer conditioning and are, therefore, out of shape and possibly at greater risk of injury.[5] However, this can also be viewed from the aspect of stress and injury. Except for the first 3 days, preseason practices are typically very fatiguing. Coaches are concerned with the teaching and practicing of plays and exposing the athletes to situations that will be experienced throughout the year. Coaches typically expect the athletes to learn a great deal in a short amount of time. The athlete's appraisal of the demands of these stressors and methods of modifying the stressors could affect the incidence of injury.

The life stress injury data from this study agree with previous research findings in collegiate football players.[10–13] These authors reported that athletes with high degrees of life change were more likely to become injured than those with low levels. Of the 121 injured athletes in this study, 73 athletes (60.3%) reported total life events scores above the median score.

Our study does not support the only previous study to our knowledge that was conducted with high school football play-

ers. Coddington and Troxell[14] noted significant relationships between injuries and those athletes scoring high on the object loss score of the Life Event Scale for Adolescents. However, they did not report an association between injury and the overall life event score. Possible reasons for this are related to the limitations mentioned earlier: small subject population, injury reporting by coaches, and use of a generalized life event measure. Our study was designed to improve on each of these limitations.

Petrie,[15] in his study of college gymnasts, reported that athletes with higher life event scores experienced more minor injuries than athletes with lower scores. The current study data regarding severity of injury support the findings of Cryan and Alles,[13] who reported that athletes with higher stress scores were not at greater risk of sustaining a serious injury. In our study, frequencies and rates of severity for the high life change group were not significantly different than those for the low life change group. They were also consistent with the total population. Our results appear to indicate that life event scores, although related to the chance of becoming injured and the chance of sustaining multiple injuries, are not related to the severity of the injury sustained.

This study's results regarding frequency of injury were also supported by the research of Cryan and Alles.[13] These authors reported that athletes in a high stress score group were more likely to become injured 1 or 2 times during the football season but not 3 or more times. They concluded that those athletes with a high degree of life change were more susceptible to multiple injuries during the season. One historical factor that acted directly on the stress response is previous injuries.[5,6] An individual's past stressors directly influence the stress response to potentially stressful athletic situations. In the model of stress and athletic injury,[5] one component of "history of stressors" is previous injury. A previous injury predisposes the athlete to reinjury; however, research suggests that the history of injury acts on the stress response, which can also predispose the athlete to a new injury.

Petrie[15] reported that athletes with higher negative life event scores missed significantly more days because of injury than those with lower scores. Our data did not support this conclusion. This difference is likely attributable to the variant and individual nature of injury. The amount of time lost due to an injury relies on many factors, such as the nature of injury, severity, position of the athlete, and individual rates of healing.[27] All of these factors interact, making it difficult to determine which is most directly influencing the rate of return to sport.

Limitations of Study

The primary limitation to application of this research is the lack of reliability and validity data for the modified LESCA. The original LESCA consisted of 69 general and athletic life events. Because it was designed for a college athletic population, we modified it for use by high school athletes. The instructions for completion and scoring were not changed. In total, 4 events were deleted, and 6 were modified to make the language applicable. As an example of the modifications made, the original "Receiving an athletic scholarship" was modified to "Being recruited for an athletic scholarship." Four questions were deleted because an appropriate modification could not be determined. An example of the events deleted was "Conflict with roommate."[15] During the review for face validity, we believed that the minor nature of the modifications had not affected the reliability of the original scale. However, because reliability testing was not performed on the modified LESCA, we do not know if this is actually the case.

CONCLUSIONS

Our study helps to establish a stress-injury relationship for high school football players. The main conclusions that can be drawn from this study include the following: (1) high school football players with high levels of total and negative life stress were more likely to become injured; (2) high school football players with high levels of total and negative life stress were more likely to sustain multiple injuries; (3) high school football players were more likely to become injured in a game than in practice; and (4) high school football players were at greater risk of injury during the preseason.

The results of this study are very important to the coach and athletic trainer, who often serve in multiple roles, including teacher, counselor, and friend. The multifaceted roles of these individuals often mean that they are in the best position to identify athletes at risk and to help the athletes. They see the athlete daily and are often aware of problems the athlete is experiencing. With the identification of the stress-injury relationship and further identification of effective moderating factors, coaches and athletic trainers will be important links in the chain of prevention of injury.

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Scholarly Productivity of Athletic Training Faculty Members

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Objective: To compare the scholarly productivity index (SPI) among the levels of academic appointment, degree type, and percentage of academic appointment of athletic training faculty members.

Design and Setting: We used a 5 × 6 × 4 factorial design for this study. A survey instrument was used to determine the number of publications and the number of years in their current appointment.

Subjects: Subjects were faculty members in Commission on Accreditation of Allied Health Education Programs-accredited athletic training education programs.

Measurements: The SPI was calculated by dividing the total number of publications (peer-reviewed and non–peer-reviewed journal articles, peer-reviewed abstracts, books written or edited, book chapters, platform presentations, published book reviews, and external funding) by the number of years in the productivity period.

Results: The SPIs were different for the levels of academic rank. Full professors had a higher SPI than all other groups (Tukey honestly significant difference, $P \leq .05$). Associate professors had higher SPIs than instructors or lecturers and clinical specialists, equivalent SPIs to assistant professors, and lower SPIs than full professors. Assistant professors had lower SPIs than full professors but were equivalent to all other groups. There were no differences among the levels of degree type or percentage of academic appointment.

Conclusions: The scholarly productivity of athletic training educators was affected by their academic rank but not by the percentage of time they were assigned to academics or their academic degree type.

Key Words: scholarly productivity index, athletic training education, tenure, promotion

The evolution of a profession can be measured through the development of its educational process. A 1997 decision by the National Athletic Trainers' Association's (NATA) Board of Directors to implement a single route to certification, although a landmark in our professional development, has increased the emphasis on hiring and retaining full-time athletic training faculty. Although beneficial to the institution, the department, and the profession, appointment in a tenure-track position carries with it a set of demands that are unique to the world of academia. No other work environment offers a concept similar to tenure, a lifetime contract.

Although scholarship is essential for the individual faculty member, it is also vital to the advancement of the athletic training profession. Faculty members who are engaged in scholarship help to integrate athletic training into the mainstream of academia and communicate the tenets of our professional practice to other allied health and medical professionals. The manner in which our profession is judged will be based largely on our contribution to the knowledge base needed for athletic health care and the scientific validation of our professional practice.[1,2]

Many new athletic training scholars are faced with the difficult task of developing and maintaining their teaching, research, and service activities while implementing or administering (or both) a Commission on Accreditation of Allied Health Education Programs (CAAHEP)–accredited athletic training program, often before or immediately after completing their doctoral programs.[3,4]

The decision to grant tenure is generally based on an individual's achievement in the areas of teaching, scholarship, and service. Although objective standards in the form of course evaluation instruments have been developed to measure an instructor's effectiveness and service contributions are often contract based, the determination of a candidate's relative scholarly accomplishments is more esoteric. Ultimately, the decision to grant tenure is heavily weighted toward the number of refereed publications and the individual's ability to obtain external funding through grant acquisition.[5–8] A scholarly productivity index (SPI) has been developed as a mechanism to objectively measure scholarly output.[2,5]

Athletic training educators possess different types of degrees, have appointments with varying percentages of time dedicated to academics, and hold various academic ranks. The extent to which these factors influence scholarly output has not been identified. Information about scholarly productivity by peers would be valuable in assisting athletic training scholars and their supervisors to develop reasonable expectations about scholarly output given the diverse responsibilities usually undertaken. Adjustments in responsibilities could then be considered to provide the athletic training faculty member with the appropriate time to meet institutional expectations for scholarly activities. The purpose of our study was to compare

scholarly productivity of athletic training faculty within NATA-approved and CAAHEP-accredited athletic training programs by degree type, percentage of academic appointment, and academic rank.

METHODS

This study used a 5 × 6 × 4 factorial design. The dependent variable was the SPI. Independent variables were academic appointment (instructor or lecturer, clinical specialist, assistant professor, associate professor, and full professor), degree type (BS, MS, MA, MEd, PhD, EdD, and other), and percentage of academic appointment (0% to 25%, 26% to 50%, 51% to 75%, 76% to 100%).

Subjects

The names and addresses of 94 undergraduate and 12 graduate directors of NATA-approved or CAAHEP-accredited entry-level programs and NATA-accredited graduate athletic training programs were obtained from the NATA World Wide Web site (www.nata.org). The program director and any faculty member returning the survey instrument served as subjects. All subjects were faculty members in CAAHEP-accredited entry-level or NATA-approved postcertification graduate athletic training education programs. Six institutions had both undergraduate and graduate programs. In those cases, individuals were represented only once in the data set. Sixty-eight (68%) of the 100 institutions responded to the survey, yielding a total of 200 faculty responses.

Survey Instrument

The data collection instrument was based on the similar studies investigating the scholarly productivity of occupational therapy and physical therapy faculty.[2,5] Demographic information regarding the respondent's highest degree, the year this degree was earned, the year the academic appointment was received, and the current academic rank and tenure status was collected for grouping purposes. The time between the date the survey was received and the most recent date since the respondent's highest degree was earned or the current academic appointment was granted was identified as being the "productivity period." For instance, a person who was employed by the institution in 1990 and received a doctorate in 1995 would have 3 productivity years, as we conducted the survey in 1998.

The remainder of the instrument ascertained the number of scholarly endeavors accomplished during the productivity period. Respondents were asked to identify the number of articles accepted for publication in peer-reviewed and non–peer-reviewed journals, abstracts in peer-reviewed journals, books written or edited, book chapters, platform presentations at district or national athletic training or sports medicine conferences, published book reviews, and the number and dollar amount of external grants awarded. Each institution was assigned a unique code number for matching returns and monitoring return rates.

To determine the instrument's face validity, athletic training and other allied health faculty members pilot tested the survey. Comments were collected, and the appropriate changes were made. The final data collection instruments and methods were approved by the Northeastern University Institutional Review Board.

Table 1. Subject Demographics by Academic Rank

		Mean (SD)		
Academic Rank	n	Productivity Years	Years Since Highest Degree Earned	Years at Current Institution
Instructor or lecturer	64	5.5 (6.8)	7.2 (6.7)	5.8 (6.9)
Clinical specialist	34	5.0 (5.1)	6.8 (5.3)	4.9 (48)
Assistant professor	53	8.3 (7.8)	7.9 (7.5)	7.7 (7.9)
Associate professor	25	14.5 (7.4)	12.1 (8.1)	14.2 (7.6)
Full professor	19	18.5 (9.4)	16.0 (9.0)	17.6 (10.7)

Table 2. Subject Demographics by Percentage of Academic Appointment

		Mean (SD)		
Percentage of Academic Appointment	n	Productivity Years	Years Since Highest Degree Earned	Years at Current Institution
0–25	35	7.2 (7.5)	8.3 (7.3)	7.6 (7.5)
26–50	45	8.3 (9.1)	8.7 (7.9)	7.9 (9.1)
51–75	13	10.0 (7.2)	9.6 (7.4)	9.8 (7.3)
76–100	107	8.8 (8.4)	9.0 (7.8)	8.6 (8.5)

Procedures

The program director was mailed copies of the survey instrument, cover letter, and institutional review board statement, along with a self-addressed, stamped return envelope. The recipient was asked to distribute the instrument to each certified athletic trainer who held a full or partial academic appointment with the program. Completed surveys were to be returned by November 15, 1998. An e-mail was sent as a follow-up to individuals who had not returned their survey by November 25, 1998.

Statistical Procedures

The SPI was calculated by dividing the total number of publications (peer-reviewed journal articles, non–peer-reviewed articles, published abstracts, textbooks and textbook chapters authored or coauthored, and professional presentations) by the number of years in the productivity period. We used a 3-way analysis of variance to detect differences in SPI among the levels of academic appointment, degree type, and percentage of academic appointment. The Tukey honestly significant difference test was used for post hoc comparisons. A probability level of $P < .05$ was used for all tests. The Statistical Package for the Social Sciences for Windows (version 9.0, SPSS Inc, Chicago, IL) was used to perform all statistical tests.

RESULTS

Sixty-eight of the 100 institutions surveyed responded, yielding a 68.0% return rate. Demographic information on the responding faculty and program directors is presented in Tables 1 through 3. The SPIs were different for the levels of academic appointment ($F_{4,121} = 2.4$, $P = .05$) (Table 4). Full professors had a higher SPI than all other groups (Tukey honestly significant difference, $P \leq .05$). Associate professors had higher SPIs than instructors or lecturers and clinical special-

Table 3. Subject Demographics by Degree Type

Degree Type	n	Mean (SD) Productivity Years	Years Since Highest Degree Earned	Years at Current Institution
MS	74	6.4 (7.5)	7.9 (6.9)	6.4 (7.5)
MA	16	11.9 (9.4)	13.9 (8.6)	13.0 (9.5)
MEd	22	6.5 (6.9)	8.6 (7.3)	5.7 (6.4)
PhD	31	9.0 (7.7)	6.6 (6.0)	8.3 (7.7)
EdD	25	14.5 (9.2)	10.6 (9.4)	14.9 (9.4)
Other doctoral degree	11	6.0 (5.2)	6.6 (5.2)	5.8 (4.4)

Table 4. Scholarly Productivity Index by Academic Ranks

Rank	n	Mean (SD)	Minimum	Maximum
Instructor or lecturer*†	52	0.21 (0.58)	0	3.00
Clinical specialist*†	27	0.18 (0.22)	0	0.56
Assistant professor*	53	0.93 (1.57)	0	7.60
Associate professor*	25	1.73 (1.73)	0	5.50
Full professor	18	2.95 (3.93)	0	14.64
Total	175	0.92 (1.88)	0	14.64

*Significantly different from full professors ($P < .05$).
†Significantly different from associate professors ($P < .05$).

Table 5. Scholarly Productivity Index by Degree Type

Degree Type	n	Mean (SD)	Minimum	Maximum
MS	74	0.24 (0.50)	0	3.00
MA	16	0.22 (0.65)	0	2.60
MEd	23	0.18 (0.32)	0	1.12
PhD	31	2.51 (3.13)	0	14.64
EdD	24	2.01 (2.16)	0	7.33
Other doctoral degree	11	0.99 (1.63)	0	5.75
Total	179	0.91 (1.86)	0	14.64

ists, equivalent SPIs to assistant professors, and lower SPIs than full professors. Assistant professors had lower SPIs than full professors but were equivalent to all other groups.

We found no statistical differences among the levels of degree type ($F_{5,121} = 2.0$, $P = .08$) (Table 5) or percentage of academic appointment ($F_{3,121} = 0.26$, $P = .85$) (Table 6). Table 7 presents the rate of various publications by academic rank.

Table 6. Scholarly Productivity Index by Percentage of Academic Appointment

Percentage of Academic Appointment	n	Mean (SD)	Minimum	Maximum
0–25	21	0.44 (0.71)	0	3.00
26–50	44	0.49 (0.92)	0	4.53
51–75	13	0.74 (0.84)	0	2.60
76–100	101	1.21 (2.31)	0	14.64
Total	179	0.91 (1.86)	0	14.64

DISCUSSION

The concept of program directors' obtaining tenured status was first discussed in our professional journal by Perrin and Lephart in 1987.[9] At that time, the authors recognized the conflicts between the criteria required to gain tenure (teaching, scholarship, and service) versus the clinical demands or the rigors of implementing a new academic program that are placed on many program directors. This conflict will become more problematic as program directors are appointed to full-time, tenure-track faculty status.

Several institutional and personal factors influence the scholarly productivity of individual faculty members. Institutional variables include the type of institution,[6] its organizational structure,[10] institutional support,[7] and the leadership style of the department chairperson.[11] Individual variables that affect scholarly productivity include career motivation,[8] academic degree,[6] tenure-track status,[6] sex,[6,7] and family obligations.[7]

The large SDs for SPI based on the type of academic degree and percentage of academic appointment indicate that some individuals were very productive. Other faculty members had zero productivity. The high SDs in both of these areas could have masked true statistical differences in these variables (ie, resulted in low statistical power). Although we did not detect different SPIs associated with academic degree or percentage of academic appointment, institutions typically have different tenure and promotion standards for these groups.

Table 7. Scholarly Productivity Index Elements of Scholarship by Academic Appointment

Scholarship	Mean (SD) Professors (19)	Associate Professors (25)	Assistant Professors (54)	Instructors (64)	Clinical Specialists (34)	Total (196)
Peer-reviewed journal articles						
Primary author	8.68 (10.65)	3.60 (3.84)	1.24 (2.32)	0.17 (0.52)	0.12 (0.54)	1.72 (4.51)
Contributing author	9.68 (19.28)	3.88 (4.60)	1.50 (3.95)	0.20 (0.62)	0.29 (0.72)	1.96 (7.01)
Non–peer-reviewed journal articles						
Primary author	4.47 (6.47)	2.72 (4.58)	1.63 (5.81)	0.20 (0.62)	0.15 (0.44)	1.32 (4.20)
Contributing author	0.26 (0.73)	0.36 (0.81)	1.11 (5.48)	0.11 (0.54)	0.00 (0.00)	0.41 (2.93)
Published abstracts						
Primary author	6.00 (7.78)	3.48 (6.02)	0.78 (1.60)	0.28 (1.88)	0.008 (0.29)	1.35 (3.92)
Contributing author	11.21 (24.44)	3.96 (8.33)	1.43 (3.27)	0.01 (0.30)	0.26 (0.75)	2.06 (8.81)
Textbooks						
Primary author	1.26 (1.76)	0.72 (1.14)	0.17 (0.54)	0.005 (0.38)	0.006 (0.24)	0.29 (0.85)
Contributing author	0.47 (0.90)	0.20 (0.58)	0.01 (0.40)	0.01 (0.12)	0.01 (0.38)	0.12 (0.45)

We were at first surprised that the percentage of academic or athletic department appointment had no influence on the SPI. However, when examining the scholarly productivity of physicians in schools of family practice medicine, Katerndahl[12] found that patient care and scholarly productivity were not dichotomous entities.

Other studies have demonstrated that institutions with graduate degree programs in athletic training tend to produce the bulk of our professional scholarship, and, overall, 15 institutions accounted for more than 42% of the scholarly activity in the *Journal of Athletic Training*.[13] Indeed, a review of the authors published in the *Journal of Athletic Training* indicates that the bulk of scholarly productivity has come from lead authors who were nonacademicians, graduate students, or both. This contribution to our professional body of knowledge should be viewed as a positive attribute, since it serves to further validate our professional practice and skills.

Our data may have been skewed by the influx of new program directors who have recently received or who are currently completing their terminal degrees and who are also responsible for developing accredited programs.[4] New program directors should attempt to negotiate recognition for gaining initial accreditation into their promotion and tenure portfolio and ensure that this effort receives appropriate weighting in the scholarship and service categories. Similarly, athletic training program directors or teaching faculty who have dual appointments but are on the tenure track should ensure that they will be well positioned for their tenure and promotion review. The proportion of time dedicated to the academic and clinical aspects should be weighted accordingly. The individual faculty member must have an appropriate amount of time built into the workload as a portion of the service component and an appropriate amount of time allocated for scholarship.

The granting of tenure is ultimately an institutional fiscal decision. A tenure award reflects the long-term allocation or reallocation of the institution's financial resources.[14] Positive tenure and promotion decisions also indicate institutional support and recognition for the athletic training education program. For most athletic training educators to gain positive promotion and tenure reviews, the expectation is that a high level of scholarship will be maintained. Academicians who have a greater proportion of their workload dedicated to scholarship should be producing research at a higher rate than those who do not.

CONCLUSIONS

We found that full professors had a higher SPI than all other groups. Associate professors had higher SPIs than instructors or lecturers and clinical specialists but not full or assistant professors. Assistant professors had lower SPIs than full professors but were equivalent to all other groups. There was no statistical difference between the type of degree or percentage of academic appointment for the scholarly productivity rate of our respondents.

Historically, many athletic training program directors were primarily employed by the institution's athletic department, shielding them from the tenure process. As athletic training faculty members are integrated into the mainstream of academia, we must be prepared to meet the rigors of the tenure process and be held to the same standards of other allied health faculty members.

Certainly we need the clinical research and scholarship generated by nondoctorally prepared athletic trainers. However, we should begin to expect that the demands of the tenure process manifest as an increased rate of productivity by tenure-track faculty relative to those faculty who are not on the tenure track.

The lack of difference in SPI based on the percentage of academic appointment or type of academic degree does not suggest all should be considered equally in the tenure and promotion process. Institutions do differentiate individuals on these factors through internal policies and procedures. As the role of the athletic trainer educator continues to evolve, we must also see improved scholarly productivity from our faculty members. In the future, our scholarly productivity should improve so that we are able to identify differences associated with the type of academic degree and percentage of academic appointment.

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The Meniscus: Review of Basic Principles With Application to Surgery and Rehabilitation

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Objective: To review basic meniscal anatomy, histology, and biomechanical principles as they apply to surgery and rehabilitation.

Data Sources: We searched MEDLINE and CINAHL for the years 1960–1999 using the terms *meniscus, surgery, rehabilitation, meniscal repair,* and *arthroscopy.*

Data Synthesis: Injuries to a healthy meniscus are usually produced by a compressive force coupled with transverse-plane tibiofemoral rotation as the knee moves from flexion to extension during rapid cutting or pivoting. The goal of meniscal surgery is to restore a functional meniscus to prevent the development of degenerative osteoarthritis in the involved knee. The goal of rehabilitation is to restore patient function based on individual needs, considering the type of surgical procedure, which meniscus was repaired, the presence of coexisting knee pathology (particularly ligamentous laxity or articular cartilage degeneration), the type of meniscal tear, the patient's age, preoperative knee status (including time between injury and surgery), decreased range of motion or strength, and the patient's athletic expectations and motivations. Progressive weight bearing and joint stress are necessary to enhance the functionality of the meniscal repair; however, excessive shear forces may be disruptive. Prolonged knee immobilization after surgery can result in the rapid development of muscular atrophy and greater delays in functional recovery.

Conclusions/Recommendations: Accelerated joint mobility and weight-bearing components of rehabilitation protocols represent the confidence placed in innovative surgical fixation methods. After wound healing, an aquatic therapy environment may be ideal during all phases of rehabilitation after meniscal surgery (regardless of the exact procedure), providing the advantages of controlled weight bearing and mobility progressions. Well-designed, controlled, longitudinal outcome studies for patients who have undergone meniscectomy, meniscal repair, or meniscal reconstruction are lacking.

Key Words: biomechanics, knee anatomy, exercise

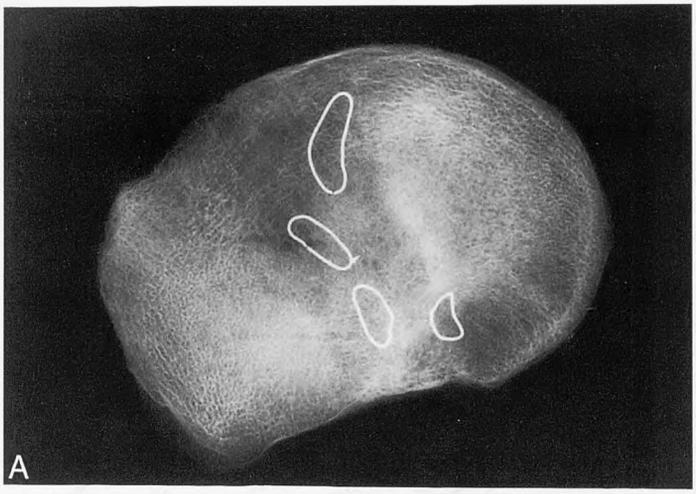
Knee injuries account for approximately 14% to 16% of all musculoskeletal injuries at the high school level.[1,2] The National Athletic Trainers' Association[1] ranked knee injury frequency second to the combined frequency of hip, thigh, and leg segment injuries, whereas the Puget Sound Sports Medicine Group ranked knee injuries second only to ankle injuries.[2] Stocker et al[3] reported that meniscal injuries accounted for 12% of all football knee injuries in a recent high school injury survey. The National Athletic Trainers' Association's high school knee injury survey projected that approximately 9000 knee surgeries are performed annually on high school athletes in the United States.[1] Injuries to a healthy meniscus are usually produced by coupled compressive and rotational tibiofemoral joint forces. These forces tend to "pinch" the menisci as they attempt to rapidly conform to the 3-dimensional joint stresses that arise as the compressively loaded knee internally or externally rotates in the transverse plane during sagittal-plane flexion-extension.[4] These coupled forces commonly occur during athletic movements that require sudden directional changes such as rapid cutting or pivoting.[4]

Instantaneous damage to both ligamentous and meniscal structures is more common than isolated injury. The *unhappy triad* was described by O'Donoghue as an injury to the medial collateral ligament, the anterior cruciate ligament (ACL), and the medial meniscus[5,6]; however, recent reports suggest that the lateral meniscus is more commonly injured.[7–10] Acute ACL disruption associated with sudden transverse-plane rotary forces more commonly damages the lateral meniscus as excessive lateral compartment compression and shear forces stress the posterolateral tibiofemoral articulation.[9] Medial meniscus injury is usually associated with repetitious anterior translation in the chronic ACL-deficient knee, disrupting articular surfaces[11] and leading to the early onset of osteoarthritis (OA).[12]

Knee injury management is a concern for most sports medicine health care providers. Our objective is to provide a review of basic anatomic, histologic, and biomechanical principles of the meniscus. This information is then assimilated with current surgical and rehabilitation methods to provide clinicians with a complete overview of the present state of meniscal injury management. The ultimate challenge is to return the athlete to sport with normal or optimal (given the extent of the initial lesion and the surgical method) meniscal function.

ANATOMY

The menisci extend the superior tibial surface, improving its congruency with the femoral condyles.[13,14] Both menisci



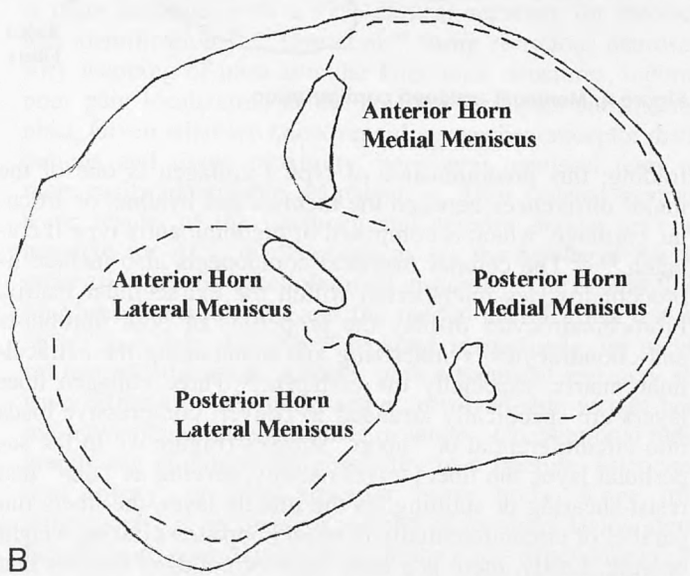


Figure 1. A, Radiographic anterior and posterior meniscal horn locations. B, Meniscal horn identification in relation to the lateral and medial meniscus (dotted outlines).

are fibrocartilaginous and wedge shaped in the coronal plane. The medial meniscus is more crescent shaped, and the lateral meniscus is more circular. The superior portions of the menisci are concave, enabling effective articulation with their respective convex femoral condyles, whereas the inferior surfaces are flat to conform to the tibial plateaus. Anterior and posterior meniscal horns attach to the intercondylar eminence of the tibial plateau (Figure 1). The coronary ligaments provide peripheral attachments between the tibial plateau and the perimeter of both menisci. The medial meniscus is also attached to the medial collateral ligament, which limits its mobility. The lateral meniscus is connected to the femur via the anterior (ligament of Humphrey) and posterior (ligament of Wrisberg) meniscofemoral ligaments, which can tension its posterior horn anteriorly and medially with increasing knee flexion.[15,16] The transverse ligament provides a connection between the anterior aspects of both menisci. The increased stability provided by the ligamentous attachments prevents the menisci from being extruded out of the joint during compression.[17–22]

Vascular Anatomy

Vascular supply is crucial to meniscal healing. The medial, lateral, and middle geniculate arteries, which branch off the popliteal artery, provide the major vascularization to the inferior and superior aspects of each meniscus (Figure 2).[23–27] The middle geniculate artery is a small posterior branch that pierces the oblique popliteal ligament at the posteromedial corner of the tibiofemoral joint. A premeniscal capillary network arising from branches of these arteries originates within the synovial and capsular tissues of the knee along the periphery of the menisci. Only 10% to 30% of the peripheral medial meniscus border and 10% to 25% of the lateral meniscus border receive direct blood supply.[23,24] Endoligamentous vessels from the anterior and posterior horns travel a short distance into the substance of the menisci and form terminal loops, providing another direct route for nourishment.[23] The remaining portion of each meniscus (65% to 75%) receives nourishment only from the synovial fluid via diffusion.[28,29]

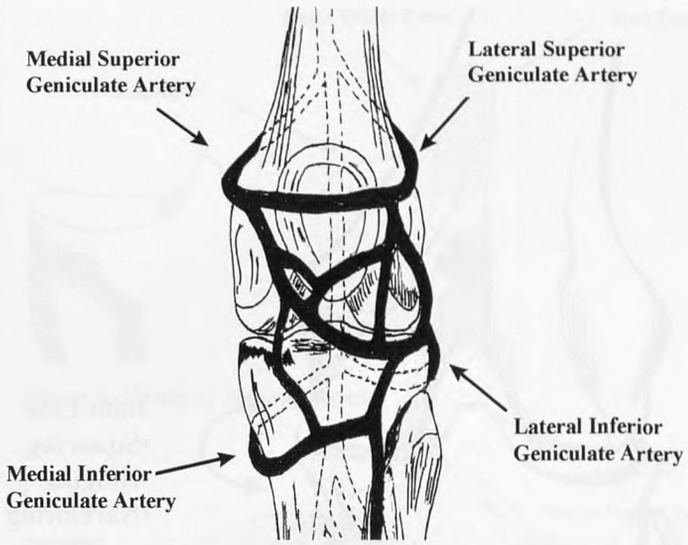


Figure 2. Confluence of geniculate arteries (anterior view).

