NATA ATHLETIC TRAINING

THE JOURNAL OF THE NATIONAL ATHLETIC TRAINERS ASSOCIATION, INC.

VOLUME 18
NUMBER 4
WINTER 1983

IN THIS ISSUE:

6 patellar stabilizers that really work.

And are clinically proven effective for ▪ Chondromalacia ▪ Tendinitis ▪ Normal patellar tracking ▪ Jumper's knee ▪ Patellar subluxation and dislocation ▪ Osgood-Schlatter's Disease ▪ Post operative therapy

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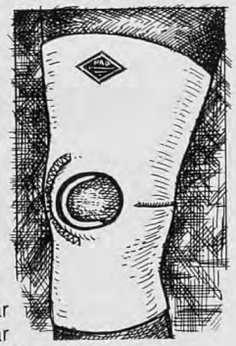
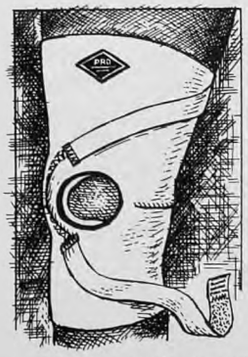


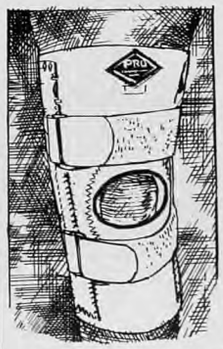
PRO 100B Deluxe Knee Sleeve

The classic Neoprene knee sleeve with a felt buttress at lateral edge of the patellar opening for patellar control and stabilization. The conservative approach to treating patellar subluxation. $24.95 *U.S. Pat. No. 4,084,584*



PRO 100B Modified Deluxe Knee Sleeve

New design features pull straps, with Velcro fasteners, attached to the ends of the felt buttress at a 45 degree angle, to provide firmer control of patellar tracking. $26.95



Dr. "M" Patellar Brace

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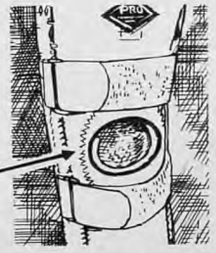
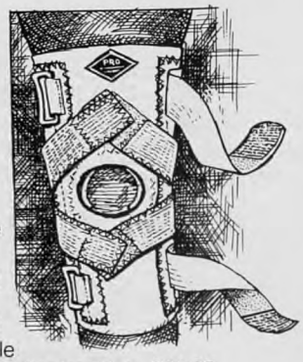


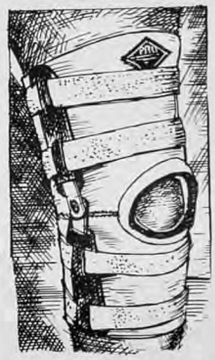
Dr. "M-I" Patellar Brace

The Dr. "M" with the felt horseshoe inverted to apply compression to the infrapatellar tendon elevating the patella. Most helpful in the treatment of chondromalacia, tendinitis and Osgood Schlatter's Disease. $32.95



Dr. "M-C" Patellar Brace (Cartilage Sleeve)

The Dr. "M" with the addition of elastic "X" straps applying specific pressure over the lateral and medial menisci while enforcing normal patellar tracking. $34.95



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ATHLETIC TRAINING

THE JOURNAL OF
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Volume 18, Number 4, Winter 1983

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Tips from the Field

Departments

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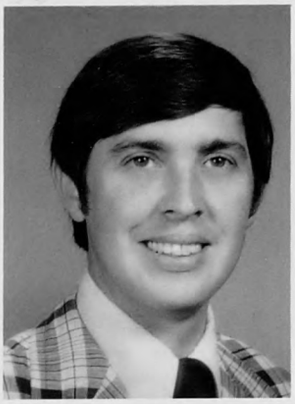
Editor's Comments



Steve Yates, ATC, M.Ed
Wake Forest University

I would like to thank the members who responded to the issues of concern in the Fall Journal. Your comments and suggestions are greatly appreciated. Some suggestions received were:

1. Student trainer section with contributions from student trainers only (This is being considered with a working title of "Student Trainer Corner.")
2. Brief synopsis or preview on manuscripts
3. Reader service card correlated to advertisers
4. Article information from back issues (Updated Index: Currently a Volume Index is published in each Winter issue, but a complete Index has not been published since Spring 1981, which was the Index 1956 through 1979.)

New Section . . . For Student Trainers Only

The following guidelines must be met for submission of papers or material to the new "Student Trainer Corner." These are essentially the same rules governing the "Student Writing Contest."

1. You must be an undergraduate student member of NATA.
2. Topics must relate to athletic training. (case reports, experimental reports, suggestions, new ideas, tips and/or specifics for a given problem)
3. Articles should be no more than 2 to 3 pages in length, double spaced.

If you wish to submit a manuscript or an item for the Student Corner, please send to: Deloss Brubaker, 103 Gill Coliseum, Oregon State University, Corvallis, OR 97331.

Licensure Update . . .

In our President's Message (see page 7) there is a listing of the states now having licensure; however, to publish each state's acts and reciprocity requirements would involve 12 to 15 pages per state. If you have particular questions contact Robert Behnke, Chairperson, NATA Licensure Committee, Indiana State University, Terre Haute, IN 47809.

Suggestion . . .

I would encourage those planning to attend the 1984 Olympic Games in Los Angeles to invest in the Travel Information Kit, c/o 1984 Games, Map Advance Research Marketing, 150 South Glenoaks Blvd., Suite 9240, Burbank, CA 91510.

Membership Drive . . .

It has been brought to my attention the need for all NATA members to solicit more physicians as advisory members of our Association. Please contact Sandra Robinson at the National Office for further information.

Personal Thanks . . .

The Journal Committee and staff members would like to thank everyone who contributed the back issues which enabled us to complete the library at the National Office. It is with a great deal of pride that we state *our Journal library is complete!* Many thanks for all the contributions.

Best Wishes . . .

It is that time of year special to all of us, whether we are in post-season football classics or basketball tournaments. The staff of the Journal wishes you and yours the best holiday season and a Happy New Year.

SY(SSSA) +

CAUTION YOUR PLAYERS

A problem for all coaches and athletic trainers is the proliferation of agents and would-be agents who seek to make agreements with players prior to the completion of their eligibility. The activity of these people will increase now that there is another professional league. It is imperative that all of the consequences of making an agreement with an agent are known by your players.

Contacts with players by agents almost always is done without any knowledge of the coach. Some agents openly admit they will continue to make contacts and agreements with players before their final season has been completed.

This could lead to forfeiture of games. Some agents are advising players not to risk injury by playing. Your players must be warned about the problem. Do it more than once.

ATTENTION!
SUPERVISING ATHLETIC TRAINERS —

Call the National Office (919/752-1725) for bulk orders of membership application forms and encourage your students and colleagues who fall into these categories to join the NATA. Please specify "Student Membership" (for Undergraduate Student Members), "Associate Membership" (for Graduate Student Members), or "Advisory" (for Team Physicians).

President's Message

Dear NATA Members:

As 1983 draws to a close, I would like to sincerely thank each of you for your support during the last two years. I believe we have achieved many common goals for the betterment and advancement of our profession.

It appears that our action regarding private sector employment has been well received. State laws have been passed in Georgia, Kentucky, Massachusetts, Missouri, New Mexico, North Dakota, Oklahoma, Rhode Island, Texas and South Carolina.

The Board of Directors of NATA, Inc. will be holding their midyear meeting in San Antonio, Texas in February, 1984. It will be our honor to host the winter meeting of the "Who Speaks for Sports Medicine" group. Our Association is an original member of this group, which consists of various related Sports Medicine associations, and has been very well received by those who are involved with the formation of these meetings. The culminating activity of this meeting will be sponsoring a Sports Medicine Congress and Exposition in 1985. Additional information regarding the "Who Speaks for Sports Medicine" meeting will be made available at Nashville.

Best wishes for a happy holiday season and prosperous new year.

Sincerely,

Bobby

Bobby Barton, ATC

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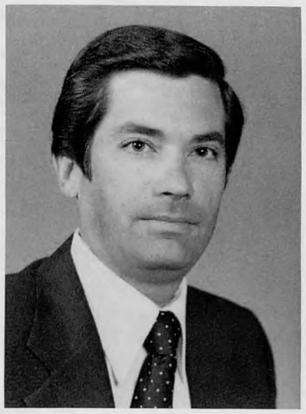

Jeff Fair, ATC, MS
Oklahoma State University

January 1984

8-14 Conference on Biomechanics and Kinesiology in Sports, Colorado Springs, CO. Contact Mary Margaret Newsom, US Olympic Committee Sports Medicine/Education Services, 1750 E. Boulder Street, Colorado Springs, CO 80909.

8 Oklahoma Athletic Trainer's Association Annual Meeting, Stillwater, OK. Contact Dan Pickett, Athletic Department, University of Oklahoma, Norman, OK 73019.

13-14 How to Implement and Manage a Sports Medicine Program, New Orleans, LA. Contact Ronald G. Peyton, Sports Medicine Education Institute, Inc., 993 Johnson Ferry Road NE, Suite 130-F, Atlanta, GA 30342.

15-17 Thirty-fifth Annual Meeting of Eastern Athletic Trainers Association, Grossinger, NY. Contact Joe Camillone, Trenton State College, CN550, Trenton, NJ 08625.

20 Sports Medicine Symposium on Adolescent Injuries, Wallingford, CT. Contact Dave Anderson, Choate Rosemary Hall School, Wallingford, CT 06492.

27-28 Eleventh Annual Sports Medicine Symposium, San Antonio, TX. Contact Jesse C. DeLee, MD, Department of Orthopaedics, University of Texas Medical School, 7703 Floyd Curl Drive, San Antonio, TX 78284.

27-29 Second Conference on Injuries to the Throwing Arm: Biomechanical and Medical Aspects, Tampa, FL. Contact Mary Margaret Newsom, USOC Sports Medicine Division, 1750 E. Boulder St., Colorado Springs, CO 80909.

February 1984

4 1984 Metroplex Athletic Trainers Clinic, Grand Prairie, TX. Contact Pat Forbis, Grand Prairie High School, 101 High School Drive, Grand Prairie, TX 75050.

4-11 Office Based Sports Medicine, Park City, UT. Contact Terrie VanAlen, Extended Programs in Medical Education, Room U-569, University of California, San Francisco, CA 94143.

March 1984

11 International Aquatic Conference, Indianapolis, IN. Contact Julie Taraba, Recreonics Corporation, 1635 Expo Lane, Indianapolis, IN 46224.

11-16 Kent State Sports Medicine Center Spring Symposium: Prevention and Rehabilitation of Baseball, Running and Skiing Injuries, Kent, OH. Contact Stephen J. Sveda MD, Room 104 Dix Stadium, Kent, OH 44240.

12-16 University of Hawaii Sports Medicine Course, Honolulu, HI. Contact Joy Lewis, Box CEP-CCECS, 2530 Dole Street, Honolulu, HI 96822.

15-17 Great Lakes Athletic Trainers Association Winter Meeting, Schaumburg, IL. Contact Roger Kalisiak, Hoffman Estates High School, 1100 W. Higgins Road, Hoffman Estates, IL 60195.

16-18 NATA District 5 Annual Meeting, Lincoln, NE. Contact Jerry Weber, Athletic Department, University of Nebraska, Lincoln, NE 68588.

18-19 Abbey Rehabilitation '84, Los Angeles, CA. Contact Gail Wetzer, 351 Hospital Road, Suite 214, Newport Beach, CA 92663. +

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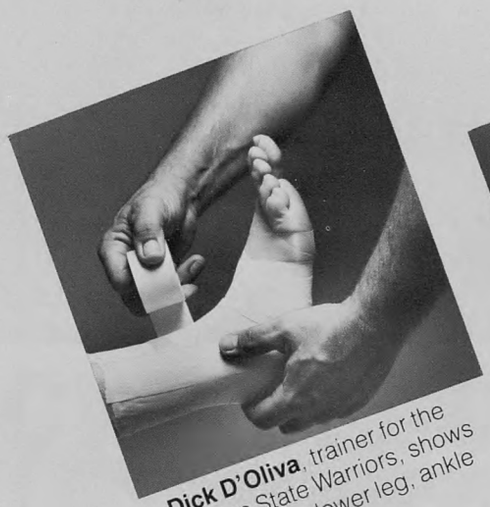

• **Dick D'Oliva**, trainer for the Golden State Warriors, shows taping of the lower leg, ankle and foot

• **Jack Curran,** trainer for the Los Angeles Lakers, demonstrates techniques for shoulder taping

• **Frank Furtado,** Seattle Supersonics trainer, deals with knee injury taping

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W.E. "PINKY" NEWELL
Chairman
Purdue University
Student Hospital
W. Lafayette, IN 47907

Dear Student Trainer:

The National Athletic Trainers Association is looking for exceptional young men and women who wish to compete for an undergraduate or graduate scholarship to help them continue their education in the athletic training field.

Since 1974, the Association has awarded grants totaling $98,250, and expects to offer 28 scholarships of $1,000 each at the Student Trainer Banquet during the NATA Annual Meeting in Nashville, Tennessee, in June 1984. These awards will be given to students who have demonstrated a commitment to academic excellence in the field of sports medicine, and will be divided equally between undergraduates and postgraduates.

In the past, applicants have been extremely well qualified by virtue of their education backgrounds, extra-curricular activities, and their demonstrated abilities and interests. We feel certain there are many potential scholarship applicant of similar qualifications who would merit consideration for one of the available NATA scholarship awards. We urge you to submit your application!

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Offered for excellence to college or university juniors only; is designed to assist the student during his/her senior year.

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Eligibility for consideration is based on the records of the student's final year in college or university. Applicants must signify their intentions to continue academic work beyond the baccalaureate degree as a full-time graduate student and plan to make athletic training the primary field of professional endeavor.

The closing date for nominations is February 1, 1984. Application folders may be obtained from the NATA National Office, P.O. Box 1865, Greenville, North Carolina 27834, or from William E. Newell, Chairman, NATA Committee on Grants and Scholarships, Purdue University Student Hospital, West Lafayette, Indiana, 47907.

Let us hear from you!

William E. "Pinky" Newell

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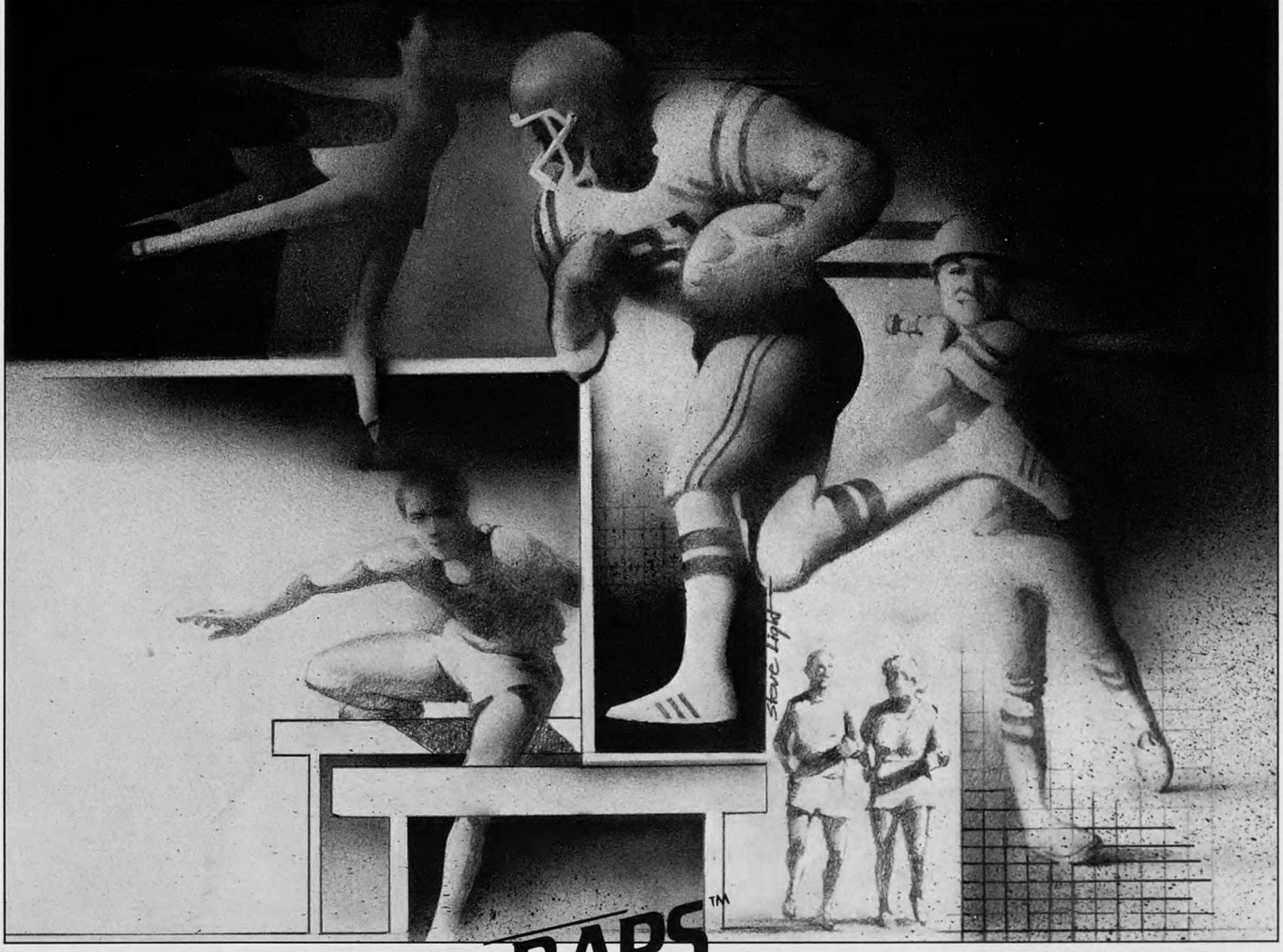

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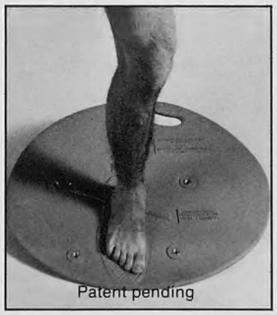

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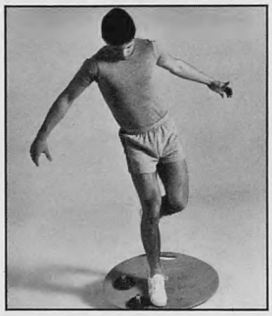

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Association Activities

David G. Yeo, DPE, ATC
Montgomery County
Community College

The AOSSM has presented a $2,000 donation in Fred W. Hoover's name to the NATA Scholarship Fund, and the Distinguished Service Award will be formally presented to Fred in the spring. The Distinguished Service Award is the ultimate honor an athletic trainer can receive from his profession and colleagues. The NATA is very privileged to recognize Fred Hoover, one of our consummate professionals in terms of dedication, leadership, and competence. We are, indeed, fortunate that he continues to serve so well his profession, Clemson University, and the National Athletic Trainers Association.

****

Chuck Medlar, retired Penn State head trainer, and one of Pennsylvania's pioneers in athletic training, was honored at an awards luncheon by the Pennsylvania Athletic Trainers' Society.

Doug May, of the Sports Medicine and Fitness Center, Doctors Hospital, Jackson, Mississippi, has been elected president of the newly created Mississippi Athletic Trainers' Association.

Mark Keppler of Carnegie-Mellon University, Pittsburgh, has assumed the office of President-Elect of the Pennsylvania Athletic Trainers Society, and Don Wheeler of Lafayette College becomes the Secretary-Treasurer of the PATS Board of Directors.

Paul Slocum, formerly of Bloomsburg State College, PA, has assumed the position of Manager of the Sports Medicine Department at NPW Medical Center, Wilkes Barre, PA.

Joe Vegso, Head Trainer at the University of Pennsylvania Sports Medicine Center, has been appointed Liaison Representative for Political Affairs of the Pennsylvania Athletic Trainers' Society. He replaces Joe Godek, West Chester State College, who resigned after spending seven years in negotiations with physical therapists in legislation relating to licensure.

Kip Smith, formerly of the University of Pittsburgh, has assumed the position of Head Football Trainer at Indiana University.

FRED HOOVER
Assistant Athletic Director
Head Trainer
Clemson University

Fred Hoover Honored With Distinguished Service Award

Fred W. Hoover, Head Athletic Trainer and Assistant Athletic Director at Clemson University, has been named the recipient of the 1983 Distinguished Service Award. This award is the highest and most prestigious honor that can be bestowed upon an athletic trainer. Sponsored by the American Orthopaedic Society for Sports Medicine, each of the ten districts of the NATA nominates one name to the Selection Committee of the AOSSM, served by six physicians and athletic trainers. The recipient of the Distinguished Service Award must be a member of the NATA Hall of Fame.

In the Spring issue of *Athletic Training,* a brief biographical sketch was presented of each of the first two recipients of the Distinguished Service Award, William E. "Pinky" Newell for 1981, and Otho L. Davis for 1982. It is an honor to share some of the background of this year's honored trainer, Mr. Fred Hoover.

A native of Jacksonville, Florida, Fred Hoover received his Bachelor of Science degree in physical education from Florida State in 1953. He served as trainer at his alma mater in 1952-53, and from 1957-58, before moving to Clemson in 1959.

Now in his 23rd year as trainer for Clemson, Hoover coordinates the training and medical needs for the University's 19 men's and women's sports programs. In addition, he was named Assistant Athletic Director in the summer of 1983, and is responsible for purchasing equipment and making travel arrangements.

Fred Hoover has served on the Board of Directors and as Convention Chairman of the NATA. He was trainer for the U.S. team in the 1963 Pan American games in San Paulo, Brazil, and served on the U.S. staff at the 1968 Mexico and 1972 Munich Olympics. He is a recipient of the NATA Twenty-five Year Award, and in 1981 Hoover was elected into the Citizens Savings - Helms Athletic Foundation Trainers Hall of Fame. In the spring of 1982, he was named consultant to the South Carolina Medical Association Medical Aspects of Sports Committee. Hoover is an affiliate member of the American Orthopedic Society for Sports Medicine, and a member of the Clemson Hall of Fame.

Fred is married to the former Elva Cook of Mt. Vernon, Illinois, and they have two children —Catherine, a Clemson graduate who is married. and Bryan, a senior at Clemson. +

National
ATHLETIC TRAINERS ASSOCIATION
Committee On Grants And Scholarships

1001 East 4th St., Greenville, NC 27834

W.E. "PINKY" NEWELL
Chairman
Purdue University
Student Hospital
W. Lafayette, IN 47907

Dear Association Member:

As our Association continues to grow and meet the challenges of a changing professional education, financial support remains an important part of reaching our goals. For this reason, the Committee on Grants and Scholarships invites you to join our list of contributing members to the Association's Endowment Fund. By doing this, you will become a partner in a scholarship program that provides hope and opportunity to the student trainer membership.

The Association recognizes the great need for the scholarship program and has funded the activity well, but the Board of Directors feels that we must increasingly help ourselves if we wish to fulfill the future identifiable goal of the scholarship program which is to recognize outstanding young student trainers by assisting them with their academic objectives.

We have been fortunate in that all scholarships are fully sponsored and none of the Endowment Fund has been used; however, there is concern regarding the danger of losing some of our sponsored awards. Our sponsors have been extremely generous in the past, but they too have been hurt by the economy. We must avoid losing our commitment to the student trainers by failing to recognize their professional preparation at a time when such recognition and encouragement is most important.

Contribution levels may begin as low as $1 per month ($12 per year), but we very much welcome and appreciate contributions of $25, $50, and $100, or more. All contributions are state and federal tax deductible.

I know we can count on your support.

Thank you, sincerely,

William E. "Pinky" Newell

William E. "Pinky" Newell

* * * * * * * * *

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Abstracts

John Wells, ATC, PT, PhD
Mars Hill College

"Hyperglycemia Physiology, Signs and Symptoms," Dolores Lake Taylor, RN, MSN, *Nursing Journal,* 52-53. February 1983.

Normally, the body regulates blood-glucose concentration so stringently that levels range only from 80 to 110 mg/100 ml of blood. But certain conditions, especially diabetes, can disrupt this control. In the diabetic, lack of insulin (a glucose-lowering hormone) allows blood-glucose concentration to soar as high as 300 to 1,000 mg/100 ml of blood. Lack of insulin inhibits transportation of glucose from the blood, across the cell membranes, into cell. Rising glucose concentration in the blood increases osmotic pressure. Water is drawn from the intercellular space, dehydrating cells causing polydipsia. When glucose concentration in the blood reaches its saturation point, glucose overflows into the renal tubules causing glucosuria. Rising glucose concentration also increases osmotic pressure in the proximal tables. Water that would normally be reabsorbed is excreted in the urine. This causes polyuria. As glucose becomes unavailable to the cells, fats, and proteins are oxidized for energy causing polyphagia. Stored fats are broken down into fatty acids. The liver metabolizes only a small portion of them, and large amounts of keto acids and acetone - by products of fatty acid metabolism - accumulate in the body. This causes ketosis.

