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PRESIDENT'S MESSAGE

The IAAF recognizes that the well-being of the athletes of our member Federations is one of its prime responsibilities. I am pleased that the members of the Medical and Anti-Doping Commission have worked hard to provide the health professionals of our member Federations with this newly-updated Medical Manual, and to design education programmes at the Regional Development Centres to aid them in caring for their athletes.

I would like to thank the members of the Medical and Anti-Doping Commission and the other contributors for their efforts in revising this Manual, and to Dr. Harmon Brown, Medical and Anti-Doping Commission member, and to the IAAF staff for their editorial work.

Lamine Diack
IAAF President

CHAIRMAN'S MESSAGE

The IAAF Medical and Anti-Doping Commission recognises that one of its major tasks is to assure the highest quality of athlete care provided by the health professionals in its member Federations, and is pleased to present this revised and updated edition of the IAAF Medical Manual. This Manual is intended for use as a resource for sports physicians and physiotherapists, and as a primary textbook for sports medicine and anti-doping courses given at the IAAF's Regional Development Centres.

We hope that this programme will continue to stimulate the continuing education of sports medicine professionals who work with all athletes in athletics, from novice to elite levels.

Juan-Manuel Alonso

Chair, IAAF Medical and Anti-Doping Commission

PREFACE

In recent years the IAAF has recognised that health care of all the athletes in its member Federations is an area which must be addressed if athletics is to continue to grow as a world-wide sport.

The role of the IAAF's Medical and Anti-Doping Commission is to aid all of its member Federations in providing for the health and safety of all athletes, from novices to world-class competitors.

The Medical and Anti-Doping Commission has prepared this revised and updated Medical Manual as part of a programme of medical and anti-doping education that is held at its various Regional Development Centres. The objectives of this programme are to aid each member in strengthening its medical organisation, and to provide its health care providers (physicians and physiotherapists) with updated information on sports medicine and doping, particularly as it relates to the sport of athletics.

We wish to thank Dr. Manuel Alonso, Chair of the Medical and Anti-Doping Commission, for his continuing support of this project, to the many members of the Commission and the other contributors for making this Manual a valuable sports medicine educational tool, and to the staff of the IAAF Anti-Doping Department for their work in assuring its finalisation. We would especially like to acknowledge Dr. Luc Magnus and Chris Butler of the IAAF for their thorough review of the manuscript and suggestions for its improvement.

A special thanks to the Technical Editor, Martha T. Brown, for making the text a readable entity. Any errors or omissions should be laid at the door of the Editor. We welcome any recommendations for future improvements.

C. Harmon Brown, M.D.

Editor

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NATIONAL SPORTS MEDICINE STRUCTURES

Each member Federation shall have a Sports Medicine Committee as a part of its basic organisational structure. The size and complexity of the Sports Medicine Committee may vary considerably, depending upon the size of the member Federation, the number of athletes served, and the geographic territory to be covered. However, the basic responsibilities of the Committee should be similar.

A. Objectives

- To raise the level and quality of health care and the quality of care provided to athletes at all levels, during both competition and training.
- To assure that the health care system is capable of delivering care uniformly, effectively, and consistently.
- To assure the highest quality of sports medicine knowledge and technical skills for the entire sports medicine team (professionals, coaches, scientists, and administrators) through the provision of training, continuing education, and other resources.

B. Responsibilities

1. Support for National Teams

- a. Prior to International Competitions
 - i. Assure the health and fitness of athletes selected for the competition.
 - ii. Provide qualified medical professionals (physicians, therapists, psychologists).
 - iii. Provide adequate supplies and equipment.
 - iv. Educate team personnel (athletes and coaches) concerning travel hygiene measures (see Chapter 12, *Infectious Diseases*).
 - v. Assure immunisations are appropriate for the site of the competition.
- b. During the Competition
 - i. Assure adequate treatment space—village and stadium.
 - ii. Establish treatment schedules and staffing—village and stadium.
 - iii. Assure proper food and water—village and stadium.
 - iv. Keep records of all illnesses and injuries, and treatments.
- c. During Year-Round Training
 - i. Assure a functioning health care system at National Team training venues.
 - ii. Develop a resource system for referral of athletes to qualified community practitioners.
 - iii. Provide Sports Sciences support services to qualified athletes:
 - Periodic physiological monitoring of the training process
 - Biomechanical analysis of technical skills

- Psychological skills training
- Nutritional analysis and education

2. Support National Competition Programmes

Provide a medical care system for national competitions such as national championships, major competitions, and international meetings (refer to *IAAF Competition Medical Handbook for Track and Field and Road Racing* for details).