Neuroanatomy

The knee joint is innervated by the posterior articular branch of the posterior tibial nerve and the terminal branches of the obturator and femoral nerves. Nerve fibers penetrate the joint capsule, along with the vascular supply, and service the substance of the menisci. Ruffini, Pacinian, and Golgi tendon mechanoreceptors have been identified in the knee joint capsule and in the peripheral menisci.[30–36] Type I (Ruffini) mechanoreceptors are low threshold and slowly adapting to changes in static joint position and pressure. Type II (Pacinian) mechanoreceptors are low threshold and fast adapting to tension changes, signaling joint acceleration.[30–36] Type III (Golgi) mechanoreceptors signal when the knee joint approaches the terminal range of motion (ROM) and are associated with neuromuscular inhibition. Concentrations of meniscal mechanoreceptors (especially Pacinian mechanoreceptors) are greatest in the meniscal horns, leading researchers to study their contributions to proprioception.[30–36]

BIOMECHANICS

The major meniscal functions are to distribute stress across the knee during weight bearing,[37,38] provide shock absorption,[37,39,40] serve as secondary joint stabilizers,[41–44] provide articular cartilage nutrition and lubrication, facilitate joint gliding, prevent hyperextension, and protect the joint margins.[17,18]

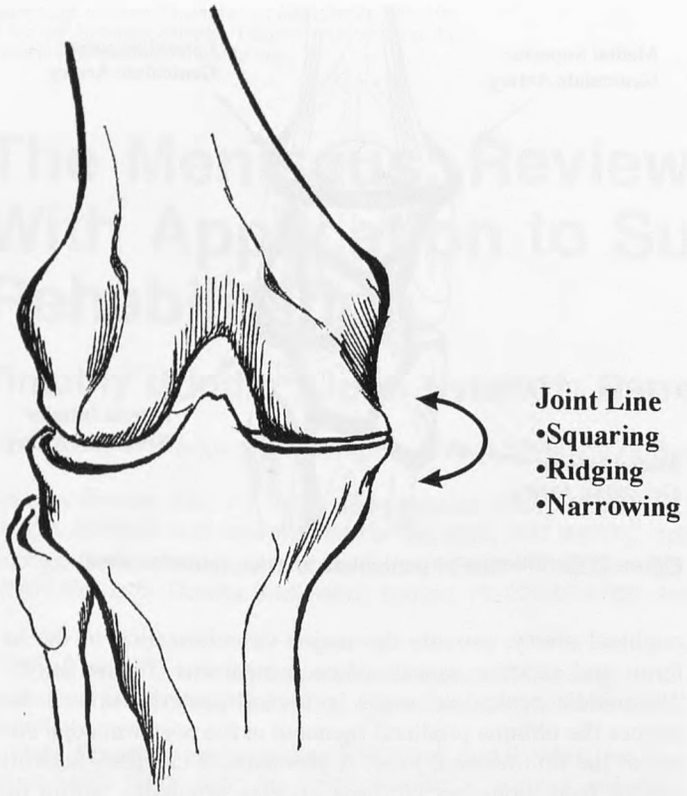


Figure 3. Medial joint line degeneration: the Fairbank sign.

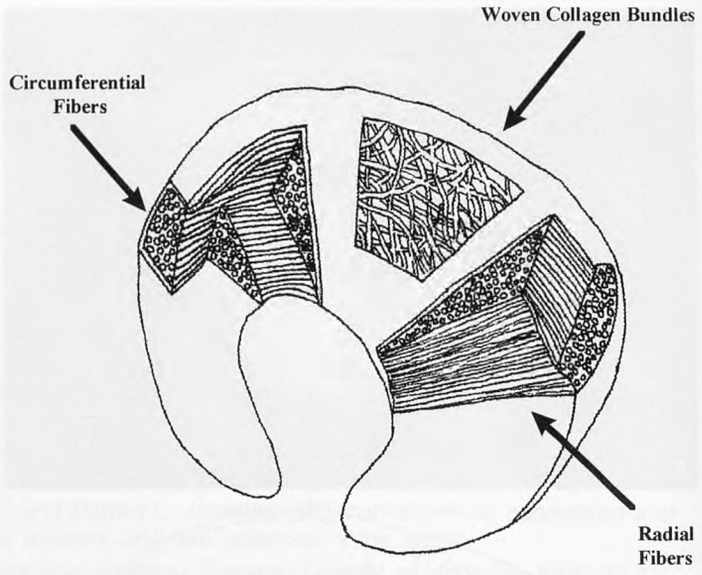


Figure 4. Meniscal collagen configuration.

Circumferential meniscal stress measurements have shown that 45% to 70% of the weight-bearing load is transmitted through the menisci when the peripheral margins are intact.[17,18,21,29,39] The biomechanical adaptations of the meniscectomized knee show a doubling of joint contact stress in conjunction with a 50% to 70% reduction in contact area.[45] A 10% reduction in meniscal contact area secondary to partial meniscectomy reportedly produces a 65% increase in peak joint-contact stresses,[45] leading to the early development of OA.[46,47] Radiographically, the Fairbank sign indicates joint space narrowing due to osteophyte formation from increased peak joint stresses (Figure 3).[46]

During knee flexion, the femoral condyles glide posteriorly on the tibial plateau in conjunction with tibial internal rotation. The lateral meniscus undergoes twice the anteroposterior translation of the medial meniscus during knee flexion (11.2 mm versus 5.1 mm).[22] This translation prevents the femur from contacting the posterior margin of the tibial plateau. The medial condyle rolling-to-translation ratio is 1:1, whereas the lateral condyle ratio is 1:4.[19] The lateral meniscus can better accommodate this mobility by translating with the femoral condyles and is thereby less susceptible to injury than the medial meniscus.[19] The congruity of the tibiofemoral articulation is maintained throughout complete knee ROM via healthy, mobile menisci.[13,48]

HISTOLOGY AND STRUCTURE

The microstructural characteristics of the menisci dictate their mechanical properties. The menisci are composed of 70% water and 30% organic matter. Collagen constitutes 75% of the organic matter, while roughly 8% to 13% of the remaining dry matter consists of noncollagenous proteins.[39,49–52] Type I collagen fibers provide the primary meniscal structural scaffolding; this predominance of type I collagen is one of the major differences between the menisci and hyaline, or articular, cartilage, which is composed of predominantly type II collagen.[48,53] The cellular meniscal components also include fibrochondrocytes interspersed within the extracellular matrix. Fibrochondrocytes display the properties of both fibroblasts and chondrocytes, synthesizing and maintaining the extracellular matrix, especially the collagen.[48] Three collagen fiber layers are specifically arranged to convert compressive loads into circumferential or "hoop" stresses (Figure 4). In the superficial layer, the fibers travel radially, serving as "ties" that resist shearing or splitting. In the middle layer, the fibers run parallel or circumferentially to resist hoop stress during weight bearing. Lastly, there is a deep layer of collagen bundles that are aligned parallel to the periphery.[54] The remainder of the extracellular matrix is composed of proteoglycans. The glycosaminoglycans (GAGs), or chains of proteoglycan aggregates, make up only 1% of the wet weight of the meniscus but contribute most to its material properties, such as tissue hydration, compressive stiffness, and elasticity.[28,55] The size of these proteoglycan macromolecules in combination with water-retention and electrostatic-repulsion properties is what gives the menisci their compressive stiffness.[10,55] Meniscal shock absorption is time dependent due to the exudation of water out of the extracellular matrix. The exudation of water from the GAG substances provides not only compressive stiffness but also joint lubrication as water is forced into the joint space. The highest GAG concentrations are found in the meniscal horns and the inner half of the menisci, coinciding with the primary weight-bearing areas.[56] Meniscal tissue also displays the time-dependent viscoelastic property of "creep," deforming over time when loading occurs with greater frequency or duration. The proteoglycans add little to meniscal tensile properties. Rather, elastin, which constitutes less than 0.06% of meniscal tissue, is believed to aid in the recovery of shape after load deformation.[55]

CLINICAL EXAMINATION

The need for surgery after meniscal injury is largely determined from the data obtained during the initial physical examination in conjunction with other diagnostic tests. A com-

prehensive examination should include a thorough injury history, regional palpation, and select special tests.[4] Athletes with meniscal tears commonly describe feeling a pop while performing a sudden running directional change, such as rapid cutting or pivoting, with or without contact with another player.[4] Johnson et al[57] reported that statistical methods applied to medical historical data were 85% to 98% accurate for predicting the presence of a meniscal tear, depending on whether 30 or 142 predictor questions were used. Knee joint-line tenderness and effusion are also associated with meniscal lesions. Knee joint-line palpation may produce equivocal results, with medial joint-line specificity of 34.5% and sensitivity of 44.9% for predicting medial meniscal tears and lateral joint-line specificity of 49.1% and sensitivity of 57.6% for predicting lateral meniscal tears in subjects with acute ACL injuries.[58] Among subjects with a nonimpaired ACL, knee joint-line tenderness is more accurate, with a 77% clinical accuracy for meniscal tear identification.[59,60] Dye et al,[61] using conscious neurosensory mapping of intra-articular knee joint structures, reported poor pain localization at the cruciate ligaments and the menisci. Given what we know regarding mechanoreceptor distributions and tissue proximity, peripheral meniscal tears are more easily identifiable via palpation. After isolated meniscal tears, results of the Lachman and anterior drawer tests are negative for ACL involvement, as are the results of the apprehension test for patellofemoral instability. The results of the McMurray rotation test and the medial-lateral grind test are 58%[60] to 83%[62] and 68%[60] accurate, respectively, for meniscal tear identification. Athletes with a meniscal tear may also have difficulty performing active, involved-side weight-bearing movements, such as squats or lunges. Conventional radiographs can eliminate the possibility of a fracture, osteochondral injury, or intra-articular loose body. Knee joint arthrography is an invasive method of meniscal lesion identification with poor diagnostic accuracy rates, depending greatly upon the skills and experience of the examiner.[63] Magnetic resonance imaging, with accuracy rates of 90% to 98% for the identification of meniscal tears, has become the radiographic procedure of choice; however, it is more costly than arthrography or conventional radiographic evaluation.[64]

MENISCECTOMY VERSUS REPAIR

The first reported surgical human meniscus repair occurred in 1885 and was described as tedious.[65] Because meniscal repair was considered technically challenging and the meniscus was viewed as a vestigial structure, total meniscectomy became the preferred operation. The early onset of OA was the long-term result of total meniscectomies, prompting the exploration of other surgical options. The partial meniscectomy has since replaced the total meniscectomy as the surgery of choice, along with other options such as repairs and transplantations. Before selecting a particular surgical technique, the surgeon considers the patient's age, health, lifestyle, and willingness to undergo major surgery and the location and type of meniscal tear.[66–70] Patients should be educated about the pros and cons of meniscal resection or repair and the extent of their rehabilitation obligation. Older or more sedentary patients are generally more effectively treated with a conventional partial meniscectomy. Patients should also be informed as to the likelihood of surgical success based on which meniscus was injured and the type of tear that occurred. Gillquist and Oretorp[71] reported that patients who underwent partial lateral

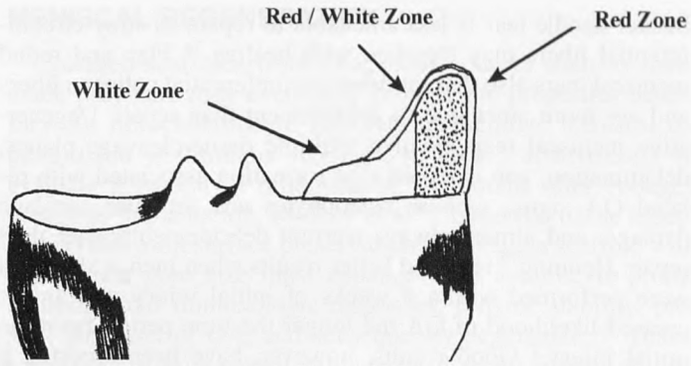


Figure 5. Meniscal healing zones.

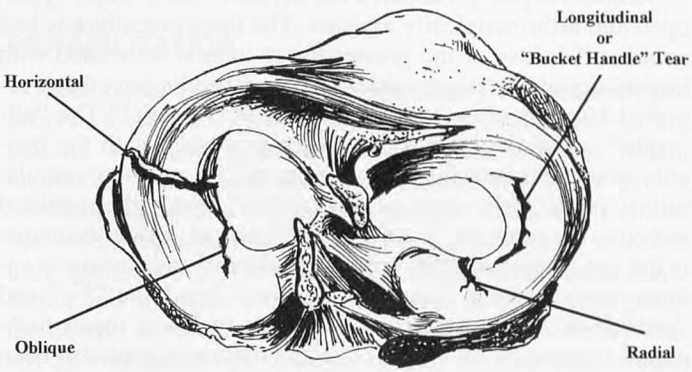


Figure 6. Types of meniscal tears.

meniscectomy did less well than those who underwent partial medial meniscectomy. Northmore-Ball and Dandy[72] reported a slightly greater frequency of excellent clinical results after partial medial meniscectomy than after partial lateral meniscectomy. Patients older than 50 years are considered ideal candidates for partial meniscectomy because they are likely to have degenerative meniscal tissue associated with OA. Degenerative meniscal tears display poor repair potential due to the insufficient tissue integrity of both the lesion site and the adjacent meniscal tissue. Some authors have also recommended partial meniscectomy as the surgical treatment of choice for patients older than 30 years.[73]

The major determinants of whether a meniscal tear is amenable to surgical repair are the location of the tear, the type of lesion, and its related vascular supply. Three zones determine the healing prognosis for meniscal lesions: red-red, red-white, and white-white (Figure 5). The red-red zone is fully vascular and therefore has an excellent healing prognosis. The red-white zone is at the border of vascular supply and has a generally good healing prognosis. The white-white zone is relatively avascular and has a poor prognosis for healing.[74] Arnoczsky and Warren[75] and Weiss et al[76] have substantiated these findings in studying the contributions of the peripheral microvascular supply to the menisci. DeHaven and Stone[77] have suggested that meniscal repairs be performed within 3 mm of the vascular periphery.

Longitudinal meniscal tears occur parallel to the direction of the circumferential fibers. A bucket-handle tear is a variant of a longitudinal tear in that the circumferential fibers are also disrupted as the tear travels from the innermost aspect of the meniscus toward the periphery (Figure 6). The vertical longitudinal meniscal tear is considered ideal for repair because of minimal circumferential fiber disruption.[74] In contrast, the

bucket-handle tear is less amenable to repair, as stray circumferential fibers may interfere with healing.[74] Flap and radial meniscal tears also disrupt these circumferential collagen fibers and are more amenable to debridement than repair. Degenerative meniscal tears involve multiple tissue-cleavage planes, delamination, and calcified cyst formation associated with related OA signs, such as osteophytes and articular cartilage damage, and almost always warrant debridement rather than repair. Henning[78] reported better results when meniscal repairs were performed within 8 weeks of initial injury and an increased likelihood of OA the longer the time period from the initial injury.[7] Good results, however, have been reported 8 weeks or more after initial injury, provided the location and meniscal lesion type met the repair criteria.[69]

Meniscal repair procedures are divided into 2 major types: open and arthroscopically assisted. The open procedure is less common because of the greater tissue trauma associated with larger surgical incisions, although good results have been reported 10 years after open medial meniscal repair.[78] The "all-inside" technique is an arthroscopic procedure with the benefits of smaller incisions and a reduced risk of neurovascular injury, particularly when peripheral tears within the meniscal red zone are repaired.[79] A variation of the all-inside technique is the use of permanent[80] or biodegradable[81] transmeniscal sutures requiring only 1 surgical incision. "Inside-out"[82,83] and "outside-in"[84] arthroscopically assisted meniscal repair techniques (named by the origin of suture delivery) have also been reported; however, they may place adjacent neurovascular structures at a greater risk of injury.[23,68] Neurovascular complications generally involve lower extremity weakness and sensory loss; therefore, regular postoperative monitoring of neurovascular integrity should be performed.

MENISCAL REPAIR FACILITATORS

The poor healing of white-white zone meniscal lesions has prompted researchers to explore options to enhance healing. Presently, 3 methods are commonly used to enhance healing after meniscal repair: fibrin clot injection, vascular access channel creation, and synovial abrasion.[37,50,77,85]

Fibrin Clot

A fibrin clot can be injected into the meniscal lesion to promote healing through hematoma chemotactic factors.[50] Arnoczky et al[50] injected a fibrin clot matrix into the meniscal defects of dog knees and reported healed tissue resembling normal meniscus 6 months later. Hashimoto et al,[86] using similar methods and mechanical testing, reported that the healed meniscus was less able to resist deformation than normal tissue. The results of both studies suggested that adjacent meniscal fibrochondrocytes or surrounding synovial fluid provided the primary biological stimulus for tissue repair. Henning et al[87] used a fascial sheath to cover fibrin clots placed in human meniscal defects and observed that 32% of the defects healed completely and 52% healed incompletely by 6 months after surgery.

Vascular Access Channels

Vascular access channels (trephination) are tunnels created from vascular portions of the peripheral meniscus (red zone) to the more central avascular area (white zone). Theoretically, trephination enables fibrovascular scar proliferation in the damaged meniscal section. Fox et al,[88] using a patient survey and clinical examination, reported good to excellent results for 90% of patients with incomplete meniscal tears treated with trephination. Using a goat model, Zhang et al[89] reported at least partial healing by 25 weeks after combined trephination and meniscal defect suturing of longitudinal tears in the avascular area.

Synovial Abrasion

Abrasion of the synovium with a surgical rasping device activates chemotactic factors that stimulate meniscal healing. Excessive synovectomy during meniscal debridement has been shown to prevent meniscal regeneration.[90] Synovial cell migration to the meniscal defect may enhance healing, with less effective healing occurring when the distance between the abraded synovium and the defect is increased.[91] Surgeons generally abrade the margins and superficial layer of the meniscal tear to further promote healing.[83,92–94]

LASERS

Lasers are more commonly being used in the ablation, or destruction, of damaged meniscal material during arthroscopic meniscectomies. The proposed mechanisms of ablation are photothermal, photochemical, and photomechanical, with each mechanism having different biological effects.[95] The photothermal effect of long-pulse, continuous-wave lasers can vaporize tissue. Photochemical effects occur from the dissociation of molecular tissue bonds, also called *photoablative decomposition*. Photomechanical effects occur from tissue exposure to short laser pulses, which stress the tissue beyond its mechanical strength. An example of an excessive photomechanical effect is the thermoelastic expansion caused by bubble formation within meniscal tissue after laser intervention.[96] Tissue cavitation in the presence of tensile stresses can lead to further degenerative changes.[96] Forman et al[97] studied the use of a laser to promote in vitro healing in human menisci that had also received a fibrin clot and reported that the laser helped prevent clot matrix displacement, allowing more time for fibrochondrocyte absorption into the meniscal defect.

MENISCAL RECONSTRUCTION

A meniscal allograft is donated from a cadaver and transplanted into an injured knee. There are 4 types of meniscal allograft preparations: fresh, deep frozen, cryopreserved and freeze dried. Each preparation has pros and cons. Although the fresh meniscal allograft contains functioning fibrochondrocytes, the window of opportunity for transplantation is only a few days. Also, the threat of human immunodeficiency virus (HIV) infection is a possibility when fresh tissue is used (approximately 1 person in 22 629 has a positive test result for the virus).[98] Both the deep-frozen and cryopreservation methods eliminate the threat of HIV infection, but they also reduce the amount of viable meniscal tissue and alter its biomechanical properties. Both alteration of the tissue biomechanical properties and improper allograft sizing can lead to postoperative failure. Velteri et al,[99] using a dog model, reported that cellular components in both cryopreserved and deep-frozen meniscal allografts were eventually fully replaced.

Fresh Allografts

Garrett and Stevensen[100] reported that fresh human allograft fixation by peripheral suturing to surrounding fibrous tissue resulted in no evidence of meniscal degeneration at 44 months after implantation; however, radiographic assessment revealed joint space narrowing on the side of the transplant. Biopsies revealed that transplanted menisci retained their original size and shape, suggesting that the chondrocytes continued to produce glycoproteins.[100] Jackson et al[101] reported normal vascular distributions in meniscal allografts up to 6 months after transplantation but noted decreased water and proteoglycan content and overall cellularity. Keating,[102] using a goat model, reported an inflammatory infiltrate at 3 months after transplantation and articular destruction at 7 months, suggesting failure linked to immune responses. Urban et al[103] emphasized proper meniscal horn placement when attempting to restore functionality to a transplanted meniscus, regardless of the preparation method. The long-term effectiveness of fresh meniscal transplants will not be fully realized until a long-term longitudinal study is completed.

Deep-Frozen Allografts

Meniscal transplantation using frozen donor tissue is probably the simplest and least expensive method; however, the process is known to destroy donor fibrochondrocytes and partially shrink the graft tissue.[104,105]

Cryopreserved Allografts

During cryopreservation (Cryolife, Marietta, GA), the graft tissue is frozen in glycerol, thereby preserving cell membrane integrity and donor fibrochondrocyte viability.[105] The graft is gradually thawed before transplantation. Cell viability may vary depending on graft size, preservation medium, and freezing or thawing rate.[106] Cryopreservation allows for longer graft storage durations, more time for serologic testing, and more precise sizing, but it is expensive and technically challenging. Arnoczsky et al[107] used a dog model to study the histologic properties of cryopreserved meniscal tissue and reported that the number of metabolically active cells after transplantation decreased and cellularity and peripheral vascularity increased by 3 months after surgery. Fissuring of the adjacent tibial condyle hyaline cartilage was also evident at 6 months after surgery.[107] Speculation continues over the long-term effects of cryopreserved meniscal allografts, and controlled longitudinal studies are needed.

Freeze-Dried Allografts

Freeze-dried grafts are frozen after vacuum dehydration. Before transplantation, the graft is thawed and rehydrated, tending to increase graft fragility. After transplantation, the graft serves as a scaffold for the ingrowth of host fibrochondrocytes. Freeze-dried meniscal allografts provide ease in handling and prolonged storage life at room temperature.[108] However, the results from freeze-dried meniscal allograft transplantation have not been encouraging because of histologic alterations, such as cartilaginous degeneration and synovitis.[108] Since the freeze-drying process does not destroy HIV in blood products, disease transmission is also possible.

MENISCAL REGENERATION

Collagen scaffolds from exogenous sources such as man-made polymers may eventually provide the properties necessary for fibrochondrocyte ingrowth to facilitate meniscal regeneration in humans. Using "re-look" arthroscopy to evaluate 8 patients a minimum of 24 months after collagen meniscus implantation, Rodkey et al[109] observed tissue regeneration and joint surface preservation. A collagen scaffold with appropriate pore size must enhance fibrochondrocyte proliferation, avoid immunologic responses, provide stability, prevent the onset of OA, and subsequently degenerate.[109] Histologic studies have shown variable results, and further clinical trials are needed to evaluate the ability of scaffolds to protect the articular cartilage of the human femoral condyle.

REHABILITATION

Rehabilitation after partial meniscectomy can generally progress as tolerated with no substantial contraindications or limitations. The main goals are to control the pain and inflammation associated with surgery, maintain ROM and general conditioning, restore or maintain isolated muscle function, and optimize integrated lower extremity neuromuscular coordination. Immediate progressive ROM and neuromuscular reeducation and strengthening are warranted. A concurrent goal is the control of effusion, pain, and inflammation with cryotherapy and the use of nonsteroidal, anti-inflammatory medication. Clinicians should be vigilant in assessing changes in patellofemoral, patellar tendon, and tibiofemoral joint line irritability via regular palpation, particularly after advancing an existing exercise program.

In a prospective, randomized study, Jokl et al[110] found that a well-planned, unsupervised rehabilitation program enables patients undergoing arthroscopic knee surgery to return to sports within the same time frame as patients who receive supervised physical therapy. In contrast, Moffet et al,[111] in a randomized, controlled study, showed that patients who received supervised rehabilitation had more rapid recovery of the quadriceps femoris muscle than did patients in an unsupervised control group; the authors concluded that early and intensive rehabilitation was vital to successful functional outcomes after partial meniscectomy.[111] Matthews and St-Pierre[112] reported that patients require 4 to 6 weeks for the quadriceps femoris and 4 weeks for the hamstrings to return to preoperative isokinetic strength levels after partial meniscectomy. St-Pierre[113] suggested that preoperative knee extensor-flexor strength deficits increase the need for supervised rehabilitation. Clinicians should be familiar with the specifics of the aforementioned surgical procedures to safely advance and, if necessary, modify rehabilitation program progressions. Most progressions focus on 2 major areas: immobilization and weight-bearing status.

Immobilization

Immobilization after meniscal repair has been recommended at or near full extension (10° to 20° of flexion) to better approximate longitudinal tears.[83,84,93,94] Zhang et al,[114] using a rabbit model, reported that immobilization has a greater influence than suture use on the healing rate of meniscal tears. Studies in dogs have substantiated this finding, with excellent healing rates using only immobilization.[115] Eriksson and Haggmart,[116] however, reported that atrophy during immobi-

lization can decrease the human quadriceps femoris mass by 40% by 5 weeks after major knee surgery. Traditionally, a brief period (4 to 6 weeks) of decreased mobility after meniscal repair is recommended; however, the authors of more recent reports recommend decreasing the immobilization period, even in the absence of sutures.[92,93,117] Those authors advocating early ROM have not reported deleterious effects on meniscal repairs[118] and have suggested that this approach improves articular cartilage health.[119]

Weight Bearing

The findings of many studies support weight-bearing limitations during the initial 4 to 8 weeks after meniscal repair.[77,83,92–94] In theory, weight bearing alone should not disrupt healing meniscal tissue, because the hoop stresses are primarily absorbed at the periphery of the meniscus. More recent reports have recommended earlier weight bearing to promote the restoration of a functional meniscus via the clinical application of Wolff's law.[120] Weight bearing in conjunction with tibiofemoral rotation during knee flexion, however, could produce shear forces capable of disrupting healing meniscal tissue, particularly if the fixation strength is inadequate.[121]

ACCELERATED REHABILITATION

DeHaven and Bronstein[122] described a meniscal repair rehabilitation protocol of an initial 2 weeks of maximum protection (immobilization at 0° of flexion, toe-touch weight bearing), 4 weeks of protected ROM (30° to 70° of flexion), and controlled knee extensor-flexor strengthening and full weight bearing after 6 weeks. Stationary cycling and moderate-intensity running were allowed between 3 and 6 months after surgery, and full return to activity was allowed at 7 months after surgery. Although the success rates for protocols similar to the protocol suggested by DeHaven and Bronstein[122] were consistently high (75% to 95%), more recent reports suggest that earlier application of controlled stress to the repaired meniscus may enhance its functionality.[68,71,83,92,94,117,123] Barber[124] reported no differences in healing rates between patients who followed a standard rehabilitation program (protective) and patients who followed a program that permitted immediate weight bearing, unbraced motion, unlimited exercise performance, and an early return to pivoting-type sports movements. Barber[124] emphasized avoiding standard "cookbook" protocols and encouraged individualized programs based on the type of surgical procedure, which meniscus was repaired, the presence of coexisting knee pathology (ie, ligamentous laxity or OA), meniscal tear type, the patient's age, preoperative knee status (including the time between injury and surgery), loss of ROM and strength, and the patient's athletic expectations and motivations.

Accelerated meniscal repair rehabilitation programs that permit full knee ROM and full weight bearing are becoming more common, with return to full activity as early as 10 weeks after surgery.[110,120,124] Although studies have documented short-term successes, controlled longitudinal studies are needed to determine long-term outcomes. Barber and Click,[125] in evaluating 63 patients with 65 meniscal repairs at a minimum of 2 years after surgery, reported successful healing in 92% of patients (53 of 58) who had an ACL reconstruction during the same surgery but in only 67% of patients (2 of 3) who were not treated for ACL deficiency. The authors suggested that patients who undergo combined ACL reconstruction and meniscal repair can safely follow the same accelerated protocol as patients who only undergo meniscal repair.[125] Shelbourne et al[120] reported that patients undergoing combined ACL reconstruction and meniscal repair who perform immediate postoperative ROM and weight bearing as tolerated have comparable clinical results to patients who follow a more restrictive rehabilitation protocol. Mintzer et al[126] assessed the return-to-activity level in 29 patients who were 17 years of age or younger at the time of meniscal repair. At an average of 5 years after surgery, 100% of the patients had excellent clinical results (full ROM, no effusion, no joint-line tenderness, no joint locking), and 85% had returned to sports with cutting or pivoting components.[126] To date, the follow-up period for accelerated rehabilitation after meniscal repair is too brief to declare its superiority over more traditional progressions.[126]

Given the concerns over attempting to provide an ideal weight-bearing and knee ROM progression, aquatic therapy may provide an excellent rehabilitation method following meniscal repair (after surgical wound healing). Tovin et al[127] and Kuhne and Zirkel[128] reported on the efficacy of aquatic therapy for patients after ACL reconstruction. By varying water depths and using flotation devices, weighted devices (vests, belts), and resistive devices (tubing, vented fins, etc), patients can safely progress from knee ROM and neuromuscular recovery activities to more aggressive muscle strength and endurance challenges and sport-specific functional movement patterns (eg, hopping and jumping tasks). An aquatic environment is also a relatively safe exercise location for patients requiring long-term restriction from the compressive forces associated with dry-land running and jumping.