George Jarrett, III

* * *

"Leg Muscle Performance of Athletes With and Without Knee Support Braces," Michael E. Houston and Paul H. Goemans, *Archives of Physical Medicine and Rehabilitaion.* 63: 431-432, September 1982.

A variety of protective and supportive knee devices have been devised because of the high incidence of injuries to this joint. Indeed, support braces can enable individuals with knee ligament instability to participate successfully in athletics. This study evaluates leg performance characteristics of young male athletes with and without their prescribed knee support braces. The effects of wearing and not wearing prescribed knee support braces of leg muscle performance were studied in 7 male athletes. All subjects wore prescribed hinged knee support braces on one leg because of medial collateral ligament instability, anterior cruciate ligament instability, or both. Testing took place over a 4 week period, with at least 1 day of rest between testing days. Mean maximal torque outputs during isokinetic knee extension contractions with braces were significantly lower by 12 to 30% compared to corresponding values without knee braces. Differences between the 2 conditions were more prominent during faster contractions. Maximal velocity measured during unloaded knee extension was 20% higher when subjects did not wear their braces. In addition, vertical velocity during a short all-out stair run was slower (p. 0.01) with braces. Marked performance reductions with braces during knee extension and stair run tests, involving both knee extension and flexion, suggest that braces had a damping effect, absorbing force output by muscles about the knee joint. Futhermore, the fact that blood lactate levels were markedly higher during the 15 minute bicycle ergometer exercise with knee braces, despite the identical workloads, suggests that braces could also interfere with blood flow and hence oxygen delivery. In conclusion, these results demonstrate that benefits of braces in terms of knee support come at the expense of impaired maximal performance, at least for young male athletes.

Lois Howard

* * *

"Inflammation, Physiology, and Symptoms," Delores Lake Taylor, RN. MSN, *Nursing Journal*, 52-53, January 1983.

Inflammation - you see it every day. But what's the physiologic process causing that redness and swelling? What is really going on? Inflammation, the body's response to injury, comprises a series of complex histologic reactions that localize and destroy the damaging substance and damaged tissue. Actually, inflammation is the first step in the healing process. The classic signs and symptoms of inflammation, described over 2,000 years ago, still hold true today: rubor (redness), color (warmth), tumor (swelling), dolar (pain), and functio laesa (loss of function). Inflammation begins with tissue injury - from trauma or disease. Almost immediately, the damaged tissue releases large quantities of histamine. The vessels dilate from the histamine released causing increased blood flow to - and congestions in - the area. Increased capillary permeability allows fluid and protein to shift from the intravascular space to the interstitial space. Triggered by immune responses in the blood, leukocytes migrate to the area via the bloodstream, injest toxic substances, and die. The dying leukocytes release endogenous pyrogens. The pyrogens are picked up by the blood and circulate to the hypothalamus, increasing body temperature. The pyrogens also circulate to the bone marrow, where they stimulate the release of leukocytes to the bloodstream.

George Jarrett, III

* * *

"The Medial Patellar Plica Syndrome," Nottage, Wm., et. al., *The American Journal of Sports Medicine* 11: 211-14, 1983.

Prior to the advent of arthroscopy, little attention was paid to the (synovial) plicae within the knee joint. Tight bands were noted at arthrotomy and dismissed as "adhesions" and were not felt to be capable of producing an internal derangement of the knee. Increased arthroscopic experience has led to the identification of several characteristic plicae which have been implicated as causing acute and chronic knee pain. There are three commonly seen plicae within the knee whose frequency,

continued on page 313

CEU Credit Quiz

Drug Interactions

Edward A. Hartshorn, PhD

Edited by: Don Kaverman, ATC

The term "drug-drug interaction" is most commonly used to describe the situation when the effects of a drug are altered by the concurrent or previous administration of another drug. The term "drug interaction" may be used synonymously, but the literature of drug interactions may be broader. It may include reports of interactions between drugs and other chemicals not always thought of as "drugs" (e.g., non-prescription medications, foods, environmental contaminants such as smoking, etc.). Furthermore, while much of the older literature described noticeable changes in the patient's response to a drug, much of today's literature describes discrete changes in how the body handles a drug without, necessarily, a clinically apparent change in the drug's effect.

The classical drug interactions involve an alteration in how the body handles a drug, that is, a change in the rate or amount of drug absorbed or in the rate of a drug's metabolism or excretion. However, the most commonly occurring drug interactions are the result of additive pharmacological effects. Such reactions are often simply considered an expected side effect by some experts. For example, the concurrent use of alcohol, an analgesic, an anti-anxiety agent, and a muscle relaxant may produce excessive nervous system depression manifested by drowsiness, slowed respirations, or slowness in response to a command or situation.

Dr. Hartshorn is currently Professor of Pharmacy, Medical University of South Carolina, College of Pharmacy, Charleston, SC 29425. He also serves as a consultant to the South Carolina Drug Advisory Committee.

Antihistamines

Antihistamines are marketed as prescription drugs (e.g., Benadryl®, Chlor-Trimeton®, and others) primarily for the relief of symptoms of allergy (i.e., for red eyes, runny nose, hives, itching, etc.). Antihistamines are also found in many non-prescription drugs ("over-the-counter" or OTC drugs). Occasionally the OTC drug is the same as the prescription drug but in a smaller dose. OTC medications containing antihistamines include allergy medicine (e.g., Allerest®), sleeping aids (e.g., Sominex®), or cough and cold medicine (e.g., Coricidin®). The latter two categories make use of two common side effects of the antihistamines; drowsiness (central nervous system depression) and dryness (anticholinergic effect). These two side effects create the majority of important drug interaction problems of the antihistamines.

CNS Depression: Not only are there many prescription and OTC products containing antihistamines (often in combination with other drugs), there are also numerous types of drugs which have a central nervous system depressant action (e.g., alcohol, antianxiety medication, antipsychotic medication, antidepressant medication, many antihypertensive drugs, muscle relaxants, pain medication, etc.). Hence, it is extremely easy to be taking several medications, all for different purposes, but all having CNS depressant effects. The result is drowsiness, dizziness on standing, confusion, difficulty in thinking or making decisions, slowed response to stimuli, and slowed respirations. A number of deaths have been reported that have been the result of an additive effect from the use of multiple drugs with CNS depressant action.

Anticholinergic Effect: Similarly, there are many drugs which have anticholinergic effects similar to the antihistamines. These include the antianxiety drugs, antipsychotic drugs, antidepressant drugs, antispasmodics, and certain drugs used to treat ulcers or intestinal motility. The additive effect from the use of several drugs with anticholinergic action is likely to be seen as dryness of the mouth, fuzziness of vision, uncomfortable feeling in hot humid weather (because of the inability to sweat), difficulty in urinating, and constipation. Several deaths due to additive anticholinergic effect (usually due to complete stasis of gut motility) have been reported.

Analgesics

There are two types of analgesics, the non-narcotic agents such as aspirin or acetaminophen (Tylenol®), and the narcotic drugs such as codeine, morphine or meperdine (Demoral®). Aspirin has anti-inflammatory properties and many of its interactions are similar to other anti-inflammatory drugs, hence will be considered in the next section. Acetaminophen has few, if any, clinically important interactions and can be eliminated at this point.

Narcotic analgesics depress respirations. The most important interaction of the narcotic analgesics is additive or potentiated respiratory depression when they are used with other agents which have a CNS depressant component. This is particluarly important with alcohol, antianxiety drugs, and antipsychotic drugs. Other reported interactions of narcotic analgesics include:

Morphine + Cimetidine (Tagamet®). Cimetidine is a popular antiulcer drug. It has been reported to enhance the depressant effect of morphine but this is not well substantiated.

Methadone + Rifampin. Rifampin, an antituberculosis drug, may decrease the effectiveness of methadone or produce withdrawal symptoms in a narcotic addict.

Propoxyphene (Darvon®) +

- Alcohol. This combination has resulted in seriously depressed respirations and death with relatively low overdoses of propoxyphene.
- Doxepin (Sinequan®). Extreme lethargy has been reported to occur in patients taking both doxepin, an antidepressant, and propoxyphene.
- Carbamazepine (Tegretol®). Increased carbamazepine serum level and toxic reactions have been reported in patients taking propoxyphene concurrently.

Meperidine (Demerol®) + Monoamine Oxidase Inhibitors. MAO inhibitors (e.g., Parnate®) are antidepressant drugs. Combination of meperidine plus MAO inhibitors has resulted in sweating, rigidity, hypotension, coma and death.

Anti-Inflammatory Agents

Aspirin. Aspirin is an analgesic, antipyretic, and anti-inflammatory agent. All of these actions may be mediated, in part, by interference with the synthesis of certain prostaglandins. Many other anti-inflammatory agents, such as Butazolidin®, Indocin®, or Motrin® are believed to act via this same effect on prostaglandins. Important interactions of aspirin include the following:

Alcohol. Prolonged bleeding time and increased GI bleeding.

Antacids. May decrease salicylate blood levels.

Anticoagulants. Increased incidence of bleeding.

Antidiabetics. Lowered serum glucose levels; enhancement of the effect of antidiabetics.

Anti-inflammatory Agents. Decreased serum level.

Corticosteroids. Increased potential for GI ulceration. Corticosteroids may lower serum salicylate levels.

Methotrexate. Increase in development of pancytopenia in subjects taking both methotrexate and aspirin.

Probenecid; sulfinpyrazone. Decreased uricosuric effect.

Non-Steroidal Anti-Inflammatory Agents (NSAIA). The NSAIA include a wide group of drugs generally characterized either as the newer aspirin-like drugs (Motrin®, Naprosyn®, Feldene®, Clinoril®, Meclomen®, Nalfon®, Tolectin®) or the older more potent agents used in gouty, rheumatoid or osteo arthritis, such as Butazolidin® or Indocin®. Interactions of the NSAIA include:

Alcohol. Prolongation of bleeding time.

Anticoagulants. Some NSAIA (e.g., phenylbutazone, sulindac) appear to enhance the hypoprothrombinemic effect of warfarin; others cause gastric irritation and enhance the chances for a bleeding episode by decreasing platelet aggregation.

Antidiabetics. Certain NSAIS may enhance the hypoglycemic effect of oral antidiabetic agents.

Aspirin. Aspirin decreases the absorption of many NSAIA. It also interferes with the uricosuric effect of some.

Beta Blockers. Many NSAIA appear to decrease the anti-hypertensive effectiveness of these drugs.

Corticosteroids. Enhancement of GI irritating effect and bleeding.

Furosemide. Decreased diuresis and antihypertensive effect.

Phenytoin. Certain NSAIA may precipitate phenytoin toxicity.

Probenecid. Probenecid blocks the excretion of indomethacin.

Smoking. Heavy smoking stimulates drug metabolizing enzymes in the liver and decreases the serum concentration of a number of drugs including phenylbutazone.

Corticosteroids. Corticosteroids are anti-inflammatory agents which act in a manner completely different from those drugs previously discussed. Steroids are most often used for short-term therapy because of serious side effects associated with long-term use.

Drugs which have been reported to interact with corticosteroids include:

Amphotericin/Thiazides. Enhanced hypokalemic effect.

Barbiturates/Anticonvulsants/Rifampin. These drugs are all enzyme inducers, likely to increase the rate of metabolism of the steroids and decrease their effectiveness.

Salicylates/NSAIA. Increased incidence of GI irritation and bleeding.

Anticoagulants. Corticosteroids may antagonize the hypoprothrombinemic effect of oral anticoagulants but increase the risk of hemorrhage due to a vascular effect.

Antidiabetics. Corticosteroids have an intrinsic hyperglycemic effect.

Anti-Infective Agents

Many of the antibiotics involved in drug interactions are those which are administered intravenously. This review will be restricted to those anti-infectives likely to be used by ambulatory patients.

Erythromycin +

Carbamazepine. Carbamazepine toxicity.
Theophylline. Theophylline toxicity.
Warfarin. Hemorrhagic episodes.

Isoniazid (INH) +

Antacids. Decreased INH absorption.
Carbamazepine. Carbamazepine toxicity.
Cheese. Headache, increased heart rate, flush.
Disulfiram (Antabuse®). Change in affect and behavior.

Metronidazole (Flagyl®) +

Alcohol. "Antabuse" reaction (flush, nausea, headache, hypotension.)

Penicillins +

Allopurinol (Zyloprim®). Increased incidence of skin rashes in patients given ampicillin.

Food. Decreased penicillin absorption.
Oral Contraceptives. Pregnancies reported in women on oral contraceptives after starting ampicillin therapy.

Sulfonamides (e.g., Gantrisin®; combinations, e.g., Bactrim®)
Methotrexate. Increased free methotrexate and toxicity.
Warfarin. Bleeding episodes.

Tetracyclines +
Antacids, adsorbents, dairy products, iron. All decrease tetracycline absorption.
Barbiturates. Decrease half-life of doxycycline.

Enzymes

The oral proteolytic enzymes used for reducing inflammation and edema, such as the bromelains (Ananase®) have not appeared in the drug interaction literature. Topical enzymes used as debriding agents (e.g., sutilains, collagenase, papain) may be inactivated by various detergents or anti-infective agents.

Muscle Relaxants

Muscle relaxants are frequently used as adjunctive therapy for relief of discomfort associated with acute, painful musculoskeletal conditions.
Typical muscle relaxants include:

Baclofen (Lioresal®)	Dantrolene (Dantrium®)
Carisoprodol (Soma®)	Diazepam (Valium®)
Chlorzoxazone (Paraflex®)	Metaxolone (Skelaxin®)
Cyclobenzaprine (Flexeril®)	Methocarbamol (Robaxin®)
	Orphenadrine (Norflex®)

All of the muscle relaxants have a CNS depressant effect which will be additive with other CNS depressants including alcohol.

Cyclobenzaprine, diazepam, and orphenadrine have anticholinergic effects which will be additive with other drugs which have an anticholinergic action.

The action of diazepam may be prolonged by cimetidine.

Alcohol

Alcohol interactions include the following:

CNS Depressants. The CNS depressant effect of alcohol is additive, and in some cases (e.g., propoxyphene), potentiated by other CNS depressants.

Tricyclic Antidepressants. Acute ingestion of alcohol inhibits liver enzymes which metabolize drugs. Elevated serum levels of certain antidepressants have been reported with acute ingestion of alcohol.

Chlorpropamide/Disulfiram/Metronidazole/Cephalosporins. Disulfiram is used to discourage alcoholics from drinking. Disulfiram-like side effects (flushing, headache, nausea, weakness) have been reported in subjects taking chlorpropamide, metronidazole, or certain cephalosporins.

Aspirin/NSAIA/Anticoagulants. Ingestion of alcohol increases the risk of hemorrhage in subjects taking these drugs.

Guanethidine/Nitroglycerin. Orthostatic hypotension may occur in subjects who drink alcohol while taking these drugs.

Cimetidine. Subjects who drink while taking cimetidine become intoxicated more rapidly and have higher alcohol blood levels than subjects not taking cimetidine.

Phenytoin/Tolbutamide/Warfarin. Chronic alcoholism results in increased activity of drug metabolizing enzymes. Reports suggest diminished effects of the above drugs in chronic alcoholics.

Anabolic Steroids

The anabolic steroids include:
Fluoxymesterone (Halotestin®)
Methandrostenolone (Dianabol®)
Nandrolone (Durabolin®)
Oxymetholone (Anadrol®)
Stanozolol (Winstrol®)

The anabolic steroids are generally testosterone derivatives which have the following reported interactions:

Anticoagulants. Increases sensitivity to the anticoagulants with reports of hemorrhage.

Antidiabetics. Enhanced hypoglycemia.

Calcium and calcium-retaining drugs. Hypercalcemia

Adrenal steroids. Edema

Laboratory Tests: Altered glucose tolerance tests, thyroid tests, and others.

Interventions and Resources

There are more than a half-dozen tests on drug interactions. In 1982, 495 drug interaction reports were reviewed in 109 different medical and pharmacy journals. An athletic trainer must keep abreast of these various interactions.

It is best, but not always practical, to give only one drug. If a patient is to receive more than one drug the possibility of an interaction should be checked with a resource. If one is familiar with what has happened before, appropriate action can be taken. In most cases of potential drug interactions, it is necessary only to monitor the subject. There may be times when it is better to substitute for one of the interacting drugs; other times one may have to adjust the dose of the drug according to the subject's response. In most cases it is necessary only to be aware of the potential problem and monitor for a change in the patient's response.

While many interactions appear to be rare or mild, they occasionally are life-threatening. Being aware of potential problems and closely monitoring the patient can reduce the risk of serious drug interaction.

Probably the most convenient resource for information on potential drug interactions is a local drug information center. Many medical universities and large hospitals have such a center. Information may also be obtained from a local pharmacy. Reference texts include *Drug Interactions* by Hansten, *Drug Interactions* by Stockley, and *Drug Interaction Facts* published by Facts and Comparisons. An annual annotated bibliography is also available to update the above texts.

References

1. Hansten PD: *Drug Interactions.* 4th ed. Philadelphia, PA: Lea & Febiger, 1979.
2. Stockley IH: *Drug Interactions.* Boston, MA: Blackwell Scientific Pub., 1981.
3. Anon. *Drug Interaction Facts.* St. Louis, MO: Facts and Comparisons, 1983.
4. Hartshorn EA: *Drug Interaction Update 1982.* Washington, D.C.: Am Soc Hosp Pharm, 1983.

Note: Many of the above interactions are listed by groups of drugs while the actual interaction may have been reported with only one member of the group. This article provides only a general outline of potential problems. Specific drug combinations require specific referencing.

+

CEU Credit Quiz

DRUG INTERACTIONS

Edward A. Hartshorn, Ph.D

As an organization accredited for continuing medical education, the Hahnemann Medical College and Hospital certifies that this continuing education offering meets the criteria for .3 hours of prescribed CEU credit in the program of the National Athletic Trainers' Association, Inc., provided the test is used and completed as designed.

To participate in this program, read the material carefully and answer the questions in the test. Mark the answers you select by placing an X in the proper square. Then tear out the test sheet, fill in your name, address and other information, and mail with $12 for processing to: School of Continuing Education, Hahnemann Medical College, 230 N. Broad St., Philadelphia, PA 19102.

The NATA National Office will be notified of all members with passing scores over 70%. CEU credit will be issued to each member's record at that time. Participation is confidential.

Questions	a	b	c	d	e
1. Which of the following types of agents when used in combination with Benadryl can cause excessive difficulty in thinking or making decisions? 1. antiaxiety agents 2. antidiabetic agents 3. antihypertensive agents 4. corticosteroids a. 1, 2, 3 b. 1, 3 c. 2, 4 d. 4 only e. 1, 2, 3, 4					
2. The additive effect from the use of several drugs with anticholinergic action is likely to be seen as 1. dryness of the mouth 2. fuzziness of vision 3. difficulty in urinating 4. constipation a. 1, 2, 3 b. 1, 3 c. 2, 4 d. 4 only e. 1, 2, 3, 4					
3. Which of the following agents has been known to decrease the effectiveness of methadone? a. Darvon b. Diabinese c. Rifampin d. all of the above e. none of the above					
4. Extreme lethargy has been reported to occur in patients taking both doxepin and acetaminophen. a. True b. False					
5. Which of the following agents have been known to cause hypoglycemia when taken concomitantly with an antidiabetic agent? a. aspirin b. non-steroidal anti-inflammatory agents c. both of the above d. none of the above					
6. Orthostatic hypotension is frequently a problem for a patient who is taking both a beta blocker and a non-steroidal anti-inflammatory agent. a. True b. False					
7. The effectiveness of corticosteroids may be decreased with the concomitant use of 1. barbiturates 2. anticonvulsants 3. Rifampin 4. antidiabetic agents a. 1, 2, 3 b. 1, 3 c. 2, 4 d. 4 only e. 1, 2, 3, 4					

FOR CREDIT, form must reach Hahnemann Medical College by March 15, 1984.

		a	b	c	d	e
8. Which of the following signs and symptoms may occur with the concomitant use of alcohol and metronidazole? 1. flushing 2. nausea 3. headache 4. hypotension	a. 1, 2, 3 b. 1, 3 c. 2, 4 d. 4 only e. 1, 2, 3, 4					
9. If an individual on Isoniazid (INH) eats cheese, he/she may develop a rapid heart beat. a. True b. False						
10. The individual who ordinarily does not become intoxicated on a six-pack of beer, may become intoxicated on that amount of alcohol if he/she begins taking a. guanethadine b. a cephalosporin c. cimetidine d. b and c above e. all of the above						
11. Which of the following muscle relaxants has/have anticholinergic effects? 1. dantrolene 2. diazepam 3. baclofen 4. cyclobenzaprine	a. 1, 2, 3 b. 1, 3 c. 2, 4 d. 4 only e. 1, 2, 3, 4					
12. There is an increased risk of hemorrhage with the concomitant use of anticoagulants with: 1. some antibiotics 2. corticosteroids 3. anabolic steroids 4. some non-steroidal anti-inflammatory agents	a. 1, 2, 3 b. 1, 3 c. 2, 4 d. 4 only e. 1, 2, 3, 4					

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Mail with $12 fee to:
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If you are interested in submitting an article to be used in the Journal quiz please contact Don Kaverman at Ferris State College. All authors of published articles will receive 1.0 CEU.

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3 These ten dollar gold Olympic coins are the first gold coins minted in the United States in some 50 years.

4 The U.S. Mints profits from the sale of the coins will go toward the Olympic effort for training expenses, the ever-rising costs of sending our team to the Olympic Games, and staging of the Games in Los Angeles.

5 These are "coins of the realm" which, as legal tender, will be highly prized by collectors.

6 The coins are works of art. The 1983 silver dollar coin was designed by Elizabeth Jones, chief engraver at the Mint. The front of the coin shows a dramatic depiction of the classic Greek discus thrower. The 1984 silver dollar coin was designed by Robert Graham, a renowned Los Angeles sculptor. The front of the coin bears a representation of the Gateway to the Olympic Coliseum. The 1984 gold ten dollar coin was designed by John Mercanti, a member of the U.S. Mint engraving staff, developed from a concept created by James Peed, a visual information specialist at the Mint. The coin captures the penetrating scene of the Olympic Torch bearers in delicate detail.

7 The 1984 Olympic commemorative coins are handsomely packaged and protected. There could be no greater thoughtfulness for special gifts than an Olympic commemorative coin. They are a memento of a great event and will become a possession of lasting value. Most importantly, they may serve as an inspiration: not everyone can be an Olympic champion, but with sufficient opportunities we can all achieve our personal goals.

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Nutritional Considerations For The Marathon Athlete

Kent Scriber, MS, PT, ATC

During the fifth century B.C. a Greek champion attributed his success to a high protein diet. Since that time it seems that athletes and coaches have been in a continuous search for the possibility of enhancing athletic performance through diet. This desire to gain an "extra edge" has caused many trained athletes to use various forms of foods and supplements for this purpose. When they do achieve a degree of success, it may reinforce their beliefs and often spreads their ideas to others. One does not have to spend a great deal of time with athletes to have their pseudo-scientific and even superstitious attitudes and beliefs toward nutrition and performance made obvious.

Contrary to what many believe, the question of nutrition for the athlete is quite straightforward and does not involve too much mystique. When one searches the literature it is clear that the basic nutritional needs of athletes do not vary significantly from sedentary individuals except for quantity of certain nutrients. The best available evidence to date suggests that an optimum diet for athletes, as for non-athletes, must supply adequate amounts of water, calories, proteins, fats, carbohydrates, vitamins and minerals in suitable proportions (16, 24, 26, 27, 33). However, a major factor that appears to enhance an endurance athlete's capacity for work is nutritionally based (7, 8, 14, 18, 19, 23, 25, 30, 32). With heavy prolonged exercise it can be of value to increase the glycogen stores in muscle groups performing the heaviest work (2, 3, 4, 13, 17, 18, 21, 26). Dehydration can also impair a runner's performance and pose serious health risks (1, 15, 24, 25). The purpose of this paper is to explore some of the scientific information presently available relating to diet as it applies to the endurance athlete, specifically the marathoner.

This topic is easily justified when one takes a cursory look at the number of present day road races and the number of participants. For example, in 1975 there were 812 runners in the United States that had run a marathon in less than three hours and during that same year 887 accomplished that feat in a single race (20). Since that time the numbers accomplishing this sub-three hour goal has gone well into the thousands for some single events. The last ten years have shown a tremendous growth in not only the number of participants in marathons, but also a great deal of scientific evidence has been gathered on the topic of nutritional considerations for the endurance athlete.