3. Grassroots Programmes Support

- a. Develop health screening, health care, and education programmes at local and regional levels for Youth and Developmental athletes.
- b. Provide health and safety education for coaches.
- c. Integrate health and safety modules into the coaching education curriculum at all levels.

Reference

1. International Association of Athletics Federation. *IAAF Competition Medical Handbook for Track and Field and Road Racing: A Practical Guide*. IAAF: Monaco, 2006.

THE FEDERATION MEDICAL OFFICER

A. Responsibilities

1. Develop and strengthen the administrative, health care, and education functions of the sports medicine structure:
 - a. Medical care for athletes and teams
 - b. Medical care operation at major competitions
 - c. Education programmes for health professionals, athletes, and coaches
 - d. General health education for athletes at all levels
2. Assure the continuing operation of all aspects of the Federation's sports medicine structure.
3. Be responsible for the Federation's communication with the IAAF and Area Organisations concerning sport medicine matters, and advising or taking appropriate actions, when necessary.
4. Maintain liaison with the Federation Doping Control Officer (Medical and Doping should not be the same person).
5. Establish and enhance relationships with other appropriate national and international sports medicine organisations (i.e., IOC, FIMS, etc.).

B. Recommended Qualifications

1. Medical professional, licensed to practice by the State.
2. Member in good standing of the Federation.
3. Possess specific medical knowledge and experience in the sport of athletics, including basic understanding of the training process.
4. Member of national/international sports medicine organisations.
5. Available to commit time to the Federation and its sports medicine structure.
6. Personal qualities:
 - Energetic and enthusiastic about Federation sports medicine programmes and duties.
 - Communication skills—has the desire to share and disseminate health information to fellow professionals, coaches, and athletes.
 - Organisational skills—capable of developing and operating an ongoing sports medicine organisation.

Reference

See Appendix 1, *Olympic Movement Medical Code*.

PLANNING INTERNATIONAL TRAVEL

A. Organisation and Planning

For local, national, and international competition, the team physician is responsible for organising the medical staff, preparing the team for travel, and ensuring that the appropriate personnel and medical supplies are available to the athletes. Frequently, the team physician will also be responsible for the medical organisation of training sites. The team physician must prepare for routine and unexpected injuries, illnesses, communicable diseases, and even major catastrophic events when serving an athletic team “on the road.” Overall factors that must be considered include the fatigue associated with travel as well as acclimatisation to heat, cold, or altitude.

The *IAAF Competition Medical Handbook for Track and Field and Road Racing* is an excellent source for guidance concerning organising medical coverage of a major athletic event. See also Appendix 2, *Planning International Travel*, for a checklist of tasks involved in international travel.

B. Food and Drink

The medical staff should ensure that familiar foods and drinks are available for the athletes and staff.

Prevention and treatment of acute infectious diseases (especially traveler’s diarrhea) is extremely important (see Chapter 12, Part 2, *Infections at Large*). Gastrointestinal infections are caused primarily by fecally contaminated water or food. Sources of potential problems are uncooked vegetables, greens and fruits, unpasteurised milk or milk products and undercooked or improperly stored meat or fish represent the highest risks. Food in the athlete’s village, restaurants, and private homes is usually safe, but it is best to eat only well-cooked foods or peeled fruits. Athletes should avoid buying food from street vendors.

Where possible, travelers should drink only bottled water or carbonated beverages. If purification is necessary, water should be boiled three to five minutes or treated with iodine or chlorine drops. Most stadia and training facilities do not have potable water. Medical staff must make sure that adequate drinking water is available for their athletes during training and competition.