SUMMARY

A comprehensive knowledge of the meniscus is necessary to effectively manage the rehabilitation of patients after meniscal injury and surgery. The unique biomechanical and histologic properties of the meniscus must be preserved to maintain knee health. Clinicians who rehabilitate patients with meniscal injuries should be familiar with normal meniscal anatomy, physiology, and biomechanics as they apply to surgery and rehabilitation. Historically, the lack of appreciation for normal meniscal function resulted in total surgical removal, prompting a proliferation of knee joint OA. Innovations in surgical techniques have led to increased meniscal tissue preservation to minimize the long-term sequelae after injury. In addition to the current interest in repair and transplantation, interest is growing in the use of exogenous materials to facilitate meniscal regeneration and the use of tissue growth factors and even gene therapy to restore a functional meniscus. Surgical innovations are progressing more rapidly than the acquisition and interpretation of long-term surgical and rehabilitation outcome data. Well-designed, longitudinal studies of surgical and rehabilitation outcomes are imperative to determine the actual efficacy of any of these procedures with regard to patient function and satisfaction.

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Madelung Deformity in a Collegiate Gymnast: A Case Report

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Objective: To present the case of a 21-year-old female collegiate gymnast with acute left wrist pain.

Background: Madelung deformity is a developmental abnormality of the wrist. It is characterized by anatomic changes in the radius, ulna, and carpal bones, leading to palmar and ulnar wrist subluxation. It is more common in female patients and is usually present bilaterally. The deformity usually becomes evident clinically between the ages of 6 and 13 years.

Differential Diagnosis: Traumatic distal radius physeal arrest, congenital anatomic variant.

Treatment: The athlete was treated with symptomatic therapeutic modalities and nonsteroidal anti-inflammatory medication for pain. She was able to continue to participate successfully in competitive gymnastics, minimally restricted, with the aid of palmar wrist tape and a commercially available wrist brace to prevent end-range wrist extension.

Uniqueness: Madelung deformity can result in wrist pain and loss of forearm rotation, leading to decreased function of the wrist and hand. This patient was able to participate successfully in elite- and college-level gymnastics with no wrist pain or injury until the age of 21 years. Furthermore, she was able to continue to participate, experiencing only periodic pain, with the aid of taping and bracing support and without the need for reconstructive surgery.

Conclusions: Although rare, Madelung deformity is typically corrected surgically in athletes with chronic pain and disability. This case demonstrates an example of successful conservative management in which the athlete continued to participate in sport.

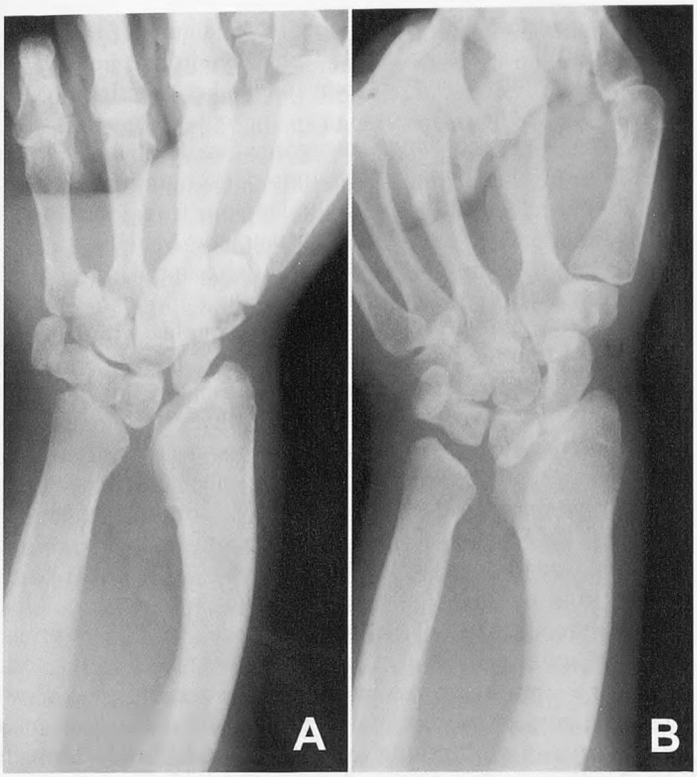
Key Words: traumatic physeal arrest, triangular fibrocartilage complex

In competitive gymnastics, the upper extremity is subject to tremendous torsional forces with axial loading due to repetitive weight bearing.[1–3] As a result, wrist pain is a common complaint among both male and female gymnasts. Of particular concern, the tremendous compressive forces gymnasts often incur at the wrists may lead to a premature, asymmetric closure of the cartilaginous distal radius physis in skeletally immature athletes.[4] Epiphyseal plate changes at the distal radius have been reported in more than 42% of male and female preadolescent and adolescent gymnasts.[1] Such changes are thought to be the direct result of forces imposed during normal gymnastics activity. Closure of the plate may produce a characteristic skeletal deformity in which the carpus is wedged between a deformed distal radius and ulna. Termed an *acquired Madelung-like deformity* because of its similar appearance to the relatively uncommon developmental malformation, such an injury typically presents early in a gymnast's career, between the ages of 6 and 13 years.[5,6] Additionally, it is more common in female than male gymnasts, usually presents bilaterally, and typically results in significant pain and disability.[5,6] In this report, I describe unilateral, nondominant left wrist Madelung deformity in a female collegiate gymnast with no history of wrist injury or pain despite 17 years of competitive gymnastics participation.

REPORT OF A CASE

A 21-year-old female art major and competitive collegiate gymnast (height, 1.39 m; weight, 49.5 kg) experienced a painful sensation of instability and "giving way" in her left, nondominant wrist while practicing an uneven parallel bar routine during a regularly scheduled team practice. The athlete had participated in competitive gymnastics since the age of 4 years and could recall no incidence of wrist pain before her college career. Furthermore, she could recall no previous injury to the left wrist but had received treatment to her right wrist more than 1 year earlier. The athlete had undergone 3 treatments during 1 week, consisting of ice and manual joint traction, for what had been assessed as a right dorsal capsular wrist sprain. During that week, her symptoms improved and treatment was discontinued without further complaint. She could not recall a particular mechanism of injury, but she did describe an abnormal sensation during the lower half of a typical giant swing. The athlete was evaluated immediately by the team's certified athletic trainer. Gross examination revealed bilateral palmar subluxation with associated radial and ulnar styloid process prominence. On evaluation, the athlete demonstrated no remarkable carpal instability compared with the contralateral side; however, she did note deep dorsal wrist tenderness that was significantly worsened with palpation. Additionally, the athlete demonstrated a loss of approximately 5° of pronation and 10° of supination compared with the contralateral side. She was removed from practice and referred to the team orthopaedist for further evaluation.

Before the scheduled consultation with the team orthopaedist, the athlete was not allowed to impact load or grip with



A, Left wrist anterior-posterior radiograph. B, Clenched-fist anterior-posterior radiograph revealing radial bow deformity and Madelung deformity.

her upper extremity but did continue to participate otherwise in regularly scheduled gymnastics practices. After the initial injury, the dorsum of the left wrist became moderately swollen and increasingly tender to palpation. A treatment protocol was instituted twice daily for pain and inflammation control and consisted of ice-bag application, cold whirlpool immersion, transcutaneous electric nerve stimulation therapy, and oral nonsteroidal anti-inflammatory medication. Manual joint traction was also attempted but subsequently discontinued because of increased pain.

Three days after the injury, plain radiographs were obtained, and the athlete was evaluated by the team orthopaedist. Plain anterior-posterior, lateral, oblique, and closed-fist anterior-posterior radiographs revealed a Madelung deformity with a widened distal radioulnar joint (Figure). Physical examination revealed palmar subluxation with associated prominence of the radial and ulnar styloid processes, marked pain with passive pronation, and manual subluxation of the distal ulna volarly but no limitation in motion compared with the opposite side. Tenderness was evident on the ulnar side of the wrist over the dorsum of the triangular fibrocartilage complex and the dorsal aspect of the distal radioulnar joint. Results of tests for carpal instability, including scapholunate dissociation, were negative. The unusual appearance of the athlete's distal radioulnar joint, both grossly and on radiographic investigation, prompted the team orthopaedist to refer the athlete to the institution's team hand surgeon for further evaluation.

Ten days after the initial injury, the team hand surgeon evaluated the athlete. Before evaluation, the athlete continued to receive the symptomatic treatment protocol as described and continued to avoid upper extremity impact loading and gripping activities during practice; however, significant dorsal wrist pain persisted. The team hand surgeon concurred with the team orthopaedist's diagnosis of a Madelung deformity and a proximal radial bow deformity. Most significantly, the widened distal radioulnar joint had allowed the lunate to migrate proximally and come to rest between the distal radius and ulna. Because of the persistent pain despite considerable activity restrictions and symptomatic treatment, a short-arm cast was applied for 10 days to more adequately allow the joint an opportunity to heal.

The athlete was again evaluated by the team hand surgeon 3 weeks after the initial injury. When the cast was removed, examination revealed only slight improvement in pain. At this time, the team hand surgeon discussed the possibility of reconstructive surgery to correct the deformity and potentially allow the athlete to return to competitive gymnastics without restriction. However, the athlete had significant reservations regarding surgical intervention for a number of reasons. First, she did not want to further disrupt her ability to participate in her art courses. Second, she was somewhat apprehensive that surgery would affect her ability to draw and sculpt as she had before the injury. Even though the involved hand was nondominant, the athlete still used it extensively during her artwork. She feared that surgery could possibly alter her abilities and adversely affect her opportunities to gain employment in the future. Third, as a senior class team member, she did not wish to forgo her final season of eligibility because of surgery and extensive rehabilitation and attempt to compete during a fifth year. Since the athlete had no carpal instability, the team hand surgeon concluded that continuing to participate without surgical intervention posed no significant threat to further injury; however, the athlete's sensation of pain would most likely persist and even worsen with continued participation in competitive gymnastics. Based on this information, the medical staff, the athlete, her parents, and the gymnastics coaching staff agreed to attempt conservative management.

The medical staff made the decision to restrict the athlete from uneven parallel bars and floor exercise for the duration of her competitive career. Relative team strength on these events meant that the athlete had no realistic opportunity to contribute to the team scores in either event. As such, the medical staff deemed participation in those events to be unnecessary and counterproductive to the athlete's performance on the balance beam and vault. The athlete was allowed to return to the balance beam immediately and was gradually released to vault. Because of the tremendous forces transmitted through the wrists during the "block" of the vault when the gymnast forcefully strikes the apparatus with both hands and pushes off, this event proved particularly painful. The athlete noted sharp pain when her wrist was forced into extension as she blocked the apparatus during the performance of her vault. To prevent forceful end-range left wrist extension, a palmar-fan tape job was constructed of two 1½-in (3.81-cm) Johnson & Johnson Zonas tape anchor strips (New Brunswick, NJ) around the distal third of the forearm, 3 strips of 2-in (5.08-cm) Johnson & Johnson Elastikon tape placed fan shaped palmarly, and a continuous closure strip of 2-in (5.08-cm) Jaylastic stretch tape (Jaybird & Mais, Inc, Lawrence, MA). Additionally, a leather wrist brace (Lion Paw, RBJ Athletics, Spanish Fork, UT) was used over the tape job. This combination of taping and bracing proved effective in preventing end-range left wrist extension while still allowing the athlete adequate mobility to successfully perform her vault.

During the season, the athlete occasionally noted periodic increases in pain. During such times, she would be restricted

from performing vault during practice until the pain subsided. Typically, such episodes would last 2 to 3 days. The athlete continued to receive symptomatic treatment consisting of ice-bag application, cold whirlpool immersion, and transcutaneous electric nerve stimulation therapy throughout the season with favorable results. Despite the injury, she was able to successfully complete the season having competed in every meet. Additionally, by season's end she had distinguished herself as one of the team's most consistent performers on both balance beam and vault. Finally, the athlete has been able to return to art classes and participate at her previous levels of ability in both drawing and sculpture with no complaints of pain.

DISCUSSION

Madelung deformity is described as an idiopathic, progressive curvature of the radius that results in a characteristic anterior subluxation of the hand with respect to the distal radioulnar joint.[4,6,7] The disorder was initially described by Madelung in 1878, before the advent of modern radiography.[8] Many authorities, including Madelung himself, credited Dupuytren with the first reference to the disorder.[6–9] The disorder is relatively uncommon, occurring in less than 2% of the general population, and is most prevalent in female patients, at a ratio of 3 to 5:1 compared with male patients.[9–13] Typically, the deformity is present bilaterally and seldom manifests clinically before the age of 7 years.[9,13,14] Some authors suggest that the typical middle to late adolescent onset of the disorder may be linked to the adolescent growth spurt.[6,8] Vickers and Nielsen[8] contended that the long-standing and progressive radial deformity gradually worsens until it is suddenly exacerbated by the increased growth rate, often occurring concurrently with a premature physeal fusion.

Individuals who present with the disorder are usually limited in pronation and supination of the involved extremity, although the source of that limitation remains controversial.[6,8] On gross examination, the athlete usually presents with a characteristic palmar migration of the carpus and hand with respect to the distal radius and ulna. Additionally, he or she often complains of pain on the ulnar side of the wrist, which can sometimes mimic or occur concurrently with a triangular fibrocartilage complex lesion, making radiographic investigation essential.[1,5] Radiographic findings most often associated with Madelung deformity include increased dorsal and radial bowing of the distal radius, a triangular-shaped carpus, an exaggerated volar and ulnar tilt of the distal articular radial surface, and positive ulnar variance.[4,6,8,10–12,14,15]

Cause

The etiology of the disorder remains unclear, but all hypothetical causes produce a local growth disturbance at the ulnar and volar parts of the distal radial epiphyseal plate.[13] Madelung deformity is commonly classified into 4 groups: idiopathic, dysplastic, genetic, and posttraumatic.[4,6,8,9,12,16] Some have suggested that idiopathic Madelung deformity is probably not a true classifying group but rather a lack of conclusive implication of 1 of the remaining 3 causative groups.[8,9] Dysplastic Madelung deformity is associated with dyschondrosteosis, multiple hereditary exostosis syndrome, diaphyseal aclasis, and onychoosteodysplasia, or HOOD syndrome.[4,11,12,17] Dysplastic Madelung deformity is a form of mesomelic dwarfism; therefore, the deformity is usually accompanied by short forearms and legs.[8,9,11,16–18] Genetic Madelung deformity has been clinically associated with Turner syndrome; however, it has also very infrequently accompanied other genetic disorders as well.[4,6,12]

Whereas the preceding 3 categories are heritable and not preventable, posttraumatic Madelung deformities are preventable because the disorder typically develops from long periods of overuse or abuse or both. Posttraumatic Madelung deformity is of particular concern in competitive gymnasts.[1,2,4,5,19,20] The impact loading to the upper extremity required during gymnastics participation, coupled with the long duration of training most gymnasts undergo, tends to close the radial epiphyseal plate prematurely, ultimately leading to a Madelung or a Madelung-like deformity.[5,6,9] Interestingly, some disagreement exists as to whether or not a posttraumatic episode with no associated genetic or dysplastic factors can actually produce a Madelung deformity. Lamb,[10] Vender and Watson,[4] De Smet et al,[1] and Mandelbaum et al[2] all referred to such a condition as a "Madelung-like" deformity, being careful to distinguish it from a true Madelung deformity. Most other authors make no such distinction.[6,8,9,12–16] The prevalence of wrist pain in gymnastics is so great that the term *gymnast's wrist* has come into widespread acceptance. However, the subject of this case report is unique in that most gymnasts with a Madelung or a Madelung-like deformity are usually diagnosed as having the disorder early in their career and are either forced to discontinue participation because of pain or undergo surgical reconstruction to continue participation.[1,2,4,5,19]

Treatment

Madelung deformity is typically treated with either rest or surgical reconstruction, depending largely on both the specific needs of the athlete and the relative severity of symptoms. A number of surgical procedures have been developed to correct the condition; however, in the absence of symptoms, surgical repair is not necessary.[10] Furthermore, surgery is typically discouraged in adolescents and young adults because symptoms tend to subside with age, thus making surgery among the general population necessary only in cases of persistent significant pain.[8,10,18] However, among symptomatic athletes who wish to prolong their athletic careers, surgical intervention provides the best chance to return to participation without pain or chronic sensations of instability.[1,4]

Surgical repair usually consists of shortening or resection of the distal ulna or a wedge osteotomy of the distal radius or both.[13] Although techniques vary considerably, each can be categorized into 1 of 3 groups.[14] The first group includes techniques applied to the radius alone, such as epiphysiodesis, corrective osteotomy, and physiolysis.[13–15] The second group involves repairs or reconstructions limited to the ulna, including epiphysiodesis, ulnar head excision, shortening osteotomy, distal ulnar resection, and creation of a pseudarthrosis with or without fusion to the radius.[14,21] The third group involves some combination of the above techniques.

Most recent investigators found significant decreases in pain, improved range of motion, and improved grip strength after surgical correction. In one investigation, 18 patients who were dissatisfied with conservative treatment measures chose to undergo surgical correction.[14] Of the 18, 9 cited pain as their primary motivator for surgical reconstruction. After surgery, 2 of those 9 continued to have pain, whereas the re-

mainder noted significant decreases in pain. Additionally, statistically significant improvement in range of motion and grip strength was apparent in all subjects. In another work by Vickers and Nielsen,[8] 17 patients underwent surgical correction, 14 primarily to reduce pain and 3 to improve appearance. All patients with complaints of pain experienced significant relief within the first 6 months after surgery; however, only 4 indicated that pain was completely alleviated. Furthermore, all patients noted substantial improvements in range of motion, particularly in supination. No long-term complications were noted in any cases.

CONCLUSIONS

Madelung deformity is a rare condition that affects the structure and function of the distal radioulnar joint. Although the disorder is commonly linked to several heritable factors, most authors suggest that trauma may also be a factor. At the same time, competitive gymnastics requires repeated impact loading of the upper extremity, thus exposing the athlete to increased risk of wrist trauma due to the tremendous torsional and compressive forces sustained during sport participation. Typically, athletes with a symptomatic deformity either undergo surgical correction or cease competitive athletic participation. This particular case demonstrates a successful conservative care approach in which activity was limited to only essential training and competitive gymnastic movements. Additionally, local modality treatments and protective taping and bracing were used to manage and prevent pain as much as possible. Furthermore, the case is particularly unique because the athlete participated through all levels of competitive gymnastics (including the elite level) without complaint for 17 years before developing severe pain during her senior season in college. Although surgical reconstruction is regarded as the solution for symptomatic Madelung deformity among athletes who wish to continue to participate, conservative management should be considered a viable option before the athlete decides to discontinue participation.

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Recalcitrant Infrapatellar Tendinitis and Surgical Outcome in a Collegiate Basketball Player: A Case Report

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Objective: To present the history, surgery, rehabilitation management, and eventual functional and surgical outcomes of a collegiate basketball player with recalcitrant jumper's knee.

Background: A 21-year-old, male collegiate basketball player had a 2-year history of anterior knee pain.

Differential Diagnosis: Injuries that often mimic symptoms of infrapatellar tendinitis include infrapatellar fat pad irritation, Hoffa fat pad disease, patellofemoral joint dysfunction, mucoid degeneration of the infrapatellar tendon, and, in preadolescents and adolescents, Sinding-Larsen-Johannsson disease.

Treatment: After conservative treatment failed to improve his symptoms, the athlete underwent surgical excision of infrapatellar fibrous scar tissue and repair of the infrapatellar tendon.

Uniqueness: This patient's case was unique in 3 distinct ways: (1) outcome surveys helped me to understand how this injury affected various aspects of this patient's life and how he viewed himself as he progressed through rehabilitation; (2) a modified functional test was used to help determine whether the athlete was ready to return to sport; and (3) the athlete progressed rapidly through rehabilitation and returned to competitive athletics in 3 months.

Conclusions: This patient was able to return to sport without functional limitations. The surgical outcome was also considered excellent.

Key Words: jumper's knee, tendinitis, tendinosis, rehabilitation

Patellar tendinitis, often referred to as *jumper's knee,* is a relatively common overuse injury often experienced by basketball, volleyball, distance running, long jumping, mountain climbing, figure skating, and tennis athletes.[1–3] However, the pathologic condition is defined as an "insertional tendinopathy"[4] that can occur at the superior or inferior patellar pole or at the tibial tubercle.[5] Common symptoms related to this condition include pain, aching, and swelling at the inferior pole of the patella and weakness around the knee, with various degrees of quadriceps atrophy.[3] In 1973, Blazina et al[6] developed a classification of jumper's knee based on pain and functional limitations (Table 1).

Stages 1 and 2 usually respond well to conservative intervention involving therapeutic exercise, ice, ultrasound, cross-friction massage, and rest.[1,3] Treatment of patients in stage 3 often includes a period of prolonged rest, reducing the number and frequency of playing sessions,[1,3] and localized corticosteroid injection. Yet continuous overuse of the patellar tendon may lead to pain that is chronic and recalcitrant in nature.[7] Surgery may be necessary for patients with stage 3 jumper's knee that has failed to heal.[2,7,8] Colosimo and Bassett[5] maintained that surgical intervention is the only treatment modality that can offer gratifying long-standing results in competitive athletes for whom long periods of inactivity are impractical.

DIFFERENTIAL DIAGNOSIS

Other conditions that may present with symptoms similar to those of infrapatellar tendinitis include injury to the infrapatellar fat pad, Hoffa disease, patellofemoral joint dysfunction, mucoid degeneration of the infrapatellar tendon, and, in adolescents and preadolescents, Sinding-Larsen-Johansson disease. With fat pad irritation, passive knee extension causes compression of the pad and usually elicits pain. The fat pad can also be "unloaded" by palpating the medial and lateral sides of the inferior pole and lifting the fat pad in a superior direction as the knee is passively extended. This generally reduces the patient's symptoms if the fat pad is irritated. Chronic inflammation of the fat pad can lead to Hoffa disease, a fibrosis of the infrapatellar fat pad.[9] Diagnostic tests and clinical examination help to determine the diagnosis for patellofemoral dysfunction. However, patellofemoral dysfunction may also predispose the athlete to patellar tendinitis.[3] Mucoid degeneration should be considered, particularly in patients who have experienced trauma, such as a direct blow to the tendon area.[10] Magnetic resonance imaging or computed tomography can also be performed to confirm the diagnosis.[10] Lastly, in Sinding-Larsen-Johansson disease, stresses are concentrated at the growth plates. Stress at the inferior pole of the patella leads to a traction epiphysitis and eventual fragmentation.[5]

EPIDEMIOLOGIC FACTORS

Ferretti[4] examined intrinsic and extrinsic factors that eventually led to jumper's knee and its progression. The intrinsic factors examined were sex, alignment of the knee and extensor mechanism, patellar position, characteristics of the tibial tu-

Table 1. Classification of Jumper's Knee Developed by Blazina et al[6]*

Stage 1:	Pain after activity only, no functional impairment
Stage 2:	Pain at the beginning of activity, disappearing after warm-up, and recurring after activity
Stage 3:	Pain during and after activity that impairs function
Stage 4:	Complete rupture

*Adapted with permission.

berosity, rotation of the femur and tibia, flexion contracture of the knee, degree of constitutional instability, characteristics of the foot, and morphotype. The extrinsic factors examined were number and frequency of training sessions, years of play, type of training, and playing and training surfaces. Hard playing surfaces and increased frequency of training sessions were the only factors that correlated positively with the incidence of jumper's knee. No correlation was noted between malalignment of the extensor mechanism or biomechanical derangement and the incidence of jumper's knee. Ferretti[4] concluded that the mechanical properties of the tendon (resistance, elasticity, and extensibility) at the bone-tendon junctions were the most significant etiologic intrinsic factors.

Kujala et al[11] agreed that the most important etiologic factors that contribute to jumper's knee were microruptures caused by jumping on hard surfaces. They also described significant correlations between leg-length inequalities and patella alta as possible predisposing factors to patellar apicitis (jumper's knee).

Almekinders and Temple[12] reviewed articles in which training errors, inappropriate shoe wear, and anatomical predisposition that resulted from inflexibility, weakness, or malposition were described as playing a role in the etiology of chronic tendon injuries. However, many of the articles lacked a control group. They noted that most studies, including the one by Kujala et al,[11] were retrospective and difficult to interpret.

My purpose is to present the case report of a collegiate basketball player with recalcitrant infrapatellar tendinitis, his clinical and surgical courses of treatment, and his eventual outcome. Proper use of the terms *tendinitis* and *tendinosis* will also be explored.

REPORT OF A CASE

A 21-year-old, male collegiate basketball player, referred by a local orthopaedic surgeon, presented to our clinic with a 2-year chief complaint of right anterior knee pain and a recent diagnosis of recalcitrant jumper's knee. The athlete was evaluated by a certified athletic trainer at our facility, who concurred with the diagnosis of infrapatellar tendinitis after taking a thorough history of the athlete's complaints and ruling out the previously described knee conditions. The athletic trainer noted right quadriceps atrophy when compared bilaterally. Assessment of the athlete's active range of motion revealed full flexion without pain and full passive extension. With a straight-leg raise, a 5° to 10° lag was present and accompanied by pain. Formal manual muscle testing was not performed because the athlete's knee was inflamed. With palpation at the inferior pole of the patella, the patient had focal tenderness and visible thickening, about the size of a quarter and raised approximately 0.635 cm (¼ in), at the inferior pole of the patella. Results of x-ray films of the knee were unremarkable. A magnetic resonance imaging scan of the knee was not obtained. Additional subjective information was gathered via 3 outcome questionnaires: Cincinnati Knee Rating Scale,[13] Quality of Life Questionnaire,[14] and Patellofemoral Knee Pain Survey.[15]

The Cincinnati Knee Rating Scale is used to assess overall function of the patient's knees at the present time, including pain, swelling, giving way, overall activity level, walking, stairs, running activity, and jumping and twisting activities. The athlete was able to perform vigorous activities but at a lower performance level, had slight or mild problems with walking, had slight or mild problems with stairs, and had moderate problems with running 1.6 to 3.2 km (1 to 2 miles). The Quality of Life Questionnaire was used to assess the ways in which the athlete's injury had affected various aspects of his life. Each of the 5 parts includes a visual analog scale; the subject is asked to place a slash on a line, which ranges from 0 to 100. The questionnaire's 5 parts address symptoms and physical concerns, work-related concerns, recreational activities and sports participation or competition, lifestyle, and social and emotional concerns. In this questionnaire, the patient revealed that he experienced close to severe pain with prolonged activity (longer than ½ hour), moderate giving-out episodes, and an extremely weak knee (Table 2). He also reported difficulty squatting and going "full out" with activity and significant limitations with sudden twisting or pivoting motions and changes in direction. He was concerned about his knee getting worse and his competitive needs. The athlete was aware of his knee problem most of the time and was apprehensive with activity.

The Patellofemoral Knee Pain Survey was also used to assess symptoms and overall function of the patient's knees. The patient had pain ascending and descending stairs, with the initiation of running and jumping, swelling in the evening, occasional painful movement of his kneecap in sporting activities, and the belief that motion and muscle mass were lost in the painful leg.

The patient's Cincinnati Knee Rating Scale and Patellofemoral Knee Pain Survey scores were 57/100 and 58/100, respectively. The preoperative Quality of Life Questionnaire scores were also calculated. After the evaluation, it was obvious that the patient could be categorized as having stage 3 infrapatellar tendinitis. His goal at this time was to finish the current season and be healthy to start playing in the end of the summer league program, the following (his junior) year, to prepare himself for conditioning, which started in mid September.

Conservative treatment was initiated, consisting of phonophoresis (0.4% dexamethasone and 0.1% lidocaine mixed in 0.47 L [16 oz] of ultrasound gel for 5 minutes at 3 MHz at 1.0 W/cm ^{2}), iontophoresis (dexamethasone at 40 mA/min with intensity as tolerated), ice with electric stimulation, and quadriceps and vastus medialis obliquus (VMO) strengthening. Quadriceps exercises included closed-chain single- and double-leg extensions on a leg machine (resistance was increased or decreased using rubber cords), weights on a leg press, terminal knee extensions on a multiple-hip exercise machine, and walking backward on a treadmill with a 5% grade. For the closed-chain double-leg extensions, the athlete squeezed a ball between his legs in an attempt to challenge the VMO. With the closed-chain single-leg extensions, a band was placed around the thigh and resistance was applied medially as the patient moved the leg into extension. Generally, the patient started with 3 sets of 10 repetitions and progressed to 3 sets

Table 2. Preoperative and Postoperative (3 and 14 Months) Results on Quality of Life Questionnaire[14]

Question*	Preoperative	3 Months	14 Months
Part I (symptoms and physical concerns)			
Severity of "giving way"? (0 = major, 100 = minor)	41	91	100
Frequency of "giving way"? (0 = constant, 100 = never)	61	86	100
Pain with ≥½ hour of activity? (0 = severe, 100 = no pain)	16	78	100
Overall knee function: how troubled by stiffness or loss of motion? (0 = severe, 100 = not troubled)	25	92	100
Overall knee function: how weak is knee? (0 = extremely, 100 = not weak)	8	88	100
Part II (work-related concerns)			
How much trouble with turning or pivoting due to knee? (0 = severe, 100 = no trouble)	Unanswered	90	100
How much trouble with squatting due to knee? (0 = severe, 100 = no trouble)	19	96	100
Concern for loss of work due to problems or knee reinjury? (0 = significant concern, 100 = no concern)	98	100	100
Concern about losing school time for treatment? (0 = significant concern, 100 = no concern)	94	100	100
Part III (recreation activities and sport participation or competition)			
Limitation with sudden twisting and pivoting? (0 = totally limited, 100 = not limited)	22	87	100
Concern for knee worsening with sporting or recreation activities? (0 = significant concern, 100 = no concern)	8	95	100
Current level of athletic performance compared with preinjury? (0 = totally limited, 100 = not limited)	30	100	100
Due to status of knee, how much have expectations lowered regarding sports? (0 = totally lowered, 100 = not lowered)	58	98	100
Play sport cautiously? (0 = always, 100 = never)	34	99	99
Fear of knee giving way in sport? (0 = extreme, 100 = no fear)	30	98	98
Concern with environmental conditions (ie, wet field, hard court)? (0 = extreme, 100 = not concerned)	17	67	88
Frustrating to have to consider knee with sports? (0 = extremely frustrating, 100 = not frustrating)	48	98	98
How difficult to go "full out"? (0 = extremely, 100 = not difficult)	12	100	100
Fearful of playing contact sports? (0 = extremely, 100 = not fearful)	65	99	99
How limited in basketball? (0 = totally, 100 = not limited)	36	97	100
How limited in track? (0 = totally, 100 = not limited)	0	99	99
Part IV (lifestyle)			
Concern with safety issues (ie, yard work) regarding knee? (0 = extreme, 100 = not concerned)	98	100	100
How limited in exercising and maintaining fitness due to knee? (0 = totally, 100 = not limited)	78	98	100
How much has enjoyment of life been limited due to knee? (0 = totally, 100 = not limited)	97	100	100
How often aware of knee problem? (0 = always, 100 = never)	15	82	98
Concern for lifestyle activities with family? (0 = extreme, 100 = not concerned)	92	98	98
Modified lifestyle to avoid damaging knee? (0 = totally, 100 = no modification)	59	100	100
Part V (social and emotion concern)			
Concern that competitive needs no longer being met because of knee? (0 = extreme, 100 = not concerned)	26	100	100
Any difficulty coming to grips with knee problems psychologically? (0 = extreme, 100 = no difficulty)	34	100	100
How often apprehensive about knee? (0 = all the time, 100 = none of the time)	35	98	98
How troubled by lack of confidence in knee? (0 = severely, 100 = not troubled)	22	98	98
How fearful of reinjuring knee? (0 = extremely, 100 = no fear)	44	100	100

*Questions have been paraphrased for brevity.

of 15 repetitions. If he completed 3 sets of 15 repetitions without pain or difficulty, the resistance was increased and the repetitions decreased. The patient tolerated these exercises well and denied any increase in symptoms or pain with the strengthening activities.