Water, Dehydration And Heat Illness

It is known that man can survive without food for 30 days, yet will not survive for more than five or six days if deprived of water. The normal human adult is 60-65% water and there is hardly a human activity in which water does not play a major role. Because of this fact, lack of water can govern performance in endurance sports (2, 28).

Water is the body's principle vehicle for transporting substances and dispersing heat. It is the only means of dissipating body heat effected by evaporation of sweat; and since production of body heat is greatly accelerated during physical exercise, water must be replaced (24). If it is not, body temperature increases and the risk of heat illness becomes obvious. Heat illness is precipitated by several predisposing factors including high temperature, high humidity, wind, poor body ventilation and water deprivation (15, 25).

The Food and Nutrition Board, NRC-NAS (25), made a statement in 1974 that there is no basis for restricting intake of water during contests or practices. Optimal physical performance is dependent upon replacement of water losses. Depriving athletes of water can and has caused avoidable tragedies. This is particularly true in distance running because its requirements place such great demands on both the circulatory regulation and body temperature regulation primarily through sweating (1, 6, 15). The resulting body water loss and accompanying weight loss may be up to 10% of the athlete's body weight. Dehydration of this proportion can significantly limit sweating, place dangerous demands on circulation, reduce capacity for exercise, and expose the athlete to the health hazards associated with hyperthermia (1, 6, 25).

In 1975 The American College of Sports Medicine published its position statement on "Prevention of Heat Injuries During Distance Running" (1) to not only alert race sponsors of the health hazards relating to distance running, but also to inform the public of preventative actions that can reduce this type of injury. These guidelines specify starting times, temperature limits, and recommendations for fluid replacement during warm weather.

In short, adequate hydration before and during an endurance event can not only be of significance in preventing impairment of performance, but also can prevent serious illness to the participant. Dehydration limits the capacity to work, largely through impaired cardiovascular function, and if water loss exceeds 10% of body weight death can result (15). Therefore, unless sweat loss is replaced at frequent intervals during strenuous physical activity, heat illness can develop.

Vitamins And Minerals

There is little question that vitamin supplements are used by many athletes. Although vitamin deficiencies

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may decrease performance (7, 33), there are no data to support the impression that excess vitamins improve physical performance (16, 26, 27, 33). Some supplements are harmless, but others, particularly the fat soluble vitamins A and D, have potentially harmful effects. Excess vitamin C and niacin have also been shown to have toxic effects (16). It seems quite safe to say that a varied, balanced diet would be adequate for sufficient vitamin supply for an endurance athlete.

Reduced plasma concentration of certain minerals has been associated with intense endurance exercise such as distance running. Marginally low or below normal plasma levels have been reported for sodium in those training in hot weather; for potassium, zinc and iron in well trained male distance runners; and for magnesium in runners following a marathon race (11). Rose (29) presented data showing that intensive exercise caused hypokalemia and recommended that the potassium loss in those engaged in an intensive aerobic program be replaced with appropriate foods. Present day thinking seems to be that although potassium losses from heavy sweating does occur, they can easily be replaced through ordinary practices and potassium supplements are not advised (26, 34). Sodium (sodium chloride) losses also occur primarily through sweating precipitated by hot, humid weather. Trained athletes do not require supplements because healthy kidneys automatically conserve sodium and potassium. Electrolytes actually become more concentrated due to dehydration so the need to replace water is greater than any immediate demands for electrolyte replacement (6). The American College of Sports Medicine recommends fluids with sufficiently diluted sugar with small concentrations of potassium and sodium eletrolytes for rapid gastric emptying (1).

A borderline low level of hemoglobin has been found in many well trained endurance type athletes (5, 10, 12, 26, 34). "Sports" anemia or "athlete's" anemia has suggested that iron supplements may be of value for this type of athlete, especially females. However, iron supplements do not necessarily cause a change in hemoglobin level (33). Some feel that lab findings that are associated with anemia in an endurance athlete may be indicative of a physiological response to training (31, 33). In other words, a highly trained athlete showing decreased blood hematocrit or hemoglobin may indeed actually have an increased total blood volume and more total body hemoglobin. In mild cases, this pseudoanemia does not impair endurance performance (10). The best approach appears to be to supplement iron only to those athletes that show an actual iron deficiency or deficient iron stores (26).

Dressendorfer, et al, (11) studied plasma mineral levels in 12 male marathoners during a 20-day race. These athletes consumed an unrestricted, isocaloric diet with no mineral supplements. They found that none of nine minerals tested (calcium, phosphorus, sodium, potassium, chloride, magnesium, iron, copper and zinc) showed tendencies to become persistently reduced over the 312 mile, 20-day road race. These findings give support to the concept that a well balanced diet will satisfy mineral requirements for endurance athletes engaged in prolonged heavy exercise.

Protein, Fats And Carbohydrates

In terms of energy expenditure during exercise the least important nutrient is protein. Protein is needed to build and repair tissue, but is used as a fuel for muscles only during starvation. Therefore, increased dietary percentages of protein are not needed by an athlete even though many athletes regard protein as having greater healthful properties than any other food (26). A high protein diet may lead to ketosis and dehydration and many studies have confirmed that there is no improved performance on high or low protein diets (33).

At low intensities of work, energy is mainly derived from lipids. The body stores fat in almost inexhaustible supplies and in conditioned athletes most submaximal exercise is fueled by fat. Once 70% of maximal aerobic effort is reached the body turns more to carbohydrates for fuel. Once 85% to 90% of maximum oxygen uptake is reached the energy system becomes primarily anaerobic and all energy is derived from carbohydrates (2). Though it is difficult for anyone other than an elite endurance athlete to work close to this level for an entire marathon, there is a good deal of evidence showing that manipulation of diet can enhance performance for endurance events.

Carbohydrate Loading And Glycogen Supercompensation

The practice of carbohydrate loading has gained so much attention during the last decade that spaghetti has become a typical pre-marathon meal. Endurance athletes commonly use this technique to attempt to improve their performance. Not only have many had success, but there has been a good deal of scientific support of this practice (2, 8, 18, 21). This practice involves manipulating diet by eating more carbohydrates to increase muscle glycogen which is an important fuel used during prolonged strenuous activity. Raising the pre-race level of glycogen is important because glycogen is not present in inexhaustible supplies within the body. The significance is that this better enables an athlete to maintain his or her optimal pace throughout the race (2, 18, 21).

The "classic" regimen for carbohydrate loading, (glycogen supercompensation), first described in the late 1960's, consists of the following. One week prior to the marathon muscle glycogen is depleted by exercising to exhaustion. During the next three days a high fat and protein diet with low carbohydrate consumption is used. Then for the three days preceding the event a high carbohydrate diet is eaten. This technique has been shown to result in a higher level of stored muscle glycogen than does a continuous diet high in carbohydrates (2, 18).

Research on glycogen depletion has suggested that "hitting the wall" at the 18 to 20 mile mark in a marathon is the point where glycogen stores have been depleted by the athlete and he must then depend on metabolizing more fat for energy. The athlete becomes less efficient, and thus he slows down significantly (21, 23, 32). Glycogen loading does not enable the runner to run faster (33), but it improves performance in that it delays glycogen depletion and the phenomenon of "hitting the wall" can be somewhat avoided (32). Though numerous studies have demonstrated undeniably that we can increase our muscle glycogen stores and prolong a high level of endurance work, the question remains as to how much stored glycogen is necessary. Why should a marathon runner load himself with enough glycogen to carry him through 4 hours when he may complete the marathon in less than 3 hours? Perhaps the classic glycogen supercompensation technique is more limited than initially thought. This diet has not been shown to significantly improve endurance performance during competition as compared to a simple

high carbohydrate diet (9, 33). Thus carbohydrate loading can be used in a less extreme regimen and still increase glycogen stores and increase endurance performance in marathon athletes (23).

The practice of carbohydrate loading has produced reason for concern as a potentially dangerous practice. Based upon individual case reports, concern has been raised that it may cause cardiac arrhythmias (22), raise serum triglycerides (13), and destroy muscle fibers by excessive storage of glycogen (18). A less severe consequence is that a sensation of heaviness and stiffness may be produced because of increased deposition (27). This is because an increased deposition of glycogen results in an increase in the amount of water in the muscle as well.

Though there may be some risk, increasing carbohydrate consumption during prolonged training periods and before endurance events is recommended. It is one of very few methods of how a thorough understanding of nutrition and its scientific implications can, in fact, be of applied significance in the area of improving performance. Because of this fact, there will be continued investigation into nutritional considerations for the endurance athlete.

Summary

For as long as man has competed he has searched for techniques to improve performance. Much of this searching has been done in the area of nutrition. This is an area where fads, superstitions, and non-scientific beliefs are, and most likely always will be, abundant. Therefore it is of great importance that sound, scientifically investigated information be conveyed to the athlete.

There is much present day evidence that encourages an athlete to maintain a well balanced diet (55% carbohydrate, 30% fats, and 15% protein) without supplementation. For the endurance athlete much has been made of the fact that nutrition can improve performance. This notion has been investigated and will continue to be investigated. There is little question that adequate hydration and eating a high carbohydrate diet will improve endurance performance in the sense that it will prevent dehydration and help delay glycogen depletion.

The athlete must still understand that there is nothing magical about improving performance. Achieving maximal performance always has and always will be a function of the best application of scientific principles and hard work.

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Marijuana and Its Effect On the Athlete

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How many times has the reader heard, "there is no valid medical evidence that marijuana is harmful to your health." Or, "there is no valid medical evidence that tobacco is harmful to your health." This last statement is perpetuated by the Tobacco Institute, the public relations arm of the tobacco industry. Questions that the reader should be asking when hearing these statements are: (1 Who keeps saying that marijuana is not harmful to your health? (2 Who would profit if it were possible to sell marijuana cigarettes over the counter? (3 Who finances the research to show that tobacco is not harmful to your health? and (4 Who finances the research to show that marijuana is not harmful to your health?

The knowledge of the health-related effects of marijuana is important and timely because marijuana is now the most widely abused of all the illicit drugs available in the United States. Substantially more high school students abuse marijuana on a daily basis than alcohol. Much of the heavy abuse of marijuana, unlike alcohol, takes place in school, where the effects on behavior, cognition, and psychomotor (athletic) performance can be particularly disturbing (101). Unlike alcohol, which is rapidly metabolized and eliminated from the body, the psychoactive components of marijuana may cause tolerance and dependence. It is imperative that the reader have reliable and detailed information about the effects of marijuana.

Chemistry and Pharmacology of Marijuana Definitions

1. **CANNABIDIOD** (CND) and
2. **CANNABINOL** (CBN) a major cannabinoids generally present in cannabis.
3. **CANNABINOIDS** are a class of 21-carbon compounds present in cannabis sativa. The basic structure contains a six-member hydroaromatic ring and a benzene ring joined by a pyran moiety. Derivatives include a number of carboxylic acids, their analogues, and transformation products, Cannabinoid is a generic term for a class of compounds.
4. **CANNABIS** is a general term for any of the various preparations of the plant.
5. **CANNABIS SATIVA** and the cannabinoids obtained from it. Cannabis sativa, also called hemp, is a herbaceous annual plant that readily grows in temperate climates. Depending upon the geographical region, and other considerations, the various natural preparations of cannabis possess different physical characteristics and concentrations of cannabinoids. Cannabis preparations may contain over 420 different compounds, of these 61 have been identified as cannabinoids, many of which possess some biological activity. Marijuana, hashish, and tetrahydro- cannabinols are examples of different forms or components of cannabis.
6. **HASHISH** is a resin generally more potent than marijuana, which is obtained from cannabis sativa by shaking, pressing, or scraping the leaves and flowers of the plant and usually contains some of the latter.
7. **MARIJUANA** is a general term for the crude preparations obtained from the plant cannabis sativa and is a mixture of crushed leaves, twigs, seeds, and sometimes the flowers of the plant. In the United States, the term marijuana has often been used interchangeably with cannabis to refer to any part of the plant or extract therefrom, of the synthetic cannabinoids that induce somatic or psychic changes in the man.
8. **SINSEMILLA** is a seedless variety of high-potency marijuana, originally grown in California.
9. **TETRAHYDROCANNABINOL** (THC) is one of the major groups of cannabinoids. Delta-9-THC is the principal active constituent in natural cannabis preparations. Delta-9-THC is also known as Delta-1-THC by the International Classification System. In the United States, the Delta-9-THC content of marijuana ranges from immeasurable amounts to about six percent. Another active isomer, Delta-8-THC, is less often present in marijuana and typically occurs in minute amounts. Many derivatives of Delta-9-THC have been synthesized (101).

Cannabis, the crude material from the plant Cannabis Sativa, contains hundreds of chemicals. Most of these are found in other plants, but 61 chemicals termed cannabinoids, are unique to the Cannabis plant. Natural and most synthetic cannabinoids are relatively insoluble

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in water, but dissolve in fats and fat solvents and are, therefore, called lipid soluble (101).

A single cannabinoid, Delta-9-Tetrahydrocannabinol (Delta-9-THC), produces almost all of the characteristic specific pharmacological effects of the complex, crude cannabis mixtures. A number of synthetic cannabinoids have pharmacological effects similar to Delta-9-THC. Other cannabinoids in the plant, for example, cannabinol, are almost inactive pharmacologically or may interact with Delta-9-THC to modify its actions. One cannabinoid, cannabidiol (CBD), can influence the metabolism of another, Delta-9-THC (114). A few cannabinoids have effects quite different from Delta-9-THC. For example, cannabidiol has relatively little psychoactive and cardiovascular effect, but is an active anticonvulsant(71).

Investigators have chemically altered the Delta-9-THC molecule in an attempt to determine which of its structural elements are required to produce behavioral or other effects. Studies of structure-activity relationships indictate that, to produce effects on behavior, a pyran ring must be part of the three-ring system, a free phenolic hydroxyl on the aromatic ring, and a lipophilic side chain (C_5H_{11}). Understanding chemical structure-effect relationships is important to guide the synthesis of cannabinoids with differing pharmacological effects. Different effects of Delta-9-THC activity by chemical design will require further syntheses and pharmacological study of a large number of cannabinoids (85).

It is impossible to understand the effects of cannabis without quantitive control of the composition and the amount of the active substances, that is, control over the dose. Systematic pharmacology must, therefore, be performed using pure compounds. In the United States, cannabis is usually smoked, which complicates the pharmacology.

The smoke from any burning plant contains hundreds of chemicals that may have biological effects. This poses a dilema for researchers because consequences of smoking cannabis cannot be fully determined by studies only of pure cannabinoids. Studies are also needed with doses of Delta-9-THC delivered, however imperfectly, by smoking.

Drug Interactions

Because cannabis is often consumed with other drugs, interactions can be expected. Other illicit drugs, tobacco, caffeine, alcohol, and OTC or prescribed medications should be considered in combination with cannabis because Delta-9-THC and its first metabolite are strongly bound to proteins in the plasma (42) and may interact with other drugs similarly bound. Cannabis and many other drugs share disposition by the hepatic metabolic enzyme systems, and there are possible interactions at the drug metabolism level. For example, drugs such as alcohol or pentobarbital can inhibit metabolism of Delta-9-THC by enzyme substrate competition. Or, after a period of inhibition one drug is removed, the enzyme activity can increase so that faster than expected metabolism follows. If given simultaneously with other drugs, Delta-9-THC can slow metabolism of drugs such as theophyllin, antipyrine, ethanol, and pentobarbitol. Cannabidiol can also inhibit metabolism of a variety of drugs normally metabolized by the shared hepatic enzyme systems (13, 70, 129).

Drug interactions also can occur by means of functional mechanisms. These can be **additive**, resulting enhancement or prolongation of behavioral and psychological effects by cannabis when combined with other central nervous system depressent drugs, such as alcohol and barbiturates. Animals less tolerant to cannabis will be less sensitive to other central nervous system depressants. This phenomenon is known as cross-tolerance (129).

Cannabis Contaminants

There is no question that large doses of paraquat by mouth or by aerosol can cause pulmonary fibrosis. Outbreaks of salmonellosis epidemiologically linked to marijuana were reported in Ohio and Michigan. Marijuana was found to be contaminated with salmonella which caused diarrhea, fever, and abdominal pain (107).

Aspergillus, a fungus, is a common contaminant of some cannabis. The spores pass easily through contaminated marijuana cigarettes, and when smoked, enter the body (78, 79).

Marijuana confiscated on the Indiana University campus was found to be adulterated with horse manure. Marijuana confiscated in New Foundland was found to be adulterated with turkey manure (82).

Abuse of Marijuana in the United States

There has been a steep rise in the abuse (129) of marijuana and other illicit drugs in the past decade. So far it is primarily a youth phenomenon. Since 1971 there has been at least a doubling of lifetime experience with marijuana in every cohort in the 12 to 24-year age group. Of all psychoactive drugs investigated (including inhalents, other hallucinogens, cocaine, heroin, stimulants, sedatives, and tranquilizers), marijuana is by far the most commonly abused illicit drug. Legal drugs for adults, such as alcohol and tobacco, are the most widely abused of all drugs among adolescents. Although substantially more students have abused alcohol in their lifetime than have abused marijuana, more high school seniors abuse marijuana on a daily basis (9 percent) than abuse alcohol daily (6 percent). Daily abusers report the abuse of marijuana in school, whereas daily abuse of alcohol tends to occur after school and on weekends (7, 28, 29, 64, 66, 67).

Some trends in the abuse of marijuana are apparent. The continuing dramatic rise in the abuse of marijuana has recently slowed. It is too early to tell whether this decrease will continue or is merely a pause in the rise. The overall prevalence of abuse of marijuana has remained at approximately 60 percent of high school seniors for the years 1978, 1979, and 1980. Between 1975 and 1978 there was an almost two-fold increase in daily abuse of marijuana from 6 percent in 1975 to a peak of 11 percent in 1978. In 1980 the daily abuse rate of high school seniors dropped by 1.2 percentage points, or more than 10 percent. This may signal a reversal of the upward trend in daily abuse unless higher absenteeism and school drop-out of daily abusers are significant factors in the decline. Multiple sources suggest that out-of-school age mates are heavier abusers than those in school. Other trends have not slowed. There was a continuing rise in 1980 of the proportion of high school seniors who during the year had abused some illicit drug other than marijuana, from 28 percent in 1979 to 30 percent in 1980 (101).

Function and Performance

Throughout the 1970's, as a correlate of continuing rise in prevalence rates, there was a trend toward younger ages of first abuse of all these drugs. For marijuana this age trend continues, but has slowed somewhat. In 1979, 23% of seniors who had abused marijuana

started their abuse in the eighth grade or below as compared to 25 percent in 1980 (101).

Effects of Marijuana on Pulmonary Function and Performance

The lungs are the natural target for the harmful effects of smoked materials. This is as true for marijuana as for tobacco. In both instances, smoke is drawn into the lungs where it can harm not only the cells that line the airways (trachea, nasopharynx, bronchii, and alveoli) and constitute the lung tissue, but also impair such cells as lung macrophages, which are part of the immune system. As a result, the smoke may inflict injury directly on parts of the system and also make the lungs vulnerable to agents that normally are held at bay by self-cleaning and self-protective mechanisms (128).

Cigarette smoking is medically considered to be the major source of preventable lung cancer. Since the smoke of the marijuana joint is held in the lungs longer than the smoke of the tobacco cigarette, Vogel extrapolates that one marijuana joint has the cancer inducing capability of 20 cigarettes (126).

Marijuana affects the control of the breathing pattern in different ways depending on the dose, the preparation, and its psychotropic effect on the abuser. One marijuana cigarette generally stimulates ventilation (air exchange between the lungs and the ambient air) in conjunction with an increase in the metabolic rate and a heightened response to carbon dioxide as a regulatory stimulant (124, 131). On the other hand, larger doses of smoked marijuana depress the ventilation and responsiveness to carbon dioxide stimulus (11, 127). Heavy marijuana smoking, at least four days per week for six to eight weeks, causes mild airway obstruction as demonstrated by conventional tests (119).

A study of 31 American soldiers stationed in West Germany who smoked large quantities of hashish (100 grams or more per month for periods of 6 to 15 months), found their ailments to be principally respiratory, including bronchitis, sinusitis, asthma, and rhinopharyngitis. In one third of the soldiers, sputum producing coughs, difficulty in breathing, and wheezing followed three to four months of regular abuse of hashish. Antibiotics failed to relieve the symptoms. The symptomatic soldiers could not work and four required hospitalization (121).

Tashkin, et al, (120) studied 74 people who had smoked marijuana for two to five years at the rate of three to six times per week. The results indicated that habitual smoking of marijuana causes a significant increase in resistance to airflow in large airways without appreciable effect on conventional tests.

Rats (38) and dogs (103) have been exposed to marijuana smoke for one year to 900 days to determine marijuana's morphological effects on the lungs. At autopsy, the animals demonstrated damage to the bronchial tubes and the lung tissue itself.

Effects of Marijuana on the Cardiovascular System

With respect to the heart and circulation, the most evident effect in human beings of smoking marijuana, or of injesting Delta-9-THC, is tachycardia (16). This rapid heart action can be harmful to the heart in which the circulation is comprised by atherosclerosis or is on the verge of failing (101). It is not uncommon for a patient to have a heart attack immediately after having a "normal" EKG. Therefore, the athlete may not really know what the true condition of his heart is if he starts abusing marijuana. Marijuana does cause EKG changes (65).

While the individual is reclining, blood pressure increases with the administration of Delta-9-THC. This has been attributed to altered autonomic function (4.43, 63). However, when the individual stands up the blood pressure may drop considerably (83). Thus, the athlete's body would not function properly when changing positions rapidly.

Exposure to Delta-9-THC affects exercise performance of athletes. Smoking 20 mg of Delta-9-THC decreased the duration of exercise, but still increased the heart rate (110). A decrease in oxygen carrying capacity of blood because of the formation of carboxhemoglobin has been shown by Aronow and Cassidy (5).

Effects of Marijuana on the Brain

Marijuana causes a temporary intoxification (128) and results in changes similar to those caused by other intoxificating drugs.

Evidence that the abuse of marijuana causes brain atrophy were obtained by pneumoencephalography on 10 patients. The size of the ventricles of the brain were measured to determine atrophy of the brain tissue. All 10 patients showed atrophy present. The usual incidence of cerebral atrophy is 1 in 100,000 subjects (17).

Three post mortem studies on monkeys have shown changes in the microscopic morphology of the brain at the ultrastructural level (50, 52, 93). Changes reported were alterations in synaptic cleft width, increased density of synaptic cleft material, a decrease in volume of rough endoplasmic reticulum, presence of clumping of synaptic vesicles in axon terminals, and an increase in intranuclear inclusions.

There are reports that epileptic seizures may be induced in known epileptics when they abuse marijuana. Studies are being done with rabbits to see if this is inherited (33, 34, 76 86).

A number of studies have been done on the effects of marijuana on neurotransmitters in the brain. The most clear cut effects have been on acetylcholine turnover, a measure of the level of activity of neurons producing this chemical. Delta-9-THC causes a reduction in acetylcholine turnover in the hippocampus (28, 29, 102) and this results from reduced activity of the acetylcholine neurons. It is noteworthy that the effect is produced by small doses and only by cannabinoids. Administration of physostigmine, a drug that enhances acetylcholine action by partially blocking its breakdown, to five healthy human volunteers produced enhancement of the lethargy and somnolence occurring late in the course of the Delta-9-THC intoxification (39). The results of this study and others (27, 28, 39, 80) have led to the conclusion that Delta-9-THC acts to inhibit acetylcholine nerve cell networks. This action may be related to the memory deficits produced (29). There have been studies on several other neurotransmitters in the brain including catecholamines, serotonin, and gamma aminobutyric acid (9, 15).

Male Reproductive Function

A group of 20 men were studied who had abused marijuana at least four days a week for a minimum of six months without the abuse of other drugs. Plasma testosterone levels in subjects who smoked five to nine marijuana joints per week were significantly lower than their controls. All but one of the men smoking more than 10 marijuana joints per week had testosterone levels below 400 mg/dl (the normal level). In addition, these men had lower sperm counts (26 million versus 68 million/ml) (21, 53, 74). Issidorides (62) showed that the head of the

sperm. the Acrosome, was abnormal in marijuana smokers.

Marijuana also has been demonstrated to be estrogenic (like female sex hormones) **in vivo** and reports indicate that the effects may be medicated via the estrogen receptor. Harmon, et al, (49) report on gynacomastia in male marijuana abusers. Gynacomastia is the excessive development of the male mammary glands, even to the point of becoming functional. The authors speculate that a metabolite of Delta-9-THC is similar to estradiol.

Female Reproductive System

There is no conclusive evidence at the present time regarding the effects of marijuana on the female reproductive system.