C. Immunisations

The medical staff should ensure that all required immunisations are updated prior to departure. These may include tetanus, immunoglobulin, diphtheria, pertussis, polio, measles, mumps, rubella, haemophilus, or others, depending upon the travel destination. The immunisation record should be kept with the athlete’s passport. (See Chapter 12, Part 1, *Infections Associated with Sports* for more information.)

D. Staff Selection

Personnel for the medical staff should be selected carefully. Team physicians and athletic trainers (physiotherapists) should have a background in athletics and familiarity with the biomechanics of the specific events. On longer trips to parts of the world where special conditions exist, teams of thirty or more athletes should be accompanied by one or two physiotherapists. At least two physicians and three or four physiotherapists are needed for teams of more than 75 athletes. Knowledgeable sports psychologists are also valuable on international trips.

E. Supplies and Equipment

The team physician must ensure that proper medical supplies and equipment are available for all possible on-the-field injuries, emergencies (including CPR), and routine medical problems both during travel and at the competition. He/she should not rely upon hosts to provide these essentials. After each trip, the supplies should be inventoried, restocked, and modified as necessary.

The medical staff should keep detailed records of all routine and emergency treatment rendered to athletes. Clear records are important for the individual athletes and for medical legal reason, and will help staffs prepare for future events.

Reference

1. International Association of Athletics Federation. IAAF Competition Medical Handbook for Track and Field and Road Racing: A Practical Guide. IAAF: Monaco, 2006.

PRINCIPLES AND ETHICAL GUIDELINES

A. Competence

Sports medicine professionals must be knowledgeable, educated, and experienced in the prevention and care of health problems, particularly those related to the sports for which they are responsible. (See also Appendix 1, *Olympic Movement Medical Code*.)

B. Sports Knowledge

Sports medicine personnel should have an in-depth knowledge of the sports for which they are responsible, including rules of competition. They must also understand the training process and the physical and mental demands training places upon an athlete. An active, wellness-oriented lifestyle enhances this understanding and improves rapport with coaches and athletes.

C. Confidentiality

An athlete has the right to expect the medical staff to observe confidentiality. When serving as a team physician, the physician is responsible to the athlete, the team administration and the coaches, so there must be clear guidelines concerning the disclosure of medical information. Disclosures should be made only to responsible personnel and only for determining the athlete's fitness to participate. Ideally, medical information should be considered privileged, until the athlete gives permission to release information to the team, the coach, or the media.

D. Communication

There must be mutual respect among all members of the medical staff and open communication concerning roles and responsibilities. In addition, medical personnel must communicate openly with coaches, athletes, parents, and family physicians.

E. Participation

The primary care team physician is responsible for determining an athlete's fitness to participate. This may occur during the pre-participation examination or after an injury is sustained in training or competition. The team physician may decide to involve special consultants to assist in these decisions.

F. Coordination

The primary care providers are responsible for coordinating the health care process among all persons involved in the sports programme, including the coach, other health professionals and para-professionals, sports scientists, administrators, families and family physicians, and the athletes themselves. Athletes must be instructed in health and safety practices and must bear a large degree of responsibility for their own welfare.

THE SPORTS MEDICINE TEAM

The sports medicine team is a group of health professionals whose major responsibility is the health and safety of the athletes. The size and scope of the health care team depends upon its locale, availability of other facilities and personnel, funding, and the number of athletes to be cared for.

A. Organisation

The health care team may be organised into a **Primary Care Team** and a **Secondary Support Team**:

PRIMARY CARE TEAM

Primary Care Sports Medicine Physician (M.D., D.O.)

Coaching Staff Physiotherapist Administration

SECONDARY SUPPORT TEAM

<i>Medical Specialists</i>	<i>Allied Health Professionals</i>	<i>Scientists</i>
Internal Medicine/ Cardiologist	Mental health practitioner Podiatrist	Physiologist Biomechanist
Orthopedist	Dentist	Strength coach
Physical Medicine & Rehabilitation	Nutritionist Nurse	
General Surgeon	Laboratory Personnel	
Ophthalmologist	Health educator	
Radiologist	Legal consultant Orthotist Chiropractor	

B. Responsibilities

1. Preventing Illnesses and Injuries

- a. Pre-participation assessment of athlete's health and fitness.
- b. Conditioning programmes—assess suitability and scientific rationale.
- c. Safety factors—inspect and approve training surfaces, equipment and personal athletic gear.
- d. Educate coach and athlete in safe practices (e.g. hygiene and safety).
- e. Environmental monitoring—check ambient temperature and humidity, assure that adequate fluids are available and that athletes and coaches are following safety guidelines.