Squeezing a ball with the double-leg extensions and the varus force applied with the single-leg extensions were selected to help recruit VMO firing during the exercises. The athlete attempted eccentric exercises to increase the tensile strength of the tissue, but he was unable to tolerate these exercises during his rehabilitation.

Stretching of the quadriceps, iliotibial band, hamstrings, gastrocnemius, and soleus was initiated prophylactically, and the athlete took nonsteroidal anti-inflammatory medication. He was seen by the athletic trainer in the clinic for 20 visits throughout 5 months and pursued a concurrent home exercise program. When the athlete was unable to travel to the clinic, the home exercise program was emphasized. Playing time was decreased, but there was no period of absolute rest.

After 5 months of conservative treatment, the patient returned to the orthopaedic surgeon. At this time, the patient was diagnosed as having recalcitrant jumper's knee and injected, in a peritendinous fashion, with a mixture of bupivicaine hydrochloride and betamethasone sodium phosphate. The patient was encouraged to continue with his rehabilitation and follow up with the surgeon in 1 month. If adequate progress was not made, an infrapatellar tendon debridement at the insertion of the patella would be considered. Conservative rehabilitation achieved limited success, and keeping the athlete's goal in mind, surgery was scheduled 1 month later, in mid April 1999.

Surgery

The patient underwent excision of infrapatellar fibrous scar and repair of the infrapatellar tendon. During the surgery, the surgeon noted a small, partial tear of the infrapatellar tendon insertion at the inferior pole of the patella. This was debrided and rasped back to a bleeding bony edge. The patellar tendon tissue contained grayish-appearing hypertrophic scar tissue, which was also debrided back to a stable margin.

Postoperative Evaluation and Treatment

The athlete returned to the clinic the day after surgery in a straight-leg immobilizer. Weight bearing was permitted as tolerated. Modalities were applied to control effusion, and the patient returned 1 week later for a formal evaluation. The initial week of "relative rest" allowed the wound and tendon to recover from the surgery.[7] Icing of the knee was emphasized daily along with heel slides and quadriceps setting. With the heel slides, the athlete was instructed not to bend his knee beyond his natural tissue tension. At the formal evaluation 1 week later, the patient complained of pain around his incision and stiffness with knee flexion. Objectively, quadriceps strength was graded 3−/5, passive knee extension was 0°, and active knee flexion was measured at 45°. The patient had moderate swelling in the patellar tendon, mild joint effusion, and a 10° extension lag when a straight-leg raise was attempted. Modalities (ice and electric stimulation) were used to control inflammation, and a pain-free exercise program was initiated.

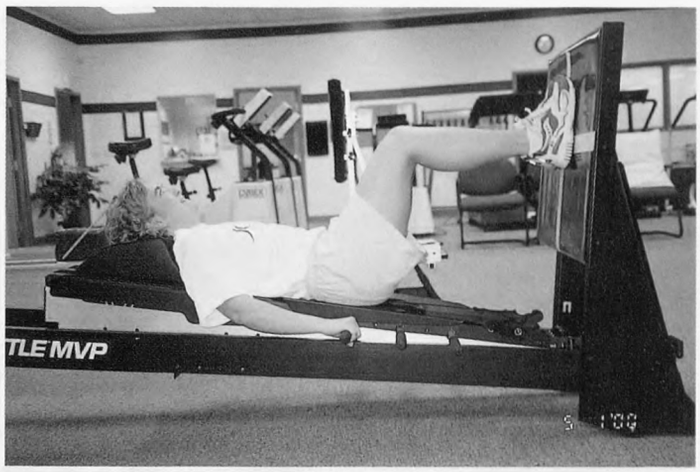

Figure 1. Knee extension exercise on the Shuttle MVP (Shuttle Systems, Glacier, WA).

Rehabilitation

Early Phase. The patient's initial rehabilitation, 1 week after surgery, consisted mostly of range-of-motion exercises, including heel slides, treadmill walking forward and backward, and biking. Strength exercises consisted of quadriceps setting using biofeedback instrumentation, straight-leg raising, leg-extension machine (both legs and single leg) (Figure 1), and multiple-hip machine (Figure 2) for terminal knee extensions and hip adduction. Passive stretching was performed on the hamstrings. Ice with interferential current concluded each session.

The patient's therapy was placed on hold when he left for vacation after the initial 3 weeks of rehabilitation. Strict guidelines were given to the patient at this time, and a home exercise program of ice, gastrocnemius and hamstring stretches, heel slides, quadriceps setting, and straight-leg raising was emphasized. The patient's active knee flexion at this time was 125°.

Approximately 1 month later, the patient returned to our clinic. He reported feeling good, being compliant with his home exercise program, and refraining from running or playing basketball to this point. Quadriceps strength was 4+/5. The evaluation also revealed full active range of motion, no quadriceps lag with straight-leg raising, and the ability to squat without pain. Rehabilitation was resumed.

Intermediate and Advanced Phases. Strengthening; progressive resistive exercise; proprioceptive, agility, and aerobic conditioning; plyometrics and eccentric loading; and functional or sport-specific drills were emphasized in this phase. Stretches (hamstrings, gastrocnemius, and quadriceps) and cryotherapy were also continued. Again, a pain-free progression was the goal. The patient began treadmill running at 8 weeks after surgery and functional or sport-specific drills on the following day. The aim was for the athlete to begin playing the following week. At 9 weeks, he was cleared for limited playing time by the orthopaedic surgeon and permitted to play 10 minutes per game at the most. Playing time was progressed as tolerated. The athletic trainer continued to monitor the patient's response to increased playing time and supervised continued strengthening and neuromuscular activities in the clinic.

Functional Testing. At 10 weeks after surgery, a functional test was used to objectively assess the patient's right lower extremity function compared with the uninvolved leg. The

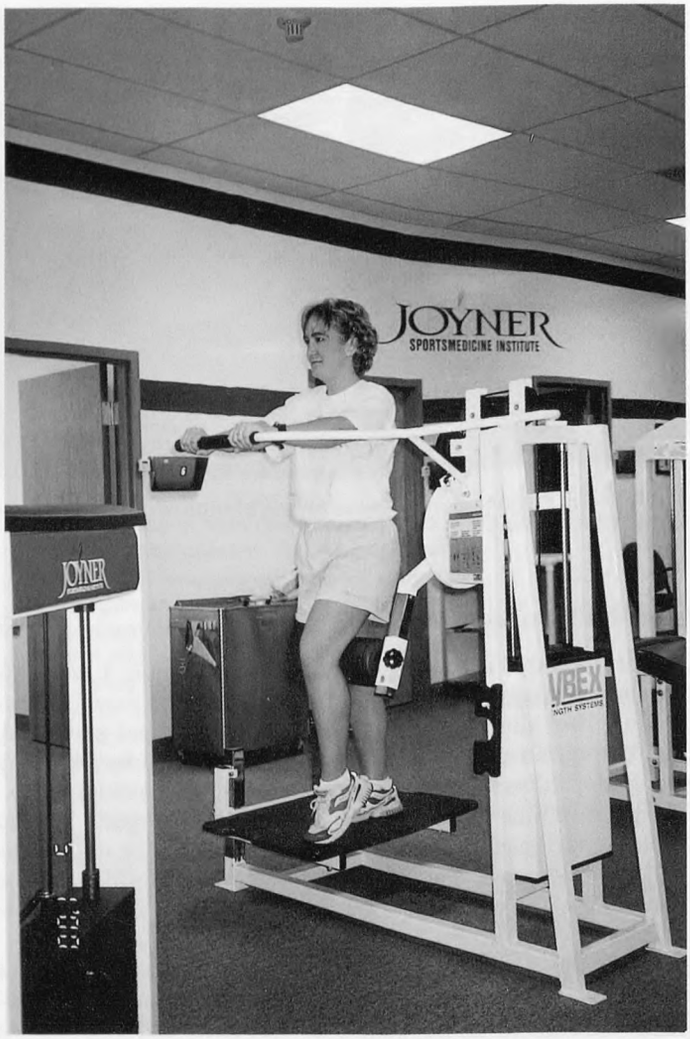

Figure 2. Terminal knee extension on a Cybex Multi-Hip Machine (Cybex International, Inc, Medway, MA).

functional test consisted of a single-leg hop test, triple hop test, lateral hop test for time, maximum-repetition leg press at body weight, and 1-repetition maximum on the leg press. (For the lateral hop test for time, the athlete stood to the side of a solid line and was asked to hop across the line, back and forth, for 15 seconds.) The number of hops was then compared bilaterally. Compared with the opposite leg, the patient scored in the 96th percentile for the single-leg hop test and lateral hop test for time and 100% on the other 3 tests. At 3 months after surgery, the patient was discharged by the athletic trainer but instructed to continue with the home exercise program.

Reevaluation of Questionnaires and Clinical Examination

At 3 and 14 months after surgery, the patient again completed the questionnaires. At 14 months, his scores escalated to 100/100 on the Cincinnati Knee Rating Scale and 98/100 on the Patellofemoral Knee Pain Survey. He denied any limitations in activity, walking, stairs, running, jumping, or twisting. Review of the Quality of Life Questionnaire at 14 months also revealed that the patient was much improved (Table 2); interestingly, the athlete's lowest rating and greatest concern was with the environmental conditions, which was in agreement with the findings of Ferretti.[4]

In addition to having the patient complete the questionnaires, I evaluated the surgical outcome using a modification of the criteria defined by Kelly et al[16] (Table 3). The athlete had full, bilateral active range of motion. Girth measurements taken at the superior pole of the patella and at 5.08 cm (2 in) and 10.16 cm (4 in) above the superior pole were equal bilaterally. Based on these objective findings and subjective reports of no pain and return to sports, the surgical outcome was considered excellent.

At 27 months after surgery, the athlete denied any knee pain. He did, however, note increased power and his satisfaction with being able to participate in the triple and long jumps the year before. These were events that he had not been able to participate in since high school because of his infrapatellar tendinitis.

DISCUSSION

Management of patellar tendinitis can be a very tedious process for the athletic trainer or physical therapist. Prevention and education are important components; however, athletes, in their desire to compete, often play through the pain and refrain from alerting the health care professional to their symptoms. In the case of patellar tendinitis, the initial symptoms tend to get worse if they are overlooked or the patient is noncompliant with treatment. The use of the word *tendinitis* in cases of true tendinosis may also lead to underestimation of the natural history of this condition by coaches and athletes.[17] Pathologic tendon conditions may occur before any symptoms are noticed.[18] If symptoms do manifest, patients in stages 1 and 2 of Blazina et al's scale[6] can usually be treated successfully with conservative treatment.[1,3] However, current treatment methods may not change the natural history of chronic tendon injuries,[12] and patients with early symptoms of pain may also take months to recover.[18] In these early stages, the practice surface and training parameters, such as frequency, intensity, and duration, should be evaluated and adjusted immediately

Table 3. Classification of Functional and Clinical Results[16]*

Result	Clinical Criteria	Functional Criteria
Excellent	Quadriceps atrophy <0.5 cm or no patellar compressive tenderness or <5° loss of flexion	No pain, full return to preinjury level of activity
Good	Quadriceps atrophy 0.5–1.5 cm or mild patellar compressive tenderness or 5–10° loss of flexion	Mild pain on heavy exertion, full return to preinjury level of activity
Fair	Quadriceps atrophy 1.5–3 cm or moderate patellar compressive tenderness or 10–15° loss of flexion	Moderate pain on heavy exertion, minimally reduced level of activity
Poor	Quadriceps atrophy >3 cm or severe patellar compressive tenderness or >15° loss of flexion	Severe pain on exertion, significant decrease in activity or no return to activities

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Table 4. Bonar's Modification of Clancy's Classification of Tendinopathies[19]*

Pathological Diagnosis	Concept (Macroscopic Pathological Condition)	Histological Appearance
Tendinosis	Intratendinous degeneration commonly caused by aging, microtrauma, and vascular compromise	Collagen disorientation, disorganization, and fiber separation with an increase in mucoid ground substance, increased prominence of cells and vascular spaces with or without neovascularization, and focal necrosis or calcification
Tendinitis or partial rupture	Symptomatic degeneration of the tendon with vascular disruption and inflammatory repair response	Degenerative changes as noted above with superimposed evidence of tear, including fibroblastic and myofibroblastic proliferation, hemorrhage, and organizing granulation tissue
Paratenonitis	"Inflammation" of the outer layer of the tendon (paratenon) alone, regardless of whether the paratenon is lined by synovium	Mucoid degeneration in the areolar tissue is seen; a scattered mild mononuclear infiltrate with or without focal fibrin deposition and fibrinous exudate is also seen
Paratenonitis with tendinosis	Paratenonitis associated with intratendinous degeneration	Degenerative changes as noted for tendinosis with mucoid degeneration with or without fibrosis and scattered inflammatory cells in the paratenon alveolar tissue

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and appropriately once symptoms are experienced. A proper rehabilitation program and progression should then be initiated to return the athlete to competition. The use of eccentric strengthening speeds tenocyte metabolism,[19] causes quadriceps muscle hypertrophy,[20] improves muscle coordination,[20] and increases tensile strength of the tendon.[21] This patient attempted eccentric exercises before surgery but could not tolerate them due to the significantly inflamed tissue. Therefore, pain-free exercises were adopted. After surgery, however, he tolerated eccentric exercises, which then became an important component in his rehabilitation and ultimate return to sport.

Also before surgery, closed-chain single- and double-leg exercises were attempted to challenge the VMO. The rationale to incorporate hip adduction exercises with a quadriceps contraction stems from the large proportion of the oblique fibers of the vastus medialis from the adductor magnus.[22] Research examining preferential activation of the VMO with hip adduction has been controversial. Hanten and Schulthies[23] reported that electromyographic activity of the VMO was significantly greater than that of the vastus lateralis during isometric hip adduction. Hodges and Richardson[24] also reported increased VMO activity compared with the vastus lateralis with the addition of hip adduction in the weight-bearing position and only with maximal hip adduction in non–weight bearing. However, more recently, other authors[25–27] demonstrated no preferential activation of the VMO with hip adduction. It is difficult to say whether the external force provided for the single-leg knee extension or the isometric squeezing of a ball with the double-leg knee extension increased the firing of the VMO in this athlete. However, based on the current literature, it seems unlikely that preferential activation of the VMO occurred. The focus, therefore, should have been on training the entire quadriceps. Although the athlete achieved gains in quadriceps hypertrophy and strength before surgery, he was unable to tolerate eccentric loads and, therefore, would have been unlikely to develop the tendon tensile strength required to meet the specific imposed demands of basketball and the long and triple jumps in track and field.

Corticosteroid injections are not recommended to curb the pain of infrapatellar tendinitis because they can have adverse affects on the tendon.[9] Repeated injections can also lead to permanent tendon injury.[3] Previous investigations of prospective placebo-controlled studies versus corticosteroid injections for chronic tendon problems failed to show differences at follow-up compared with oral nonsteroidal anti-inflammatory drugs (NSAIDs) or placebo.[12] Although NSAIDs may help with pain relief, possible healing effects on the tendon have not been studied.[12] Using NSAIDs may also mask early symptoms, potentially leading to further tendon damage.[19] With stage 3 tendinitis, which is perhaps more appropriately termed *tendinosis,* the conservative pathway is not so successful. Current conservative treatment methods, such as those for stages 1 and 2, may not significantly alter the patient's now-chronic state. At this point, the histopathology should be the focus of treatment[19] and possible surgery. If surgery is indicated, the use of procedures such as a lateral release or VMO advancement may be questionable based on Ferretti's epidemiologic findings.[4]

In this athlete, initial symptoms were ignored and not reported. As a consequence, a self-limiting, manageable acute or subacute injury progressed to a chronic injury that eventually required surgery. In essence, the tendinitis progressed to a tendinosis. Because these terms can be confusing, classifications for tendinopathies have been developed.[19,28] Bonar's classification of tendinopathies has been considered the most reliable (Table 4).[19]

The term *tendinitis* refers to symptomatic degeneration of the tendon along with an inflammatory response and vascular disruption.[19,28] Tendinitis, unlike tendinosis, does not encompass histologic changes occurring in the tendon.[28] The term *tendinosis* histologically refers to noninflammatory degeneration due to aging, microtrauma, and vascular compromise.[19,28] Furthermore, noninflammatory collagen degeneration occurs along with fiber disorientation, hypocellularity, and scattered vascular ingrowth, with possible calcification and necrosis.[19,28] Given the surgeon's observation of a small tear in the tendon and a gray hypertrophic region, it was obvious that the athlete's injury was not self-limiting and would not have resolved with further conservative treatment.

With an emphasis on a pain-free progression and a proper rehabilitation program after surgery, the patient progressed rapidly and returned to competitive activities in 3 months. This time frame was similar to that described by Popp et al[7] and Scranton and Farrar[10] but shorter than the time frame reported by Cook et al[29] (7 to 12 months), who noted that returning to jumping sports prematurely may contribute to a poor outcome.[29] However, the rehabilitation for the athletes in this ret-

rospective study was not discussed, nor were the details of the surgical technique.

Although this case study represents a successful surgical outcome and rehabilitation of infrapatellar tendinosis, there appear to be many unanswered questions relating to the management and the efficacy of treatment of tendinitis and chronic tendon injury. Many times, the patient is told to rest and pursue conservative treatment. However, how long the patient should rest and continue with conservative care before undergoing surgery remains unclear. In the case of this athlete, conservative treatment was continued for 6 months. The athlete experienced an exacerbation of symptoms during the season and opted to have surgery toward the end of the season. For any athlete, it is difficult to determine when surgery is the preferred option over continued conservative treatment. Although more difficult to influence and assess, intrinsic factors, such as the vascularity of the affected area and the rate of tenocyte repair, should be considered. With tissue damage, estimates are 2 to 3 weeks for a tissue response to occur.[30] If the rate of tenocyte repair can be estimated in the individual, it would then be helpful in developing a hypothesis to determine when the patient should start to develop a healing response. If the patient then demonstrates a failure to heal, perhaps other interventions, such as sonography, may be helpful, in correlation with the clinical examination, in determining the extent and severity of the patellar tendinosis.[5,7]

Lastly, a modified functional test was incorporated to help determine the patient's readiness to return to sport. The use of a functional test, such as the one used in this case report, does not appear to be a common practice in helping to determine when to return to sport after patellar tendon surgery. Some authors have reported a goal of returning to sport in 12 weeks[7] after an eccentric strengthening program or 7 to 12 months after rehabilitation.[29] However, no data pertaining to the athlete's strength, endurance, or readiness to return to sport were reported. An adaptable functional test can provide meaningful information regarding the athlete's actual readiness throughout the rehabilitation progression. The single-leg hop and triple hop tests for distance are reliable measures of lower extremity performance compared with the uninvolved leg.[31,32] The lateral hop test helped to provide information regarding the athlete's ability to change direction quickly in the frontal plane and the athlete's overall comfort level when performing a multiple lateral hop task on the involved leg. The single-leg 1-repetition maximum on the leg press was performed to assess bilateral leg strength. The maximum number of repetitions at the athlete's body weight on the leg press was performed to assess muscle endurance. However, the reliability and validity of these last 3 tests have not been examined in the literature. Although these functional tests are often reserved for helping to determine the readiness of an athlete to return to sport after anterior cruciate ligament reconstruction, they were helpful in the decision-making process to return this athlete to sport, in conjunction with the athlete's self-reports of function via outcome questionnaires and objective measures.

My purpose in describing this case study was to present the outcome of surgery and rehabilitation of recalcitrant infrapatellar tendinitis in a collegiate basketball player. The rehabilitation presented was developed to meet the needs of the injured athlete with the resources available. I did not personally supervise the rehabilitation of this athlete and, thus, do not necessarily endorse all aspects of the program. It was, however, apparent that this program of rehabilitation concluded with the athlete's successful return to high-demand activities.

CONCLUSION

Patellar tendinitis is a progressive disease that results from repetitive microtrauma at the bone-tendon junction, particularly at the inferior pole of the patella. If symptoms are ignored, the tendinitis eventually progresses to a tendinosis, which may then require surgical intervention. Therefore, it is important for the athletic trainer and physical therapist to emphasize prevention to athletes, coaches, and parents. Further scientific examination relating to early intervention, progression of tendinitis into tendinopathy, efficacy of treatment, and rehabilitation by certified athletic trainers and physical therapists for all stages of tendon injury is clearly warranted. Applicability and usefulness of functional testing in patients with chronic tendinosis and patellar tendon surgery should be further explored as well.

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An Unusual Scalp Lesion in a 15-Year-Old Girl: A Case Report

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Objective: To present an unusual congenital malformation of the central nervous system.

Background: Neural tube defects (NTDs) are potentially serious congenital malformations. When undiagnosed in childhood, such lesions may later be mistaken for a variety of other soft tissue abnormalities. Athletic trainers should be aware of the clinical findings associated with NTDs and the potential for infection in the event of an injury, thus ensuring proper treatment for injured athletes and referral of any athletes with suspicious lesions.

Differential Diagnosis: Atretic meningocele, hemangioma, lipoma, sebaceous nevus, dermoid cyst, scar tissue, aplasia cutis congenita, and hematoma.

Treatment: The consulting pediatric neurosurgeon thought that repairing the atretic meningocele was only necessary if symptoms recurred and persisted. This young woman is at increased risk for having a child with an NTD and will benefit from high doses of folic acid early in a future pregnancy.

Uniqueness: Typically, NTDs are diagnosed in infancy or early childhood. This case represents a young woman whose NTD was not properly diagnosed until adolescence. In addition, NTDs can be mistaken for a variety of other skin lesions. The location and appearance of an NTD are typically distinctive to the knowledgeable examiner.

Conclusions: Although NTDs are unusual, athletic trainers should be aware of such pathologic conditions to avoid mistaking these lesions for traumatic sequelae and to identify those athletes who may need further evaluation to rule out a potentially serious condition.

Key Words: atretic meningocele, scalp lesion, neural tube defect, congenital abnormalities

Neural tube defects (NTDs) are developmental anomalies of the brain and spinal cord that range from fatal malformations to minor skin blemishes.[1] They are typically diagnosed prenatally or shortly after birth. However, benign-appearing variants, such as an atretic meningocele, may go undiagnosed into adolescence or beyond. The purpose of this case report is to introduce a relatively rare (1 to 2 cases per 10 000 people[2]) but important congenital abnormality to the athletic training literature. A thorough review of the available literature and a MEDLINE search uncovered no prior reports of athletes with any of the several forms of meningocele. The proper identification of these lesions is important because NTDs may communicate directly with the central nervous system, and a traumatic injury has the potential to cause a devastating infection. When discovered incidentally, the lesions may be mistaken for scars or other skin lesions, but neuroimaging is still paramount to make a proper diagnosis.[3]

REPORT OF A CASE

The patient was a 15-year-old girl (height, 175 cm; weight, 83 kg) who presented to the team physician (M.C.K.) with complaints of headaches and a persistently painful, tender, and swollen lesion on the back of her head. The symptoms began after she received a direct blow to the area during basketball practice approximately 4 weeks earlier. Her headaches often developed around noon, with a band of pain wrapping completely around her head. The headaches occurred 2 to 3 times per week and were well controlled by over-the-counter analgesics (acetaminophen and ibuprofen). She reported no visual disturbances, nausea, vomiting, fevers, or discharge from the lesion.

Further historical review with the patient's mother revealed that she was born with a small, dimpled lesion on the back of her head. Her mother was told that this resulted from a fetal scalp electrode. Since birth, the lesion would occasionally enlarge without significant pain or persistence.

Physical examination revealed a 3.5-cm, well-demarcated, fluctuant mass in the midline of the occipital bone, with a slight defect palpable in the skull at the lesion's borders (Figure 1). The mass was tender, without erythema or expressible fluid. The skin was dimpled in the middle of the lesion, with a high density of hair follicles within the dimple. Results of her neurologic examination were unremarkable. An initial diagnosis of encephalocele was made based on the history and physical examination.

Magnetic resonance imaging of the brain revealed several minor anomalies of the posterior cranial fossa, including an enlarged fourth ventricle and a small (less than 1 cm) midline occipital bone defect (Figure 2). A portion of the cerebellar meninges appeared to protrude through the defect. The cerebellum, although grossly normal, was tethered to the involved portion of the skull.

The patient was referred to a pediatric neurosurgeon for consultation, and her headaches and scalp tenderness sponta-

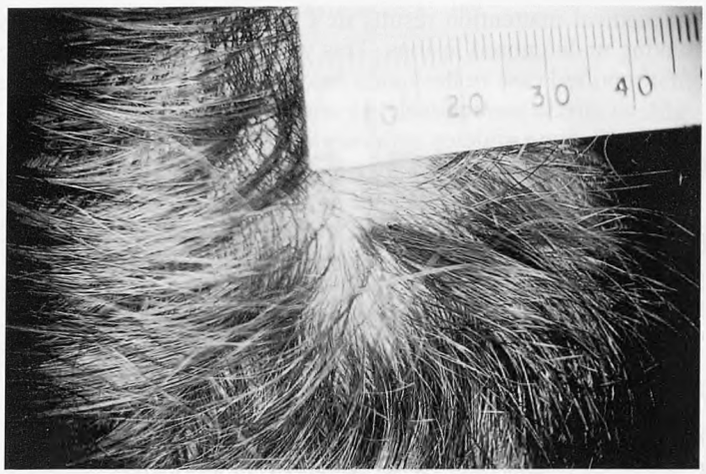

Figure 1. Atretic meningocele. Note the high density of hair follicles within the dimpled area of skin; 10 equals 10 mm (original magnification ×4).

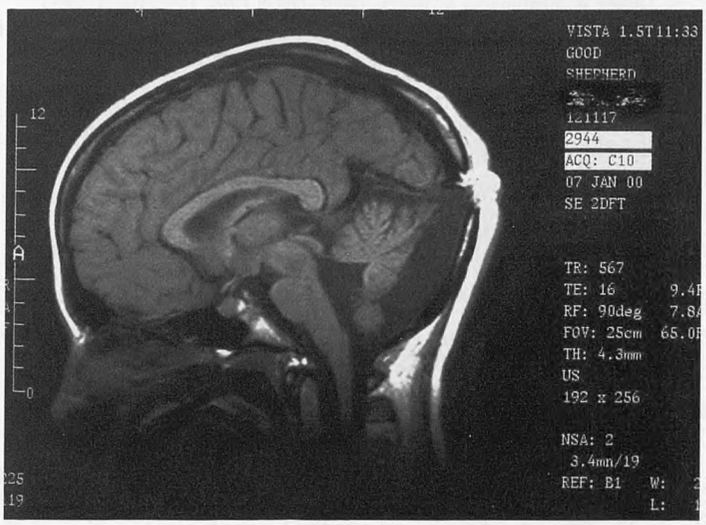

Figure 2. Magnetic resonance imaging scan of the patient's brain showing atretic meningocele, skull defect, and minor malformation of the cerebellum.

neously resolved before her appointment. The consulting surgeon made the diagnosis of atretic meningocele and recommended nonoperative treatment of observation as long as she continued to remain asymptomatic. She has remained asymptomatic for the past 12 months.