Birth Defects and Teratogenicity

Because Delta-9-THC crosses the placenta it is a potential teratogen, an agent that causes defects in the developing embryo. This effect could occur in either of two ways: (1) exposure to cannabis prior to conception could harm the ova and sperm, or (2) the fetus could be harmed directly during organogenesis (the development or growth of the fetus) (35, 40, 59, 101).

The potential genetic effects of marijuana are of major concern because of its prevalent abuse by young people in their reproductive years. In a retrospective study of college students by Stenchever (116) chromosome breaks were found in blood cultures of 49 light (one or less exposures per week) and heavy (more than two exposures per week) abusers of marijuana.

Studies of lymphocytes cultured from human marijuana smokers defined either as moderate abusers (at least one marijuana joint per week, range 1-10 for a minimum of two years) or heavy abusers (more than three times per week) turned up a significantly larger number of cells with less than 30 chomosomes (48 is normal) than would be found in control cultures (90).

Body Temperature

In a hot environment (40C.) marijuana caused inhibition of sweating and a consistent rise in body temperature (60). During physical activity the athlete's body would not be able to cool itself, thus, possibly leading to heat stress syndrome.

Behavioral and Psychological Effects

Marijuana seriously impairs psychomotor function in the areas of:

1. Coordination as examined by hand steadiness, body sway and accuracy of execution of movement.
2. Tracking performance.
3. Perceptual tasks.
4. Vigilance.
5. Performance on automobile driving and flying simulators.
6. Operating automobiles on test roadways.

Simpler and well-practiced skills are less susceptible to disruption by the effects of marijuana than are novel or complex tasks.

The studies reported here cover the range of commonly abused doses from very low up to 0.250 mg/kg of Delta-9-THC in marijuana cigarettes at a single sitting. These are acute effects, that is, changes which can be seen after a single dose. The effects begin to be seen at about the same dose level at which a "high" is perceived (0.050-0.150 mg/kg of Delta-9-THC). Certainly the effects are dose related (161).

Marijuana has been found to impair motor coordination at doses commonly abused in social settings by both naive and chronic abusers. The functions studied include: hand steadiness (20, 89) and body sway (35, 72, 89). Studies have also shown a dose-related increase in impairment of posture stability as measured by increased body sway (72).

Most studies examine reaction time as the time it takes the subject to respond to a visual or auditory signal. The effects of marijuana on either speed of initial detection of the signal or speed of response have been used. When a subject has been intoxificated with marijuana he is less likely to attend to the reaction time task (14, 20, 30, 91, 92, 95, 106, 117).

Tests that measure a subject's ability to detect a brief flash or light show significant impairment by low to moderate doses (2-3mg) of smoked marijuana. Sustained attention is required in signal detection tasks. The large reduction in signal detection that occurs under the influence of marijuana suggests a substantial risk for abusers who are operating machines (18, 68, 91, 92, 111, 112, 113).

Several studies have demonstrated that a single moderate dose of marijuana impairs short-term memory. This effect is especially noticeable in the phases of short term memory that are heavily dependent on attention, such as information acquisition and storage (1, 2, 12, 27, 20, 87). Examples of the types of impaired tasks would be remembering a sequence of directions. What about an athlete trying to learn plays, audibles, or assignments while under the influence of marijuana? Miller and Cornett (88) found that increases in heart rate are produced by marijuana to about the same degree as impairment on intellectual tasks.

Another intellectual function influenced by marijuana is time sense. Under the influence of moderate doses of the drug, most investigators report that subjects consistently overestimate the amount of time that has elapsed. Thus, under the influence of marijuana, a given event is reported to last longer than it actually does (20, 122, 125).

State-dependent learning refers to a situation in which material that is learned while the subject is under the influence of a drug is remembered best in the state of drug intoxification in which it was originally learned. A series of studies have been conducted with oral doses of 20 mg. (0.3 mg/kg) of Delta-9-THC to investigate the extent to which learning and memory are linked to the state of intoxification. This modest dose of marijuana caused learning to take place more slowly than when the subject was drug free. Once learned, recall of learning that occurred during intoxification was best when the subject was again under the influence of marijuana. Although state-dependent learning occurs with marijuana, the quality of learning and recall is impaired because the information of problem-solving skills learned in the marijuana intoxification state will be reduced or impaired, The major deficit is in the attention, storage phase of learning (22, 23, 24, 25).

Studies of the effects of marijuana on closed course automobile driving performance show that this skill is impaired by marijuana. Car handling skills were reduced. It should be noted that these studies, involving subjects under the influence of marijuana, examined performance in less complex situations than are actually met in real-life driving situations. However, a closed course has the advantages of standard conditions and safety factors. In real life driving situations, the perceptual and cognitive demands are considerably more com-

plex (6, 48, 73). The Klonoff (73) study of driving performance on city streets indicates that smoked marijuana (5-10 mg Delta-9;THC) impairs judgement and concentration in addition to impairing car handling skills. The time that it takes to apply the brakes of the automobile, braking time, is 22-63 percent longer under the influence of marijuana than it is under the influence of alcohol (128).

Surveys show that marijuana and alcohol are frequently consumed together. (37, 64). Thus, it is important to determine the interaction between these two drugs. As both drugs have sedative properties, an additive effect would be expected and has been found. Mann, et al, (84) found that 0.05 percent blood alcohol level contration (BAC) increased the impairment produced by 5 mg. of smoked Delta-9-THC on tracking behavior. Hansteen, et al, (48) used two doses of alcohol and two doses of marijuana. Even the low dose of alcohol (0.07 BAC) and the low dose of Delta-9-THC (1.4 mg) impaired complex tracking in an additive effect on the ability to perform on a psychomotor test. This additive effect would be of concern to those operating a motor vehicle (10).

Amotivational Syndrome

Clinicians coined the term "amotivational syndrome" to describe a characteristic set of personality changes seen in daily marijuana abusers. The changes include apathy, loss of ambition, loss of effectiveness, diminished ability to carry out long-term plans, difficulty in concentrating, a decline in school work, a decline in athletic performance, failing grades, difficulties in concentration, intermittent confusion, and impaired memory. As usually described, these changes are seen in frequent or daily abusers, and thus they must be considered a form of chronic intoxificaton (64, 115).

Baker and Lucas (8) describe the case of a man whom friends describe as previously conscientious, capable and effective. But after smoking marijuana daily for three years, he changed into an individual for whom the abuse of marijuana was a way of life and in whom a serious deterioration of social function was observed. Other reports consist of groups of cases with similar histories (113).

A variety of other data support such a condition. In a large survey Johnson, et al, (64) asked daily marijuana abusers about adverse effects. The most common answer was "loss of energy", 42 percent. Nearly a third (32 percent) thought that marijuana caused them to be less interested in other activities than they had been before, and 34 percent thought that it hurt their job and/or work performance. Several studies (36, 77, 114) found marijuana abusers had increased levels of psychological disturbances, lower academic performance, and lower performance on scales measuring attitudes toward achievement and purpose in life.

Therapeutic Potential and Medical Uses of Marijuana

There has been growing reinterest in the possibility that cannabis and its derivatives will be valuable for the treatment of several medical and psychiatric conditions. The 97th Congress introduced H.R. 4498 "to provide for the therapeutic **use** of marijuana in situations involving life-threatening or sense-threatening illness and to provide adequate supplies of marijuana for such use" (101).

Cannabis (the crude drug), Delta-9-THC (the pure compound), and some other cannabinoid derivatives lower intraocular pressure when administered by various routes, such as inhalation, oral, or intravenous. However, adverse side effects of cannibis and Delta-9-THC have been reported. Most patients with glaucoma are elderly, and have reduced tolerance for many of the side effects. Even without the adverse side effects, smoking, oral, and intravenous routes of administration are not suitable for long term. For example, to have adequate control for intraocular pressure, four marijuana cigarettes per day of two percent Delta-9-THC would be necessary. This amount is considered heavy usage and could pose a serious health problem in long-term use. Therefore, topical application would be the most salutary route of administration for the patient who needs continuous treatment (44, 45, 46, 47, 55, 56, 61, 96, 99, 101, 109).

The suggestion is that cannabis might have some useful antiemetic effect. Interest arose in 1973 when cancer patients receiving intensive chemotherapy observed that their social abuse of cannabis appeared to reduce their customary nausea and vomiting.

Several controlled studies have been reported. The antiemetic effect of Delta-9-THC against those chemotherapeutic agents that are moderate in their effects is pronounced (e.g. Methotrexate), but that Delta-9-THC is less effective against those agents with severe emetic properties (e.g. Cisplatin) (19, 26, 31, 41, 54, 81, 104, 105).

There are widespread anecdotal reports that cannabis is effective in relieving muscle spasm of spasticity. Petro (97) has reported such effects and has carried out a double-blind study of the administration of Delta-9-THC on spasticity. Petro and Ellenberger (98) reported that 10 mg. of Delta-9-THC significantly reduced spasticity by clinical measurement and that the quadriceps electromyograms demonstrated a decrease in interference patterns in patients with primarily extensor spasticity.

Marijuana has not been shown to be an effective medication in asthma (60, 118), anxiety (32, 94, 100), depression (75, 100), pain (58, 100), alcoholism (103), or tumors (51, 130).

Legalization

There is a cry from some people to legalize marijuana. The international agreement entitled "Single Convention on Narcotic Drugs" was written in 1961 and signed by the United States in 1967. Article 4 requires the signature parties to "take such legislative and administrative measures as may be necessary . . . to limit exclusively to medical and scientific purposes the production, manufacture, export, import, distribution of, trade in, use, and possession of drugs." Lawyers are still arguing the question of legalization (108).

Conclusions

Marijuana is not the safe, harmless, or innocuous substance that some people are lead to believe. If an athlete abuses marijuana and suffers the harmful anatomical, physiological, and psychological effects described in this report, there is no way that the athlete's performance will not be affected.

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Case Report

Compression of the Lateral Femoral Cutaneous Nerve: Meralgia Paresthetica

Joseph J. Kolb

Hip and groin injuries have varying degrees of occurence depending on the respective participating sport. Trauma can include contusions, bursitis, strains, stress fractures or referred pain from lumbar spine pathology.

The athletic trainer or clinician must be aware and alert to the fact that an unsuspected mechanism of injury and related pathology should always be considered. This is especially important because the groin/hip complex is the pathway for the nerves of the lower extremities emanating from the lumbar and sacral regions.

It is because of this fact that the evaluator should never compromise a complete evaluation and differential diagnosis.

Evaluation

This report deals with a 19 year old college junior playing his third season as a defensive back in an NCAA Division III football program.

Two days after receiving his pads and gear the athlete reported a sharp pain in the right groin region to the athletic training staff.

On examination there were no visible signs of edema or inflammation. Slight point tenderness was elicited in the anterior groin. Passive range of motion tests were negative. During manual resistive muscle testing through the joint range of motion, the only positive test was that in hip flexion with the knee extended.

Initially the diagnosis was thought to be an iliopsoas strain until the athlete also reported a slight numbness over the anterolateral aspect of the thigh.

It was at this point that the athlete was referred to the team orthopaedist. In addition to the structural evaluation, the physician also conducted a cutaneous sensory test. The results on the pin prick and brushing tests showed a slightly desensitized area over the L2 and L3 areas of the thigh.

Upon further interview, the physician was informed by the athlete that upon applying his contact pads and uniform, he habitually "extra" tightened his pant belt. With this pressure in addition to the hip pad girdle, the physician diagnosed the problem as Meralgia Paresthetica involving a compression of the Lateral Femoral Cutaneous Nerve.

Anatomy and Pathomechanics

Anatomically, the Lateral Femoral Cutaneous Nerve, which is from L2 and L3, supplies cutaneous sensory function to the anterolateral half of the thigh from the buttocks to the knee (2).

As the nerve comes from within the pelvis, it courses medially from the anterior superior iliac spine and moves beneath the lateral end of the inguinal ligament (1,5).

Little was reported in the literature of a single direct traumatic episode as being a prime mechanism of injury (1,2,3,4,5,6,7). The references did include reports on the following as causes of Meralgia Paresthetica: obesity, sudden weight loss rendering the nerve more superficial than the patient is accustomed to, increased demand of the abdominal muscles, exercising the lower extremities after inactivity and tight undergarments (1,2,3,4,5,6,7).

In this entrapment neuropathy of this nerve behind the inguinal ligament, the common signs include pain or numbness, hyalgesia to pin prick and dusting tests, point tenderness can be elicited where the nerve emerges into the thigh below the inguinal ligament (1,2,3,4,5,6,7). Hip extension has also been reported to cause pain (5) but was negative in this case. It can be hypothesized that the pain on flexion was elicited by the contraction of the surrounding muscles which might have contributed to the impingement.

Treatment

The athlete was informed to avoid excessive tightening of his belts. Ice was applied for two days and the athlete was permitted to practice within pain tolerance.

One week after the evaluation, the subsequent adjustments were made and the athlete became asymptomatic.

Conclusion

It is evident that had it not been for a complete evaluation the athlete would have continued to tighten his pants while being frustrated with a never-healing groin strain.

Additional considerations in preventing such a condition would be to remain aware of the proper fitting of the athlete's equipment and its relationship to his/her weight and size.

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Mr. Kolb is currently Executive Director of the East Coast Athletic Safety Council located in New York.

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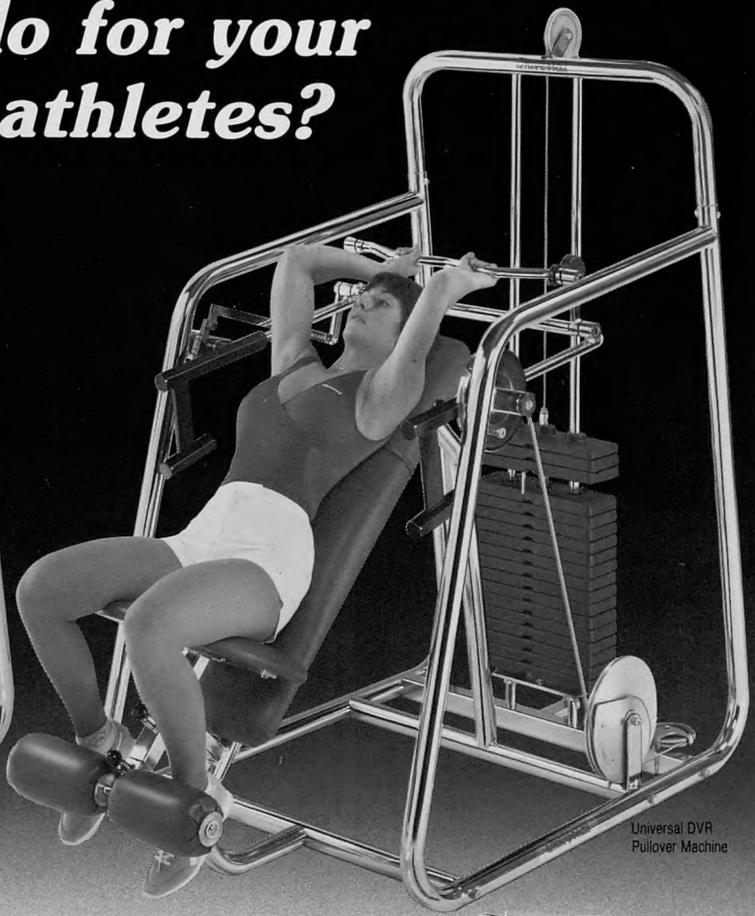

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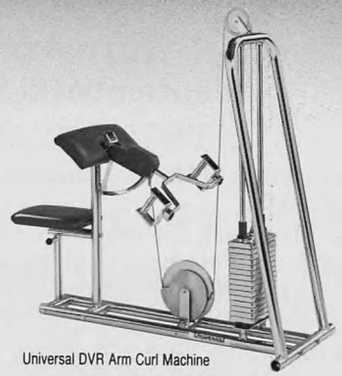

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In Memoriam

Edward "Ed" Block
September 3, 1913 - May 9, 1983

Ed Block, trainer emeritus of the Baltimore Colts and the most beloved person ever associated with the team, died May 9, 1983.

Born in St. Louis, Missouri, he received his B.S. and M.A. from the University of Missouri, a doctorate of education from Teachers College, Columbia University and became a registered physical therapist through Columbia University Medical Center in New York. Ed was once asked why he had not become a medical physician. His reply: "Doctors treat the dying, I wanted to treat the living."

As a tank commander under General George Patton, Ed was decorated with a purple Heart during his military career (1941-1946).

Ed returned to St. Louis where he was a faculty member and head trainer under then head Coach Weeb Ewbank at Washington University. In 1954, Ewbank retained Ed as trainer with the Baltimore Colts. Of Ed's death, Weeb Ewbank tributed Ed by writing "Ed was the type of man our world needs more of, and he may be equaled as a great trainer, but not exceeded."

During the off season while with the Colts Ed worked with Kernan's Children Hospital and Johns Hopkins Hospital in Baltimore.

For seven years he contributed his knowledge to the in-space conditioning program with N.A.S.A..

A heart attack before the 1977 season forced Ed into semi-retirement, but to hundreds of players whose bumps and bruises passed under his scrutiny, he will always be remembered.

Ed was a "mother hen", "father confessor" and confidant. He ministered to the players' wants and pains with a sheltering wing of compassion far beyond the normal servitude of a trainer.

An admission Ed once made, "The real secret of training is something every mother knows. It's an observation thing. How did your mom know when you lied to her? It's the same thing in training — a feeling."

Honoring Ed, the Eastside Athletic Club inaugurated the Ed Block Courage Award in 1978. Acknowledging the tribute to Ed, the late Carroll Rosenbloom wrote, "When God gave you your wonderful talents as an athletic trainer, he decided to surpass them only with the capacities he accorded you for loyalty, courage and compassion. His final result, a great man!"

In Memoriam

Tasha E. Bolton
June 24, 1956 - May 30, 1983

Tasha Bolton, assistant athletic trainer at Northwestern University, died May 30, 1983 at University Hospital in Madison, Wisconsin.

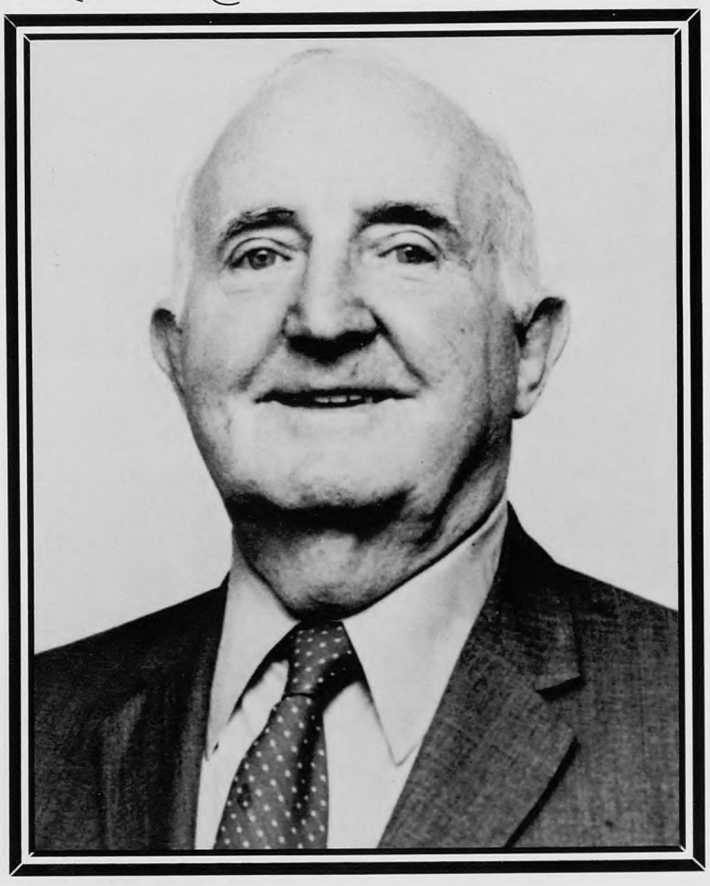
Tasha graduated from Beloit High School in Wisconsin after serving 13 months as an American Field Service student in Thailand. In 1979 she graduated with a degree in Physical Education from the University of Wisconsin - Madison. Tasha continued her education as a certified graduate assistant at Michigan State. She received her Masters degree in 1982.

Tasha was hired as an assistant athletic trainer at Northwestern following graduation. Shortly after this time she was stricken with leukemia.

Tasha is survived by her parents Carolyn and Clarence Bolton, sisters Cassandra and Karen, brother Edward and grandmothers Mrs. Doris Bolton and Mrs. Carrie Alexander.

Tasha Bolton was a young woman who will be remembered by all who knew her. Her energy and sense of humor seemed endless. She always enjoyed life to its fullest. The profession of athletic training suffered a loss when Tasha was taken too soon from us.

In Memoriam



Edward D. O'Donnell
October 7, 1900 - January 12, 1983

After a long illness Edward O'Donnell died early this year. He had been active in physical therapy and athletic training for more than half a century.

Edward O'Donnell was born in New Haven, Connecticut. He attended Roxbury school, attended Springfield College, received an honorary doctorate in physical education from Arnold College and studied at Johns Hopkins Childrens Hospital. Mr. O'Donnell worked the great majority of his career at Yale University. For 44 years he served as Chief of Physical Therapy and Rehabilitation. During his tenure at Yale he also served as Head Wrestling Coach for 17 years and trainer for the football program for 15 years. He left Yale University in 1970.

Edward O'Donnell was nationally known in his field. He authored a number of articles, and lectured on physical therapy and athletic injuries at Yale Medical School and the Hospital of St. Raphael.

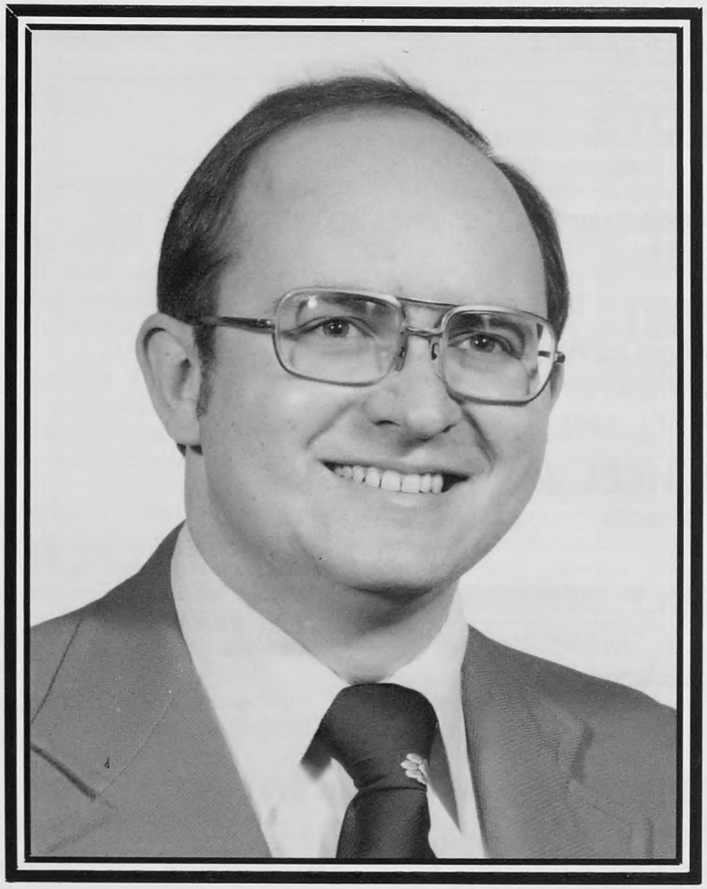
Among his many other accomplishments he served as olympic team trainer to the 1948 Olympics in London and was President of the Connecticut Board of Physical Therapy for 10 years.

Mr. O'Donnell is survived by his wife Harriet Maliff O'Donnell, two sisters, eight nieces and eight nephews.

Edward D. O'Donnell was a pioneer in the field of athletic training and injury care. Many in the profession today are building on his work.

For his contributions and efforts he will be greatly missed by all who knew him.

In Memoriam



Larry Sutton

February 2, 1952 - August 30, 1983

Larry Sutton, assistant trainer and basketball trainer at Clemson University, died August 30, 1983, in Emory Hospital after being hospitalized twelve weeks with pancreatitis and other complications.

Larry began his career in athletic training as a high school student at Bethel High School in Hampton, Virginia. Following graduation he attended East Tennessee State University where he majored in Health and Physical Education. Larry received his Bachelor of Science degree in 1975.

The following year he became a graduate assistant trainer at East Tennessee State University and began pursuit of a masters degree in instructional communication.

In September 1976 Larry Sutton joined the athletic staff of Clemson University. He served as an assistant trainer in all sports and basketball trainer until his untimely death. He was known as "The Healer" by the athletes and staff at Clemson.