2. Evaluating and Treating Illnesses and Injury

- a. Initial management
- b. Referral network—a referral system with written policies and procedures for referral must be in place.

3. Rehabilitation

Written guidelines must be in place covering an athlete's return to activity, including practice and competition.

4. Record-Keeping

Clear patient records, referral forms and consultation reports, and a daily treatment log must be maintained.

5. Legal

- a. Standards of care for sports medicine practices must be followed.
- b. Consent for care must be clearly spelled out, and informed consent obtained (in writing) for procedures; parental consent must be obtained for care of minors.
- c. Confidentiality of information and disclosure of information guidelines must be in place.
- d. Medications should only be prescribed with informed consent and explanation of possible side-effects; avoid prescribing medications banned by IAAF and the World Anti-Doping Agency (WADA).

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1. Herbert, D. L., and W. G. Herbert. Medico-legal issues. *In ACSM's Guidelines for the Team Physician*, R. C. Cantu, and L. J. Micheli (eds.). Philadelphia: Lea & Febiger, 1991.
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PRE-PARTICIPATION EXAMINATION

Every participant in sports should receive a thorough pre-participation medical examination (PPE) by a knowledgeable sports physician. The examination may vary depending on the local system and administrative/legal requirements, but should encompass a detailed medical history and complete physical examination. The IAAF also recommends a physical examination prior to participation in major international competitions.

A. Primary Objectives

There are several primary objectives in conducting the pre-participation examination. These include:

1. Detect Potentially Disabling or Life-threatening Conditions (both medical and musculoskeletal)

Such conditions are primarily cardiovascular, some of which may be clinically silent and not readily detected (see also Chapter 14, Part 1, *Cardiovascular Evaluation of Athletes*).

2. Screen for Conditions that Predispose to Illness or Injury

The PPE allows the physician to screen for medical and musculo-skeletal conditions that may predispose to illness or injury during training and competition. These may include acute or chronic conditions (especially those that have been untreated), injuries that have not been completely rehabilitated, and congenital-developmental abnormalities. Detection of these conditions allows for rehabilitation or other therapeutic interventions such as management of exercise-associated asthma.

3. Meet Administrative Requirements

The PPE is conducted in order to meet administrative requirements of the appropriate sports or governmental agencies.

B. Secondary Objectives

1. Assess General Health

The PPE may be the only contact that the athlete has with a health care provider, especially those athletes with limited access to health care. Many young athletes may have chronic and untreated health problems that may require on-going management.

2. Entry into the Health Care System

The PPE may establish a relationship with the health care system, and provide an opportunity to develop an ongoing health resource for the athlete.

3. Discuss Health and Lifestyle Issues

The interview offers an opportunity to discuss and counsel regarding a wide variety of topics, including nutrition and use of supplements, weight control practices, alcohol and substance abuse, proper training methods, and sexually transmitted diseases. Confidentiality must be assured by having a private interview room, either in a private office or in a private examination room as a part of group processing.

C. Personnel Qualifications

The examination should be carried out by a qualified physician, which may include a primary care sports physician, family practice physician, pediatrician, or orthopedic surgeon. Allied health professionals that may support the physician include the physiotherapist, nurse, and in some cases such specialists as physiologists and nutritionists.

D. Examination Formats

1. Private Office

The primary care physician's office is best for the PPE, especially if the physician is the athlete's personal physician. This allows for the use of complete medical records, knowledge of the athlete's history, and better physician-athlete rapport.

2. Medical Team

A team approach to the PPE can be valuable if there are cost considerations, the athlete does not have a personal physician, the athlete's personal physician does not feel qualified, or if the institution or team requires an organised system for a large number of athletes.

The team physician may organise primary care physicians, pediatricians, specialists such as orthopedists, and various para-professionals (physiotherapists, nurses, etc.).

a. Organisation of the Medical Facility (see Table 2-1)

Table 2-1. Medical facility organisation.