DISCUSSION

An atretic meningocele represents one end of the spectrum of NTDs. These lesions range from benign-appearing skin lesions, which may go undiagnosed for years (such as with our patient), to fatal malformations of the brain and spinal cord. The nomenclature of NTDs can be confusing, and the specific defects are numerous. For the purposes of this discussion, we need go no further than an understanding of cephaloceles, which are congenital herniations of intracranial contents through a defect in the skull.[4] If the herniated portion contains meninges or cerebrospinal fluid, it is termed a *meningocele,* whereas an *encephalocele* contains any amount of brain tissue, as well as meninges or cerebrospinal fluid.[5,6] Cephaloceles most often occur along the midline of the skull, with most being located over the occiput. The term *atretic meningocele* denotes a small, noncystic, flat or nodular lesion, in contrast with the larger pedunculated masses also seen. The atretic form represents about half of all such lesions.[2,7]

Neural tube defects arise during the embryonic phase of human development, in the first 54 to 60 days after ovulation.[1] The exact pathogenesis is unknown, but folic acid supplementation has been shown to have a protective effect.[8–11] A genetic predisposition has also been described.[4,12] The prevalence of NTDs varies widely with ethnic and geographic variations, ranging from a high of 3.05 to 6.79 per 1000 live births in the British Isles to a low of 0.1 to 0.6 per 1000 live births in continental Europe and Japan.[1] The frequency of cephaloceles ranges from 1 in 5000 to 1 in 9000 live births,[6] with meningoceles comprising approximately 40% of those defects.[6,13]

Ideally, cephaloceles and related lesions are diagnosed prenatally by fetal ultrasonography or shortly after birth. Early diagnosis allows for assessment and evaluation of any associated anomalies. We had a low index of suspicion for any serious neurologic condition in our patient. However, in one study, 36 (78%) of 46 children with cephaloceles had occult central nervous system anomalies, including hydrocephalus, posterior fossa cysts, and abnormalities of the ventricles.[2] As described previously, our patient had only minor brain anomalies.

In infants and children, the focus of the initial examination and neuroradiologic evaluation is the determination of the child's long-term neurologic prognosis. When the condition is discovered during adolescence or adulthood, the focus shifts to making the correct diagnosis and defining the underlying anatomy. The lesions may be discovered incidentally during an examination or after trauma to the head. Although a direct blow to the lesion could theoretically cause it to rupture, a laceration contiguous to the NTD presents the highest potential for infection. If a person with an NTD has grown past childhood without developing any neurologic sequelae, he or she is at low risk for future neurologic problems.[2]

Scalp lesions similar to our patient's can be difficult to diagnose; however, a thorough history and physical examination quickly narrowed the differential diagnosis. Dermoid cysts are made up of epithelial cells, sweat glands, or hairs forming subcutaneous collections. They are typically nontender, firm, and noncompressible and have normal overlying skin.[3] None of these characteristics were apparent with our patient's lesion. Hemangiomas are collections of blood vessels just below the skin. They are rarely tender and are typically dark blue or red in appearance.

Lipomas are nontender, subcutaneous collections of fat cells. They are deep in the dermis, and the overlying skin glides smoothly over the nodule with palpation. Sebaceous nevi and aplasia cutis congenita are hairless lesions, each presenting with a bald patch of skin. Sebaceous nevi are a yellow-orange color and become bumpy with new growth as the child passes into adolescence. The lesion of aplasia cutis congenita represents a developmental anomaly that culminates in the loss of dermis, epidermis, fat, or all overlying skin tissue and is usually quite distinctive.[14]

Scalp lesions may also be confused with recent trauma or scar tissue. The location and associated findings, such as skin dimpling and increased concentration of hair follicles (hypertrichosis), should steer the examiner toward a broader differential diagnosis. In general, hypertrichosis, dimples, lipomas,

hemangiomas, or cysts overlying the spinal column or the midline of the skull should raise suspicion for underlying neurologic abnormalities.[15] A palpable skull defect should trigger serious concerns and lead to a thorough evaluation. Leakage of clear fluid from a scalp lesion should be treated as a medical emergency; it likely represents direct communication with the intracranial vault, and the athlete is at risk for a developing meningitis.

Our patient's lesion was mistakenly attributed to the scar from a fetal scalp electrode. However, the position of the lesion, low on the occiput, made this an unlikely cause because this site cannot be accessed to place a scalp monitor before delivery. Other characteristics should have raised the suspicion for some form of NTD. Excessive concentration of hair follicles, midline location, and dimpling of the skin increased the probability of a pathologic neurologic condition.[7] The lesion was painful, a common finding with meningoceles.[7] In one European study, 20% to 30% of all scalp lesions connected with the central nervous system in some fashion.[15]

After neuroimaging and evaluation by the consulting pediatric neurosurgeon, our patient was managed conservatively, forgoing surgical repair because she was asymptomatic at the time of the consultation. Resolution of her symptoms likely resulted from the healing of local trauma and inflammation that followed her initial traumatic insult. Indications for surgical repair of atretic meningoceles include cosmesis, persistent pain or tenderness, and avoiding rupture or ulceration.[7] Owing to a genetic link associated with some NTDs,[4] she started taking 800 μg of folate to decrease the risk of an NTD should an unplanned pregnancy occur. She was counseled to undergo further genetic counseling before any future planned pregnancy.

In this case, the potential for rupture or ulceration is low because the lesion is small and relatively flat. Any athlete with a pedunculated or bulging NTD is at higher risk for traumatic injury; however, these more obvious lesions are typically diagnosed and repaired in infancy. Given the low risk for rupture, our patient did not require any special protective equipment and continued her basketball season without interruption. The certified athletic trainer at her high school was told the diagnosis and its significance by the team physician. Had she experienced any trauma that resulted in breakage of the skin over or near the lesion, she would have required copious irrigation with sterile isotonic sodium chloride solution to the area. She would then have been immediately evaluated by the team physician if possible. Her risk for subsequent infection would be addressed at that time.

CONCLUSIONS

Neural tube defects are congenital anomalies typically diagnosed during infancy and childhood. However, misdiagnosis or medical inattention results in a few adolescents and adults having undiagnosed lesions. This young woman represents the first reported case in the sports medicine literature of an athlete with an atretic meningocele. Given the rate of occurrence, hundreds of young athletes nationwide likely have similar lesions. If athletic trainers are aware of the existence of such lesions, they can refer individuals with undiagnosed conditions to physicians for further evaluation. Affected women can then be given important genetic counseling and prescribed supplemental folic acid. Most importantly, errors will not be made in the evaluation and treatment of traumatic injuries, thereby avoiding potentially devastating infections of the central nervous system.

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Qualitative Inquiry in Athletic Training: Principles, Possibilities, and Promises

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Objective: To discuss the principles of qualitative research and provide insights into how such methods can benefit the profession of athletic training.

Background: The growth of a profession is influenced by the type of research performed by its members. Although qualitative research methods can serve to answer many clinical and professional questions that help athletic trainers navigate their socioprofessional contexts, an informal review of the *Journal of Athletic Training* reveals a paucity of such methods.

Description: We provide an overview of the characteristics of qualitative research and common data collection and analysis techniques. Practical examples related to athletic training are also offered.

Applications: Athletic trainers interact with other professionals, patients, athletes, and administrators and function in a larger society. Consequently, they are likely to face critical influences and phenomena that affect the meaning they give to their experiences. Qualitative research facilitates a depth of understanding related to our contexts that traditional research may not provide. Furthermore, qualitative research complements traditional ways of thinking about research itself and promotes a greater understanding related to specific phenomena. As the profession of athletic training continues to grow, qualitative research methods will assume a more prominent role. Thus, it will be necessary for consumers of athletic training research to understand the functional aspects of the qualitative paradigm.

Key Words: qualitative paradigm, naturalistic inquiry, interpretive research

In a recent publication, Knight and Ingersoll[1] suggested that the growth of the athletic training profession depends in part on the scholarly activity performed by its members. Research, as one form of scholarly activity, plays an essential role in revealing cause and effect, making associations among concepts, making comparisons, gaining insights, guiding decision making, and developing a sound knowledge base. As Weissinger et al[2] stated, one potential influencing factor involved with the development of a body of knowledge in a profession is an expansion of the methods used to collect and analyze data.

An informal appraisal of the past athletic training research in the *Journal of Athletic Training* reveals that quantitative research methods are currently a widely used form of inquiry. This is certainly not surprising given the scientific nature of the profession and the research questions that have been asked and answered within this paradigm. Although quantitative research has surely contributed to the advancement of knowledge and subsequent health care delivery in athletic training, we must recognize that both researchers and clinicians ask many questions that warrant the use of alternative methods. The purpose of our article, therefore, is to offer a first step in facilitating an understanding of qualitative inquiry within the field of athletic training. This article is divided into 3 main sections. In the first section, we will explain the primary characteristics of qualitative research. The second section focuses on common data collection and data analysis procedures. Finally, in the third section, we will discuss the future directions of qualitative research in athletic training. Throughout this article we will provide practical examples and possibilities, including how qualitative research can inform athletic trainers.

PRIMARY CHARACTERISTICS OF QUALITATIVE RESEARCH

The quantitative research paradigm takes a positivistic stance. That is, this paradigm assumes that a single objective reality exists,[3] which is ascertainable by our senses and logical extensions of our senses[4] (eg, microscopes, electrocardiograms, electromyograms). We can, therefore, measure and observe components of this single reality and test hypotheses about how one component affects another. The qualitative research paradigm, on the other hand, is based on the postmodern philosophical idea that multiple realities exist. Consequently, rather than our world being one objective and measurable entity, it is a subjective phenomenon that needs to be interpreted.[3] The qualitative paradigm recognizes that the meaning people give to situations and phenomena is crucial for understanding a particular context.[5] However, qualitative and quantitative methods are more than just different ways of researching the same items. Rather, they answer different types of questions, have different strengths, and use different techniques.[6]

Qualitative researchers are especially concerned with how people develop meaning out of their lived experiences.[7] Moreover, qualitative research is based on the idea that meaning is socially constructed. That is, meaning is created based on personal interactions with others and our environment and the

perceptions we give to our lived experiences. Therefore, qualitative researchers rely on a combination of textual data from interviews, conversations, and field notes rather than attempting to reduce meaning to numbers for comparative purposes.

Qualitative research can also be known as naturalistic inquiry, interpretive research, phenomenologic research, ethnography, and even descriptive research. Although qualitative inquiry can be performed in a variety of ways, common tenets are shared in this paradigm. Patton[8] discussed these common tenets as themes of qualitative inquiry. At a fundamental level, Patton[8] stated, qualitative inquiry is based on naturalistic inquiry, a holistic perspective, a focus on processes, inductive analysis, qualitative data, personal insights, case orientation, empathetic neutrality, and flexibility of design.

Qualitative researchers prefer natural or real-world settings. They do not attempt to control variables, manipulate procedures, create research or comparison groups, or isolate a particular phenomenon. Rather, qualitative researchers immerse themselves in a naturally occurring setting to observe and understand it. Thus, qualitative research tends to take a holistic perspective to inquiry. As such, the entire phenomenon under investigation is understood as a complete system rather than isolated events.

Qualitative research is most appropriate for answering questions relative to processes, site-specific phenomena, contexts, programs, or situations in which little is already known. As an example, "by what processes and in what ways have athletic trainers improved health care delivery in a rural school district?" is a question that is best answered using qualitative methods. "What is the economic impact of athletic trainers working in a rural school district?" is best answered using quantitative methods because economic factors are best measured with numbers.[9] An additional example is "in what way does approved clinical instructor status improve the educational delivery to student athletic trainers during their clinical education?" Such a question warrants qualitative methods because the approved clinical instructor programs will be new in the near future and little is known about the influence such programs will have on student learning.

Additionally, qualitative research is flexible and dynamic in that a researcher can choose which data to collect and how during the research process. In fact, qualitative research has metaphorically been compared with jazz music[10] because of the improvisation and flexibility needed to appropriately adapt the methods as findings unfold. Therefore, once researchers initiate a qualitative study and collect data, they need to be prepared to change their procedures and tactics as the process evolves and new insights are gained.

Qualitative research is inductive as opposed to deductive. The researcher begins with specific data and moves toward building general patterns.[8] That is, whereas an experimental design requires that a hypothesis be stated before the study in an attempt to either prove or disprove it, a qualitative study allows various dimensions to unfold or emerge, thus permitting hypotheses to become a product of the research. Moreover, qualitative inquiry is interpretive in that a researcher gathers a large amount of data with the intent of theorizing about the problem or phenomenon under investigation. Qualitative methods are a fundamental research strategy for many of the social sciences, including sociology and anthropology. Although qualitative research is derived from various epistemologic, philosophical, and methodologic traditions,[8] at its foundation are phenomenology and symbolic interactionism.[3]

Comparison of Qualitative and Quantitative Research Attributes

Qualitative Research Attributes	Quantitative Research Attributes
Inductive reasoning: drawing reasonable conclusions from one or several pieces of evidence; essentially moving from specific observations to general explanations or patterns[8]	Deductive reasoning: a conclusion is reached by following specific premises that are true; essentially moving from general explanations to creating facts
Generates a hypothesis[3]	Attempts to prove or disprove a hypothesis
Attempts to demonstrate trustworthiness[4]	Attempts to establish validity and reliability
Attempts to gain insight and understanding related to specific phenomena, cases, or situations	Attempts to determine cause and effect and predict outcomes[11]
Purposeful and small sample of participants relative to phenomenon to be investigated[8]	Random and large sample of subjects to ensure generalizability

Phenomenology focuses on an individual's experience, how people create their view of the world around them, and how they interact with their environment.[11] Researchers using a phenomenologic approach seek both a rich description of a context and a depth of understanding and meaning related to specific phenomena but from the participants' perspectives.[12] In athletic training, for example, such an approach could be used to address a phenomenon related to rehabilitation noncompliance, practitioner burnout, or nontraditional student experiences in an athletic training education program. As a more specific example, practitioner burnout could be investigated qualitatively to identify stress-coping strategies. Thus, practitioners could share their perspectives and describe how they attempted to cope with stress in a specific context. Such a qualitative investigation may uncover contextual issues that facilitated the burnout process.

Symbolic interactionism is a reaction to psychology's focus on intrinsic factors (eg, motivation or stress) and sociology's emphasis on extrinsic factors (eg, social class and structure) causing a specific behavior.[13] According to Blumer,[13] the symbolic interactionist framework suggested that (1) human beings act toward objects based on the meaning that the items have for them, (2) meaning is a product of social interaction in our society, and (3) the attribution of meaning to objects through symbols is a continuous interpretive process. An example of using a symbolic interactionist's framework in athletic training is examining the professional socialization process of various contexts (eg, intercollegiate athletics, high school, or professional ranks). Additionally, such questions as how medical decisions are made in a clinical context or how athletic trainers in various subcultures develop professionally over time are potential research topics that could be addressed from the symbolic interactionist perspective. At its foundation, however, qualitative inquiry is interpretive, relies on inductive analysis, and is concerned with the meaning created by participants. The Table identifies the key differences between qualitative and quantitative research.

QUALITATIVE DATA COLLECTION AND ANALYSIS

As with any research project involving human participants, a qualitative researcher must receive approval from an institutional review board. The review board ensures that the data collection and data analysis procedures protect the partici-

pants' anonymity. This is accomplished by giving any participants, institutions, or programs a pseudonym before any portion of the report is published. Qualitative researchers collect data in many ways, including interviews, observations, document analysis, artifacts (eg, photographs, videotapes, and tools), and surveys. Interviews and observations, however, are 2 of the most commonly used methods of gathering data in qualitative research. The following section will explain the observation and interview process and then describe how the textual data are analyzed.

OBSERVATION

Qualitative researchers often immerse themselves in a particular context and observe participants. Observation involves recording interactions among subjects, various events, a participant's behavior, and even a description of the context by taking field notes.[11,14] Such observations allow the qualitative researcher not only to recognize the essence of a context but also to identify particular behavior patterns and meanings.

Observation can be participatory or nonparticipatory. With participant observation, a qualitative researcher becomes involved in the actual activity being studied. For example, an athletic training researcher interested in understanding the contextual influences and dynamics of patient interaction within the professional ranks might volunteer with a professional team during practices. During this time, the researcher could not only provide health care services (ie, participate in the setting) but also observe the natural setting to further understand the dynamics involved. Nonparticipatory involvement means that the researcher does not participate in the activity while obtaining data. Rather, he or she watches a phenomenon in its natural setting.

INTERVIEWS

Interviews are conducted when a researcher needs to understand factors that cannot be observed.[8] For example, for a study conducted to gain insight into and understanding of why particular athletes play through pain, interviews would be necessary because the athlete's thoughts, feelings, and perceptions cannot be observed.

Interviews are also conducted when information about past events needs to be obtained. For example, a researcher investigating the professional socialization of intercollegiate athletic trainers may attempt to learn about the initial experiences and challenges they faced when first entering their work environment. Obviously, these experiences and challenges are not observable, so participants would need to be asked to reflect on these past events.

An interview can take many forms, including an unstructured, semistructured, or structured format.[3,12] Generally, however, a semistructured format is most commonly used[12] and directed by an interview guide. That is, based on the research question, an interview guide is designed to formulate a list of questions related to specific phenomena. A less structured interview guide is often preferred because it assumes that interviewees will explain, characterize, and define their contexts in unique ways.[3] Regardless of the type of interview conducted, the conversation is recorded (with the participant's permission) and transcribed. The data are then considered textual and the written words can be analyzed.

DATA ANALYSIS

Qualitative data analysis is interpretive in nature. Harris[15] reviewed the literature regarding interpretive research and identified 3 levels of interpretation that are necessary for drawing appropriate conclusions. First, the project must be grounded in the collective understandings of the culture created among the participants. Second, the project must include the researcher's insights. Third, the project must be well linked to other research. Harris[15] added that combining interpretations at each of these 3 levels into an integrated whole is paramount in qualitative research. The researcher interviews and observes participants (or specific behaviors if watching videotapes of social interactions) and then examines the data for meaning. We must make clear, however, that with qualitative research, the data analysis is a continuous and ongoing activity that occurs simultaneously with data collection. From the moment the first interview is conducted or the first observation is made, the researcher obtains a deeper understanding of the phenomenon being studied and may, accordingly, make modifications and adjustments to the data collection techniques.

Qualitative researchers have a preference for grounded theory, that is, developing theory based on the data obtained in a study.[16] According to Strauss and Corbin,[17] textual data are initially analyzed by creating concepts and categories. The researcher reads a sentence or paragraph and then gives this incident a name or label that represents it. These conceptual statements are then reviewed and grouped into categories according to their similarities. This is similar to Lincoln and Guba's[18] process of identifying units of data, such as sentences, paragraphs, or comments, that can provide information about a particular concept in and of itself. These "units of data" are then categorized according to their similarities with other units. The following is a useful sequence based on the literature[3,4,6,12] that helps a reader to understand how qualitative data are analyzed. Qualitative data analysis involves (1) identifying meaningful concepts (meaning condensation), (2) grouping similar concepts together (meaning categorization), (3) labeling groups of concepts (defining the categories), (4) developing theory, (5) negatively testing the theory, and (6) comparing the theory with the relevant literature.

Initially, the transcripts and observation notes are read and a participant's meaningful statements are identified, rephrased, and abridged. For example, if a student athletic trainer hypothetically suggested in an interview that he or she "spends a great deal of time each day having student-athletes tell them about their frustrations," this could be labeled as "listening." Therefore, meaning is condensed, and larger portions of text are reduced and made more succinct.[12] Essentially, the concepts identified are then considered to be units of data.

Once various concepts are identified and condensed, they are compared with one another. At this time, the like concepts are grouped together into categories. The various categories, or groups of concepts, are then given labels that describe the categories. For example, using the hypothetical situation above, if a researcher had several different concepts from interviews with student athletic trainers, such as "listening," "giving advice," and "empathizing," these could be categorized as "student athletic trainers' social support schemes." The researcher then examines the categories and interprets their relationship, subsequently creating a tentative theory. As Thomas and Nelson[19] stated, the researcher attempts to

"merge" categories into a holistic portrayal of the phenomenon under investigation.

The generated theory, however, must then be negatively analyzed. This means that the generated theory is tested for its plausibility. For example, after conducting 3 interviews and observing student athletic trainers for 4 weeks, a researcher identified and documented a particular sequence of social support schemes displayed by the participants. It would be necessary for this researcher to investigate the experiences of other student athletic trainers in the same or similar contexts to determine whether the theory or explanatory concepts are applicable. Moreover, a negative case analysis involves being skeptical about findings and searching for alternative explanations that link the various categories. Once the theory is developed, it is then compared with the related literature.

Although the data analysis can be done by hand using concepts printed on note cards, many computerized data analysis programs are currently available to qualitative researchers. Examples include the NUD*IST (Non-numerical, Unstructured Data require ways of Indexing, Searching and Theorizing) program, produced by QSR International (Melbourne, Australia), and The Ethnograph, produced by Qualis Research Associates (Amherst, MA). These programs offer qualitative researchers a structured database to organize concepts and categories and quickly find units of data in the transcripts.

Qualitative research is based on human interest and actively seeks to fully understand human behavior by becoming close to those being studied to expose factors that may not be identified with instruments or surveys.[8] Moreover, qualitative research tends to humanize data, problems, and issues,[20] presupposing that a phenomenon cannot be understood without empathy and introspection.[8] The researcher, however, is the primary data collection and data analysis instrument and is capable of extreme sensitivity and flexibility with regard to thoughtfully examining and organizing the data. Quantitative research, alternatively, attempts to be objective through blind experiments and collecting data with instruments that do not rely on human sensitivity.[8] A qualitative researcher's intimate involvement with participants and data often prompts the questions of researcher bias and how the reader of a qualitative research study can trust the interpretation of data.

Although quantitative research would be concerned with aspects of validity and reliability of data collection and analysis, these terms are not typically used in qualitative research. Rather, qualitative researchers are concerned with the "trustworthiness" or "authenticity" of the study. Trustworthiness of a qualitative study can be established in many ways, including triangulation,[4,6,11] peer reviews, and member checks.[3]

Triangulation refers to a researcher's cross-checking information from multiple perspectives. This can entail using different investigators, different methods (ie, observations and interviews), or even different data sources.[8] Using the previous example, if a researcher was gathering data related to student athletic trainer's social support schemes, it would be wise to interview not only student athletic trainers but also student athletes, supervising staff, and clinical educators. Thus, there are multiple sources from which to collect data and subsequently triangulate the findings to ensure that the findings are accurate and make sense in a given context.

Peer review requires that a highly skilled external researcher examine the transcripts, concepts, and categories generated from the study. The examination is performed to ensure that the study was performed in a logical manner and that the insights and discoveries uncovered in the investigation are credible. A member check refers to the qualitative researcher's sharing the initial results of the study with a few participants and asking them to examine the findings relative to their own experiences to ensure that the findings are plausible from the participants' perspectives.

Although quantitative studies concern themselves with sample size, this is not the case with qualitative research. Because a goal of quantitative studies is to attempt to generalize, large sample sizes are desirable. Qualitative research seeks to gain insight and understanding about particular phenomena, cases, processes, or programs. As such, qualitative research may be conducted with one participant or multiple participants, depending on the context or phenomenon under investigation.

FUTURE DIRECTIONS AND POSSIBILITIES IN ATHLETIC TRAINING

Many professions have affirmed the value and impact that qualitative inquiry can have on professional practice. In fact, many journals have committed to publishing qualitative research projects. Examples relative to athletic training include *Qualitative Health Research, Social Science and Medicine, The Gerontologist, Family Medicine, Culture, Medicine and Psychiatry, Advanced Nursing Science,*[21] *Research Quarterly for Exercise and Sport, Sociology of Sport Journal, International Review of Sport Sociology,* and the *British Medical Journal.* Although athletic training is largely a scientific field of study, we must recognize the potential promise qualitative research offers to help us further understand our professional roles in a social context.

The delivery of patient care is itself a social act that results in many interactions, which create shared meanings.[15] Athletic trainers associate with other professionals, patients, athletes, and administrators and, therefore, function in a larger society. Moreover, we cannot divorce ourselves from our context and the influences that affect us as health care providers. Consequently, we are likely to face critical influences and phenomena that affect the meaning we create. Qualitative research can facilitate a better understanding of phenomena and allow athletic trainers to better navigate their socioprofessional environments.

Arguments about whether quantitative or qualitative research has more merit have raged for many years[19] and have produced many debates and propositions. An either-or relationship, however, should not exist between qualitative and quantitative methods because, as we have discussed in this article, they answer different types of questions that facilitate an understanding of our professional roles and responsibilities. In many instances, a study can use both quantitative and qualitative methods in a mixed-methods approach. As an example, Hughes et al[22] used a mixed-methods approach to study the appeal of designer drinks among young people. These authors conducted group interviews (focus groups) to explore attitudes related to drinking and then used the qualitative results to inform the development of the questionnaire for the quantitative portion of the study. Furthermore, when a quantitative study uncovers a nuance or unexpected finding related to the human condition, a qualitative analysis could be integrated to gain a better understanding of the situation. The idea of combining methods, however, is not without debate. The multimethod approach is often contended because of the broad theoretical differences.[23]

When both the quantitative and qualitative paradigms are understood, valued, and sometimes integrated, the breadth and depth of knowledge in athletic training can expand and positively influence the lives of patients, clinicians, educators, and student athletic trainers. We have written this article to provide an initial step toward a better understanding of the basic principles of qualitative research for the readership of the *Journal of Athletic Training*. For a more comprehensive understanding of the qualitative research paradigm, we direct those interested to investigate the references and suggested readings listed below.

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Auscultation of the Chest and Abdomen by Athletic Trainers

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Objective: To present a practical overview of the methods and techniques of auscultation of the chest and abdomen for use during the physical examination of athletes. Our intent is to provide information on this clinical technique to assist athletic trainers in recognizing and referring athletes presenting with potentially serious internal organ conditions.

Background: Use of the stethoscope is a clinical skill increasingly necessary for athletic trainers. Given the expanding breadth of both the assessment techniques used by athletic trainers and the populations they care for and the fact that clinical instruction guidelines have changed in the newly adopted National Athletic Trainers' Association Educational Competencies, our goal is to provide a framework upon which future instruction can be based.

Description: This review covers the use of a stethoscope for auscultation of the chest and abdomen. Auscultation of the heart is covered first, followed by techniques for auscultating the breath sounds. Lastly, auscultation of the abdomen describes techniques for listening for bowel sounds and arterial bruits.

Clinical Advantages: During the assessment of injuries to and illnesses of athletes, knowledge of auscultatory techniques is valuable and of increasing importance to athletic trainers. Athletic trainers who do not know how to perform auscultation may fail to recognize, and therefore fail to refer for further evaluation, athletes with potentially serious pathologic conditions.

Key Words: murmurs, vesicular breath sounds, rhonchi, rales, borborygmi, bruits

The athletic training profession continues to grow and diversify. Clinical athletic training education has recently undergone a significant change as described in the newly adopted National Athletic Trainers' Association Educational Competencies.[1] In addition to the populations that certified athletic trainers (ATCs) work with in the typical scholastic, intercollegiate, and clinical settings, ATCs now are being placed in novel work settings (eg, industrial, secondary school, and performing arts settings) to work with nontraditional and varied populations. Furthermore, these populations (which number in the millions and include children, older adults, and individuals with disabilities) are participating in various forms of exercise training.[2] Certified athletic trainers are now serving all age groups of the physically active. As allied health professionals, ATCs are in a position to recognize potentially threatening conditions requiring referral, and, therefore, they need to possess the clinical skills necessary to perform this task.

The use of a stethoscope for auscultation of the chest and abdomen is a clinical skill that must be taught to all entry-level athletic trainers. Within the psychomotor domain, candidates for national certification will likely have to evaluate athletes who exhibit signs and symptoms associated with cardiopulmonary and gastrointestinal conditions and will have to use a stethoscope correctly for auscultation of the heart, lungs, and bowel. To identify abnormal sounds on auscultation, ATCs should be familiar with normal chest and bowel sounds. The auscultatory examination may provide information that can assist the ATC in making a medical referral that may point the way to necessary therapy.

The organs of the chest and abdomen to be auscultated are of importance individually, but they are anatomically and physiologically linked to other organs and must be viewed in a comprehensive fashion.[3] During auscultation, the examining ATC will recognize that sounds vary according to the underlying normal or pathologic anatomy or disease state of the organ. Sounds that are heard originate from movements of the organs or parts thereof, the flow of air, or movement of fluids through the organs; these sounds may vary greatly in normal as compared with abnormal or diseased organs. The purpose of this clinical techniques paper is to present a practical overview of the methods and techniques of auscultation of the chest (ie, the heart and lungs) and abdomen for use during the examination of athletes. The findings from such an examination may warrant referral. This paper is not intended to provide ATCs with the knowledge to diagnose any condition present in the heart, lungs, or abdomen but rather to detect abnormalities and report these abnormalities to a physician for confirmation. Our intent is to provide information on this important clinical technique to help ATCs recognize and refer athletes with potentially serious internal organ conditions.

THE AUSCULTATION EXAMINATION

To become proficient at auscultation, ATCs must examine many athletes under the guidance of an experienced instructor or physician to learn the skills needed to identify the physical

abnormalities associated with various conditions. The use of audiotapes presenting recorded auscultatory sounds will aid in this learning process but will not substitute for live examination. Also, the use of a cardiology teaching device, such as the Harvey Cardiology Patient Simulator (Center for Research in Medical Education, University of Miami School of Medicine, Miami, FL), facilitates learning by demonstrating numerous pathologic heart sounds.

Before auscultation of the chest and abdomen, the ATC should obtain a thorough subjective history from the athlete, targeting his or her personal and family health history. The personal health history should address (but is not limited to) issues such as past injuries and illnesses, allergies, childhood illnesses, current medications, diagnostic tests, diet, exercise level, general health (including any current symptoms), immunization status, surgeries and hospitalizations, sleeping habits, and substance abuse. The family history should address (but is not limited to) the age, health, and cause of death of family members. The family history should also address the presence of allergies, anemia, arthritis, asthma, cancer, diabetes, epilepsy, heart disease, hypercholesterolemia, hypertension, psychiatric illness, renal disease, stroke, substance abuse, and tuberculosis.

After taking the history, the examining ATC should measure the 4 basic vital signs to provide a foundation for the assessment: core temperature, arterial pulse rate, respiratory rate, and sitting blood pressure. Only by measuring these basic vital signs can the correct assessment and interpretation of the auscultatory findings be made. Additionally, the examining ATC should observe the athlete for pallor, cyanosis, jaundice, cough, and dyspnea and should note the athlete's general demeanor in an attempt to gather information on his or her specific medical condition.[4]

Auscultation simply requires the use of a good acoustic stethoscope. The stethoscope head should be equipped with a rigid diaphragm for effective transmission of high-frequency sounds and a bell for effective transmission of low-frequency sounds. The tubing that transmits sounds to the earpieces should be of heavy gauge, to better transmit all sounds, and from 31 to 40 cm in length.[4]

Auscultation of the Heart

The indications for an ATC to perform cardiac auscultation are numerous and include, among others, the following 9 items. Any athlete presenting with any of these signs or symptoms should be referred for examination by a physician.