Clemson basketball coach Bill Foster said, "Larry Sutton was as dedicated and competent an employee as Clemson University has ever had. His contributions to this department over the last seven years were immense." Fred Hoover, Head Athletic Trainer at Clemson, remarked, "As a working individual who gave so much of himself to Clemson, the NATA, and our District Association, Larry will be very difficult to replace. He was a fine human being and will be painfully missed by the entire training profession."

At the time of his death Larry was serving as Secretary-Treasurer for District 3 of the NATA.

He is survived by his parents, Mr. and Mrs. Carlton Sutton, of Yorktown, Virginia.

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Book Reviews

1982 Year Book of Sports Medicine
J.L. Anderson, F. George, L.J. Drakauer, R.J. Shephard, and J.S. Torg
Year Book Medical Publishers, Inc.
35 E. Wacker Dr., Chicago, IL 60601
1982
387 pp., illustrated
$39.95

The 1982 Year Book of Sports Medicine provides a brief summary of information gathered from over 500,000 possible sports medicine articles. Printed in numerous journals that originate from a wide variety of professional fields, each entry is a concise report on research articles published through January, 1982. While full credit is given to each article's original author and information on where the article first appeared is provided, each summary is initialed by one of the book's editors.

Sections of this book will not be of equal interest to everyone because of the wide variety of subjects covered. Chapters include exercise physiology, general medicine, biomechanics, sports injuries, pediatric sports medicine, women in sports, and athletic training. The book gives a fair representation of contrasting views which thoroughly cover the above topics. Written in an understandable style this text would be of special use to any trainer who wishes to supplement current knowledge with technical data. Illustrated with many graphs, charts, and tables, one should be familiar with statistical probability values, level of significances, and statistical tests to fully profit from the information provided.

The primary value of this book is that it provides a yearly collection of quality articles published in more than ninety-three journals. To subscribe to each individual journal would require a large subscription budget, extensive library space, and more time than is feasible with the time restraints associated with the profession of sports medicine. In short, ask not how much the book will cost but consider how much it will save you in time, money, and space - a rare advantage in any publication.

Stephen M. Forney, ATC

The Physician and Sports Medicine Guide to Running
Allan J. Ryan, MD
McGraw-Hill Book Company, New York
1980
95 pp., illustrated
$7.95

When a running program is begun, particularly by the novice untrained in sports medicine, many people wish to learn about the changes that will take place in their bodies, both physiologically and mentally. Dr. Ryan has written a well-informed, easy to understand synopsis of the effects of running and why. In these few short pages he acquaints the reader with a basic understanding and love for running.

Dr. Ryan begins by discussing some of the physiological evidence which explains "What Happens When You Run" (Ch. 2) and continues with a very good program for the beginning runner to follow (Ch. 3). His program stresses the gradual building up of tolerance and endurance and includes descriptions and illustrations of stretching exercises that are particularly helpful to the runner both as warm-up and as cool-down exercises.

Perhaps the best section of this work is the discussion of running equipment and the running environment. Here Dr. Ryan covers everything from headbands to a very informative section on the different types of running shoes and the advantages and disadvantages of each. Dr. Ryan explains the hazards and pitfalls of running surfaces, climatic conditions and diet in the runners' performance.

Unfortunately the brevity of the book, which proved so useful in the beginning chapters, is continued when Dr. Ryan explains many of the injuries common to runners. A particularly worthwhile aspect of this chapter, however, is that after each injury description he gives the reader an estimate of the recovery time and suggests several alternative activities that the runner can do to maintain fitness during the off time from running.

In the academic setting this book will be helpful as a recommended reading for a course in coaching track and cross country. Its main audience, however, is the beginning runner who wants to know the mechanical, physical and mental changes that occur when a running program is started. For the professional in sports medicine little advanced information will be gained and there is no new research. The effectiveness of the book lies in its ability to communicate difficult concepts and its overall upbeat and compact style. This makes it a good reference for the novice runner.

Paul Concialdi, ATC

Training and Conditioning of Athletes, 2nd Edition
Max M. Novich, MD and Buddy Taylor, MS, ATC
Lea and Febiger, Philadelphia, PA
1983
320 pp., illustrated
$16.50

In 1970, Dr. Max M. Novich and Certified Athletic Trainer Buddy Taylor, both pioneers in sports medicine and Hall of Famers in the World Boxing Hall of Fame and the NATA respectively, combined their multifaceted experience in athletics at all levels including professionals and wrote *"Training and Conditioning of Athletes"* which was published by Lea and Febiger. This was the first time that a famous orthopedic surgeon and his counterpart in athletic training, emphasized the preventative aspects of athletic injuries as well as the emergency and therapeutic care of the injured and ill athlete. The book has dozens of easily understood illustrations showing the anatomy of specific joints, musculature of specific areas, and illustrations showing taping for injured areas. The book was a best seller and sold out.

Now, Dr. Max Novich and Buddy Taylor have written a second edition which promises to be another best seller. It is still written in compact, informative step by step and down to earth readable English. The book provides a tremendous amount of information that is relevant to the athlete, trainer and physician.

The second edition clarifies, and updates prior information on common athletic injuries and disorders

continued on page 313

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MEMBERSHIP INFORMATION

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NATA Membership
P.O. Drawer 1865
Greenville, N.C. 27834

The membership year is from January through December, with no reduction of dues for a partial membership year. As part of your membership dues, you will receive a subscription to *ATHLETIC TRAINING*, The Journal of the National Athletic Trainers' Association, Inc.

CERTIFIED CODE 1

NO person may join the NATA as a Certified member. Those persons that are Student or Associate members and meet all other requirements, will be charged member's fee to take the exam, all others must pay non-member's fee to take the Certification examination.

ASSOCIATE CODE 2

A. Persons applying for Associate membership are individuals who are pursuing the profession of Athletic Training.
B. Qualifications for Membership:
   1. Have a bachelor's or advanced degree from an accredited college or university.
   2. Currently making progress toward the fulfillment of the requirements for NATA Certification under a and/or b as follows:
      a. Enrollment in one or more academic courses in an accredited college or university that are related to the fulfillment of the requirements for NATA Certification.
      b. Currently acquiring acceptable supervised athletic training work hours under the supervision of an NATA Certified Athletic Trainer. Such hours must conform to the description of the character of supervised work hours indicated in the requirements for Certification. (See acceptable hours guidelines.)

STUDENT CODE 4

A. Qualifications for Membership:
   1. Enrollment in one or more academic courses in an accredited college or university as an undergraduate student (Bachelor's degree not yet earned.)
   2. Making progress toward the fulfillment of the requirements for NATA Certification under a and/or b as follows:
      a. Enrollment in one or more academic courses, in an accredited college or university, that related to the fulfillment of the requirements for NATA Certification.
      b. Currently acquiring acceptable supervised athletic training work hours under the supervision of an NATA Certified Athletic Trainer. Such hours must conform to the description of the character of supervised work hours indicated in the requirements for Certification. (See acceptable hours guidelines.)

AFFILIATE CODE 5

A. This membership class is open to individuals who are interested in the relationship of Athletic Training to education, biological sciences, psychology, athletics or sports medicine.
B. Qualifications for membership:
   1. Bachelor's degree from an accredited college or university or certification in physical therapy.
   2. Professionally working in athletics, education, research, or medicine.

ADVISORY CODE 6

A. Qualifications for membership:
   1. Physicians (MD or DO) who are directly associated with a sports program and are providing medical care and advice to members of teams and advising the Athletic Trainer in regard to his/her duties.

ALLIED CODE 7

This membership class is open to individuals whose business interests or employment are related to athletics in general.

AFFILIATE-INTERNATIONAL CODE 10

This membership class is open only to persons who reside in an area not included in any NATA District. This is the only membership class open to such residents.

ACCEPTABLE HOURS GUIDELINES

(Student and Associate Members)

Please Note:

In order for hours to be credited for certification purposes, they must be attained under the direct supervision of an NATA Certified Athletic Trainer.

The hours must be worked in connection with programs by which the supervising certified athletic trainer is employed.

A. Acceptable Hours:
   1. Hours spent at organized team practices and contests (professional, collegiate, or interscholastic)
   2. Hours spent teaching or lecturing in athletic training*
   3. Hours spent in practicums and labs related to athletic training*

4. Hours spent working in sports medicine clinics or centers**
5. Hours spent in other allied health areas**

* Verification from the department chairperson and supervising certified athletic trainer must state amount of hours spent, course content and mode of measurement

** In order to receive credit for hours spent in sports medicine clinics or centers, please request that the establishment send for the NATA Athletic Training/Sports Medicine Clinical Affiliation application. Please note: No hours will be accepted by applicants from centers that were not first approved by the Board of Certification.

B. Non-Acceptable Hours:
1. Hours spent in a program not supervised or directed by an NATA Certified Athletic Trainer
2. Time spent travelling with teams
3. Academic hours
4. Hours spent in sports medicine clinics or centers that are not first approved by the NATA Board of Certification. +

ABSTRACTS *from page 285*

appearance, and anatomic attitude are quite variable. The most common of these is the infrapatellar plica, or ligamentum mucosum, which runs parallel to the anterior cruciate ligament. The second most common plica is the suprapatellar and commonly acts as a tethering band in the superior portion of the quadriceps bursa and may separate the quadriceps bursa into two separate segments. The medial patellar plica is the least commonly occurring plica. When fully developed this band runs distally along the medial sidewall of the knee joint from the level of the superior pole of the patella to insert into the fat pad. By virtue of its location it lies flat against the medial femoral condyle when the knee is flexed and is normally asymptomatic. As the knee is passively flexed from 30° to 60°, the medial patellar plica can be seen arthroscopically to glide over the medial femoral condyle, beneath the patella while external rotation of the tibia causes tight wedging of the medial patellar plica between the medial facets of the patellar plica causing vulnerability to direct blows. Following blunt trauma, localized synovitis and edema may occur, followed by progressive fibrosis and loss of elasticity producing an unyielding structure which may be painful and cause chondromalacia of either the medial facet of the patella or the medial femoral condyle. Symptoms from a pathological medial patellar plica are most commonly seen in the adolescent, but can be seen at any age. Pain is the primary complaint, localized medial to the patella, commonly one finger breadth proximal to the inferior pole of the patella, and along the medial fat pad at the insertion of the plica. The medial patellar plica is palpable adjacent to the medial aspect of the patella, and pressure over this area should reproduce the patient's symptoms. Medial patella plica pain is increased with repetitive activities and is commonly aggravated by a quadriceps strengthening program, but will subside with rest. The initial evaluation of the patient and formation of the diagnosis should lead to a trial of nonoperative management. Nonoperative treatment modalities should include rest, hamstring stretches, and quadriceps strengthening exercises. The only surgically effective treatment of this syndrome is excision of the medial patellar plica. +

D.A. "Bru" Brubaker

Book Reviews, *from page 311*

and the current standard of care. In addition clinical athletic conditions of the shoulders, knees, lower legs and feet are discussed using current terminology, diagnostic techniques and treatment. The authors try to develop the pathomechanics of the injuries and how the tissues are damaged. The use of the arthroscope is gone into in detail.

There are now sections on judo, ice hockey, rules and equipment changes, new medical diagnostic techniques such as nuclear imaging, CAT scans, and other equipment not in vogue when the first edition was published. Massage and therapeutic cold are discussed in detail. A new addition is the psychology of performance and mentioning of endorphins, which are the body's natural pain antidotes.

It is the only book on the market of this nature that carries a comprehensive section on the preparations of an athlete for boxing, a sport that most physicians and physical educators avoid. Many trainers are now getting into the training aspects of boxing and see it is not unlike other sports which they service.

This book has a 16 page chapter on drugs in sports. This chapter explains why athletes get into the drug thing. This is now very timely because of the recent explosion of drugs in professional sports and the recent drug scandal in the 1983 Pan American Games. The fact that drugs do not help performance and that drugs may lead to some dangerous side effects should warrant reading this chapter to learn how to rid teams of this menace.

The preparticipation or preseason examination along with water and electrolyte balance are two of the most important contributions for Sports Medicine. These are covered in depth. Nutrition is covered well and helped with nutritional charts that point to a necessary well-balanced diet as part of training and conditioning of the athlete.

Chapter X and XI, titled Examinations and Decisions on the Field and Training Room Tips respectively, have special significance to the trainer. Both of these chapters have been enlarged and new clinical conditions discussed in chapter XI. In most athletic events, save boxing, the trainer initiates emergency care of the injured athlete. Usually the trainer has to work alone in the training room before a physician shows. These two chapters have a depth of information that will hold a sensible trainer in good stead while waiting for the team doctor to arrive.

There is also a well written glossary which converts training room language into more precise medical language which is now the vogue in most training rooms. There is also a well written index.

This book is an excellent buy for athletic trainers, physical educators, family physicians, and nurses who service injured and ill athletes. +

"Pinky" Newell, ATC

Potpourri

Dennis Aten, ATC, RPT, MS
Eastern Illinois University

Ultrasound

A new bibliography on ultrasound is now available. Compiled by Phyllis M. Quinn, coordinator of APTA's Information Central, the bibliography lists nearly 70 sources on the subject of ultrasound. The listing is available free by writing Information Central, APTA, 1156 15th St. N.W., Washington, D.C. 20005.

Illinois State Medical Society Inaugurates Team Physician Awards

1983 News Release

BLOOMINGTON — The first recipients of the inaugural "Team Physician Awards" to be presented on an annual basis by the Illinois State Medical Society will be guests of the Illinois High School Association (IHSA) and will be honored at the annual Class AA Boys State Final Basketball Tournament press buffet.

The purpose of the award program is twofold: to recognize physicians who are continually contributing their time and skills to young athletes, and to encourage other physicians to assist local athletic teams and recognize the athletic injury concerns of local school districts.

Named 1983 recipients by the Society are: Dr. Edward Grogg of Mahomet, Dr. Robert Hart of Peoria, Dr. William Hart of Peoria, Dr. Kenneth Malmberg of Auburn, Dr. William Schiffbauer of Streator and Dr. George Wilkins of Granite City. Dr. H. Bates Noble of Chicago, chairman of the Illinois State Medical Society Medicine Committee, will introduce the recipients and other guests at the buffet.

To be eligible for the "Team Physician Award" a physician must:

- Maintain a license to practice medicine in all of its branches.
- Have tenure as a team physician at a high school or college level for a minimum of 10 years.
- Maintain current membership in the Society.

Retired physicians may be nominated for past team physician activities. Nominations may come from school officials, coaches, athletes, parents, civic organizations and/or other physicians.

The 1983 award winners have combined 154 years experience working with young men and women as team physicians.

Dr. Kenneth Malmberg has been the team physician at Auburn High School for the past 40 years. Nominated by Auburn football coach John Rigg, Dr. Malmberg, according to Rigg, has "not only supplied immediate care at our ball games, but has taken time to visit practice and observe our coaching techniques, as well as observe and comment on the rehabilitation of athletes who have been injured. His support of our program does not end here, for he is Rotary program chairman for the annual 'Meet the Coaches Night.' "

Dr. Robert Hart and Dr. William Hart have worked with the athletes at Peoria (H.S.) and the surrounding community since the 1947-48 school year, and have been instrumental in forming Sports Medicine clinics and workshops for area coaches and physical educators. "Their dedication to sports medicine goes above and beyond the Hippocratic Oath," said Peoria (H.S.) Principal William C. Robertson, who served as PHS football coach from 1957-64. "They have given freely of their time and talents when they were asked. Their presence at all football games, home and away, for 67 (combined) years is a tribute to their dedication to sports medicine."

For more than 25 years Dr. William Schiffbauer has provided medical support to the athletes of Streator (Twp.) High School. In addition, he has arranged for speakers for the LaSalle County Medical Society, as well as being a speaker in the local continuing medical education seminars at St. Mary's Hospital. In addition, he has been the driving force behind the establishment of the Streator (Twp.) High School training facility.

Dr. George Wilkins serves as team physician for both high schools in the Granite City system, and has been the team physician in the system for more than 20 years. Through his efforts and financial support, the system maintains a student athletic trainer scholarship program. He administers all senior high school athletic physical examinations, and the (minimal) fees are returned to the system's sports medicine program. He also handles arrangements with the St. Louis Sports Medicine Clinic to staff all home football games for both schools.

Dr. Edward Grogg of Mahomet is much, much more than the Mahomet-Seymour team physician. In 1978, he started the Center for Athletic Injury Research (C.A.I.R.) through the Carle Foundation, and in August of 1982 the new C.A.I.R. facility adjacent to the high school was dedicated. He also organized the Annual Carle Foundation Sports Medicine Seminar in 1973, and it provides annual instruction and information to coaches, trainers, team physicians and others responsible for the prevention, recognition and treatment of athletic injuries. For the past several years, Dr. Grogg has served as on-site physician for the IHSA Boys State Final Wrestling and Boys State Final Basketball Tournaments at the Assembly Hall.

Prescription Trends

Good Health Digest
October, 1982

American pharmacies today are filling fewer prescriptions for all kinds of drugs and medications than they were in the mid 1970's. Experts attribute the change to negative effects of certain drugs that are often widely publicized. According to Dr. David Bennett of the American Medical Association's division of drugs,

medical schools are now teaching drug prescribing more cautiously, and there is a strong attempt to emphasize such nondrug therapy as hot baths, jogging, counseling and behavior therapy.

Weight Lifting May Pose Danger

Progress Report
February, 1983

Weight lifting may pose a threat to teenagers, while weight training, a physical conditioning exercise, is reasonably safe, according to the American Academy of Pediatrics.

Young people who lift weights "are extremely competitive and want to outdo their peers; therefore, they are prone to attempt lifts beyond their capabilities," the Academy said.

In weight training, the athlete repetitively lifts weights which are lighter than his or her maximum capability. The Academy endorses such training because the exercise is an effective means of improving athletic performance in most sports.

Weight lifting, in contrast, is a competitive sport whereby the athlete attempts to lift the maximum weight without rest intervals.

More than 32,000 injuries related to weight lifting required hospital visits in 1979, according to the Consumer Product Safety Commission. Approximately half of the injuries occurred to those in the 10 to 19 year old age group.

Injuries which may occur in weight lifting include minor sprains and strains, fractures of long bones, shoulder, low back, and knee injuries, and weightlifter's blackout, caused by a temporary decrease in blood reaching the brain.

A temporary but significant increase in blood pressure can also occur in weight training especially to those teenagers with cardiovascular problems.

Exercise - In Monkeys

Reader's Digest

The question of whether exercise really pays off continues to intrigue medical scientists as well as those who jog, swim and sweat in the hope of lessening their chances of a heart attack. There is considerable evidence from population studies to support the theory that cardiovascular exercise - some form of exertion that can elevate the pulse rate in sustained fashion at least three times per week for at least 30 minutes - will indeed reduce coronary risk. Laboratory studies to date have been somewhat confusing, however. Most results have come from lower vertibrates. Thus, there was interest in a recent Boston University Medical Center study suggesting that regular exercise on a treadmill enabled monkeys to withstand the ravages of a coronary-artery-disease producing diet, as compared with monkeys on the same diet who did not exercise. Clearly this study needs to be reproduced and refined before such animal results can be translated into firm recommendations for humans. Nonetheless, it represents the first laboratory evidence from higher primates that objectively supports the role of exercise in preventing coronary heart disease. In this instance maybe we should change the parties in the adage "Monkey see, monkey do."

continued on page 318

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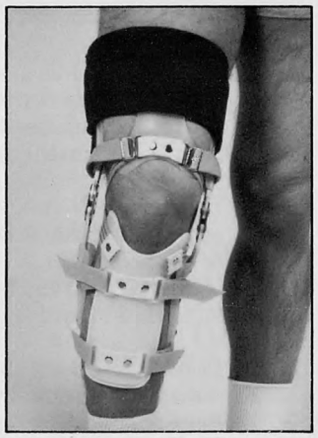

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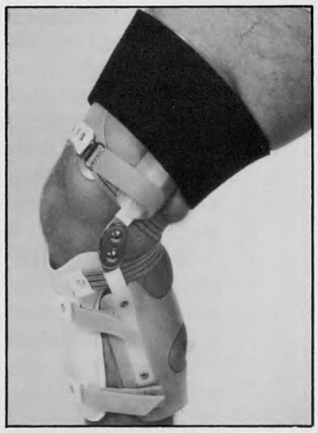

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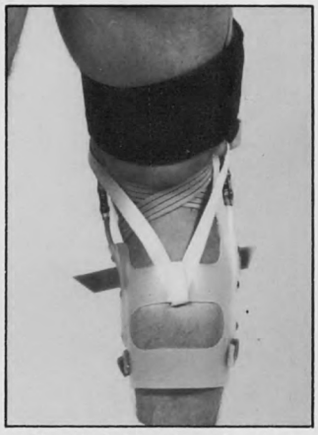

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Protective Padding for the Female Fencer

Nancy Miraldi and Elisa Cangialosi, ATC

During the 1982-83 women's fencing season, three out of four of the first team members received hits to the pubic area. To date, there are no protective pads on the market for females except breast plates for breast protection. These plates are inserted in the jacket. To reduce the possibility of pubic injury, some manufacturers have doubled and even tripled the material in the pubic area but this is not always enough protection.

A simple solution is to modify the fencer's pants. Trace the outline of a football coccyx pad or football kidney pad on a piece of cardboard allowing an extra 3/4 of an inch around the pad. These pads in turn will be used as the protective pad (Fig. 1). The pattern will serve to cut out a piece of material. This will be the pocket into which the protective pad will be placed. Polyester material was found to be the material of choice because of its elasticity.

Turn the pants inside out. Place the pocket so that the middle of it is centered on the seam of the pants and the small of the pocket is positioned just above the crotch (Fig. 2).

Stitch the pocket, keeping the wider part opened. Insert the pad with the smaller part first (Fig. 3). The kidney pad has been found to be more comfortable. To protect a larger area the coccyx pad should be used and easily slips into the pocket patch made for the kidney pad (Fig. 4). When the lame is put on, the bulge from the pad will be covered. A small piece of adhesive velcro, with a few sewn stitches for security, will close the top of the patch.

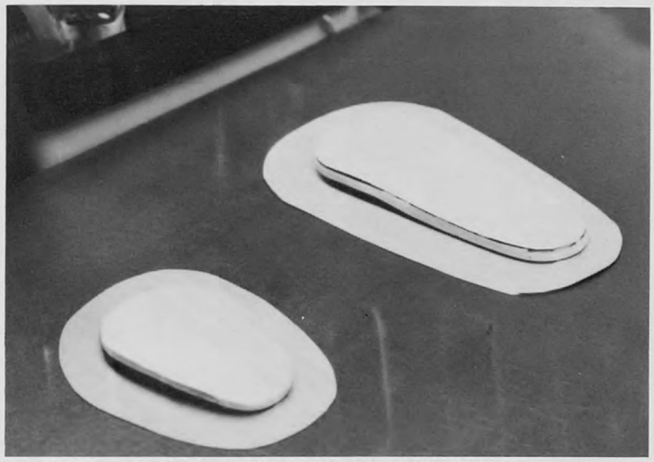

Figure 1 A. Kidney Pad B. Coccyx Pad

Ms. Nancy Miraldi coaches and teaches fencing at Rutgers University, Newark Campus, Newark, New Jersey. Ms. Elisa Cangialosi, ATC, presently works as an Assistant Trainer at Saint Peter's College in New Jersey City, New Jersey.

Figure 2

Figure 3 Pants are shown inside out for illustration only.

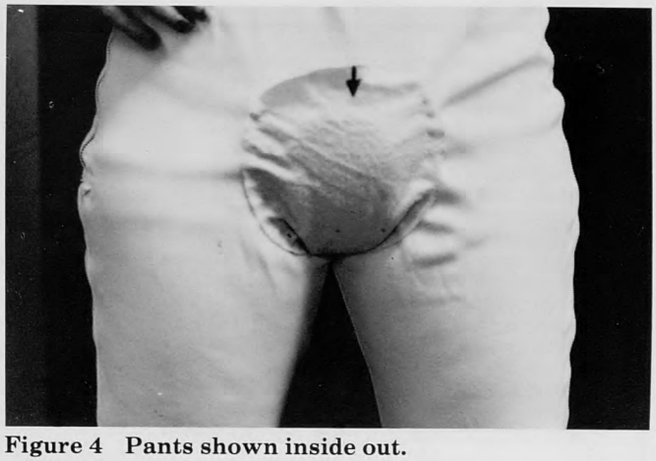

Figure 4 Pants shown inside out.

New Products

Cybex Division of Lumex Inc. has announced its entry into the fitness market with the purchase of Eagle Performance Systems of Owatonna, Minnesota. Cybex, can now strongly complement its own fitness products with Eagle's 16-piece line of variable-resistance, selectorized exercise machines, plus free weights and accessories. These 16 units, which offer variable resistance muscle loading for all the major muscle groups of the body, share several superior features that set them apart from similar exercise equipment.