Stage	Purpose
Waiting area	Sign-in, registration, including careful instructions about filling out required forms
Vitals station	Height, weight, blood pressure, vision
Office examination	History review, physical examination performed by one physician for a given student-athlete
Specialty offices	Orthopedic assessment, cardiology evaluation, etc.
Optional stages	Educational and rehabilitation areas

b. Facilitating the Examination Process (see Table 2-2)

Table 2-2. Steps to facilitating the examination process.

Provide athletes in advance with information about the detailed nature of the examination and the appropriate attire to wear to lessen privacy concerns and increase efficiency.
Ensure separate areas for examining male and female athletes
Have a private individual counseling room for discussion of sensitive issues to maintain confidentiality and facilitate better communication
Enhance familiarity and continuity of care by enlisting the assistance of as many primary physicians as possible for the athletes being examined
Establish a clear protocol for referral to primary physicians, specialists, rehabilitation, or other medical evaluations for every athlete who is not cleared for participation because of illness or injury. If there is a team physician, he or she should keep—and follow up on—a list of athletes who are disqualified or who require further evaluation before final clearance. If the athlete is not cleared for the desired sport, the evaluating physician should counsel the athlete concerning alternate permissible activities.

E. Medical History (see Appendix 3, *Pre-participation Physical Evaluation*)

A complete medical history is the most important part of the PPE, and should be obtained from the athlete and, when appropriate, the athlete's parents. The physician must ensure that the athlete and parents understand the questions and provide help if there is a possibility of illiteracy, or if interpreters are required.

The medical history should include the following specific information:

1. A history of any significant medical diseases including cardiovascular disease, pulmonary disease, diabetes, mononucleosis, hypertension, anemia, hepatitis, ulcers or sexually transmitted diseases. Any weight changes, especially in the past six months, should be noted.

2. A history of any medications for acute conditions or chronic disease. The proper use of this medication should be reviewed with the athlete to ensure that the therapy is maintained. Unintentional misuse of the medications, especially of non-steroidal anti-inflammatory drugs, should be discussed. Athletes should be aware that prolonged use of non-prescription medications may cause significant bleeding disorders, liver or kidney injury, or other side effects. Supplement use should be determined, and the athlete made aware of their potential untoward use, including the possibility of their containing banned substances.

3. Medications for exercise-induced bronchospasm or other pulmonary disease should be discussed with the patient. Athletes with asthma or exercise-induced bronchospasm should be made aware of the need to obtain a Therapeutic Use Exemption (TUE) if involved in national or international competition (see also Chapter 14, Part 2, *Asthma and Exercise-Induced Bronchospasm*).

4. For female athletes, a history of any oral contraceptive use should be obtained and the age of menarche and the menstrual cycle should be discussed. A history of pregnancy, deliveries, and sexually transmitted disease should be obtained. Determine if there is a relationship between missed periods and heavy training activities. Determine if severe menstrual cramps regularly limit routine activities or athletic participation.

5. A history of drug use, including alcohol, recreational drugs, and/or tobacco. Short- and long-term effects of these drugs should be discussed.

6. Banned substances should be discussed with the athlete to prevent any inadvertent use. The athlete should be provided with a list of banned substances or a resource where information concerning banned substances can be obtained. (See Chapter 15, *Drugs in Sports/Doping Control*.)

7. A history of allergies. This should include allergies to medications, foods, and stings (Hymenoptera, e.g. bees, wasps, yellow jackets; ants). Appropriate actions and medications for life-threatening emergencies use should be addressed. Seasonal environmental allergies should also be discussed.

8. A cardiovascular history, particularly for athletes under the age of 30. Any history of cardiovascular disease, syncope or near syncope, arrhythmias, chest pain, fatigue, hypertension or pertinent family history should be obtained. Hypertrophic cardiomyopathy, outflow tract obstruction, conduction abnormalities and arrhythmias or valvular problems including aortic stenosis or mitral valve prolapse should be diagnosed when possible. A history of Marfan's syndrome, sickle cell anemia, Wolff-Parkinson-White syndrome or other syndromes should be obtained.