1. Evidence of cyanosis of the skin and mucous membranes accompanied by clubbing of the nails could indicate congenital heart disease, which would reveal cardiac murmurs upon auscultation.[5] An ATC who finds such cyanosis should immediately refer the athlete to a physician. In addition, cyanosis can be caused by severe chronic obstructive pulmonary disease in older adult athletes, which also requires immediate referral. Cyanosis, no matter what the cause, may be the result of an acute life-threatening condition warranting the immediate administration of emergency medical techniques and activation of the emergency medical system.
2. Any signs of Marfan syndrome, such as very tall stature, spider fingers, a high and arched palate, excessive arm length, chest-wall deformity, or a family history of sudden cardiovascular death at a young age.[6,7]
3. Palpitations, a history of syncope, or dizzy spells (discussed subsequently).
4. A very rapid pulse (tachycardia) over 100 beats per minute at rest or a very slow pulse (bradycardia) of less than 40 beats per minute.
5. Unusual and severe dyspnea after routine exercise.
6. Sustained arterial hypertension.
7. Any symptoms of hypertrophic cardiomyopathy (HCM), which can cause sudden death in athletes after severe exertion. The average age of presentation for this condition is 26 years, and such athletes usually have a family history of sudden death. They commonly present with dyspnea (due to an elevation in pulmonary venous pressure secondary to elevated left-ventricular diastolic pressure) and may also present with angina, syncope, fatigue, and palpitations. Unfortunately, many patients are asymptomatic until the sudden death event.[8,9] A harsh systolic murmur on auscultation is evidence for HCM, and the athlete should be referred to a physician immediately.
8. Complaints of effort-related retrosternal pain relieved by rest (ie, angina pectoris). Angina heralds the probable occurrence of an acute heart attack and is a manifestation of coronary heart disease. Recent studies, including autopsy studies, have shown that coronary heart disease is a cause of sudden death in athletes, including marathon runners.[10]
9. Traumatic chest injuries. Blunt, nonpenetrating chest trauma can cause cardiac concussion, "commotio cordis," or myocardial contusion, which can result in cardiac arrest. Such trauma can occur in young athletes from a blow to the chest (including blows from projectiles such as baseballs and even hockey pucks) and can cause sudden death. The survival rate is very low; Maron et al[11] reported 25 fatal cases. Specifically, the traumatic precordial blow is delivered at an electrically vulnerable period of ventricular excitability, thus inducing cardiac arrest. Almost all victims are under 20 years of age.[11–13] Experimental models are now being developed to study this serious problem.[14]

For optimal auscultatory examination of the heart, an athlete should be approached from the right side. A right-sided approach facilitates effective observation, percussion, and auscultation of the precordial areas as well as assessment of cardiac size.[4] The athlete should be at rest and recumbent with the head and chest elevated to 45°. This optimum position is preferred over the upright seated position. We recommend this position because it facilitates the observance of chest movements associated with cardiac function as well as correct placement of the stethoscope head during auscultation. When auscultation is performed in the athletic training quarters, the examination room should be warm, very quiet, and well lighted. Patient modesty is to be maintained, and a same-sex chaperone should be present when necessary. Obviously, in cases of trauma, the ATC may need to modify the examination position or improvise, according to the severity of the situation.

The examiner should begin by determining the approximate size of the heart. Except in very obese patients, heart size can be determined by noting the point of maximum impulse (PMI), otherwise known as the apical impulse. The PMI is produced by an early, brief, left-ventricular pulsation as the heart moves anteriorly during systole and comes in contact with the chest wall. The PMI should also be assessed by palpation. The PMI is the most inferior and lateral position at which the apical impulse can be clearly viewed and palpated, and its location

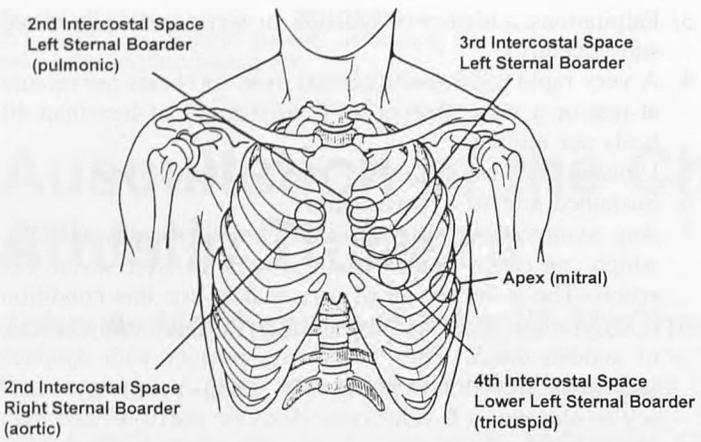

Figure 1. Points of cardiac auscultation. Reprinted with permission from Crawford M. *Heart Sounds: A Practical Guide.* Point Pleasant, PA: Merck, Sharp & Dohme; 1989:10.

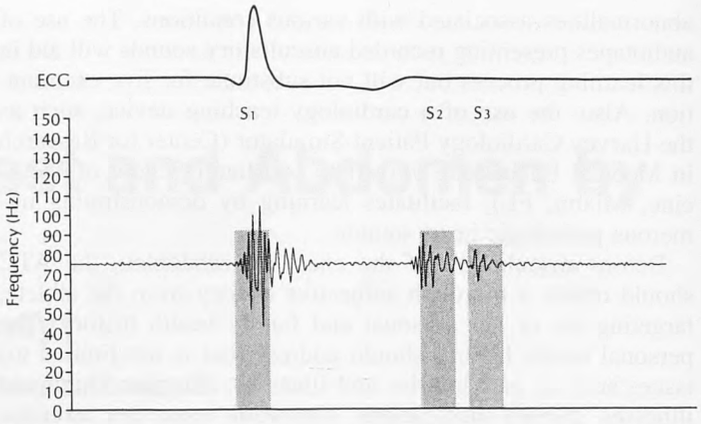

Figure 2. The physiologic heart sounds of a 21-year-old athlete. Reprinted with permission from Crawford M. *Heart Sounds: A Practical Guide.* Point Pleasant, PA: Merck, Sharp & Dohme; 1989: 16.

reflects heart size.[4] Its position normally corresponds to a point in the midclavicular line near the level of the left fifth intercostal space at the apex (Figure 1). If the heart is enlarged, the PMI may be found lower, near the sixth intercostal space and at the anterior axillary line. A cardiac pulsation detected to the right of the sternum is always abnormal and may indicate aneurysmal dilatation of the ascending aorta, situs inversus, or right-ventricular enlargement. Heart enlargement should alert the examining ATC that the athlete may have congenital heart disease, cardiac dilatation due to longstanding hypertension, pericardial effusion, or some serious valvular heart disease.[15] All of these conditions also present with abnormal heart sounds.

Examples of some of these abnormal sounds include the diastolic murmur of aortic valve insufficiency, which sounds like a decrescendo whispered letter *R*. The systolic murmur of mitral valve regurgitation has a blowing sound.[16] Systolic murmurs vary in character: some sound like cooing, whereas others sound like honking. Another systolic murmur frequently heard in young people is caused by mitral valve prolapse and is usually accompanied by a clicking sound.[17] In contrast, in individuals with a pericardial effusion, the heart sounds are distant, and any murmur may be difficult or impossible to hear due to the intervening pericardial fluid. Please refer to the later section on murmurs for a more complete discussion of these sounds.

The examiner then begins auscultation by putting the fingers of one hand on an arterial pulse of the athlete, usually the radial or carotid pulse, while listening to the heart.[3,18] Palpation of the carotid artery is preferred because it provides the most accurate representation of the central aortic pulse.[19] Palpation of the pulse can be very valuable for determining the timing (systolic or diastolic) of the murmur being simultaneously auscultated (discussed subsequently). Palpation of the pulse is also fundamental in determining the pulse rate as well as the pulse rhythm. A regular rhythm is a sinus rhythm, whereas irregular rhythms can occur in a variety of cardiac conditions such as atrial fibrillation.

The examiner should lightly and evenly press the diaphragm end of the stethoscope to the exposed precordium to auscultate the chest. The hand holding the diaphragm should be positioned such that the heel of this hand rests on the chest and the fingers are free to hold the stethoscope head stable. The auscultation then proceeds with application of the stethoscope to the 4 main valve areas (Figure 1). The examiner must also listen at the valvular area where the cardiac sounds are best heard: the aortic, pulmonic, tricuspid, and mitral valve listening zones. Simultaneous arterial palpation during auscultation allows the ATC to determine the timing of any cardiac murmurs. The importance of the timing cannot be overemphasized. If the murmur is heard simultaneously with the arterial pulse, then the murmur is systolic. If the murmur is heard after the pulse, then the murmur is diastolic, a finding of certain pathologic significance. This technique of simultaneous pulse palpation during auscultation is widely used in medical education for determining the timing of murmurs and, therefore, is appropriate for use by ATCs. It is very helpful to any clinician faced with identifying murmurs accompanied by atrial fibrillation, for example.

The 4 valvular areas are auscultated to assess the following 4 heart sound components:

1. Cardiac rhythm. Note if the rhythm is regular and constant or irregular and skipping beats.
2. Heart sounds (S_1 and S_2). These sounds are commonly referred to as the lub-dub sounds. The S_1 sound, or lub, is caused by the closure of the mitral and tricuspid valves, whereas the S_2 sound, or dub, is due to closure of the aortic and pulmonic valves.[3] These sounds should be clear and distinct. They are depicted in Figure 2. In young athletes with high cardiac outputs, a third, normal heart sound (S_3) may be heard. The S_3 sound occurs during the rapid-filling phase of the left ventricle with some recoil of the left ventricle; it sounds like lub–dub-dub and is called ventricular gallop. Although this third heart sound is often of no concern, when it is detected by an ATC, referral or physician consultation is warranted.
3. Murmurs. These sounds are produced by turbulent blood flow through partially obstructed or incompetent cardiac valves or septal defects within the heart.[20] The resultant vibrations range from 50 to 500 Hz and vary greatly. An example of a murmur caused by obstruction of valves is the systolic murmur of partial aortic or pulmonic valve stenosis. Murmurs due to valvular incompetence include the systolic murmur of mitral valve regurgitation and the diastolic murmur of aortic valve incompetence, which sounds like a whispered, prolonged letter *R*.[16] Systolic murmurs are heard as abnormal sounds between S_1 and S_2, whereas

diastolic murmurs are heard after S_2 and before S_1. These murmurs not only vary in timing (systolic and diastolic) but also in length, pitch, and intensity of loudness. They may present as sounds that are rough, blowing, musical, cooing, or honking or sound like a whispered, prolonged *R* sound (the latter in individuals with aortic valve incompetence).[16] Recall that as one listens with the stethoscope, the fingers are to remain on an arterial pulse to assess the timing of these murmurs. High-pitched murmurs, as well as S_1 and S_2 sounds, are best heard with the diaphragm of the stethoscope, whereas those with low pitch are better heard with the bell head.[4] In certain hyperkinetic states, a cardiac murmur may be physiologic in origin and benign. However, a note of caution is called for here. Wood,[21] in a landmark cardiology text, stated that to dismiss a murmur as "functional" is inappropriate and that functional murmurs are not insignificant and are not meaningless.[21] It is beyond the scope of an ATC to determine whether or not a murmur is pathologic. All athletes presenting with previously undiagnosed murmurs must be referred to a physician.

4. Extracardiac sounds. These sounds are harsh, rough, to-and-fro, and rubbing sounds due to pericarditis and are sometimes heard during auscultation. They must be heard in both systole and diastole in order to be attributed to pericarditis. Unless the friction rub sounds are heard in all phases of the cardiac cycle, the sounds may be arising from structures other than the pericardium. An example is pleuropericardial rubs. Pleural rubs disappear when the patient holds his or her breath, whereas pericardial rubs do not.[22] Extracardiac sounds due to pericarditis are heard more clearly and are termed "close to the ear." Pericarditis is frequently due to viral infections or may be present during acute myocardial infarction. Athletes presenting with extracardiac sounds should be referred.[22]

Auscultation of the Lungs

Auscultation of the lungs should be performed on athletes who exhibit 1 or more of the following complaints or signs, which are given only as examples and are not intended to be a complete listing. These signs and symptoms are reasons for referral to a physician for further examination.

1. Dyspnea, or shortness of breath, which is present at rest or disproportionate to the subject's exercise activity.[23] Dyspnea can indicate conditions such as (but not limited to) pneumonia, pneumothorax, asthma, or heart failure.
2. Cough, with or without hemoptysis, which can indicate pneumonitis, bronchitis, fibrotic lung disease, or even bronchial carcinoma.
3. Pleuritic pain, which can be due to acute inflammation of the parietal pleural surface, herpes zoster involving intercostal nerves, or rib fracture.
4. A resting respiration rate of 22 per minute or more. The athlete's lungs should be auscultated to rule out underlying lung disease.
5. Cyanosis or finger clubbing, which can indicate a number of pulmonary diseases, including chronic suppurative disease or pulmonary carcinoma.[24,25]

During auscultation of the lungs, the athlete should be seated upright on an examination table, with his or her legs over the edge. The athlete's neck should be slightly forward flexed with his or her arms folded. This position allows the scapulae to move laterally, which enlarges the lung examination area of the posterior and lateral thorax. When the examiner completes the examination of the posterior thorax (including the lung apexes) and moves to the anterior areas, the athlete's arms are then returned to the sides.

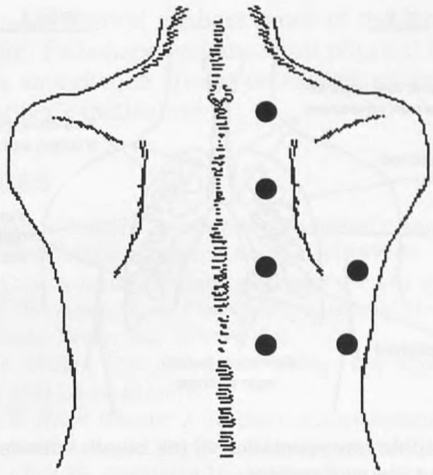

Figure 3. Points of breath sound auscultation on the patient's posterior chest.

The athlete is then asked to take slow, deep, long breaths while the ATC looks for symmetric lung expansion. The diaphragm head of the stethoscope is applied lightly over the posterior and lateral aspects of the chest to hear the breath sounds. The lungs are auscultated over the posterior chest wall, beginning at a point midway between the superomedial border of the scapula and the spine and descending to the level of the diaphragm. Then the ATC listens over the posterolateral chest (Figure 3). Upon completion, the anterior chest is auscultated.

Areas over major bronchi and the posterior chest midline should be avoided during auscultation because normal bronchial breath sounds are typically heard in these areas. This caution in technique is stressed because bronchial sounds are usually heard over areas of lung consolidation but are also heard over the trachea and major bronchi during examination of the thorax in normal individuals. Normal breath sounds are soft and low pitched and caused by the flow of air through the bronchi and alveoli. These breath sounds are termed vesicular; they are heard through inspiration and fade away during expiration. Types of normal (well) and abnormal (ill) breath sounds are shown in Figure 4. The ATC should listen for any abnormal breath sounds, including the following sounds:

1. Crackles, or rales, are short, discontinuous sounds heard at the end of inspiration and are of 2 types. Dry or fine rales sound like rubbing dry strands of hair together between the thumb and finger close to the ear and are usually heard in individuals with pneumonia. The other type of rales are termed moist or coarse rales. These rales mimic the sound of a hook-and-loop fastener opening and are frequently heard in heart failure.[26] Pathologic rales are associated with serious pulmonary disease and do not disappear after a sharp cough. Benign rales, which are not associated with pulmonary disease, ordinarily disappear after coughing.
2. Wheezes, also called sibilant wheezes, are high-pitched musical sounds heard during inspiration and expiration. These sounds are caused by the passage of high-velocity air through bronchi narrowed by bronchospasm, tenacious mu-

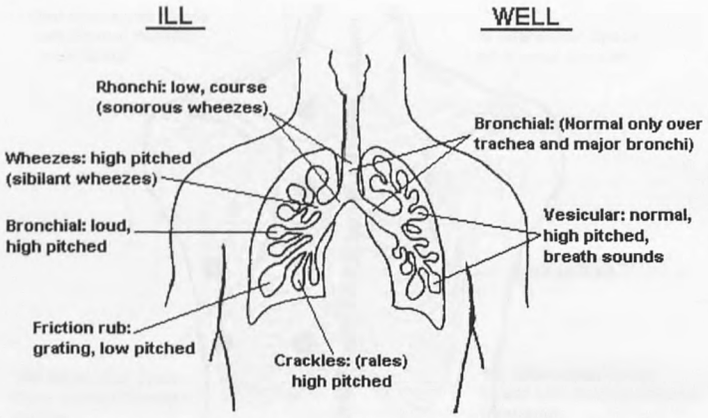

Figure 4. Graphic representation of the breath sounds found in the ill patient and the well patient.

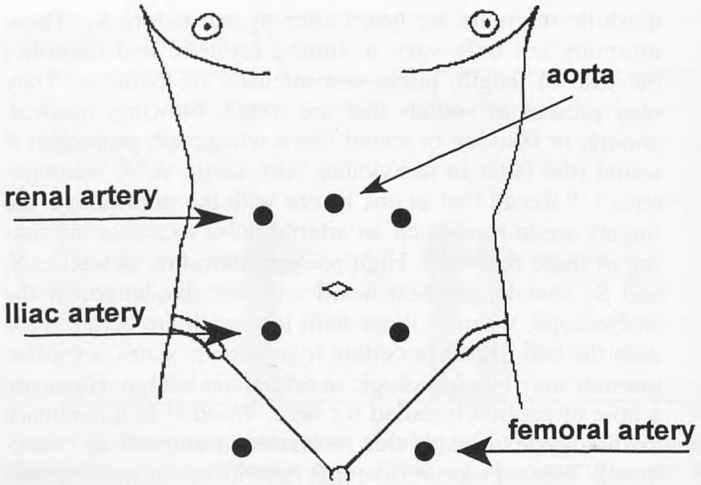

Figure 5. Points of abdominal auscultation.

cus, or foreign bodies. They are heard frequently in individuals with asthma and chronic bronchitis.[27]

3. Rhonchi, also called sonorous wheezes, are deeper-sounding, more rumbling breath sounds. They are usually caused by the passage of air through bronchi obstructed by thick mucus. Lower-pitched rhonchi arise from the larger bronchi (for example, those affected by tracheobronchitis) and usually disappear after coughing.[27]
4. Bronchial breath sounds are loud, relatively high-pitched sounds with a short pause between inspiration and the longer expiratory component. These sounds may be heard in areas of lung consolidation (solidification), which may occur in cases of consolidated pneumonia or some cases of atelectasis (a shrunken and airless lung). These bronchial breath sounds are transmitted to the examiner through a solid medium, the consolidated lung. This phenomenon produces the same sounds as those heard when auscultating over a bronchus. When sounds are transmitted through air-filled lungs, the sounds are less intense.
5. A pleural friction rub may be auscultated in the presence of inflammation of the pleural surfaces. A grating, low-pitched sound is heard during inspiration and is caused by friction between the 2 inflamed pleural surfaces. It is heard in cases of pleurisy in which the patient most often complains of chest pain on inspiration.
6. Absence of breath sounds may be due to pleural effusion, pneumothorax, hemothorax, or blockage of a major bronchus.

Auscultating the chest provides the ATC with information concerning the pitch, length, and intensity of an athlete's breath sounds during inspiration and expiration. Normally, clear, vesicular breath sounds are heard when auscultating the athlete's lungs. Abnormal and questionable findings should prompt an ATC to refer the athlete for further examination.

Auscultation of the Abdomen

With the high incidence of collision in sports, the number of athletes complaining of abdominal pain, and the relative frequency of abdominal injuries to athletes, this straightforward examination technique for auscultating the abdomen can be very useful to ATCs. After the athlete's history has been obtained, he or she should be placed in a supine position with the hips and knees flexed for abdominal auscultation. After taking a relevant history and observing the athlete with a potential abdominal injury or illness, the examiner should proceed to auscultate over the 4 quadrants of the abdomen. Palpation of the abdomen is performed after auscultation. During abdominal auscultation, the ATC listens for abnormalities of intestinal motility using the diaphragm head of the stethoscope. Normal bowel sounds include sounds that are scattered and low pitched and loud stomach growls (borborygmi) in the abdominal quadrants. These sounds can be heard at times, as we all are aware, in normal individuals both with and without the aid of a stethoscope. Bates[28] noted that normal bowel sounds are quite variable in number, ranging from 5 to 35 sounds per minute. Bowel sounds may be increased in individuals with gastroenteritis and diarrhea. Tinkling bowel sounds may be heard in individuals with bowel obstruction. Decreased or absent bowel sounds may occur in individuals with peritonitis, internal organ injury, and paralytic ileus. Before determining that bowel sounds are absent, one should listen in the right lower quadrant for at least 2 minutes.[29]

Additional sounds that can be heard in the abdomen are arterial bruits, which resemble harsh, rough, murmur-like sounds. These sounds are best heard with the bell head of the stethoscope, which can be pressed more deeply into the wall of the abdomen than can the diaphragm, bringing the bell closer to the stenosed artery emitting the bruit sound. This technique has been used over many years by the primary author and has proven successful and rewarding in confirming diagnoses.

These bruits are not only heard in partially stenosed arteries, but they also arise from abdominal arterial aneurysms; the incidence of such aneurysms is increasing as the general population ages.[30] These bruits are heard in older adults and occasionally in younger adults with Leriche syndrome (partial abdominal aortic occlusion).[31] Although the incidence of abdominal vascular stenosis pathology is not high in the traditional young athlete population, ATCs' patient populations have expanded to include large numbers of physically active older adults. These older individuals have a much higher incidence of arterial stenosis and aneurysms, which may be detectable by abdominal auscultation. The ATC should be able to recognize and refer these vascular problems that, not infrequently, are life threatening. The renal, iliac, and femoral artery areas of the abdominal quadrants should be auscultated for arterial bruits (Figure 5).

The presence of any bruit is obviously of great importance.

Patients with bruits may present with symptoms of intermittent claudication of the lower extremities when the common iliac or more distal arteries are involved in the stenotic process.

Of great importance also are renal bruits auscultated in patients with hypertension caused by partial stenosis of a renal artery, the Goldblatt kidney.[32] This bruit is sometimes heard near the abdominal flank. The associated hypertension is termed renovascular hypertension, and in a large study, these bruits were heard in 46% of the patients.[33] When renovascular hypertension occurs in young adults (under age 35), it is usually due to fibromuscular dysplasia, which partially obstructs a renal artery, causing a bruit; in contrast, in older adults, the stenosis typically results from atherosclerosis.[34,35] For both causes, corrective surgery can reestablish the normal blood pressure. Certified athletic trainers should always be alert to this serious, remediable problem in any hypertensive athlete. The ATC should always refer any athlete presenting with sustained hypertension (with or without an abdominal bruit). Information on renovascular hypertension and the frequency of bruits is being included because of the diversity of the patient populations seen by ATCs. Although renovascular hypertension is a cause of hypertension in athletes of all ages, it is treatable and curable.

CONCLUSIONS

Since its invention by Rene Laennec in 1819, the stethoscope for auscultation of the chest and abdomen has proven to be a necessary and indispensable tool for the examination of patients. The technique of auscultation unmasks pathology in the chest and abdomen that would otherwise remain undiagnosed and untreated. Using auscultatory techniques, structural abnormalities that are causing an athlete's complaints and disability can be identified.

The modern ATC should acquire this skill in order to adequately examine physically active individuals. A wide variety of people of all ages, from the pediatric population through the geriatric population, depend on ATCs to provide competent primary examinations and care before they are seen by a physician and to refer them as needed. In response to this need, the National Athletic Trainers' Association now mandates that ATCs be able to auscultate the chest and abdomen and identify the normal heart, lung, and bowel sounds. This worthy clinical proficiency requires training in the recognition of both normal and pathologic states, and it takes much practice. The athletic trainer who learns these techniques must hear both the normal and abnormal sounds, either from an apparatus or from a patient-athlete with the abnormality, in order to know what is normal and what is abnormal. In the same way that an ATC must understand ligamentous injuries in order to competently assess the normal state of capsuloligamentous stability, so must an ATC have knowledge of pathologic heart, lung, and bowel sounds in order to effectively identify the normal sounds of the chest and abdomen upon auscultation.

We hope that this paper will provide a framework upon which future instruction in these clinical techniques can be based and a stimulus for the further study and observation of patient-athletes. This overview is not intended to enable the ATC to render the type of evaluation rendered by physicians but rather to offer knowledge on techniques that will allow the ATC to gather information on possible conditions afflicting athletes. We in no way imply or suggest that the ATC should act as a physician but rather that the ATC should gather information on the clinical medical status of the athletes under his or her care. Pathologic and abnormal physical findings detected during auscultation always necessitate referral to a physician for further examination.

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A Review of Sudden Cardiac Death in Young Athletes and Strategies for Preparticipation Cardiovascular Screening

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Objectives: To provide the reader with an overview of the many causes of sudden cardiac death in young athletes and to present various strategies for preparticipation cardiovascular screening.

Data Source: A MEDLINE search using the phrase *sudden cardiac death* and the key word *athlete* for the years 1980 to 2000.

Data Synthesis: Sudden cardiac death is a rare event in athletics. More than 20 different causes have been described, but most cases result from a few distinct entities. Most afflicted athletes have no symptoms before death. Many attempts have been made to detect those at risk for sudden cardiac death before athletic participation. At this time, a thorough history and physical examination are the most efficient screening methods for detecting cardiovascular abnormalities. Studies show that the current status of preparticipation cardiovascular screening of high school and college athletes nationwide is poor.

Conclusions and Recommendations: The use of diagnostic tests to screen for cardiovascular abnormalities is ineffective and inefficient. The most prudent and effective methods of preparticipation screening for cardiovascular abnormalities at this time are a history and physical examination in accordance with the American Heart Association guidelines. Athletic trainers must ensure that their institutions comply with these minimum standards.

Key Words: sudden cardiac death, hypertrophic cardiomyopathy, preparticipation athletic evaluation, coronary artery anomalies

The sudden, unexpected death of a young athlete is a tragedy unparalleled in sports. Aside from the grief of friends and family members, shock waves reverberate as the community, institution (high school, college, or professional organization), and sports medicine team all cope with the death. Instinctively, those involved wonder what intervention might have prevented the death. Occasionally, the search for answers may spawn a parallel search to assign blame.[1] Every case of sudden death in a young athlete garners large amounts of media attention. Indeed, such publicity may influence the public perception as to the frequency of these events. Although sudden cardiac death (SCD) is a long-recognized entity, the deaths of Hank Gathers and Reggie Lewis in the early 1990s focused new attention on the condition.[1–6]

Fatal sport-related injuries can result from head and cervical spine trauma, but most sudden deaths in athletes are cardiac in origin.[7,8] An enormous amount of research has been generated during the past 10 years evaluating the causes and events surrounding SCD and potential screening mechanisms for identifying those at risk. Concomitantly, the 1990s ushered in a renewed interest and changing focus in the preparticipation athletic evaluation (PAE), as researchers and sports medicine practitioners implemented a variety of methods to improve the long-standing and often controversial "sports physical." Currently, these approaches vary, but several thorough and practical evaluation models have been proposed.[9–15]

The purpose of this article is to present the athletic trainer with a practical base of knowledge from which to develop the cardiovascular portion of the PAE for young competitive athletes. With this knowledge, the athletic trainer, in conjunction with the team physician, can review their institution's current PAE format. If necessary, changes can be implemented to develop a more prudent and effective means of screening for potential cardiac anomalies. Although SCD affects all age groups and can occur in any setting, my discussion will focus on athletes younger than 35 years.

INCREASING AWARENESS

The first recorded sudden death of an athlete was that of Pheidippides, a young long-distance messenger, in 490 BC. On arrival in Athens, he reported the defeat of the Persian army and then fell dead.[15] The more recent deaths of several well-known athletes have brought SCD into the public consciousness. Athletes who experienced SCD include marathon runner Jim Fixx (1984), Olympic volleyball player Flo Hyman (1986), former basketball star Pete Maravich (1988), college basketball star Hank Gathers (1990), professional basketball All-Star Reggie Lewis (1993), and Olympic figure skating champion Sergei Grinkov (1995). All died from cardiac causes.

The deaths of Hank Gathers and Reggie Lewis spurred an increased awareness of SCD in the sports medicine community and the public.[1,2,16] As a student athletic trainer in the early

Table 1. Conditions Linked to Cases of Sudden Cardiac Death*

Hypertrophic cardiomyopathy
Idiopathic left ventricular hypertrophy
Arrhythmogenic right ventricular dysplasia
Congenital coronary artery anomalies
Atherosclerotic coronary artery disease
Hypoplastic coronary arteries
Tunneled coronary arteries
Marfan syndrome
Myocarditis
Wolff-Parkinson-White syndrome
Prolonged QT syndrome
Severe valvular heart disease (aortic stenosis, pulmonic stenosis)
Idiopathic ventricular tachycardia
Congenital heart disease
Coarctation of the aorta
Coronary artery disease
High-grade ventricular arrhythmias
Illicit drugs (eg, cocaine, methamphetamines, inhalants)
Performance-enhancing drugs (eg, anabolic steroids, erythropoietin)
Commotio cordis

*Adapted from Reisdorff and Prodinger.[30]

1990s, I recall the impact of the Gathers' tragedy as SCD became a frequent subject of athletic training room and classroom discussions. What was once thought of as an exceedingly uncommon occurrence at the time took on the appearance of a commonplace event. Fortunately, SCD remains rare.