Eagle machines are designed for women and men alike, thereby eliminating the need for separate machines. Their simplicity of design allows convenient entry and exit and can be used by orthopedically and neurologically involved individuals as well as the general population. Weight stacks are conveniently located so they can be selected from the exercise position without leaving the machine. Eagle's compact design maximizes use of available floor space and allows the machines to be safely placed back to back, side to side, against a wall, or in some cases, in a corner.

Together Cybex and Eagle can now offer a comprehensive line of exercise systems for sports medicine clinics, rehabilitation facilities, training rooms, and fitness centers. +

Potpourri, *from page 314*

"Anorexic Joggers?"

From Reader's Digest

Some middle-aged men who run compulsively appear to be driven by dangerous motives, three University of Arizona researchers write in the *New England Journal of Medicine*. Psychiatrists Alayne Yates and Kevin Leehey and psychologist Catherine M. Shisslak interviewed 60 runners and found that certain mature men among them showed surprising resemblances to young women with anorexia nervosa, the starvation disorder. The men were introverted, often lonely, depressed, self-effacing and "uncomfortable with the direct expression of anger."

Social pressures may explain the similarities, the scientists say. Girls often are judged by beauty, and men by athletic ability. When insecure girls diet to make themselves slim, they may become anorexic; when men run to show their athletic prowess, they may become compulsive marathoners. "Only a small percentage of runners or dieters" are affected, the study reports. "Their behavior becomes pathological as a result of extreme constriction, inflexibility, repetitive thoughts, adherence to rituals and need to control themselves and their environments." +

Athletic Training Educators/Students

Available for Purchase

COMPETENCIES IN ATHLETIC TRAINING

Developed by the NATA Professional Education Committee and approved by the Board of Directors, the competencies included in this nineteen page manual clearly identify the body of knowledge and technical skills to be developed by the entry-level athletic trainer. Specific competencies are identified for each of seven "major tasks" comprising the role of the athletic trainer and are categorized according to knowledge and intellectual skills (cognitive domain), manipulative and motor skills (psychomotor domain), and attitudes and values (affective domain).

The manual is intended to assist both instructional personnel and students in identifying knowledges and skills to be mastered. Individual students preparing for careers in athletic training will find the list of competencies helpful as a guide to self-evaluation of their strengths and weaknesses.

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Current Literature

Paul Concialdi, ATC, MA
Kiskiminetas Springs School

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The Current Literature Editor invites the submission of bibliographic material of NATA members that is published in other journals. Submit information to Kiski School, Saltsburg, PA 15681. +

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Available for Purchase

GUIDELINES FOR DEVELOPMENT AND IMPLEMENTATION OF NATA APPROVED UNDERGRADUATE ATHLETIC TRAINING EDUCATION PROGRAMS
1983 Edition

Recently developed by the NATA Professional Education Committee and approved by the Board of Directors, these **Guidelines** govern NATA evaluation of undergraduate athletic training education programs as academic majors. Directors of current NATA approved athletic training education programs and college and university personnel contemplating development of new undergraduate programs will find these **Guidelines** essential to program development according to NATA standards.

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ANNUAL SURVEY OF FOOTBALL INJURY RESEARCH 1931 - 1982

Frederick O. Mueller **Richard D. Schindler**

Frederick O. Mueller, Ph.D.
Chairman, American Football Coaches Committee on Football Injuries
National Collegiate Athletic Association Committee on
Competitive Safeguards and Medical Aspects of Sports

and

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Section I

INTRODUCTION

In 1931 the American Football Coaches Association initiated the First Annual Survey of Football Fatalities. The original survey committee was chaired by Marvin A. Stevens, M.D., of Yale University, who served from 1931-1942. Floyd R. Eastwood, Ph.D., Purdue University, succeeded Dr. Stevens in 1942 and served through 1964. Carl S. Blyth, Ph.D., University of North Carolina at Chapel Hill, was appointed in 1965 and served through the 1979 football season. In January 1980, Frederick O. Mueller, Ph.D., University of North Carolina at Chapel Hill, was appointed by the American Football Coaches Association and the National Collegiate Athletic Association to continue this research under the new title, Annual Survey of Football Injury Research.

The primary purpose of the Annual Survey of Football Injury Research is to make the game of football a safer and, therefore, a more enjoyable sports activity. Because of these surveys, the game of football has realized many benefits in regard to rule changes and improvement of equipment.

Data Collection

Throughout the year, upon notification of a suspected football fatality, immediate contact is made with the appropriate officials (coaches, administrators, physicians and trainers.) Pertinent information is collected through questionnaires and personal contact. In every case during the past year the appropriate officials responded to the inquiries. Data collection forms were returned and, in many cases, covering letters giving valuable information were included.

Football fatalities are classified for this report as direct and indirect. The criteria used to classify football fatalities are as follows:

Direct — Those fatalities which resulted directly from participation in football.

Indirect — Those fatalities which are caused by systemic failure as a result of exertion while participating in football activity or by a complication which was secondary to a non-fatal injury.

In several instances of reported football fatalities, the respondent stated that the fatality should not be attributed to football. Reasons for these statements are that the fatally injured was not playing football when the accident occurred or that the fatality was attributed to physical defects that were unrelated to football injuries.

Dr. Mueller compiled and prepared the survey report on college, professional and sandlot levels, and Mr. Richard D. Schindler of the National Federation of State High School Associations assumed complete responsibility for collecting and preparing the senior and junior high school phase of the study.

As the conclusion of the football season, both reports are compiled into this Annual Survey of Football Injury Research. This report is sponsored by the American Football Coaches Association, the National

Collegiate Athletic Association, and the National Federation of State High School Associations.

Acknowledgements

This 1982 report was compiled with the assistance of association executive officers, high school and college coaches, athletic directors, school administrators, physicians, a national newspaper clipping agency, and professional associates of the authors. Dr. Carl S. Blyth served as a consultant for the 1982 report.

Section II
SUMMARY

1. Nine fatalities were directly related to football during the 1982 football season. Seven of the fatalities occurred in high school. There were no direct fatalities in college. The two remaining fatalities occurred in organized youth league programs. These last two fatalities will be listed as sandlot. (Table I)

2. The incidence of direct fatal injuries is very low on a 100,000 player-exposure basis. For the approximately 1,575,000 participants in 1982, the number of fatalities was .57 participants per 100,000 players.

3. The incidence of direct fatalities in high school football was .53 participants per 100,000 players. The incidence of direct fatalities in college was 0.00 participants per 100,000 players. (Table III)

4. Most direct fatal injuries usually occur during regularly-scheduled games. During the 1982 season eight fatalities occurred in games and one in practice.

5. The 1982 survey shows that of the nine direct fatalities one occurred in August, two in September, five in October, and one in November.

6. The major activities in football would naturally account for the greatest number of direct fatalities. In 1982 tackling incurred three fatalities, being tackled resulted in four, blocking a punt resulted in one, and the cause of one fatality was unknown.

7. In 1982 seven of the direct fatalities resulted from injuries to the head, one resulted from internal injuries, and the cause of one was unknown.

8. In many cases football cannot be directly responsible for fatal injuries (heat stroke, heart failure and so forth). In 1982 there were eleven indirect fatalities. Two of these were the result of heat stroke, five were the result of heart failure, one resulted from an asthmatic attack, one resulted from a sickle cell crisis, one from a congenital brain defect, and one from natural causes. Seven of the indirect fatalities were associated with high school football, three in college football, and one sandlot.

Section III
DISCUSSION AND RECOMMENDATIONS

The 1982 survey shows that the number of direct fatalities has remained stable with no dramatic increase or decrease in any area. The trend for fewer football fatalities, when compared to fatality data collected for the past 25 years, has continued in 1982. Progress has been made and an all-out effort must be made to continue this trend and to avoid another rise in direct fatalities.

Past efforts that were successful in reducing fatalities to the level indicated in the 1979 data should again be emphasized. Rule changes for the 1976 football season which eliminted the head as a primary and initial contact area for blocking and tackling are of utmost importance. Since 1960 most of the direct fatalities have been caused by head and neck injuries. The 1982 survey shows that seven of the direct fatalities resulted from injuries to the head. We must continue to reduce head and neck injuries. It has been suggested that the increased protection afforded by recent advances in protective equipment encourages young athletes to take undue risks when executing football skills.

Several suggestions for reducing head and neck injuries are as follows:

1. Athletes must be given proper conditioning exercises which will strengthen their necks so that participants will be able to hold their heads firmly erect when making contact.

2. Coaches should drill the athletes in proper education in the fundamentals of football skills, particularly blocking and tackling.

3. Coaches and officials should discourage the players from using their heads as battering rams when blocking and tackling. The rules prohibiting spearing should be enforced in practice and in games. The players should be taught to respect the helmet as a protective device and that the helmet should not be used as weapon.

4. All coaches, physicians, and trainers should take special care to see that the players' equipment is properly fitted, particularly the helmet.

Enforcement of the rules prohibiting "spearing," properly fitted helmets, and excellent physical condition are the factors which will help reduce fatalities and serious head and neck injuries.

Another important effort has been and continues to be the improvement of football protective equipment under the guidance of the National Operating Committee on Standards for Athletic Equipment (NOCSAE). The NOCSAE organizations continue their research on improving helmets for football. It is imperative that old and worn equipment be properly renovated or discarded and continued emphasis be placed on developing the best equipment possible. Manufacturers, coaches, trainers, and physicians should continue their joint and individual efforts toward this end.

In addition to the above, it is important, whenever possible, for a physician to be on the field of play during game and practice sessions. When this is not possible, arrangements must be made in advance to obtain a physician's immediate service when emergencies arise. Each institution should have a team trainer who is a regular member of the institution's staff and who is qualified in treating and preventing injuries.

The authors of this study are convinced that the current rules which eliminate the head in blocking and tackling, the helmet research conducted by NOCSAE, and better medical supervision have played the primary role in reducing fatalities and serious head and neck injuries in football.

A continuous effort should be made to eliminate heat stroke deaths associated with football. Since the beginning of the survey through 1959 there were five cases of heat stroke deaths reported. From 1960 through 1982 there have been sixty-nine heat stroke cases which re-

sulted in death (Table IV). Since 1974 there has been a dramatic reduction in heat stroke deaths with the exception of 1978 when there were four. Two deaths were associated with heat stress in 1982. All coaches, trainers, and physicians should continue their efforts toward eliminating athletic fatalities which result from physical activity in hot weather.

Heat stroke and heat exhaustion are prevented by careful control of various factors in the conditioning program of the athlete. When football activity is carried on in hot weather, the following suggestions and precautions should be taken:

1. Each athlete should have a complete physical examination with medical history and an annual health history update. History of previous heat illness and type of training activities before organized practice begins should be included.

2. Acclimatize athletes to heat gradually by providing graduated practice sessions for the first seven to ten days and other abnormally hot or humid days.

3. Know both the temperature and humidity since it is more difficult for the body to cool itself in high humidity. Use of a sling psychrometer is recommended to measure the relative humidity and any time the wet-bulb temperature is over 78° practice should be altered.

4. Adjust activity level and provide frequent rest periods. Rest in cool, shaded areas with some air movement and remove helmets and loosen or remove jerseys. Rest periods of 15-30 minutes should be provided during workouts of one hour.

5. Provide adequate water replacement during practice. Water should always be available and in **unlimited quantities** to the athletes.

Give Water Regularly.

6. Salt should be replaced daily, and liberal salting of the athletes' food will accomplish this purpose. Coaches should not provide salt tablets to athletes while they practice. Attention must be directed to water replacement.

7. Athletes should weigh each day before and after practice and weight charts checked in order to treat the athlete who loses excessive weight each day. Generally, a three percent body weight loss through sweating is safe, and a five percent loss is in the danger zone.

8. Clothing is important and a player should avoid use of long sleeves, long stockings, and any excess clothing. Never use rubberized clothing or sweatshirts.

9. Some athletes are more susceptible to heat injury. These individuals are not accustomed to work in the heat, may be overweight, and may be the eager athlete who constantly competes at his capacity.

10. It is important to observe athletes for signs of heat illness. Some trouble signs are nausea, incoherence, fatigue, weakness, vomiting, cramps, weak rapid pulse, flushed appearance, visual disturbance, and unsteadiness. If heat illness is suspected, seek a physician's immediate service. Recommended emergency procedures are vital.

Recommendations

Specific recommendations resulting from 1982 survey data are as follows:

1. Mandatory medical examinations and medical history should be taken before allowing an athlete to participate in football. The NCAA recommends a thorough medical examination when the athlete first enters the college athletic program and an annual health history update with use of referral exams when warranted. If the doctor or coach has any questions about the athlete's readiness to participate, the athlete should not be allowed to play. High school coaches should follow the recommendations set by their state high school athletic associations.

2. All personnel concerned with training football athletes should emphasize proper, gradual, and complete physical conditioning. Particular emphasis should be placed on neck strengthening exercises.

3. A physician should be present at all games and practice sessions. If it is impossible for a physician to be present at all practice sessions, emergency measures must be provided.

4. All personnel associated with football participation should be cognizant of the problems and safety measures related to physical activity in hot weather.

5. Each institution should strive to have a team trainer who is a regular member of the faculty and is adequately prepared and qualified.

6. Cooperative liaison should be maintained by all groups interested in the field of Athletic Medicine (coaches, trainers, physicians, manufacturers, administrators, and so forth).

7. There should be strict enforcement of game rules, and administrative regulations should be enforced to protect the health of the athlete. Coaches and school officials must support the game officials in their conduct of the athletic contests.

8. There should be a renewed emphasis on employing well-trained athletic personnel, providing excellent facilities, and securing the safest and best equipment possible.

9. There should be continued research concerning the safety factor in football (rules, facilities, equipment, and so forth).

10. Coaches should continue to teach and emphasize the proper fundamentals of blocking and tackling to help reduce head and neck fatalities. Keep the head out of football.

11. Strict enforcement of the rules of the game by both coaches and officials will help reduce serious injuries.

12. When a player has experienced or shown signs of head trauma (loss of consciousness, visual disturbances, headache, inability to walk correctly, obvious disorientation, memory loss), he should receive immediate medical attention and should not be allowed to return to practice or game without permission from the proper medical authorities.

Section IV
CASE STUDIES DIRECT FATALITIES

High School

A 14-year-old high school sophomore was fatally injured while participating in a practice scrimmage on August 25, 1982. He was tackled in a head-on collision while carrying the ball on a kickoff. He collapsed and lost consciousness after

the play and died on August 28, 1982. He was reported as having his "bell rung" in a scrimmage on August 6, 1982. Cause of death was a subdural hematoma.

On September 10, 1982, a 17-year-old high school football player was tackled after catching a pass. He fell to the natural turf hitting his hip first, shoulder second, and head last. He died September 15, 1982, from a head injury as a result of his head hitting the ground after the tackle. It was noted that earlier in the season he was struck by a ball in the head that gave him a headache and he was hit in the head during a scrimmage and was dizzy for a short time.

A 16-year-old high school player was seriously injured in a game while being tackled. The injury took place on October 22, 1982, and after being in a coma died on October 28, 1982. Cause of death was a subdural hematoma.

A 14-year-old high school athlete was kicked in the stomach while making a tackle in a game on October 23, 1982. The player was helping on a tackle and as he approached the ball carrier the legs of the ball carrier swung around and hit the player in the stomach. The player was not in pain and went about normal activities after the game. Six hours after the game, the injured player died from internal bleeding that led to congestive heart failure.

On October 28, 1982, a 17-year-old high school junior varsity football player received head injuries received in a game. Information as to how the accident took place was not available. He died on October 30, 1982.

A 17-year-old high school football player died on October 31, 1982, after receiving a head injury in a game. The injured player was making a tackle and his head made contact with the thigh of the ball carrier. He was playing defensive back and tackling the pass receiver in the open field. Cause of death was a subdural hematoma.

On November 6, 1982, a 16-year-old high school football player died of a subdural hematoma received in a football scrimmage on October 25, 1982. The injured player was a linebacker making a tackle.

Sandlot

On September 12, 1982, a 13-year-old football player was injured while playing in a youth football game. The player was injured while trying to block a punt when he hit another player head-on. He died September 14, 1982. The injured player did not play during the 1981 season due to severe headaches. Cause of death was a subdural hematoma.

A youth league football player, age 15, was injured while playing in a game October 21, 1982. He collapsed at the game and was in a coma. Details on cause of death were not available at this writing.

Section V
CASE STUDIES
INDIRECT FATALITIES

High School

On August 19, 1982, a 13-year-old middle school student died from heat stroke. The player was practicing in full uniform and the temperature was 90°. Humidity information was not available. Practice involved two 45-minute work periods with a 15-minute water break between the work sessions. The injured player was reported as not having any water during the break. He collapsed in the locker room after practice, was packed in ice, and taken to the emergency room.

A 17-year-old high school football player died on August 27, 1982, after a severe asthmatic attack led to cardiac arrest.

A 15-year-old high school football player died in August, 1982, when he collapsed and died after hitting a blocking sled. There were no warning signs and the medical examiner said the heart just stopped pumping.

On October 15, 1982, a 17-year-old high school football player died from natural causes related to the heart. He had a breathing problem while standing on the sidelines at practice after being involved in stretching and warm-up drills.

A 14-year-old football player died on August 24, 1982. He collapsed during a run in practice and died in the emergency room from cardiac arrest.

A 17-year-old high school football player died on September 24, 1982. He took himself out of the game with one minute left and complained of blurred vision. He collapsed in the locker room. The autopsy report stated it was not a specific injury but was due to a congenital blood vessel defect in the brain.

A high school football player did from cardiac arrest but specific information was not available at this writing.

College

A college football player collapsed after completing a 12-minute run. He was taken to the emergency room and died that same day—August 15, 1982. He died from heart failure precipitated by sickle cell crisis.

A college football player died of heat stroke on October 15, 1982. He was a 230-pound linebacker practicing in mild weather. Exact temperature and humidity readings were not available. Additional information was not available at this writing.

A 21-year-old college football player died from natural causes in October, 1982. The medical examiner stated it was an arteriovenous malformation at the back of the brain and the death was not related to football. The player was a punter and punted twice in the game. He was not involved in any contact. He died in the emergency room.

Sandlot

A 9-year-old youth league football player died two hours after he collapsed at practice. It was the fifth day of practice and the player had just completed one lap of the field. The medical examiner stated the player died of cardiac arrest from natural causes. The player died on August 6, 1982.

TABLE I

Fatalities: Directly Due to Football - 1931-1982*

Year	SANDLOT Direct	PRO AND SEMIPRO Direct	HIGH SCHOOL Direct	COLLEGE Direct	TOTAL Direct
**1931-1959	115	68	262	41	486
1960	1	1	11	1	14
1961	3	0	10	6	19
1962	6	1	12	0	19
1963	1	1	12	2	16
1964	4	1	21	3	29
1965	4	0	20	1	25
1966	4	0	20	0	24
1967	5	0	16	3	24
1968	4	1	26	5	36
1969	3	1	18	1	23
1970	3	0	23	3	29
1971	2	0	15	3	20
1972	3	1	16	2	22
1973	2	0	7	0	9
1974	0	0	10	1	11
1975	1	0	13	1	15
1976	3	0	15	0	18
1977	1	0	8	1	10
1978	0	0	9	0	9
1979	0	0	3	1	4
1980	0	0	9	0	9
1981	2	0	5	2	9
1982	2	0	7	0	9
TOTALS	169	75	568	77	889

* No study was made in 1942
** Yearly totals available from past reports

TABLE II

Fatalities: Indirectly Due to Football - 1931-1982*

Year	SANDLOT Indirect	PRO AND SEMIPRO Indirect	HIGH SCHOOL Indirect	COLLEGE Indirect	TOTAL Indirect
**1931-1959	72	12	112	28	224
1960	0	0	2	2	4
1961	4	1	11	0	16
1962	0	1	4	2	7
1963	2	0	4	2	8
1964	3	0	12	1	16
1965	4	1	14	5	24
1966	0	0	6	2	8
1967	0	0	4	1	5
1968	2	0	8	2	12
1969	3	1	8	3	15
1970	0	0	12	2	14
1971	2	1	7	2	12
1972	0	0	10	1	11
1973	0	0	5	3	8
1974	0	0	5	3	8
1975	2	0	3	3	8
1976	1	0	7	2	10
1977	0	0	6	0	6
1978	0	0	8	1	9
1979	1	0	8	1	10
1980	0	0	4	0	4
1981	0	0	6	0	6
1982	1	0	7	3	11
TOTALS	97	17	273	69	456

* No study was made in 1942.
** Yearly totals available from past reports.
Based on 1,300,000 junior and senior high school players and 75,000 college players.

TABLE III

Direct Fatalities Incidence Per 100,000 - 1931-1982*

YEAR	HIGH SCHOOL	COLLEGE
**1931-1959		
1960	1.78	1.53
1961	1.62	9.23
1962	1.94	0.00
1963	1.94	3.04
1964	2.23	4.56
1965	2.00	1.33
1966	2.00	0.00
1967	1.60	4.00
1968	2.60	6.60
1969	1.64	1.33
1970	1.92	4.00
1971	1.25	4.00
1972	1.33	2.67
1973	0.58	0.00
1974	0.83	1.33
1975	1.08	1.33
1976	1.00	0.00
1977	0.53	1.33
1978	0.60	0.00
1979	0.23	1.33
1980	0.69	0.00
1981	0.38	2.67
1982	0.54	0.00

* No study was made in 1942.
** Yearly totals available from past reports.

TABLE IV

Heat Stroke Fatalities 1931-1982*

YEAR	TOTAL	YEAR	TOTAL
1931-1954	0	1970	8
1955	1	1971	4
1956-1958	0	1972	7
1959	4	1973	3
1960	3	1974	0
1961	3	1975	0
1962	5	1976	1
1963	0	1977	1
1964	4	1978	4
1965	6	1979	2
1966	1	1980	1
1967	2	1981	2
1968	5	1982	2
1969	5		

* No study was made in 1942.

TABLE V

DIRECT FATALITIES 1982: TYPE OF ACTIVITY ENGAGED IN

Type of Activity	Number
Blocking Punt-Collision	1
Collision with Person(s)	1
Tackled	3
Tackled-Head Hit Ground	1
Tackling	3
Totals	9

TABLE VI

DIRECT FATALITIES 1982: CAUSE OF DEATH

Causes	Sandlot	Pro	High School	College	Total
Head Injury	1	0	6	0	7
Abdominal-Internal Injuries	0	0	1	0	1
Not Specified	1	0	0	0	1
Totals	2	0	7	0	9

TABLE VII

DIRECT FATALITIES 1982: POSITION PLAYED

Position	Sandlot	Pro	High School	College	Total
Running Back	1	0	3	0	4
Linebacker	0	0	1	0	1
Interior Defensive Lineman	0	0	1	0	1
Defensive Back	0	0	1	0	1
Offensive Lineman	1	0	0	0	1
Not Specified	0	0	1	0	1
Totals	2	0	7	0	9

TABLE VIII

INDIRECT FATALITIES 1982: CAUSE OF DEATH

Causes	Sandlot	Pro	High School	College	Total
Heat Stroke	0	0	1	1	2
Heart Failure	1	0	4	0	5
Asthmatic Episode	0	0	1	0	1
Sickle Cell Crisis	0	0	0	1	1
Natural Causes	0	0	0	1	1
Congenital Brain Ailment	0	0	1	0	1
Totals	1	0	7	3	11

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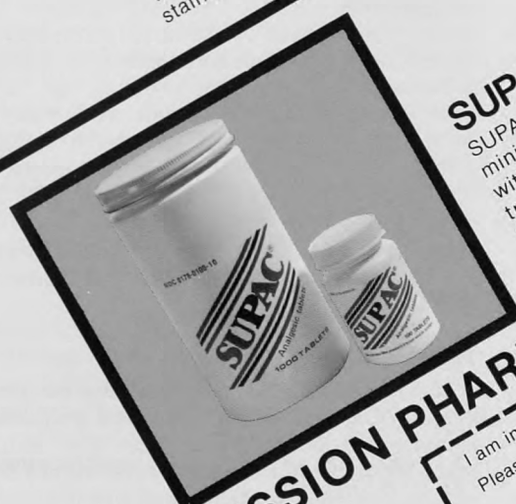

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Treatment and Prevention of Hypothermia and Frostbite

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Hypothermia usually affects mountain climbers, hikers, skiers and hunters, rather than athletes from athletic teams which compete in weather conducive to mild hypothermia and/or localized freezing of body tissue. Athletic trainers should, however, be familiar with the etiology, diagnosis and treatment of these cold weather emergencies.

Hypothermia is a condition of generalized body cooling that results from exposure to inclement weather or immersion in cold water. Although hypothermia is associated with cold weather, the ambient temperature need not be excessively low to initiate body cooling. Hypothermia can occur at air temperatures up to 65 degrees Fahrenheit (1). The effect of environmental temperature is exacerbated by windchill and wetness. Wet clothes conduct heat away from the body more readily than dry clothes. Moisture from perspiration, rain, sleet, or snow further contributes to this conduction. Dehydration, starvation, vasodilation from alcohol consumption, electrolyte depletion, and exhaustion also predispose an exposed athlete to hypothermia.