9. A neurological history. Determine if the athlete has ever been unconscious, had a severe head injury or concussion, or has a history of seizures, cervical spinal cord neuropraxia with transient quadriplegia, cervical spinal stenosis, congenital fusions, cervical instability, or cervical or lumbar disc disease.

10. A history of environmental trauma including hyperthermia, hypothermia, altitude sickness or other. Heat problems may include frequent muscle cramps, heat exhaustion, heat stroke and/or difficulty acclimatizing to heat.

11. A pulmonary history including any history of asthma, exercise induced bronchospasm, and seasonal/environmental allergies.

12. A history of skin problems should include any infectious diseases such as herpes simplex, scabies, pubic lice, molluscum contagiosum, impetigo, and sexually transmitted disease.

13. A history of previous injuries. Most athletic injuries are recurrent injuries, so it's important to ask, "Were you injured last season?" Inquire about the mechanism of injury, treatment obtained, and whether the injury was rehabilitated adequately. Ask about any fractures, dislocations, or significant joint disease. If any current abnormalities are found on examination, provide the athlete with a plan to obtain a definitive diagnosis and treatment.

14. An ophthalmologic history to determine whether the patient wears glasses, contact lenses, or protective eye wear. Problems with vision or eyes should be discussed. Especially important is whether there have been previous eye injuries including orbital fracture, hyphema, eyelid or globe laceration, or surgery. Depth of field is very important in triple jumpers, long jumpers, and relay runners.

15. A history of immunisations. Diphtheria, tetanus, and pertussis (DTP), and polio are standard children's vaccines in many countries. Measles, mumps, rubella (MMR), and hemophilus are also frequently given. Everyone should have an updated tetanus immunisation. Hepatitis B immunisation may be considered for sexually active individuals and immunoglobulin for non-immunised individuals exposed to infections. Requirements for additional immunisations will vary depending upon the travel destination. As an example, hepatitis A immunisation is highly recommended for athletes travelling to endemic regions.

16. The use of any protective equipment such as braces, pads, and eye protection should be ascertained and discussed.

17. A history of current training volume and intensity (if any). E.g. how many hours training per week, how many kilometres run per week, how many strengthening sessions per week, etc.

F. Physical Examination (see Appendix 3, *Preparticipation Physical Evaluation*)

The examining physician should start by measuring the athlete's height and weight; for junior athletes these measurements should be compared to a standard growth chart. The measurement of body composition can be important in determining the need for counseling about diet, disordered eating, and weight management (gain, maintenance, or loss). The IAAF book *Too Thin to Win?* is an excellent resource about proper and disordered eating. Counseling about the Female Athlete Triad may be provided to the athlete. The following components should comprise the remainder of the examination:

1. Visual acuity should be determined using a standard Snellen Eye Chart. Cases of unusually poor vision (including eye loss) should be discussed with the athlete and his/her parents. Options for visual correction, protective lenses, or eye wear should be discussed. Anisocoria (unequal size) of the pupils should be noted, as this could be important following a head injury.

2. The cardiovascular system should be evaluated. Blood pressure should be measured and the pulse should be checked for rate and rhythm. A large blood pressure cuff should be used for athletes with biceps greater than 33 cm in diameter.

3. The heart should be auscultated in both the seated and supine positions. Functional murmurs can often be differentiated from pathological murmurs such as hypertrophic cardiomyopathy and aortic stenosis by maneuvers such as deep inspiration, Valsalva's maneuvers, rising from a squatting position, and lying on the left side. Although murmurs are frequent in adolescents, questionable murmurs should be evaluated to obtain a definitive diagnosis. Athletes exhibiting signs

of mitral or aortic valve stenosis, aortic insufficiency, prolapsing mitral valve or coarctation of the aorta, or any suspicion of post-infectious carditis should be examined by a cardiologist. (See also Chapter 14, Part 1, *Cardiovascular Evaluation of Athletes*.)

4. When equipment and personnel are available, an electrocardiogram at rest may be done during the initial evaluation.

5. The lungs should be auscultated and any abnormalities recorded. The abdomen should be examined for pain, masses, or organomegaly with particular attention given to the liver and spleen.