EPIDEMIOLOGY

Although not a universally accepted definition, SCD can be considered a nontraumatic, nonviolent, unexpected death due to cardiac causes within 1 hour of the onset of symptoms.[17] The exact incidence of SCD is difficult to ascertain, because many studies have relied on the self-reporting of physicians and media accounts of deaths.[7,8] The National Federation of State High School Associations estimates 10 to 25 cases of SCD per year in individuals younger than 30 years.[18] Although not representative of the general population, given the presumed state of good health, data compiled from 1965 to 1985 show the incidence of sudden death to be only 1 per 735 000 screened US Air Force recruits between 17 and 28 years of age.[19] A study of Minnesota high schools revealed 3 individuals succumbing to SCD during a 12-year period, translating to a risk of 1 death per 200 000 athletes per year.[20] The incidence of sudden death during exercise in unscreened men younger than 30 years was estimated to be 1 death per 280 000 men per year in Rhode Island.[21]

In a landmark study, Maron et al[7] detailed the clinical, demographic, and pathologic profiles of 134 young, competitive athletes experiencing SCD from 1985 through 1995. The mean age was 17 years (range, 12 to 40 years); 90% were male and 44% were black. Basketball and football players accounted for 68% of the deaths. The National Center for Catastrophic Sports Injury Research[8] identified 160 athletes dying from nontraumatic causes (78% of deaths were from cardiac causes) in high school and college sports between June 1983 and June 1993. The estimated death rate of male athletes was 5-fold higher than for female athletes (7.47 versus 1.33 per 1 000 000 athletes per year), and 65% of the deaths occurred during participation in basketball or football. Interestingly, male college athletes had twice the estimated death rate of their high school counterparts (14.5 versus 6.6 per 1 000 000 athletes per year).

Table 2. Most Common Causes of Sudden Cardiac Death in 134 Athletes*

Cause	No. (%)
Hypertrophic cardiomyopathy	48 (36)
"Possible hypertrophic cardiomyopathy"	14 (10)
Aberrant coronary arteries	17 (13)
Other coronary anomalies	8 (6)
Ruptured aortic aneurysm	6 (5)
Tunneled coronary artery	6 (5)
Aortic valve stenosis	5 (4)
Lesion consistent with myocarditis	4 (3)
Idiopathic myocardial scarring	4 (3)
Remaining 8 causes	22 (15)

*Adapted from review by Maron et al.[7] Reprinted with permission from *JAMA*. 1996; 276. 199–204. Copyrighted (1996), American Medical Association.

THE ATHLETE'S HEART AND CARDIOVASCULAR RESPONSE TO EXERCISE

A brief overview of adaptive cardiovascular physiology is necessary before a discussion of the causes of SCD. Exercise results in both hemodynamic and electrophysiologic changes within normal myocardial tissue. During intense aerobic exercise, the oxygen consumption of muscle tissue increases markedly, and cardiac output must rise to meet the demands. Over time, aerobic training results in increased left ventricular mass, increased heart rate during exercise (decreased resting heart rate), increased ventricular stroke volume, and increased cardiac output, among other effects.[22]

First demonstrated in 1935, physiologic hypertrophy of the heart in response to cardiovascular conditioning is prevalent among well-trained athletes.[23] The amount of adaptation depends on the intensity of training and can be assessed through changes on physical examination, electrocardiography (ECG), and echocardiography.[15,23,24] The left ventricle hypertrophy is typically symmetric (ie, hypertrophy of the septum and left ventricular free wall is equal), plateaus early in conditioning, and rapidly diminishes back to baseline within weeks of inactivity.[22]

The electrophysiologic changes brought on by exercise are also enhanced by emotion and competitive stress. The release of circulating catecholamines stimulates heart rate, myocardial contractility, and blood pressure, all resulting in increased myocardial oxygen consumption. Such changes may perturb underlying myocardial ischemia and trigger a variety of cardiac arrhythmias if a pre-existing abnormality is present. In one study,[7] 90% of athletes collapsed during or immediately after exercise, highlighting this vulnerable period.

CAUSES OF SUDDEN CARDIAC DEATH

More than 20 pathologic entities have been identified as causes of SCD in young athletes (Table 1). However, a few lesions are responsible for most deaths. McCaffrey et al[25] reviewed 7 studies and found hypertrophic cardiomyopathy (HCM) responsible for 24% of the deaths, whereas coronary artery abnormalities were present in 18% of patients. Coronary artery disease and myocarditis accounted for 14% and 12% of cases, respectively. The review by Maron et al[7] found HCM or "possible HCM" to account for 46% of all deaths, with coronary artery anomalies leading to an additional 19% of SCD cases (Table 2). Other studies have found HCM responsible for nearly half of all deaths.[26,27]

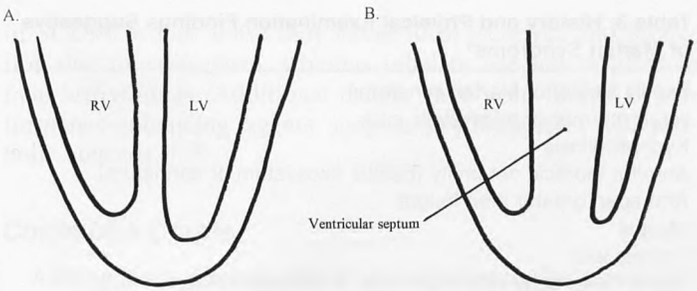

Figure 1. Cross-sectional views. A, Normal ventricular septal anatomy. B, Asymmetric septal hypertrophy found in hypertrophic cardiomyopathy. RV indicates right ventricle; LV, left ventricle.

Although a thorough discussion of all causes of SCD is beyond the scope of this review, I will present an overview of the most common causes and those less common causes that lend themselves to clinical detection through preparticipation screening.

Hypertrophic Cardiomyopathy

Although rare in the general population (0.1% to 0.2% prevalence),[28] HCM is the most common cause of SCD in young athletes.[7,25–27] It was HCM that claimed the life of Hank Gathers in what has certainly been the most notorious case of SCD.[1] Typically, HCM is inherited as an autosomal dominant condition; more than 100 individual genetic defects can result in the characteristic pathologic findings.[16] Sixty percent of individuals with HCM have an affected first-degree relative.[26]

On autopsy, patients with HCM are found to have a larger-than-normal heart with a distinctively enlarged left ventricle. The total mass of the left ventricle is increased without compensatory dilatation of the chamber; thus, ventricular filling is decreased during diastole. The thickness of the ventricular septal wall may be markedly increased, from 15 to 50 mm, with less than 13 mm being considered normal, even in a trained athlete.[22] Hypertrophy of the ventricular septum is disproportionate to that of the left ventricular free wall, an asymmetry not seen in physiologic hypertrophy (Figure 1). In addition to the increased size, the asymmetric thickening of the septum may act as an obstruction to the flow of blood into the aorta during diastole. Microscopic changes include abnormalities of the small arteries and "myocardial disarray," a bizarre arrangement of muscle cells with diffuse interstitial fibrosis.[29]

The hallmark physical examination finding in HCM is a systolic murmur that decreases in intensity with the athlete in the supine position. This contrasts with functional outflow murmurs common in athletes, which increase in intensity with lying down. Approximately 90% of patients with HCM have abnormal ECG results.[30] In many, but not all, cases, HCM can be diagnosed by echocardiographic findings of marked, asymmetric left ventricle wall thickening, diminished left ventricle chamber size, and abnormal diastolic filling.[22]

Despite the aforementioned information, individuals typically present with SCD as their first and only symptom of HCM. A few may have a family history of a sudden or unexplained death. In one study,[7] just 10 (21%) of 48 athletes who died of HCM had signs or symptoms of cardiac disease (chest pain, exertional dyspnea, syncope, dizziness) before death. The mechanism of death is not fully understood but is most likely secondary to a malignant arrhythmia arising within the abnormal myocardial fibers. Syncope may result from either dynamic obstruction of the left ventricular outflow tract or a nonsustained arrhythmia.

Idiopathic Left Ventricular Hypertrophy

Idiopathic left ventricular hypertrophy (ILVH) is an ill-defined and poorly understood condition responsible for up to 10% of SCD cases. Although ILVH is difficult to diagnose during life, on autopsy the heart shows symmetric enlargement, unlike that seen in HCM but with hypertrophy far in excess of that seen in trained athletes.[27] In addition, the coronary arteries and the myocardial tissue are normal.[30] The family history reveals no HCM or prior events of SCD. At this time, the cause of ILVH is undetermined. Possible causes include adaptation to undiagnosed systemic hypertension, extreme physiologic hypertrophy,[16] and a nonfamilial variant of HCM.[30]

Coronary Artery Anomalies

A variety of congenital coronary artery anomalies combine to represent the second leading cause of SCD in young athletes. The most common abnormality consists of the left main coronary artery arising from the right sinus of Valsalva (Figure 2). The artery comes off the sinus at an acute angle, which is thought to contribute to diminished blood flow as the aorta dilates during exercise.[31] The aberrant artery then courses between the aorta and pulmonary trunk, making it prone to compression as the great vessels enlarge with the increased cardiac output of exercise.[32] Other anomalies have been described, including reports of single coronary arteries.[22]

Only about one third of affected individuals are thought to be symptomatic (experiencing angina, syncope, or exertional dyspnea) before SCD.[33] However, 10 of 12 athletes with coronary artery anomalies in the United States and Italy had symptoms before death.[34] All 9 athletes who underwent ECG testing had normal results, including 6 individuals with normal exercise stress test results. The mechanism of SCD in all cases is thought to be an arrhythmia triggered by myocardial ischemia or infarction. Some cases may be suspected on echocardiography, but a definitive diagnosis is made by coronary angiography, computed tomography, or magnetic resonance imaging.

Myocarditis

Acute myocarditis is an inflammatory condition of infectious origin. Coxsackie B virus causes more than 50% of all cases, but a variety of pathogens have been implicated.[35] Affected individuals may present with dyspnea, orthopnea, cough, and exercise intolerance. When present, such symptoms are often overshadowed or preceded by symptoms of viral illness such as vomiting, fever, nausea, diarrhea, and myalgias. However, many individuals are asymptomatic, and SCD may be the only presenting sign.[15,22,30] The infected myocardium becomes inflamed, creating an unstable site where a potentially terminal arrhythmia may arise. In other cases, involvement of the conduction system may lead to a fatal heart block.

Myocarditis is suspected based on the clinical symptoms in conjunction with physical examination findings of congestive heart failure (eg, audible S_3 gallop, distended neck veins, peripheral edema, hepatomegaly). Although chest radiographs,

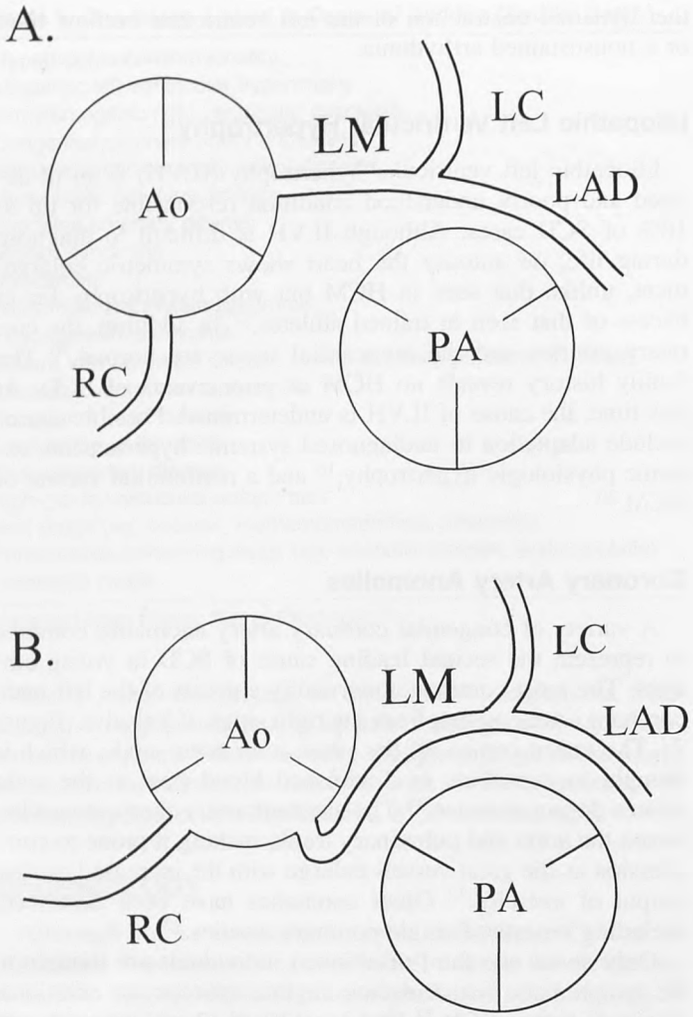

Figure 2. A, Normal coronary artery anatomy. B, Most common congenital cardiac artery anomaly with left main (LM) coronary artery arising from the right sinus of Valsalva and coursing between the aorta (Ao) and pulmonary artery (PA). This anatomic variation also results in the right coronary artery's (RC) originating in a different region than normal, but its function is not affected. LC indicates left coronary artery; LAD, left anterior descending artery.

ECG, and echocardiography may suggest the disease, diagnosis can only be confirmed by myocardial biopsy. Maron et al[7] described 4 cases of SCD secondary to acute myocarditis and 4 other deceased athletes with pathologic findings of isolated idiopathic myocardial scarring. These cases may represent an arrhythmogenic potential for healed myocarditis.[36]

Marfan Syndrome

Marfan syndrome is an autosomal, dominantly inherited connective tissue disorder occurring in about 1 in 10 000 people. Affected individuals are at increased risk for SCD as the result of progressive dilatation of the aortic root, culminating in complete dissection or rupture of the aorta with subsequent mediastinal hemorrhage, pericardial tamponade, coronary artery dissection, or acute aortic insufficiency with rapid congestive heart failure.[30] Aortic root dilatation results from cystic medial necrosis, a condition that weakens the walls of the aorta because of a decreased number of elastic fibers.

The diagnosis of Marfan syndrome is based on clinical criteria (Table 3), although genetic testing may be appropriate in families with several affected members. Clinical features include tall stature, long and thin limbs (dolichostenomelia), an arm span substantially greater than height, diminished upper body–to–lower body ratio, and long, thin facies. Additional findings include anterior thorax abnormalities (pectus excavatum or carinatum), hyperextensible joints, and the ability to significantly overlap the thumb and fifth digit while encircling the thin wrist.[37] The defective connective tissue also may place those affected at risk for a dislocation of the eye lens.

Table 3. History and Physical Examination Findings Suggestive of Marfan Syndrome*

Family history of Marfan syndrome
Heart murmur or midsystolic click
Kyphoscoliosis
Anterior thoracic deformity (pectus excavatum or carinatum)
Arm span greater than height
Myopia
Ectopic lens
Upper-lower body ratio more than 1 SD below mean

*Adapted with permission from McKeag.[37]

Electrophysiologic Abnormalities

Abnormalities of the conduction system may lead to fatal cardiac arrhythmias. Their incidence is likely underreported since autopsy findings may be inconclusive.[7] The 2 most commonly encountered abnormalities in the general population are Wolff-Parkinson-White syndrome (WPW) and long QT syndrome (LQTS). WPW represents an accessory conduction pathway within the myocardium, which, when triggered, results in symptomatic, but typically benign, atrial or ventricular arrhythmias. Rarely, these arrhythmias may degenerate into ventricular fibrillation and prove fatal. Often, WPW can be recognized by specific ECG findings, but a significant number of accessory pathways are "concealed," that is, the ECG results are normal despite the presence of the pathway. The condition may be treated with medication, but the treatment of choice in competitive athletes is radiofrequency ablation of the accessory pathway.

An inherited cardiac disorder, LQTS occurs in about 1 in 10 000 individuals, with 60% of those having a positive history of LQTS or SCD in a family member.[38] Approximately 60% of patients present with symptoms related to physical activity or strong emotional response, primarily syncope, seizures, or heart palpitations. One third of previously "healthy" young adults present with SCD.[38] The mechanism of demise is a distinctive fatal arrhythmia triggered by catecholamine release. The ECG results are abnormal in nearly all affected individuals. Treatment generally involves β-blocker medication (sometimes in conjunction with permanent cardiac pacing) and avoidance of intense physical exertion.

Other Causes

A number of more rare causes have also been implicated in SCD. Aortic stenosis and mitral valve prolapse are often described as risk factors for SCD, but the mechanism of death in each is uncertain. Mitral valve prolapse is a relatively common condition, and its association with SCD is controversial. Athletes who had congenital heart malformations, such as tetralogy of Fallot, repaired in infancy are at risk for fatal arrhythmias. A number of illicit drugs have also been implicated

in SCD. Cocaine abuse may cause local ischemia and infarction due to vasospasm, whereas inhalant use has resulted in fatal arrhythmias. Additional deaths have been linked to performance-enhancing agents such as erythropoietin and anabolic steroids.[15,30]

Commotio Cordis

Although not directly related to a cardiovascular abnormality, commotio cordis deserves mention. Literally meaning "concussion of the heart," commotio cordis death results from a fatal dysrhythmia induced by the transfer of kinetic energy by a nonpenetrating projectile (baseball, softball, or hockey puck) striking the chest. It is thought that the blow must occur at a particularly vulnerable phase of the cardiac cycle (ventricular repolarization) to be fatal.[39,40] Fortunately, episodes of commotio cordis are rare, because the rate of resuscitation is remarkably low.[30]

ON-FIELD RESPONSE

Any athlete who collapses on or off the field should be assessed for the presence of respirations and pulse. If these are absent, cardiopulmonary resuscitation should be initiated and the emergency medical system activated. The final common pathway in nearly all cases of SCD is a fatal ventricular arrhythmia.[41] Chances for successful resuscitation are remote, even if cardiopulmonary resuscitation is started immediately and defibrillation equipment is readily available.[16] With survival unlikely once an arrhythmia is triggered, the only potential opportunity to prevent such tragedies is to identify susceptible individuals before a fatal event.

PAST EXPERIENCES WITH CARDIAC SCREENING

As I have presented, numerous potential causes of SCD exist; however, only a few abnormalities claim most lives. Many of the conditions present with ominous symptoms such as exercise-related syncope, exertional dyspnea, or chest pain.[7,15,16,42] In addition, some individuals with HCM may have a family history of SCD. However, two thirds of athletes with potentially fatal cardiac disease or cardiac anomalies present with SCD.[7,43] Disturbingly, Maron et al[7] found that of the 115 persons experiencing SCD who had undergone a preparticipation medical evaluation, only 4 (3%) were suspected of having cardiac disease. The correct diagnosis was made in only one athlete before death.

Large population studies have revealed that the standard history and physical examination result in an extremely small number of significant cardiac findings.[44,45] Therefore, efforts have been made to discover potentially serious cardiovascular abnormalities before athletic participation begins. Diagnostic evaluation beyond a thorough history and physical examination may identify a small percentage of individuals at risk for SCD. A number of studies have been conducted in the United States using echocardiography and ECGs as screening instruments. In one of the larger studies to date,[46] 2997 athletes were screened by echocardiography; no disqualifying cardiac abnormalities were discovered. In another study,[47] 501 college athletes were screened with a family and personal history and an ECG. Ninety athletes (18%) then underwent an echocardiogram to rule out potential abnormalities (75 due to an abnormal ECG result). No athletes were restricted from participation. The screening ECG has been further studied and is widely believed to be ineffective because of a significant false-positive rate.[47–50]

Lewis et al[49] performed echocardiography on 265 black athletes at Howard University, revealing 14 cases (5.3%) of mitral valve prolapse but no cases of HCM or other disqualifying pathologic condition. Murry et al,[48] using screening echocardiography, found no lesions to preclude activity in the 125 athletes screened. Interestingly, one screened athlete had an episode of syncope the next day at football practice. Despite extensive testing, no diagnosis had been established before the publication of their study.

Results from Italy's long-running screening program (details discussed herein) show that such endeavors can uncover significant pathologic findings.[32,51] From 1979 to 1996, 33 735 young athletes (all younger than 35 years) were screened at a regional sports medicine center in the Veneto region of Italy.[51] A total of 3016 individuals (8.9%) were referred for echocardiography because of family history, abnormal physical examination findings, or ECG abnormalities. In 22 individuals (20 male and 2 female athletes), HCM was diagnosed, disqualifying them from competition. In all, 621 athletes (1.8%) were disqualified for cardiovascular conditions including rhythm and conduction abnormalities, systemic hypertension, valvular disease, and HCM.

POTENTIAL SEQUELAE OF SCREENING PROGRAMS

Although diagnostic tools such as ECGs and echocardiography may identify a small number of individuals at risk for SCD, the emotional and financial costs are high given the limits on the screening instruments currently available. With the low prevalence of cardiac anomalies, it has been estimated that 200 000 individual athletes would need to be screened to identify the single individual who would die suddenly.[52] This means that even if a near-perfect screening test existed, with a specificity and sensitivity of 99%, we would be left with only 1 truly positive test result for the 1999 false-positive results generated. We can easily extrapolate the number of false-positive results that would be created nationwide, considering the millions of high school and college athletes who participate in sports each year.

Many may argue that any effort is worthwhile if it saves lives, regardless of cost. The Italian government has screened all athletes ages 12 to 35 years on an annual basis since 1971.[32,51] The screening battery includes a history and physical examination, exercise and pulmonary function testing, and an ECG.[16] The program is funded by the country's National Health Service. Interestingly, in the event of an incorrect diagnosis that leads directly to death or impaired health, the physician who cleared the athlete for competition may be held liable in civil and criminal court.[16] Although Italian researchers report a much lower rate of SCD due to HCM,[51] the overall SCD incidence of 1.6 in 100 000 young athletes in Italy is similar to the rate seen in the United States.

Screening all athletes with echocardiography would certainly identify many potential causes of SCD but at an astounding financial cost (average echocardiogram cost, $857). Even if economically possible, instituting screening nationwide would be difficult because of limited availability of testing in many rural areas. Screening by ECG is less expensive, but as dis-

cussed, the high false-positive rate would necessitate further testing in up to 10% of screened athletes.

More importantly, the emotional costs of such testing would be far greater. The large number of false-positive results generated would create worry and stress in parents and athletes as they awaited further testing. Such worry may affect later participation in competitive sports and recreational activities, with the specter of sudden death haunting the athlete. Similar effects have been described in children diagnosed as having innocent heart murmurs[53] and the well-known "cardiac cripple" syndrome seen after a myocardial infarction.

Another pitfall of screening is that we are unable to stratify risk based on the severity of HCM or any other lesion discovered. Therefore, the universal recommendation is the avoidance of "intense" physical activity. Of course, the presence of HCM or other potentially fatal heart pathologic condition does not uniformly result in SCD.[54,55] The diagnosis of a potentially fatal heart condition is no guarantee that a previously healthy, active athlete will suddenly and willingly adopt a sedentary lifestyle. Disqualification from sanctioned athletics based on the presence of a potentially fatal heart condition has been legally challenged.[2] Recently, however, a US Court of Appeals cited the 26th Bethesda Conference[56] as the guidelines physicians should rely on when formulating decisions on participation for athletes with cardiovascular disease, perhaps setting a precedent for future court cases.[57] Unfortunately, even athletes who understand their risk of SCD will sometimes continue to participate in intense recreational activity, occasionally with fatal results.

CURRENT RECOMMENDATIONS FOR CARDIAC SCREENING

Recognizing the significant limitations and great costs of mass population screening of athletes for cardiovascular abnormalities, the American Heart Association (AHA) assembled a panel of experts in 1996 and developed "recommendations and guidelines for the most prudent, practical, and effective screening procedures and strategies."[9] Although not specifically discouraging the use of screening ECGs or echocardiography, the panel pointed out that, given the low prevalence of cardiac disease, the implementation of such programs on a wide scale would likely result in a greater number of false-positive than true-positive results.

The panel did recommend that all high school and college athletes undergo a cardiovascular evaluation before athletic participation, performed by a health care worker trained in the evaluation of cardiovascular disease, preferably a licensed physician. Such screening should be repeated every 2 years. The evaluation should consist of the following elements (Table 4): (1) family history of premature or sudden death or heart disease in any surviving relatives; (2) personal history of heart murmur, systemic hypertension, excessive fatigue, exertional chest pain, exertional syncope, or excessive shortness of breath; (3) physical assessment for heart murmur, femoral pulses, stigmata of Marfan syndrome (Table 3), and brachial artery blood pressure; and (4) parental verification of history form by signature for high school athletes.

If cardiac disease is identified through screening and referral results in a definitive cardiac diagnosis, recommendations regarding further competitive and recreational activity should be formulated in accordance with the consensus panel guidelines of the 26th Bethesda Conference.[56]

Table 4. American Heart Association Recommended Screening Items[9]

Family history
Premature death (sudden or otherwise) or significant disability from cardiovascular disease in a close relative younger than 50 years
Specific knowledge of close relatives with certain cardiovascular conditions (eg, hypertrophic cardiomyopathy, dilated cardiomyopathy, long QT syndrome, Marfan syndrome, or clinically important arrhythmias)
Personal history
Excessive, unexpected, and unexplained fatigue associated with exercise
Excessive, unexpected, and unexplained shortness of breath associated with exercise
Exertional chest pain or discomfort
Exertional syncope or near syncope
Heart murmur
Systemic hypertension
Physical examination
Auscultation of heart in both supine and standing positions
Palpation of femoral pulses
Recognition of physical stigmata of Marfan syndrome
Brachial artery blood pressure measurement
Parental verification of personal and family history for high school athletes

Table 5. Primary and Secondary Objectives of the Preparticipation Athletic Evaluation

Primary Objectives	Secondary Objectives
1. Detect medical or musculoskeletal conditions that may predispose an athlete to illness or injury during the competition.	1. Determine the general health of the athlete.
2. Detect potentially life-threatening or disabling medical or musculoskeletal conditions that may limit an athlete's safe participation.	2. Counsel the athlete.
3. Address legal or insurance requirements.	3. Assess fitness level for specific sports.

CURRENT STATUS OF CARDIAC SCREENING

Unfortunately, many physicians, athletic trainers, and administrators continue to face the annual "sports physical" event with a certain loathing. Although there is no legal precedent for conducting such examinations, there is an implicit moral and ethical responsibility on the part of the institution (high school or college) to provide such a service.[9] The objectives of the PAE have been well described (Table 5). Despite this, all involved often view the examination as an inconvenience. The manner in which most PAEs are conducted nationwide reflects this sentiment.

The preparticipation cardiovascular screening process at the high school and college level in the United States is currently not meeting the standards set by the AHA.[58–60] This is despite the increased focus on SCD during the past decade by many in the sports medicine community and numerous publications,[10,13–15,42] including a well-publicized monograph,[12] which have highlighted the need for improvement. When compared with the AHA guidelines, only 26% of National Collegiate Athletic Association (NCAA) schools were found to be

using "adequate" history and physical examination forms (containing at least 9 of 12 recommended items).[58] NCAA Division II and Division III schools (28% and 30%, respectively) were found to have inadequate forms (containing 4 or fewer of the 12 recommended items) more often than Division I schools (14%). Interestingly, 75% of NCAA team physicians were orthopaedic surgeons, physicians with little or no postgraduate training in the evaluation of cardiac disease.

The status of examinations in high schools nationwide is predictably even more dismal. In the absence of national standardization of PAE forms, only 17 of 43 state forms recently evaluated were found to be "adequate" with regard to AHA recommendations.[60] Gomez et al[59] surveyed a sample of certified athletic trainers at high schools nationwide and found that only 17.2% of returned PAE forms contained all elements of the cardiovascular history recommended by Lombardo et al.[11] In Oregon, the state athletic association has only recently recommended a form for use by member schools.[61] In a survey conducted by our institution (unpublished data, 2000), we found that a large number of schools continued to use suboptimal forms despite the recommendation.

Reviewing the forms used during the PAE process gives insight into what history is obtained and the scope of the physical examination performed. Such guidelines are important, since the practitioners performing PAEs nationwide vary markedly. Five states have no specific recommendations, requirements, or restrictions with regard to who may perform the PAE.[60] Of the remaining 46 jurisdictions (including the District of Columbia), 21 allow physician assistants and nurse practitioners to perform examinations, whereas chiropractors are approved to do so in 10 states and naturopathic clinicians in a single jurisdiction. No state offers specific qualifications for examiners or guidelines for the examinations. Although the cardiovascular system training and experience of physician assistants and nurse practitioners may vary greatly, chiropractors and naturopaths have limited training in the detection of cardiovascular disease.[60]

When examinations are conducted in accordance with AHA recommendations by appropriately trained practitioners, potentially serious cardiac pathologic conditions may be detected. As previously discussed, few athletes have symptoms or physical examination findings before SCD, and the current method of screening has a very low yield. However, because almost all institutions currently "go through the motions" of the PAE on a yearly basis, most need to make only minimal changes (use of appropriate forms and properly trained examiners) to improve the ability to detect many clinically significant cardiac conditions in the young athlete.

CONCLUSIONS

Fortunately, most athletic trainers will never experience the sudden death of an athlete, because it is an extremely rare event. Despite the variety of causes of SCD, only a few conditions are responsible for most deaths. Most athletes who eventually succumb to SCD have no history of cardiac problems (family or personal) and no symptoms before death. Hence, the detection of at-risk individuals poses a significant challenge to the sports medicine team. Population screening by diagnostic testing is not currently economically or practically feasible, but a significant proportion of at-risk athletes can be identified through a thorough history and physical examination.