If an exposed athlete loses more heat than he or she is producing, body temperature begins to drop. As temperature falls (from 98.6° to 81°F) the athlete moves through phases of shivering, apathy and fatigue, to a state of impaired physical and mental functioning (2). Loss of consciousness, freezing of the extremities, and death ensue when body cooling progresses to a severe state and the temperature drops below 80°F.

Physiologic Response

The physiologic response to falling body temperature is a combination of vasoconstriction, which decreases heat loss, and shivering, which increases heat production (3). Shivering provides short term heat maintenance, but cannot be sustained for extended periods of time. If the athlete continues to be exposed to the cold, body temperature begins to fall again, at which point the basal metabolism rate is increased. If these compensatory mechanisms are insufficient, and the core temperature falls below 94°F, then the regulatory systems begin to fail (4). In particular, the respiratory control center becomes depressed, consequently ventilation is reduced and anoxia results. Anoxia leads to slowing of metabolism, which is followed by a further drop in temperature. Thus a cycle of falling temperature, reduced ventilation, anoxia, and metabolic slowing is established.

As anoxia progresses, lactic acid is produced (5). This acid and other metabolic wastes accumulate because liver and kidney efficiency diminish in hypothermia (3,5). Hepatic metabolism of lactic acid is reduced resulting in metabolic acidosis. Furthermore, the kidneys, which normally excrete hydrogen ions during metabolic acidosis, fail to excrete sufficient hydrogen ions to maintain homeostasis because sodium excretion is enhanced in hypothermia (4,5). This impaired sodium-hydrogen exchange exacerbates the metabolic acidosis. (Hypothermia apparently does not result in increased reabsorption of bicarbonate ions.) Kidney inefficiency

Recognition of Hypothermia

The signs and symptoms of hypothermia are related to the falling body temperature (2). As the core temperature falls from 98.6°F to 95°F, shivering and goose bumps are often noted. The athlete will probably move around and jump up and down in an effort to warm up during this mild phase of hypothermia. He or she may seem mentally withdrawn or may complain of dizziness. As the body temperature drops below 95°F, the athlete will lose coordination. As a result, use of the hands will be difficult, stumbling will be frequent, and speech will be slow and slurred. The athlete will seem lethargic during this moderate phase of hypothermia. As the temperature falls to the mid eighties, blood pressure may become imperceptible and heart sounds may become inaudible because sound conduction through the tissues is reduced (4). Pupillary reflexes may be blocked. The skin will probably look pale and waxy and will feel semi-rigid. The muscles will be tense and rigid. The athlete will appear to be very sleepy, and perhaps irrational. If the temperature reaches 80°F the pulse will become weak and irregular. Loss of consciousness will occur, heralding the severe phase of hypothermia. As the temperature falls below 78°F, respirations and pulse will cease.

Treatment

The basic principles of treatment for hypothermia are, first, to prevent further heat loss and, second, to safely rewarm the body. An athlete who exhibits signs or symptoms of mild hypothermia should be moved into a warm, dry environment. All wet uniforms or clothing should be removed, and replaced with dry clothing. Additional insulation such as a jacket, parka, or blanket should be put on, as well as a hat and gloves. The athlete should be given warm, preferably carbohydrate-loaded, liquids to drink. Alcoholic drinks are contraindicated. The athlete should be encouraged to exercise. If he or she is unable to exercise, then external heat should be added by means of an electric blanket. Alternatively, the

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athlete should be placed in a hot 110°F whirlpool.

The treatment for moderate hypothermia is similar to that for mild hypothermia. An athletic trainer should attempt to keep the lethargic or semiconscious athlete awake. If the athlete is aroused enough to drink, warm liquids should be provided.

Treatment for the unconscious athlete with severe hypothermia is more complicated. Fortunately, most athletic trainers will not be called upon to treat these critical patients. Nonetheless, athletic trainers should be familiar with the correct emergency care. In brief, an airway should be established, the temperature should be stabilized, and the severly hypothemic athlete should be transported quickly, but carefully, to a medical center emergency department. Although a patent airway should be maintained, the athlete should not be hyperventilated because the associated pCO2 drop may trigger ventricular fibrillation (4). Wet clothing should be removed, and the athlete's body should be dried and covered with blankets.

Rewarming of the athlete should be delayed until he or she arrives at the medical center. About 65% of these hypothermia patients die if they are rewarmed improperly (1). Rapid or improper rewarming can result in rewarming shock, cardiac dysrythmia, or myocardial infarction as the peripheral circulatory system dilates and blood is shunted away from heart and lungs.

Peritoneal dialysis and/or inhalation of heated, humidified air or oxygen can be used in the hospital to rewarm the athlete from the core out (6). Intravenous therapy can also be instituted to restore blood volume and electrolytes (6). The athlete with severe hypothermia should therefore be transported swiftly but gently to the medical center. Rough treatment enhances movement of cold, acidic blood from the peripheral cardiovascular network to the heart, where it can induce ventricular fibrillation (2).

If the athlete becomes pulseless, then cardiopulmonary resuscitation must be instituted immediately. It is unlikely that cardiac function will return to normal before the body is rewarmed and the electrolytes are corrected. Thus CPR will have to be maintained while the athlete is moved to a hospital.

The hypothermic athlete who is found pulseless is not necessarily beyond hope. Prolonged cardiac arrest may be salvageable because the brain can survive for longer periods of time without perfusion. It can survive for about ten minutes at 86°F, and perhaps 25 to 30 minutes without perfusion at 68°F (4). Successful resuscitation has been reported for a patient with a core temperature of 50°F who had been in cardiac arrest for an hour when found (4). Clearly, the pulseless hypothermic athlete merits an extraordinary attempt at resuscitation.

Prevention

Athletic trainers should advise coaches and athletes regarding hypothermia prevention. This advice should encompass proper nutrition and dress.

Pregame or prepractice meals should be planned to provide ample carbohydrates for heat production in addition to activity related energy needs. Hot and cold drinks for rehydration should be available on the sidelines. The hot drinks will help warm players as well as the coaches, manager, and trainers. The cold drinks will be thirst quenching for players as they come off the field. Alcoholic drinks are contraindicated because they actually reduce body temperature even though the individual may initially feel warmer. Smoking should also be avoided as nicotine causes arterial constriction which restricts blood flow to the periphery that may result in frostbite in subfreezing environments (4). Athletes and coaches should be told to open or remove clothing if they feel they are going to sweat. Although it sounds unusual, athletes should not be encouraged to "work up a sweat" before they play outside on a cold day. Indeed, they should avoid sweating if at all possible. Athletes should wear thermal underwear rather than insulated underwear because the former permits perspiration escape. Ventilated net underwear is even better. Many teams use athletic panty hose. Mittens provide better protection than gloves, and should be available for players who are on the sidelines, as should wind-resistant and water-repellant parkas. Hats should be mandatory as 50% of the body's heat is lost through the head and neck (1). Extra towels should be available to wrap around the players' necks as they wait to enter the game.

Special preparations should be made on the sidelines for games in particularly cold weather. Insulated indoor-outdoor carpeting can be placed in the bench area, as well as kerosene fired industrial heaters. Shelters can be constructed around the bench, using polyethylene plastic. At the very least, insulated containers of warm liquids should be provided.

Frostbite

Frostbite, unlike hypothermia, only occurs in subfreezing weather. When isolated parts of the body are exposed to extreme cold, the tissue can freeze. Typically, numbness is first felt at ambient temperatures near 40°F. Freezing begins at air temperatures ranging from 32°-29°F.Gradual freezing results in ice crystal formation in the interstitial space which increases interstitial osmolarity. The increased osmolarity leads to cell dehydration (5). Rapid freezing, which may occur during ethyl choloride application, can cause ice crystal formation within cells.

Three degrees of frostbite have been described (1). First degree or incipient frostbite usually only involves the tips of the fingers, toes, ears or nose. The onset of this incipient frostbite is slow and painless. The athlete or coach with first degree frostbite probably will not realize that his tissue is freezing unless someone mentions that his skin looks white. This frostbite can be treated by blowing warm air over the affected tissue. The tissue should never be rubbed as friction can damage the tissue. The area will probably tingle as it is warmed.

Second degree or superficial frostbite presents as firm, waxy looking skin with soft, pliable subdermal tissue. This frostbite should be thawed rapidly with immersion in a warm, 105°F. bath (5). The tissue may tingle or burn, swell, and become discolored as it thaws.

Third degree or deep frostbite, rarely seen in athletes, involves tissue beneath the skin, and may involve an entire hand or foot. The skin will appear gray or waxy white in color and will be firm when palpated. The athlete will probably recall a painful, stinging or burning sensation before the tissue became numb. The treatment for third degree frostbite begins with gentle removal of clothing from the frozen area. The tissue should be thawed rapidly in warm water (106°-110°F). However, the tissue should not be warmed if there is any chance of refreezing. Water can be poured over the nose or ear; extremities should be warmed by immersion until the skin turns deep red or purplish. This warming will probably take twenty minutes. The water temperature should be monitored while the part is warming and should be maintained in the 106° - 110°F range. The athlete should be given hot drinks, if possible, but smoking is contraindicated, as the nicotine causes

arterial contriction and subsequently diminished blood flow in the extremities.

After the area is warmed it should be gently dried and bandaged with a sterile dressing. Athletes with second and third degree frostbite should be referred to a physician for follow-up care.

Frostbite can be prevented by use of gloves or mittens and appropriate head gear.

Summary

The signs and symptoms of hypothermia progressively develop as body temperature drops. Hypothermia treatment focuses on reduction of heat loss, and total body rewarming. Rewarming of the rare athlete with severe hypothermia should take place in a hospital. Cardiopulmonary resuscitation of a pulseless hypothermic athlete may be successful even after relatively prolonged periods of cardiac arrest. Hypothermia prevention requires nutritional support as well as appropriate dress.

Frostbite develops when body tissues freeze. It is best treated with rapid rewarming. Frostbite prevention is facilitated by use of protective clothing.

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Index to Volume 18

Traumatic Edema and the Lymphatic System

Peter Kolb, MEd, ATC

Craig Denegar, MEd, ATC

The treatment of edema is fundamental to athletic training. Following injury, the athletic trainer applies ice, compression, and elevation to limit edema. Then treatments are undertaken to remove remaining edema so that rehabilitation can be performed and competition resumed. Often, edema is misunderstood and the key to its resolution, the lymphatic system, ignored.

This paper reviews the basic physiological causes of edema and the role the lymphatic system plays in its reabsorption. Finally, it summarizes treatments and their rationale.

Traumatic Edema

Traumatic edema results from a loss of the gradient that causes filtrated plasma to be reabsorbed. Normally, only .2% of the plasma proteins filter through the arterial capillary walls into the interstitial space (Fig. 1) because the protein diameters generally exceed that of the capillary pores (9). The oncotic (pertaining to swelling) (6) osmotic pressure is then favorable for reabsorption of the fluid. Trauma causes a disruption of the capillary endothelium and a greater amount of plasma proteins enter the interstitial space. (Fig. 2) This is aggravated by vasodilation due to histamine release following cell death (12). There is also an increase in the enzymatic proteins, lactic acid dehydrogenase (LDH) and glutamic-oxalo-acetic acid transaminase (GOT), released by the tissue cells (22). The combination of these proteins locally raises the oncotic osmotic pressure in the interstitium (Fig. 3) and the resulting retention of fluid is edema. A small increase in interstitial oncotic osmotic pressure causes interstitial fluid volume to increase several hundred percent (9).

The interstitium is composed of two major structures, long collagen fibers and proteoglycan molecules. The proteoglycan molecules are long, thin, coiled mucopolysaccharides and can only be seen by electron microscopy. The collagen and proteoglycans combine to trap the interstitial fluid in a sort of gel. This gel prevents free fluid flow. Despite this, diffusion in the gel has been shown to be very rapid and average sized plasma proteins can migrate with 95 to 98% effectiveness (5,9).

Guyton states that rivulets and vesicles of free fluid do exist in the interstitium and in acute edema they expand tremendously (9). Following trauma, the enzymes LDH and GOT are mostly found in the gel, while the fluid and proteins leaving capillaries enter free fluid vesicles (22). Once the gel has swollen more than 30%, the proteoglycan matrix begins to break up, allowing free fluid channels to form throughout the tissue (9).

Edema is almost undetectable until interstitial fluid volume has risen more than 30%. There are two types of edema. Pitting edema can be recognized by the ease with which the fluid can be depressed. Non-pitting edema is hard to the touch and believed to be from a coagulation of fluid (9).

The Lymphatic System

The only way that protein can be removed from the interstitium to restore the oncotic osmotic pressure gradient is through the lymphatic system. Small lymphatic capillaries with bulbous ends originate near the capillary beds and flow undirectionally into increasingly larger vessels that parallel and empty into the veins (12). Essentially all lower body lymph coming from the left arm, chest, and side of the head pass through the thoracic duct to the left internal jugular and subclavian veins. Lymph from the right arm, chest, and side of the head flows into the right jugular and subclavian veins (9).

Proteins and interstitial fluid enter lymphatic vessels easily because of large pores between the vessels' endothelial cells (Fig. 4) (10,8). The cells overlap to form flaps and are "held in place by anchoring filaments that attach to connective tissue (9)." These flaps allow fluid to enter the vessel, but will not allow it to leak back out. Increased pressure on the connective tissue stretches the pore's anchoring filaments. This opens the pore and creates a suction that enhances flow into the lymph vessel (9). Thus the increase in interstitial fluid volume from edema causes greater flow into and within the lymphatic vessels.

The interstitial fluid, now lymph, activates a stretch reflex of smooth muscle in the lymphatic vessel which automatically contracts. Although this contraction would tend to move the lymph in both directions, backflow is prevented by valves (3). The lymph moves forward, distending another portion of the vessel and the smooth muscle there contracts. This system is known as the lymphatic pump and propogates flow of the lymph to the veins (19).

There is a maximum rate limit to lymphatic flow, however. Too great an edematous pressure causes overdistention of the vessels, separating the endothelial cells and making the pores ineffective. Interstitial

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Mr. Denegar is the Head Athletic Trainer at Virginia Military Institute, Lexington, Virginia 24450.

Illustrations by Mr. Rick Parker of Washington and Lee University.

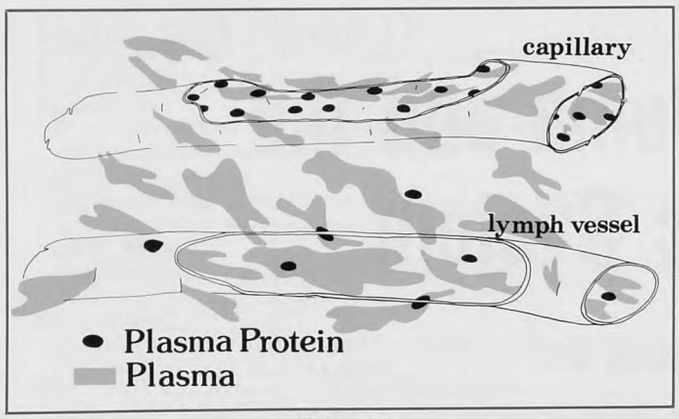

Figure 1
Dissolved proteins are the only constituents of plasma that do not readily diffuse through the pores of the capillary membrane.
The difference of protein concentrations within the capillary and interstitium is responsible for the oncotic osmotic pressure at the capillary membrane.

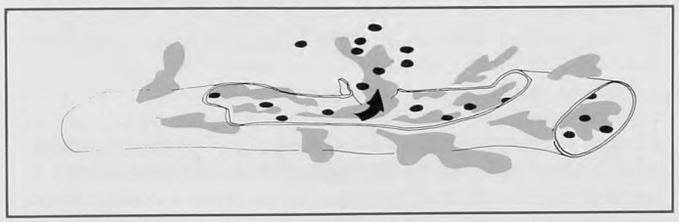

Figure 2
Damage to the capillary allows plasma and dissolved proteins to spill into the interstitium. Osmotic pressure at the capillary membrane is greatly diminished.

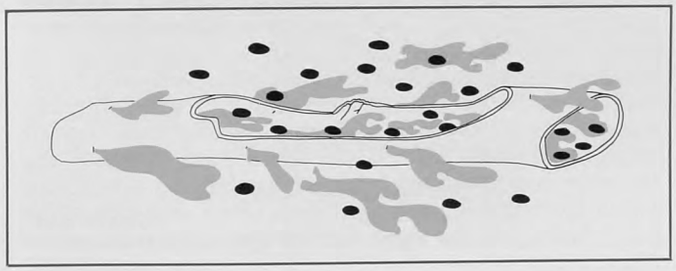

Figure 3
When the rent in the capillary is closed, high concentrations of plasma proteins remain in the interstitium.

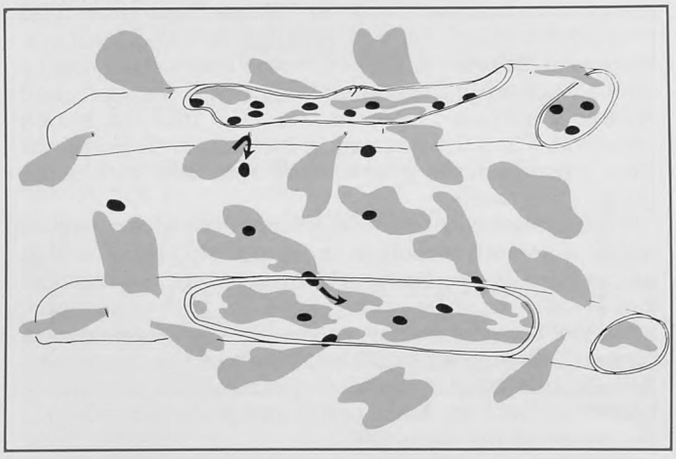

Figure 4
As proteins diffuse into the lymphatic vessels, excess interstitial fluid (edema) is absorbed into the lymph vessel and venous capillaries.

pressure can also impose resistance on larger lymphatic vessels, opposing the flow from lymphatic capillaries (9). This demonstrates the importance of early and proper treatment to limit edema formation.

Treatments to Facilitate Protein Removal by Increasing Lymph Flow

The restoration of the oncotic osmotic pressure gradient is the initial phase of edema reduction. This is accomplished by removing the excess proteins from the interstitium through their mobilization to the lymphatic vessels. Any treatment which improves lymph flow will expedite this process. Elevation, external compression, and muscular contraction are established methods for this (7, 9, 23).

Elevation of an edematous area uses gravity to augment lymph flow. This is a simple, but effective treatment and is often used in conjunction with other therapies.

Passive external pressure helps increase lymph flow. This is why intermittent compression, massage, and elastic support bandages are effective. The compression from these modalities moves the lymph in the vessels and may also move pitting edema over a larger area, allowing more lymphatic vessels to become involved in reabsorption (2, 4, 7, 17, 23, 24, 25).

Similarly, active internal pressure from muscular contraction improves lymph flow and protein mobilization. Therapeutic exercise, especially cryokinetics, and high voltage galvanic stimulation (HVGS) are two means by which this is accomplished (1, 13). These modalities also relieve muscle spasm that can occlude lymphatic and veinous return (1, 14, 20).

Contrast and HVGS at subthreshold currents are two methods that empirically show results in reducing edema. Although contrast treatment is clinically accepted, the literature offers no satisfactory rationale for its effectiveness (11, 25). The same is true of subthreshold HVGS (1, 4, 18, 20, 21) The authors have theorized that negative current repels the negatively charged proteins. This protein migration disperses the edema over a larger area and therefore employs more lymphatic vessels in its reabsorption. This effect may be enhanced by the positive polarity of the ground pad.

The oncotic osmotic pressure gradient is restored once the interstitium's concentration of protein is returned to normal. Fluid in the interstitial space will then be reabsorbed by the circulatory system. Treatments that primarily increase circulation, such as a hot whirlpool bath, can now be used. Prior to removal of the excess proteins from the interstitium, such treatments have minimal benefit (7, 15).

Summary

Traumatic edema is the result of an increased number of proteins in the interstitial space. These proteins elevate the interstitium's oncotic osmotic pressure and capillary filtration exceeds reabsorption. The only means to remove these proteins is through the lymphatic system. Elevation, intermittent compression, massage, elastic bandages, and muscular contraction are all effective treatments to increase lymph flow and therefore, reduce edema.

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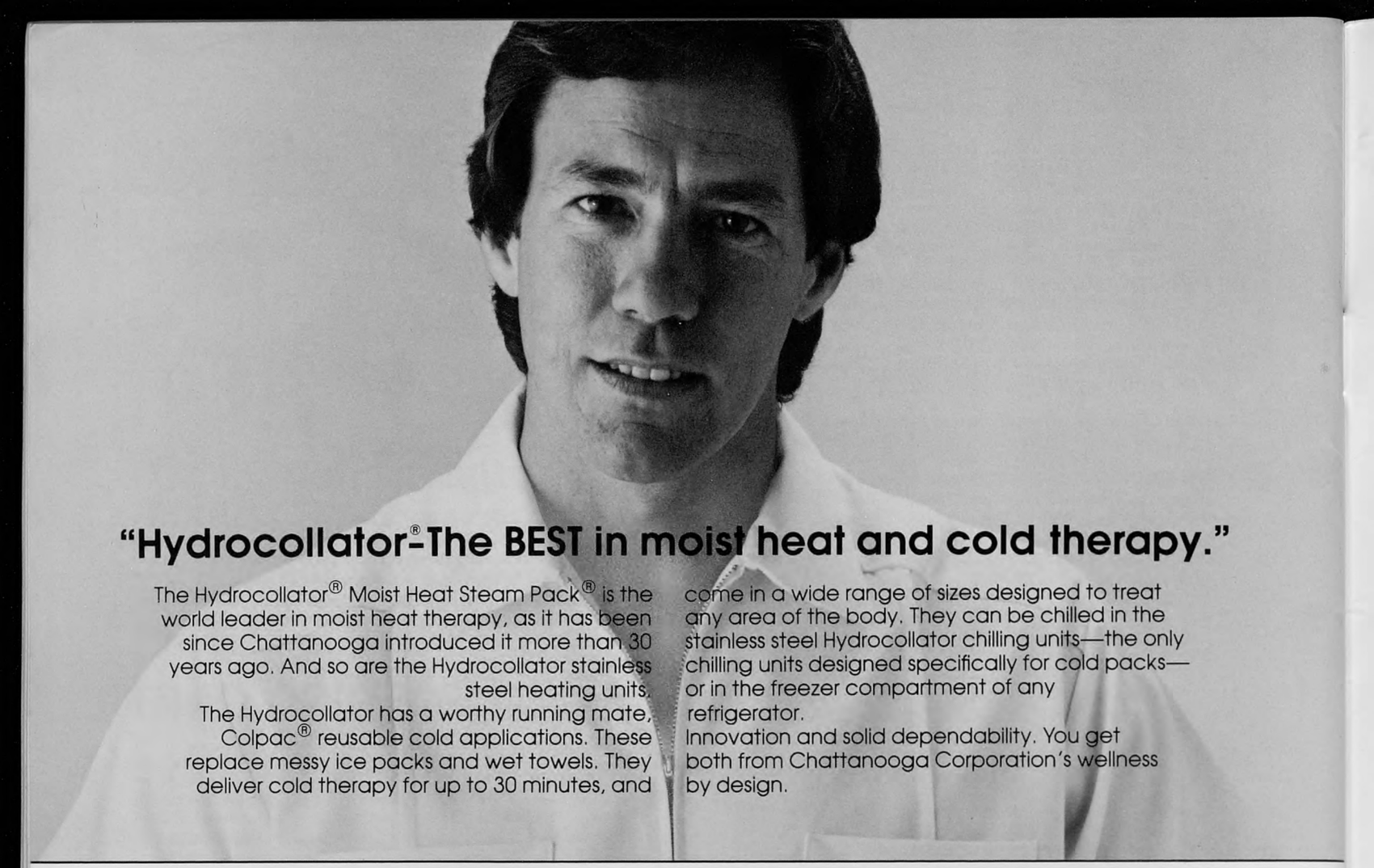

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Announcements

CONGRATULATIONS

It gives me great pleasure to announce the re-election of Dr. Bobby Barton as President of the NATA, Inc. The voting membership of our Association is to be commended for their outstanding participation in the 1983 Presidential Election. We have just completed counting the largest number of votes cast in any Presidential election. This is an indication that our membership is working to achieve the professionalism that they so rightfully deserve.

Otho Davis, ATC
Executive Director

Schedule of Future Sites and Dates NATA Certification Examination

All regional sites are subject to a **minimum** of six candidates per site and limited to a maximum of **thirty** candidates.

Completed applications must be received in the Certification Office by the deadline for the date chosen. However, this does not guarantee the first choice if site and date. Applications are accepted and scheduled in order of remittance.

January 8, 1984

Deadline for returning applications is 11-26-83

New Britain, CT	Albuquerque, NM
Philadelphia, PA	Costa Mesa, CA
Raleigh, NC	Richmond, KY
Chicago, IL	Portland, OR
Ft. Worth, TX	

March 18, 1984

Deadline for returning applications is 2-4-84

Boston, MA	Tucson, AZ
Pittsburgh, PA	Sacramento, CA
Springfield, VA	Richmond, KY
Chicago, IL	Seattle. WA
Lincoln, NE (March 16)	

June 24, 1984

Deadline for returning applications is 5-12-84

New Britain, CT	Cedar Falls, IA
Syracuse, NY	Ft. Worth, TX
Raleigh, NC	Denver, CO
Anderson, IN	Costa Mesa, CA
Madison, WI	Nashville, TN

August 5, 1984

Deadline for returning applications is 6-23-84

Boston, MA	Lawrence, KS
Harrisburg, PA	Costa Mesa, CA
Raleigh, NC	Chattanooga, TN
Anderson, IN	*Eugene, OR
Dayton, OH	

* indicates change from previous listing

Application requests must be in written form. Telephone call requests cannot be honored. To obtain an application write to:

NATA Board of Certification
Application Request
Post Office Drawer 1865
Greenville, NC 27835-1865

Note: 1985 dates will approximate 1984 dates on a regional basis.

Sixth Annual N.A.T.A. Student Writing Contest

In an effort to promote scholarship among young athletic trainers, the National Athletic Trainers Association sponsors an annual writing contest. This contest is open to all undergraduate student members of the NATA. Papers for this contest must be on a topic germane to the profession of athletic training and can be case reports, literature reviews, experimental reports, analysis of training room techniques, etc. The deadline for these reports is March 1, 1984. Further information is available in the Journal of the NATA. Any inquiries need to be sent to: Deloss Brubaker, 103 Gill Coliseum, Oregon State University, Corvallis, OR 97331.

Notes *from the* National Office

"To inform and update the membership on various subjects of interest and answer the most frequently asked questions."

MISSION ACCOMPLISHED

***We are very pleased to report that the National Office's back issue reference library is now complete. In fact, it has been complete for quite some time. We just didn't realize it. The following letter explains.

Dear Barbara,

In the last issue of ATHLETIC TRAINING you asked for some back issues of the Journal. In checking, I think the September 1956 was the **First Issue**. On page 1 (Sept. 1956) the editor begins with an article, "Why the Journal?" and states the "Journal Begins." Also on page 12 (Sept. 1956), under Part "P" of the Secretary's Report, it states "It was decided to have a magazine, *'Journal of the National Athletic Trainers Association'*." The meeting was June 17-19, 1956.

I don't think the Summer 1960 and Summer 1961 issues were ever published. I checked through the Author/Subject Index 1956-1979 and found no authors or articles for Summer 1960 or Summer 1961. Also, starting in the 1959 Fall issue it says "Published 3 Times Yearly" and continues to make this statement until the Fall 1961 issue where it says "Published 4 Times Yearly."

I am sending you the September 1956 issue and maybe someone else will respond or do some checking.

Sincerely,

Walter O. Koch, ATC (Retired)
1753 Leland Avenue
Lima, OH 45805

Thank you, Mr. Koch, for your interest and help. Now we know that we do, in fact, have every issue of the Journal on file here in the National Office and can supply copies of any article requested.

FROM THE MEMBERSHIP OFFICE

***The new procedure for handling district transfers for members (other than students) is as follows: When a member sends an address change to the National Office, and the address change is into another district, the district correction will automatically be made. There will be no need to submit the District Transfer form as in the past. If you are a student member, however, remember you must request that your District be changed.

—If you became a member of NATA after August, 1983, your 1984 dues are paid. While your membership became effective immediately, the dues payment was applied to 1984 and your membership card should reflect 1984. You will not be billed again until October of 1984 when the 1985 statements will be mailed.

HERE'S WHERE TO FIND IT

*Certificate and Plaque Order Form - p. 14, Spring '83.
*CEU Report Form (this issue) - p. 337, Winter '83.
*Competencies in Athletic Training (this issue) - p. 319, Winter '83.
*Constitution - p. 75, Spring '83.
*Continuing Education Requirements and Appeals Process (this issue) - p. 336, Winter '83.
*"Guidelines for Development" order form (this issue) - p. 321, Winter '83.
*Membership Guidelines (this issue) p. 312, Winter '83.
*Role Delineation Study order form - p. 14, Spring '83.
*Storage Cases for ATHLETIC TRAINING -p. 80, Spring '83.
*Student Documentation of Internship Hours (Forms A & B) - p. 248 & 249, Fall '83.
*Student Writing Contest Guidelines - p. 243, Fall '83.

***ONE MORE TIME

The following was printed a year ago in the Winter 1982 Journal. Many NATA members may not have seen that particular issue and since we still regularly receive duplicate notices of address change, we feel it is worth repeating.

ADDRESS CHANGE

NATA members who have an address change within their district need notify the National Office *only once*. We often receive two notices of address change from a member because he or she believes a separate notification must be sent to the Journal. This is not the case. Each NATA member has ONE address of record on file at the National Office computer. When an address change is processed, that member's address is corrected on *all mailing lists*. This includes the mailing list for ATHLETIC TRAINING. So you may save 20¢ postage, as well as some time, by sending only one notification of address change to the National Office. Your mailing label for the Journal will automatically be updated. Please be reminded, however, that while your regular first class mail will be forwarded by the post office after your move, your Journal will not be forwarded due to its second class status. ALWAYS advise your post office to forward your second class mail as well as the first class mail. There is a small charge for the second class mail forwarding — usually 30¢ to 40¢ — but this is much more economical in the long run than replacing a missing Journal for $5.00.

FROM THE CERTIFICATION OFFICE

***Although there is a noted deadline for returning application for each examination date, the sites may fill prior to this date. Thus, it is important that you file an application with the Certification Office as soon as an examination date and site can be targeted.

Please DO NOT schedule for an examination site and date until you are certain of the commitment. There is a $50.00 rescheduling fee required if one reschedules the examination after having made arrangements with the Certification Office.

Work experience hours can not be greater than five years old when applying for the Certification Examination. Only the most recent five years are reviewed for the required number of work experience hours (800 hours for curriculum students and 1800 hours for internship students).

REMEMBER, the Internship Hours Reporting forms must reach the Certification Office by the end of January 1984. The proper forms are located in the Fall 1983 issue of ATHLETIC TRAINING. DO NOT send any hours logs — only the proper form from the internship student and the supervising athletic trainer.

***EXTENDED BENEFITS

Effective October 1, 1983 and extending for a period of one year thereafter, death benefits involving insured members participating in the National Athletic Trainers Association Group Term Life insurance program will be increased at no additional premium. In the event loss experience during this policy period continues to be favorable, extended death benefits may be continued for the following year. Riders will be issued to all current insureds as soon as they become available. All new insureds will be told of this additional benefit and their certificates, too, will include the new rider.

GRAFFITTI

***Thanks to all of you who take the time to note the department to which your communication is directed on the outside of your mailing envelopes. This helps so very much in routing the tremendous amount of mail received in the National Office and saves a great deal of time and clerical expense for your Association.

***If your school library does not subscribe to ATHLETIC TRAINING, you may want to remind the librarian that the Spring '84 issue will be the beginning of a new volume and now is an excellent time to contact the National Office for a New Subscription Form.

***Your National Office Staff wishes you a safe and happy Holiday Season!

NATA Professional Education Committee DISTINGUISHED ATHLETIC TRAINING EDUCATOR AWARD

Nominations are being received for the first **Distinguished Athletic Training Educator Award** to be presented annually by the NATA Professional Education Committee in recognition of excellence in athletic training education:

I. Qualifications

To be nominated for the award, educators must have the following qualifications:

1. Current member of the National Athletic Trainers Association, Inc.
2. Member of a teaching faculty in the area of athletic training/sports medicine for at least ten (10) years.
3. Minimum of ten years of outstanding service in the area of athletic training education and research.
4. Recognized excellence in the field of athletic training education.
5. Outstanding service in district, state or national professional organizations concerned primarily with the field of athletic training.
6. Evidence of quality in publications and public speaking on topics in athletic training/sports medicine.

II. Nomination Procedures

Nominations may come from any certified athletic trainer, athletic training student, or faculty member of a college or university. The nominator must submit the following materials.

1. the candidate's current personal resume which includes:

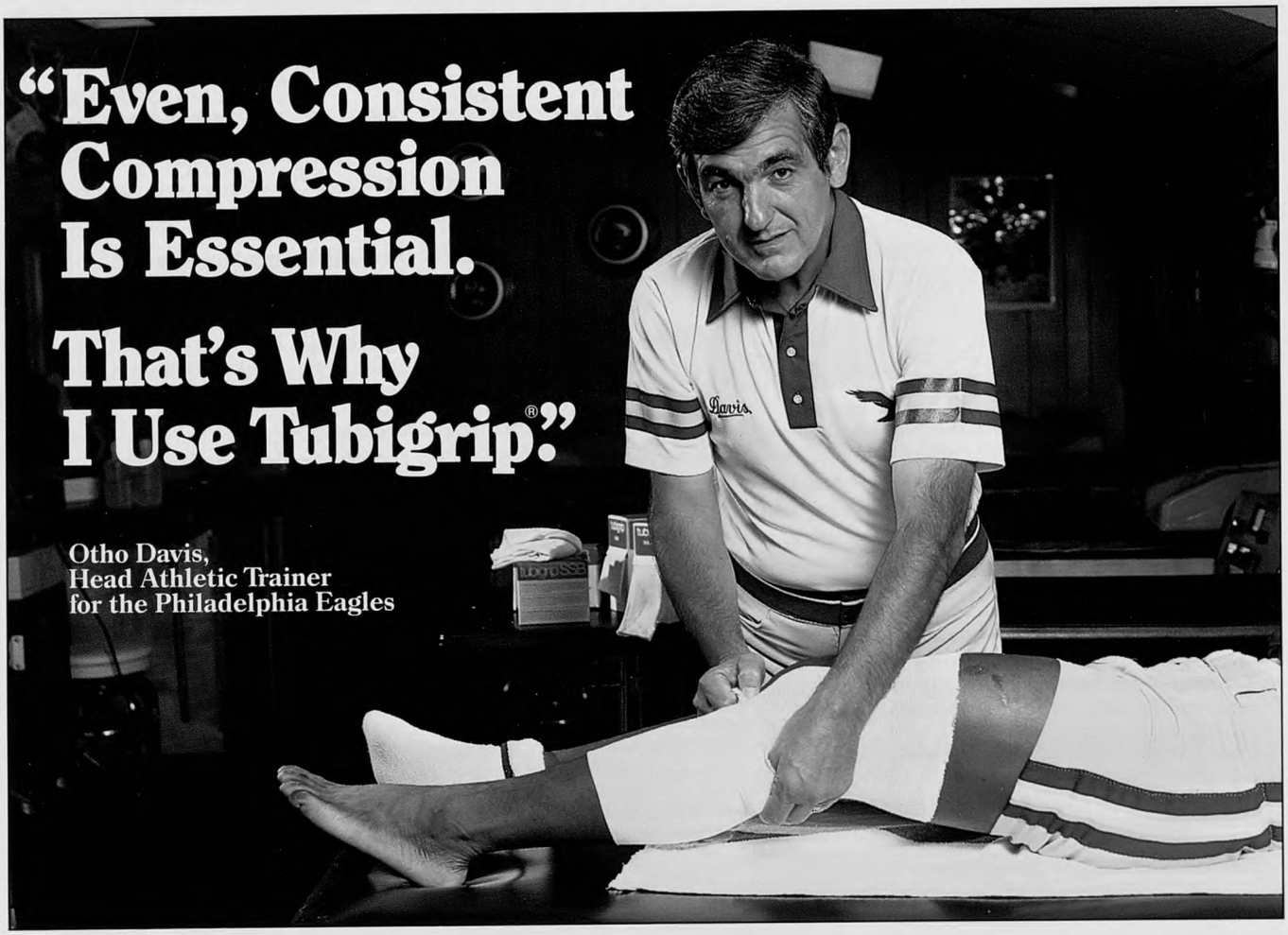
    a. academic background
    b. employment background
    c. published research and other publications (journal articles, books, etc.)
    d. course work taught (during past five years)
    e. classroom teaching innovations
    f. course work/curriculums developed
    g. professional memberships
    h. positions on state, district, or national level of the National Athletic Trainers Association, Inc.
    i. positions on state, district, or national level of related sports medicine professional organizations
    j. consultant work
    k. speaking engagements on community, state, regional, and national levels
    l. community service
    m. college or university service (i.e. committee involvement, thesis advising, etc.)
    n. any other pertinent materials

2. A minimum of three letters (additional letters may be submitted) from professional colleagues, administrators, or students providing detailed rationale in support of the candidate's nomination.

Nominations including the above materials should be sent to the Professional Education Committee Project Director, Honors and Award, and must be received by **May 1, 1984**. Presentation of the award will be made to the recipient at the 1984 NATA Annual Meeting and Clinical Symposium in San Antonio, Texas. Send nominations to:

Ken Murray
Athletic Department
Texas Tech University
P.O. Box 4199
Lubbock, Texas 79409

"Even, Consistent Compression Is Essential. That's Why I Use Tubigrip®."



Otho Davis,
Head Athletic Trainer
for the Philadelphia Eagles

"In my opinion, the advantage of Tubigrip® is that it gives you firm, even pressure. Tubigrip doesn't overlap the way an elastic wrap does.

With some of those elastic wraps, you get more cotton and less elasticity. You end up pulling it too tight, and then you're left with nothing there!

Also, if an elastic wrap is left on too long, too tight, you can very easily get a tourniquet effect.

If it's too loose, it's ineffective. With Tubigrip, you get a firm, even type of compression every time.

We use Tubigrip on new injuries to help control edema and reduce swelling. We also use the bandage to protect against turf burn, particularly on the forearms and elbows.

For injuries, I usually cut the desired length of Tubigrip and put it on double. But for some applications, such as an arm sleeve for protection against turf burn, the athlete knows what size he wants and likes, so he can come in and apply his own.

Tubigrip is also very effective at holding a dressing or ice bag in position. For example, we take an ice bag and use a double thickness of Tubigrip to make an envelope to secure the ice.

I've even used Tubigrip under adhesive tape. Sometimes a player needs the extra support or compression that Tubigrip provides. And when you have a player with some arthritic changes in the joint, Tubigrip helps hold the heat in.

Our players wear Tubigrip both at practice and in games. It's very comfortable and it's always worked effectively for us.

I think it's important for students and professionals alike to find out about new products and to keep informed on the latest topics in the athletic training field.

I take a position about keeping informed on the latest in sports medicine. It all comes down to how much time you devote to your educational reading.

Educational reading familiarizes me with new techniques and new products such as Tubigrip support bandages."

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For more information on Tubigrip and tubular support bandaging become a member of Tubigram™ an education service of Mark One® Healthcare Products Inc. Write to:

SETON® PRODUCTS DIVISION, MARK ONE HEALTHCARE PRODUCTS INC.,
TUBIGRAM EDUCATIONAL SERVICES, P.O. BOX 906, PARAMOUNT, CA 90723.

Guide to Contributors

Athletic Training, The Journal of the National Athletic Trainers Association, welcomes the submission of manuscripts which may be of interest to persons engaged in or concerned with the progress of the athletic training profession.

The following recommendations are offered to those submitting manuscripts:

1. Four copies of the manuscript should be forwarded to the editor and each page typewritten on one side of 8½ x 11 inch plain paper, triple spaced with one inch margins.
2. Good quality color photography is acceptable for accompanying graphics but glossy black and white prints are preferred. Graphs, charts, or figures should be of good quality and clearly presented on white paper with black ink in a form which will be legible if reduced for publication. Tables must be typed, not hand written. Personal photographs are encouraged.

   All art work to be reproduced should be submitted as black and white line art (either drawn with a Rapidograph [technical fountain pen] or a velox stat or PMT process) with NO tonal values, shading, washes, Zip-a-tone — type screen effects, etc. used.

   All artwork to be reproduced in black plus a second (or more colors) should be submitted as black and white line art (see above paragraph), with an Amberlith® or similar-type overlay employed for each area of additional color(s). Also, all areas of tonal value, shading, "washes", etc. should be supplied on a separate clear or frosted acetate or Amberlith® overlay. In addition, all areas to be screened (a percent or tint of black or color) should be supplied on an Amberlith® overlay.
3. The list of references and citations should be in the following form: a) books: author, title, publisher with city and state of publication, year; b) articles: family names, initials and titles of all authors, title of article, journal title, with abbreviations accepted as per Index Medicus, volume, page, year. Citations in the text of the manuscript will take the form of a number in parentheses, (7), directly after the reference or name of author being cited, indicating the number assigned to the citation bibliography. Example of references to a journal, book, chapter in an edited book, and presentation at a meeting are illustrated below:
   a. Knight K: Preparation of manuscripts for publication. *Athletic Training* 11 (3):127-129, 1976.
   b. Klafs CE, Arnheim DD: *Modern Principles of Athletic Training*. 4th edition. St. Louis, CV Mosby Co. 1977 p. 61.
   c. Albohm M: Common injuries in womens volleyball. *Relevant Topics in Athletic Training*. Edited by Scriber K, Burke EJ, Ithaca NY: Monument Publications, 1978, pp. 79-81.
   d. Behnke R: Licensure for athletic trainers: problems and solutions. Presented at the 29th Annual Meeting and Clinical Symposium of the National Athletic Trainers Association. Las Vegas, Nev, June 15, 1978.
4. In view of *The Copyright Revision Act of 1976*, effective January 1, 1978, all transmittal letters to the editor must contain the following language before manuscripts can be reviewed for possible publication: "In consideration of the NATA taking action in reviewing and editing my submission, the author(s) undersigned hereby transfers, assigns or otherwise conveys all copyright ownership, to the NATA in the event that such work is published by the NATA." We regret that transmittal letters not containing the foregoing language signed by all authors of the manuscript will necessitate return of the manuscript.

   Manuscripts are accepted for publication with the understanding that they are original and have been submitted solely to *Athletic Training*. Materials taken from other sources, including text, illustrations, or tables, must be accompanied by a written statement from both the author and publisher giving *Athletic Training* permission to reproduce the material. Photographs must be accompanied by a signed photograph release form.

   Accepted manuscripts become the property of the Journal. For permission to reproduce an article published in *Athletic Training*, send requests to the Editor-in-Chief.
5. Manuscripts are reviewed and edited to improve the effectiveness of communication between the author and the readers and to assist the author in a presentation compatible with the accepted style of *Athletic Training*. The initial review process takes from six to eight weeks. The time required to process a manuscript through all phases of review, revision, and editing, to final publication is usually six to eight months depending on the timeliness of the subject. The author accepts responsibility for any major corrections of the manuscript as suggested by the editor.

   If time permits, galley proofs of accepted papers will be sent to the author for corrections prior to publication. Reprints of the article may be ordered by the author at this time.
6. It is requested that submitting authors include a brief biographical sketch and acceptable black and white glossy photograph of themselves. **Please refrain from putting paper clips on any photograph.**
7. Unused manuscripts will be returned, when accompanied by a stamped, self-addressed envelope.

   Address all manuscripts to:
   Clint Thompson
   Jenison Gym
   Michigan State University
   East Lansing, Michigan 48824

The following recommendations are offered to those submitting CASE HISTORIES:

1. The above recommendations for submitting manuscripts apply to case studies as well but only two copies of the report need be sent to the Editor-in-Chief.
2. All titles should be brief within descriptive limits. The name of the disability treated should be included in the title if it is the relevant factor; if the technique or kind of treatment used is the principal reason for the report, this should be in the title. Often both should appear. Use of subtitles is recommended. Headings and Subheadings are required in the involved report but they are unnecessary in the very short report. Names of patients are not to be used, only first or third person pronouns.
3. An outline of the report should include the following components:
   a. Personal data (age, sex, race, marital status, and occupation when relevant)
   b. Chief complaint
   c. History of present complaint (including symptoms)
   d. Results of physical examination (Example: "Physical findings relevant to the physical therapy program were . . .")
   e. Medical history — surgery, laboratory, exam, etc.
   f. Diagnosis
   g. Treatment and clinical course (rehabilitation until and after return to competition) use charts, graphs when possible
   h. Criteria for return to competition
   i. Deviation from the expected
   j. Results — days missed
4. **Release Form**

   It is mandatory that *Athletic Training* receives along with the submitted case a signed release form by the individual being discussed in the case study injury situation. Case studies will be returned if the release is not included.

The following recommendations are offered to those submitting material to be considered as a TIP FROM THE FIELD:

1. The above recommendations for submitting manuscripts apply to tips from the field but only two copies of the paper need be submitted.
2. Copy should be typewritten, brief, concise, in the first or third person, and using high quality illustrations and/or black and white glossy prints.

Journal Deadlines

In order to avoid confusion and delays for any contributions to the Journal the deadlines for various sections of the Journal are provided below.

Send material for "Announcements", "Case Studies", "Letters to the Editor", and "New Products" to:

Steve Yates, Editor-in-Chief
P.O. Box 7265-Sports Medicine Unit
Wake Forest University
Winston-Salem, NC 27109

Manuscripts must be sent to:

Clint Thompson
Jenison Gym
Michigan State University
East Lansing, MI 48824
(517)353-4412

Information on upcoming events for the "Calendar of Events" section should be sent to:

Jeff Fair, ATC
Athletic Department
Oklahoma State University
Stillwater, OK 74074

"Tips From the Field" should be sent to:

Dave Burton
Duncanville High School
Duncanville, TX 75116

The Editorial Board will review papers submitted on an individual basis, work with the authors and prepare these papers for publication.

The deadlines are:

Journal	**Deadline**
Spring Issue	December 15
Summer Issue	March 15
Fall Issue	June 15
Winter Issue	September 15

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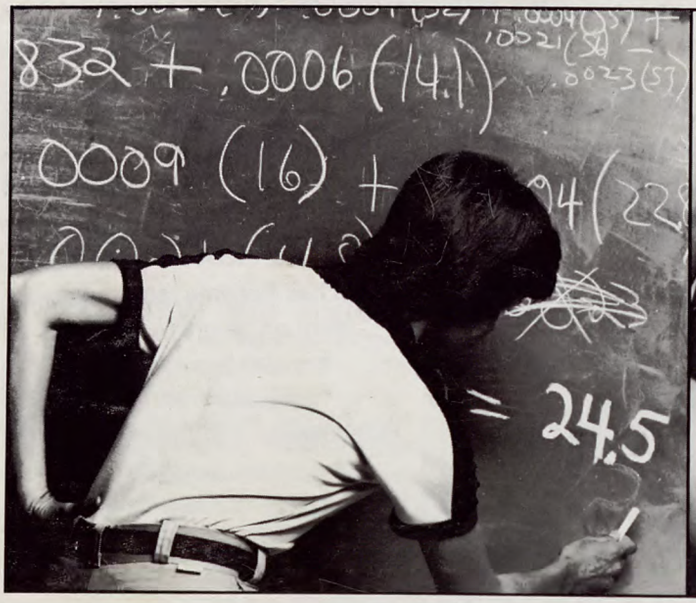

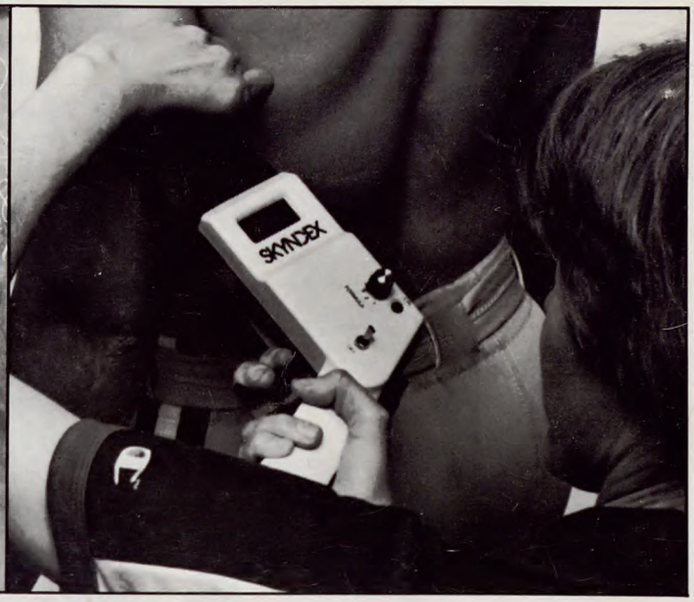

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