Although no perfect screening instrument is currently available, a moral and ethical obligation exists for physicians and athletic trainers to ensure that athletes are assessed in the most prudent and efficient manner available. Therefore, the recommendations of the AHA should be considered the minimum standard for the cardiovascular screening of high school and college athletes nationwide. Current data show that we are doing a woeful job of implementing these standards. Athletic trainers are often responsible for the organization and administration of the PAE and must work with their team physicians to implement such changes. In addition, all athletic trainers should offer their expertise to administrators at high schools without sports medicine services. As physicians and athletic trainers, we recognize that athletic participation carries inherent risks. However, when we are capable of minimizing those risks, we must have the professional integrity to act within our capacity to do so.

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Sudden Cardiac Arrest in Athletic Medicine

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Objective: To emphasize the importance of decreasing the response time by a trained target responder to increase the survival rate among athletes experiencing sudden cardiac arrest at an athletic event.

Background: Death due to sudden cardiac arrest that is witnessed is preventable in many cases. However, most people who experience this condition die because of a prolonged response time from onset of the fatal arrhythmia to defibrillation by trained treatment providers. If athletic trainers or other members of the athletic care medical team are trained as target responders and equipped with automated electronic defibrillators, they can immediately treat an athlete who experiences a sudden, life-threatening tachyarrhythmia. This prompt response to the life-threatening emergency should result in a higher survival rate.

Description: We review the causes of sudden cardiac arrest during athletic events, note some unusual clinical presentations, discuss improved methods of response and new equipment for treatment, and define the athletic trainer's role as a target responder trained to treat people experiencing sudden cardiac arrest at an athletic event.

Clinical Advantages: An athletic care team willing to become part of an emergency response team can help improve the survival rate of athletes experiencing sudden cardiac arrest at an athletic event.

Key Words: target responder, medical emergency plan, defibrillation

In the United States each year, sudden cardiac arrest kills 350 000 people, which is approximately 1000 people per day.[1] Before 1990, cities with established advanced and basic life support programs reported that their efforts to resuscitate those people experiencing sudden cardiac arrest resulted in a 4% to 11% survival rate.[2] The American Heart Association has challenged cities to improve this survival rate to 30%.[1] These statistics are derived from death certificates of people of all age groups who experienced sudden cardiac arrest. The exact incidence of sudden cardiac arrest in athletes is unknown because no universal, standard surveillance method is used.

We do know that death during an athletic event can result from direct and indirect causes.[2] The direct causes of death primarily are traumatic (eg, a closed head injury). The major indirect cause of death during athletics is from an arrhythmia resulting in sudden cardiac arrest. From 1931 to 1986, direct causes of death in high school athletes outnumbered indirect causes by a ratio of 2:1.[3] Since 1982, that ratio has reversed to 1:2 due to an increase in the indirect causes of death.[2] Unfortunately, much of the information on the indirect causes of death has been obtained from newspaper reports.

The American Heart Association[4] reviewed 158 cases of death due to sudden cardiac arrest in athletes that primarily were reported in American newspapers from 1985 to 1995. Of these deaths, 138 (68%) occurred in players who had a mean age of 17 years and who participated in either football or basketball. The occurrence of sudden cardiac arrest in 70 whites (52%), 69 African Americans (44%), 8 Asians (2%), 1 Hispanic (0.6%), and 1 Native American (0.6%) demonstrated that it is nondiscriminatory. Although football and basketball were the most common sports, 18 different competitive sports were represented.[5] The fact that most of these reports originated in the news media has helped focus public awareness on the problem; however, these reports do not represent the actual incidence of sudden cardiac arrest in athletes because no universally used, standardized surveillance method, which would allow statistical evaluation of these problems at a national level, is required.

The clinical presentation of sudden cardiac arrest in athletes is abrupt, and, unfortunately, the player usually has had no prodrome before the cataclysmic event.[6] In the report of Maron et al,[4] 90% of the 158 athletes experiencing sudden cardiac arrest collapsed during or immediately after a training session or scheduled athletic contest. Only 12 of the 158 athletes reported symptoms before the fatal event. At their preparticipation physical examination, athletes may not report the symptoms that alert medical personnel to their risk of sudden

cardiac arrest. Thus, the arrhythmia is usually instantaneous in an asymptomatic athlete and occurs during or at the end of intense physical activity, with death quickly following.[7] The first people to respond must be well prepared to reach, evaluate, and treat the athlete before he or she dies.

Our purpose is to review the causes of sudden cardiac arrest during athletic events, to note some unusual clinical presentations, to discuss improved methods of response and new equipment for treatment, and to define the athletic trainer's role as a target responder trained to treat sudden cardiac arrest.

CAUSES OF SUDDEN CARDIAC ARREST

The most common cause of sudden cardiac arrest in athletes younger than 35 years is a consequence of an underlying cardiac abnormality. According to a study by The American Heart Association,[4] the cardiac problems that can cause sudden cardiac arrest include hypertrophic cardiomyopathy (36%), hypertrophic cardiomyopathy–like structural changes (10%), anomalous origin of the left main coronary artery (10%), other coronary anomalies (9%), myocarditis (6%), ruptured aortic aneurysm (5%), tunneled left anterior descending artery (5%), aortic valve stenosis (4%), dilated cardiomyopathy (3%), and arrhythmogenic right ventricular dysplasia (2%). Other causes of sudden cardiac arrest include cardiac concussion (commotio cordis),[8,9] drug-related (arrhythmia-allowing or arrhythmia-inducing)[6] difficulties,[1,2] and underlying coronary artery disease causing myocardial infarction with an associated fatal arrhythmia.

Prevention of Cardiac Causes of Sudden Cardiac Arrest

Preventing sudden cardiac arrest is difficult if the preexisting cardiac condition is not recognized. To try to achieve this end, physicians should include a thorough history and cardiac examination as part of the preparticipation physical examination. A 12-lead electrocardiogram (ECG), a stress rhythm ECG, or both should be performed if any risk factors are identified at the initial assessment.[10] Those risk factors include a history of chest pain, shortness of breath, or arrhythmia; a history of cardiac problems requiring medication; and a family history of death due to sudden cardiac arrest at a young age. Findings on clinical examination of bruits, murmurs, high blood pressure, and arrhythmias deserve more thorough evaluation.

Athletes who have a recognizable or a known preexisting physical condition, such as Marfan syndrome, need to be well screened because Marfan syndrome is associated with heart valve anomalies and aortic aneurysm (which may rupture).[4] They also are at risk for sudden cardiac arrest.

Evaluation beyond this baseline may require special testing following a cardiology evaluation and may use a 12-lead ECG stress test. A limited echocardiogram is the best screening test, but a full echocardiogram is required if symptoms and risk factors are identified. It is useful in diagnosing hypertrophic cardiomyopathy, myocarditis, left ventricle dysfunction, valve abnormalities, and dilated aortic root. In addition, electron-beam computed tomography scanning can help the cardiologist diagnose coronary artery anomalies, such as coronary artery aneurysm or left coronary artery origin from the right side, but is not commonly used as a screening test.[11,12] This test and coronary artery angiography can demonstrate coronary artery abnormalities; however, they are not cost effective and thus should not be considered "screening" tests.

Because using these special tests to routinely screen large numbers of athletes is not cost effective, these more advanced screening tests usually are limited to athletes with cardiac symptoms or signs determined by history, examination, or ECG rhythm abnormalities. Therefore, the athletic care medical team needs to be well prepared to treat sudden cardiac arrest in athletes when it occurs.

Unusual Clinical Presentations of Sudden Cardiac Arrest

Commotio Cordis. The first unusual clinical presentation is commotio cordis or cardiac concussion.[8,9,13] Blunt trauma to the chest can interrupt the electric function of the heart, resulting in ventricular fibrillation. The subsequent electric dysfunction results from the impact being delivered during the diastolic phase of the cardiac cycle (at the peak of the T wave electrically), which affects the repolarization of the heart and creates the fatal arrhythmia.[14] This problem can occur in noncontact (eg, baseball) or contact (eg, hockey) sports.[9] Since 1995, 60 deaths in children and young adults have been attributed to this cause (B. Maron, unpublished data, July 1999). Awareness of this condition and the development and use of protective padding and softer youth baseballs could aid in preventing the problem.[8,9,13,15]

Sentinel Seizure. The second unusual clinical syndrome is cardiac arrest occurring in combination with a sentinel seizure. The seizure is usually the first symptom that is obvious to a bystander and probably results from oxygen deprivation in the brain because of the fatal arrhythmia. As the athlete collapses due to loss of consciousness, the seizure activity decreases. Prompt cardiac assessment at this time is critical because further delay can result in death from the unrecognized fatal arrhythmia. The incidence of a sentinel seizure in association with sudden cardiac arrest is uncertain. However, through reviewing 10 news media reports of sudden death and contacting athletic department personnel involved in these cases during 1997 and 1998, we determined that seizure activity was cited as the initial clinical presentation in 3 of the 10 patients. To our knowledge, this complex issue of seizure associated with sudden cardiac arrest has not been reported in the literature, but this association should be considered in the future.

Drug-Related Difficulties: Illegal or Legal. The third clinical condition is a fatal arrhythmia related to intense physical activity combined with illegal or legal drug use.[6,16] Cocaine is reported to be associated with an increased risk of a myocardial infarction by 24 times in the first hour after taking it.[16] Certainly, a fatal arrhythmia can result from the myocardial infarction. Inadequate dosage of legal medications, such as β blockers, can result in arrhythmia and sudden cardiac arrest. The beneficial effect of the medication on the heart's ability to withstand the stress of intense activity can be reduced due to an inadequate level of the drug in the blood.[6] This situation was thought to contribute to the cause of Hank Gathers' death while playing basketball.[6] With respect to athletes taking legal medications, a medication history, cardiology evaluation, maintenance evaluation, and possibly measurement of blood levels of medication may be required. The team cardiologist should counsel all athletes and their parents regarding the risk of participation in the face of both cardiac conditions and cardiac-affecting medications. A cardiac-monitoring program is usually established for these at-risk athletes.

It would seem that the way to prevent problems with illegal

drugs is to include drug screenings as part of the preparticipation evaluation to determine illegal drug use. However, these screenings are not cost effective for any school and, in fact, could financially penalize all schools, especially those with limited financial resources.

Unfortunately, the incidence and survival data for these unusual clinical syndromes are unknown because no single national sentinel data tracking system is used consistently. The universal acceptance of and compliance with such a system might provide data to improve our understanding of these complex issues and their prevention.

CHAIN OF SURVIVAL

The effective treatment of an athlete experiencing sudden cardiac arrest depends on a sequence of responses by well-prepared providers, so that the steps, when linked together, form a "chain of survival" for the successful management of sudden cardiac arrest.[1,2] The chain of survival includes the following: (1) prompt emergency medical system (EMS) activation; (2) early cardiopulmonary resuscitation (CPR) by a first or target responder (less than 2 minutes); (3) early defibrillation (2 to 4 minutes); (4) early advanced life support (less than 8 minutes); and (5) late advanced life support.[17] The first 4 links must have as short a time delay as possible to significantly increase survival rates.

Prompt EMS Activation

The first link in the chain of survival begins with summoning EMS directly or through a bystander as a trained target responder begins early intervention of basic life support (BLS). The chance of survival of a person experiencing sudden cardiac arrest is reported to decline by 5% to 10% each minute the condition is left untreated; therefore, the sooner BLS, including defibrillation, is activated, the better the athlete's chance of survival.[1,2] The EMS response time in most communities ranges from 12 to 15 minutes.[2] This delay has horizontal and vertical components.[18] The horizontal delay begins with activation of EMS by telephone (ie, dialing 911 where available) and ends with the arrival of EMS at the site. This time delay is similar for most communities with well-developed EMS programs (eg, 4.5 to 5.5 minutes for New York and 4.9 to 5.6 minutes for Las Vegas).[18,19] The vertical time delay begins with the time required to park the rescue vehicle and ends with initiation of the first defibrillation shock. This vertical component seems to affect the total time from EMS activation to defibrillation more than the horizontal component affects it, with the total response time in Las Vegas averaging 9.8 minutes in people who lived (29.3%) and 12.4 minutes in people who died.[18] The additional delay of EMS response caused by the vertical component, when not coupled with an aggressive, community-oriented, trained target-responder BLS system with defibrillation capabilities, may be why many large communities, including Chicago and New York, have reported only a 1% to 2% survival rate in people experiencing sudden cardiac arrest.[18–21]

White et al[22] reported on a community in which "target responders," including EMS personnel and police, were equipped with automated external defibrillators (AEDs). Additionally, the target responder response time was improved to 5.4 minutes. These 2 changes resulted in a 49% survival rate in patients with sudden cardiac arrest. In 58% of the people who lived, police first arrived as the target responder, and in 42% of the people who lived, EMS personnel first arrived as target responders.[22] This study demonstrates the success achieved by combining EMS activation with target-responder BLS, including early defibrillation. It also demonstrates that the use of this model in athlete care is promising.

Giving athletic trainers access to an AED as a university-supplied piece of equipment outside the normal budget of the athletic department, providing a university-supported training program that integrates them as target responders for athletic care, and facilitating response drills with local EMS representatives would enhance survival probabilities if such an event occurred during a university-sanctioned athletic practice or competition. Certainly, this coordination between the athletic care emergency plan of the university and that of the community should be motivated by the same endpoint—improving survival in athletes experiencing sudden cardiac arrest.

Early CPR and Early Defibrillation

The second and third links in the chain of survival are inseparable from the first. Early CPR, which is a capability of a first responder trained in BLS, can enable the heart to survive longer when it is in fibrillation.[1,17] Larsen et al[23] calculated a 5.5% per minute decrease in survival and attributed 2.2% of that decline to each minute of delay in CPR initiation. However, CPR alone cannot reinstitute the normal cardiac output or normal electric activity for a heart in ventricular fibrillation.[1,17] The quality of CPR is important; authors of one study[24] have suggested that when CPR is administered by bystanders, it is administered effectively less than half the time. However, at best, CPR delivers 60 to 80 mm Hg of pressure to the heart, only generating approximately 30% of the cardiac output.[1,17] As a consequence of these factors, the American Heart Association has included early defibrillation training as a necessary addition to CPR in BLS certification.[1]

Combining the first 2 links with the third link in the chain of survival for treating sudden cardiac arrest (ie, early defibrillation), thus, is deemed necessary. The sooner an athlete in sudden cardiac arrest due to a tachyarrhythmia receives defibrillation, the better his or her chance of survival. More than 95% of patients who receive defibrillation shock in the first minute of arrest survive.[2,17] Each minute of delay in initiating defibrillation after an arrest lasting 9.4 minutes leads to a 5% to 10% decrease in a person's survival—even if the person had been receiving CPR during that period.[2,17] One theory proposed is that ventricular defibrillation decays to asystole as the heart becomes ischemic.[25] Certainly, the early target-responder's quick response, effective CPR, early defibrillation, and a shortened EMS response time are responsible for the improvements in survival rates noted here,[2,17,20,22,26] with White et al[22] reporting the most significant improvement in survival rate (49%).

In athletic medicine, certainly the early target responder should be the athletic trainer who is well prepared to respond rapidly and is trained in both CPR and early defibrillation.

Defibrillators. Two types of defibrillation equipment can be used for this purpose: a manual defibrillator or an AED. A manual defibrillator must be used by a physician or paramedic because it requires an analysis or interpretation of the cardiac rhythm by the skilled responder. When used, the manual defibrillator's specificity is 96% for the appropriate shock to be delivered to treat the arrhythmia. This specificity, however, is

dependent on the responder's ability to recognize a shockable arrhythmia.[1,2] In addition, the time delay for this treatment application equals the average EMS response time in a given community plus the average 1 minute required for rhythm interpretation.[1,2]

Unlike manual units, the recently introduced AED, which can be used by a trained target responder,[1,2] can automatically analyze the cardiac arrhythmia through an internal diagnostic algorithm and can determine the need for defibrillation. The specificity is 100% for appropriate shock delivery, and the unit may take 1 minute less than a manual defibrillator to use because the responder does not have to evaluate or interpret a rhythm strip.[2] This 1-minute difference between manual defibrillation and AED use translates into a 10% improvement in survival rate. In addition, the lithium battery in an AED can be used for years and does not lose its charge between uses—another benefit over a manual defibrillator, which must have its battery repeatedly recharged.[2] Finally, the defibrillation of patients by a broader target responder group than EMS personnel alone has been shown to improve survival rates.[22] From an accounting basis alone, the cost of a basic LIFEPAK 500 (Medtronic Physio-Control, Redmond, WA) AED is approximately $3000, but the cost of a LIFEPAK 10 defibrillator/monitor/pacemaker is approximately $9000.

Prompt Response. The main fact learned from the data obtained from such community efforts is that an improvement in the survivorship of athletes experiencing sudden cardiac arrest is possible if the athletic care program includes prompt response by target responders who are trained in BLS to include defibrillation and who are equipped with AEDs. Target responders should include EMS personnel, police, sheriffs, security guards, medical personnel, athletic trainers, and administrators involved in organizing medical care for sporting and civic events. Certainly, medical "control" is necessary for a cohesive program, but the stimulus for development can come from any interested participant. The "Casino Project" in Las Vegas illustrates appropriate application of these principles. After casino security guards were trained as target responders and the vertical response time of EMS was improved, the survival rate of casino patrons experiencing sudden cardiac arrest improved significantly.[18]

Not all athletes in sudden cardiac arrest can be treated successfully with electric defibrillation shocks. Because asystole, or flatline ECG activity, indicates that the heart has no electric activity, no mechanical pumping effect is possible.[2,17] In addition, the opposite condition of pulseless electric activity (PEA) can occur.[25] In this condition, electric activity is present, but the heart muscle does not respond to it; therefore, no mechanical pumping occurs, and no pulse is palpable. In these cases, CPR instead of defibrillation is required to provide some pumping effect, and intravenous access for treatment with epinephrine and bicarbonate should be established. The cause of the PEA must be sought quickly for survival to be ensured. The most common correctable causes of PEA include hypovolemia, cardiac tamponade, tension pneumothorax, hypoxemia, and acidosis.[25]

The target responder can use BLS, including defibrillation capabilities, to promptly treat the athlete experiencing a ventricular fibrillation and can use CPR to sustain the athlete with PEA until EMS paramedics arrive to provide advanced life support. The incidence of the initial arrhythmia, as well as whether it was witnessed, has much to do with survival.[18] Most occurrences of sudden cardiac arrest in athletes are thought to be due to tachyarrhythmia; therefore, they most commonly can be treated with defibrillation.[1,2,4,6,8,9] This cause differs from the initial arrhythmia that is reported to cause the condition in an older at-risk group, which includes officials, referees, coaches, and spectators. The fatal arrhythmia in this older patient population most likely parallels the findings of Sedgwick et al,[27] who implicated ventricular fibrillation in 64% of patients, ventricular tachycardia in 4%, bradycardia in 28%, and PEA in 4% as the initial arrhythmia.

Early Advanced Life Support

The fourth link in the chain of survival is early advanced life support. Defibrillation, early intubation techniques, external pacemakers, cardiac medications, and early transport to cardiac care units or to emergency departments equipped and staffed for advanced life support complete this link of survival.[1,2] For this fourth component of any athletic care medical program's acute emergency plan to work, EMS personnel, including paramedics with advanced life support capabilities, must be notified when a cardiac arrest is first identified to shorten the horizontal component of time delay. Then, in addition to arriving on site promptly, they must try to shorten the vertical component of their response time delay to assume patient care. The smooth transition of the care of the athlete from the certified athletic trainer as the target responder to the EMS responder depends on the training of both responders. High-quality CPR, as well as defibrillation with an AED, is important also. The leads that the athletic trainer as the target responder uses for the AED must be compatible with EMS equipment, and replacement protocols must be developed. The EMS personnel then assume care of the athlete and begin early advanced life support treatment.

An athletic program's medical staff, including certified athletic trainers and physicians, needs to be involved in target-responder training since that staff becomes a satellite target-responder group in the EMS plans of the university and the community. Although the athletic care medical team may only be responsible for providing the first 3 links in the chain of survival, this group, as well as police, sheriffs, security guards, and any other group designated as target responders, must be integrated into the community's emergency plan and must be trained and recertified as necessary in BLS, AED use, and advanced life support transfers to the community EMS team to significantly improve survival of the affected athlete.

Late Advanced Life Support

The fifth link in the chain of survival is late advanced life support. This link is provided by the full spectrum of advanced life support care offered through emergency departments and cardiac care units, their staffs, and cardiologists and cardiovascular surgeons. The treatment can include in-dwelling pacemakers or defibrillators, medications, and other surgery. Certainly, this late advanced life support capability is outside the spectrum of care that the athletic care medical team can provide. However, if the athletic care medical team is not well prepared, the athlete experiencing a life-threatening arrhythmia will not survive to take advantage of late advanced life support treatments.

In order for any athletic care medical team to improve its ability to enhance survival of athletes experiencing sudden cardiac arrest, each of these links of survival must be forged, or

combined, into a dependable chain for survival—the purpose of any emergency medical plan. Any athletic program's emergency plan thus becomes a vital peripheral link in this chain of survival.

MEDICAL EMERGENCY PLAN

After recognizing the many problems that result in a poor chance of survival for athletes experiencing sudden cardiac arrest, the athletic care medical team should develop a comprehensive and well-integrated plan to address these emergencies. The first, second, third, and fourth components of the emergency plan should proceed with near-simultaneous activation of EMS with a practiced response time (less than 8 minutes to initiation of treatment). This includes planning prompt EMS access to the field of play, which might otherwise be delayed wherever a large concentration of people gathers (eg, a civic event, an athletic stadium, or a sports arena). This EMS response should be combined with a more rapid (less than 2-minute) response by the athletic care medical team's target responders equipped with an AED. In practice, the athletic care medical team trained as target responders should immediately start BLS, including airway management, CPR (within 1 minute), and early defibrillation (within 2 minutes). Advanced lifesaving measures follow when EMS paramedics arrive (within 5 minutes) to further stabilize and then transport the patient to a designated advanced life support facility. The transfer of the athlete's care from the athletic care medical team to EMS requires integration of the athletic care emergency medical plan with the community EMS emergency care plan.

CONCLUSIONS

In summary, a well-organized athletic care emergency plan should include all medical persons, regardless of discipline, capable of contributing to an improvement in the survival rate of an athlete having a sudden cardiac arrest. That plan also should provide training and recertification programs and assistance in supplying equipment for implementation of this type of comprehensive program. This funding should be a financial project of the university and not a budget item of the athletic department. Linking the school's athletic medical coverage to the community EMS programs will benefit everyone involved. It has been demonstrated that a community willing to altruistically become "its brother's keeper" through such a program has witnessed improved survival rates for a treatable cause of death in our society—sudden cardiac arrest.[22]

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(Revised January 2001)

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19. The body or main part of the manuscript varies

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according to the type of article (examples follow); however, the body should include a discussion section in which the importance of the material presented is discussed and related to other pertinent literature. When appropriate, a discussion subheading on the clinical relevance of the findings is recommended. Liberal use of headings and subheadings, charts, graphs, and figures is recommended.

a. The body of an **Original Research** article consists of a methods section, a presentation of the results, and a discussion of the results. The methods section should contain sufficient detail concerning the methods, procedures, and apparatus employed so that others can reproduce the results. The results should be summarized using descriptive and inferential statistics and a few well-planned and carefully constructed illustrations.
b. The body of a **Literature Review** article should be organized into subsections in which related thoughts of others are presented, summarized, and referenced. Each subsection should have a heading and brief summary, possibly one sentence. Sections must be arranged so that they progressively focus on the problem or question posed in the introduction.
c. The body of a **Case Report** should include the following components: personal data (age, sex, race, marital status, and occupation when relevant—not name), chief complaint, history of present complaint (including symptoms), results of physical examination (example: "Physical findings relevant to the rehabilitation program were . . . "), medical history (surgery, laboratory results, examination, etc), diagnosis, treatment and clinical course (rehabilitation until and after return to competition), criteria for return to competition, and deviation from expectations (what makes this case unique).
d. The body of a **Clinical Techniques** article should include both the *how* and *why* of the technique: a step-by-step explanation of how to perform the technique, supplemented by photographs or illustrations, and an explanation of why the technique should be used. The discussion concerning the *why* of the technique should review similar techniques, point out how the new technique differs, and explain the advantages and disadvantages of the technique in comparison with other techniques.

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

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

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

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

    Internet Sources:
    1. Knight KL, Ingersoll CD. Structure of a scholarly manuscript: 66 tips for what goes where. Available at http://www.journalofathletictraining.org/jat/66tips.html. Accessed January 1, 1999.
    2. National Athletic Trainers' Association. NATA blood borne pathogens guidelines for athletic trainers. Available at http://www.journalofathletictraining.org. Accessed January 1, 1999.
25. Table Style: 1) Title is bold; body and column headings are roman type; 2) units are set above rules in parentheses; 3) numbers are aligned in columns by decimal; 4) footnotes are indicated by symbols (order of symbols: *, †, ‡, §, ‖, ¶); 5) capitalize the first letter of each major word in titles; for each column or row entry, capitalize the first word only. See a current issue of the *Journal* for examples.
26. All black-and-white line art should be submitted in camera-ready form. Line art should be of good quality; should be clearly presented on white paper with black ink, sans serif typeface, and no box; and should be printed on a laser printer—no dot matrix. Figures that require reduction for publication must remain readable at their final size (either 1 column or 2 columns wide). Photographs should be glossy black and white prints. Do not use paper clips, write on photographs, or attach photographs to sheets of paper. On the reverse of each figure attach a write-on label with the figure number, name of the author, and an arrow indicating the top. (Note: Prepare the label before affixing it to the figure.) Authors should submit 1 original of each figure and 4 copies for review.
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29. The *Journal of Athletic Training* follows the redundant publication guidelines of the Council of Science Editors, Inc (*CBE Views.* 1996; 19:76–77; also available on the *JAT* web site at http://www.journalofathletictraining.org). Authors found in violation of redundant publication will have sanctions invoked by the Journal Committee of the National Athletic Trainers' Association, Inc.

PUBLICATION POLICIES

30. Original Research manuscripts will be categorized under the following table of contents subheadings: clinical studies, basic science, educational studies, epidemiologic studies, and observational/informational studies.
31. Only Case Reports and Clinical Techniques that define and establish the optimal standard of care or the practice of athletic training will be considered for publication in *JAT.* All other Case Reports and Clinical Techniques will be considered for publication in the *NATA News.*
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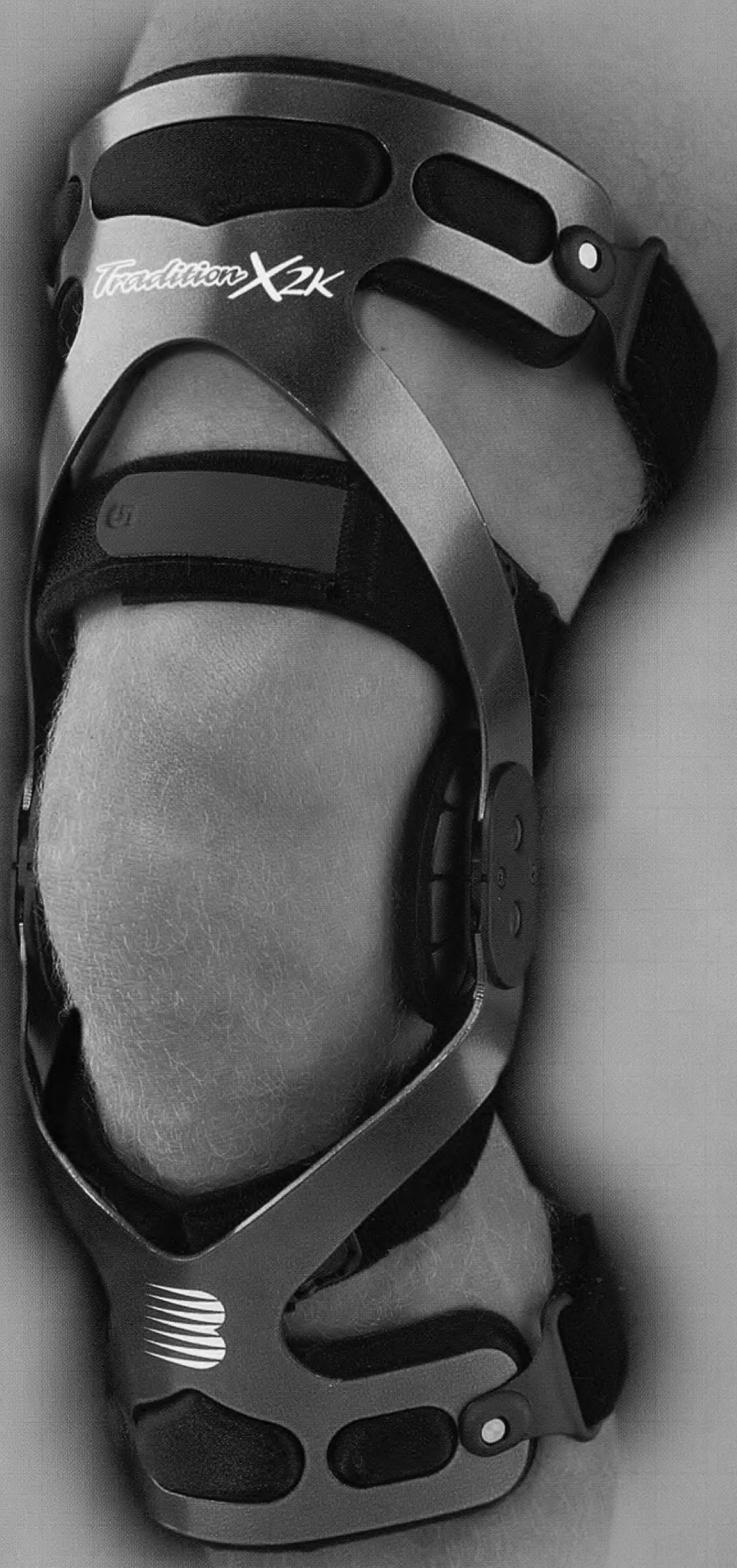

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