6. The skin should be observed and palpated. Particular attention should be given to any infectious disease or suspicious nevi or lesions.

7. Male athletes should be examined for any undescended testicles, presence or absence of the testicles in the scrotum, or any masses. Testicular cancer is the leading cause of cancer deaths in men 20–35 years of age. The examiner should check for inguinal, femoral, or lower abdominal hernias.

8. Whether a genitourinary examination is performed on a female athlete will depend on the athlete's age, cultural practices, and whether she has other routine medical care. Adult and sexually active females should have a Pap and pelvic examination at least once a year. All women should receive instructions about self breast examination.

9. Tanner-Whitehouse staging of pubertal status should be a part of the examination for all pre-pubertal and pubertal athletes.

10. The musculoskeletal examination should be thorough. The cervical-thoracic and lumbar spine, hips, and all extremities should be checked for normal range of motion, strength and endurance. The general habitus should be observed and any asymmetry, acute or chronic swelling or joint enlargement, surgical scars, or other abnormalities should be noted. Leg length inequality with a disparity in strength or flexibility makes an athlete more susceptible to acute strains and overuse syndromes.

The athlete should have full range of motion of the spine. A neurological examination should be performed including Spurling's axial compression test, Lhermitte's sign, deep tendon reflexes of the biceps, triceps, brachial radialis, patella and achilles, and sensory examination of the upper and lower extremities. Special attention should be given to any indication of peripheral nerve entrapment or nerve root injury.

A shoulder examination should include tests for full range of motion with emphasis on the rotator cuff, deltoid, and trapezius. The cervical, thoracic, and scapular musculature should be evaluated.

The elbows should be checked for full range of motion and particularly in throwers, the medial and lateral compartments examined for pain, asymmetries, or neurological entrapment syndromes. For throwers, the hands and wrists should also be checked.

Knee examination should test for full range of motion and especially for ligamentous stability.

Ankle and foot examination should include tests for stability, significant gait abnormalities due to pronation, genu valgus or varus, leg length differences, or other manifestations of malalignment syndromes.

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SPORTS RULES AND ATHLETE SAFETY

The rules of athletics are designed primarily to ensure that competition is fair and equitable for all participants. However, IAAF rules concerning the competition facilities, equipment, and environmental conditions have been put into place in an effort to provide optimal safety and health for the athlete. The health care team that is involved in athletics should be familiar with these rules, and be prepared to evaluate the competition venues, equipment, and meeting conduct, and ensure that rules pertaining to safety and health are enforced. Further, many incidents that occur during the competition may cause specific and serious injuries that will require prompt, appropriate action by the health providers (see Chapter 8, *Emergency Care*). Staff, equipment, supplies, and evacuation services must be in place to meet these emergencies, even though they are uncommon.

A. Administration

1. International Officials (Rule 105)

At Olympic Games, World Championships, World Cups, Continental, Area, and Regional Championships, the IAAF shall appoint a Medical Delegate (Rule 113) and a Doping Control Delegate (Rule 114). Their responsibilities are to ensure that adequate facilities, staff, and equipment are available for medical care and emergency management, and that doping control facilities and staff are suitable to meet IAAF requirements. For more details, the Medical Delegate and the medical care organisation should refer to the *IAAF Medical Handbook for Track and Field and Road Racing: A Practical Guide* (2006). The Doping Control personnel should refer to the current edition of the *Procedural Regulations for Doping Control*.

B. Competition Rules with Medical Implications

1. Shoes (Rule 143.2)

Competitors may compete in bare feet or with footgear on one or both feet. (Athletes who run barefoot on surfaces to which they are not accustomed may develop severe blisters. Running barefoot during road or cross-country races subjects athletes to possible puncture wounds, lacerations, or frost-bite during cold weather.)

The shoes must not be constructed so as to give an athlete any unfair additional assistance, including the incorporation of new technology that will give the athlete any unfair advantage.

The shoe may have up to 11 spikes; spikes must not project more than 9 mm, except in the high jump and javelin throwing where it shall not exceed 12 mm.

The sole may be up to 13 mm thick in the high jump and long jump, and the heel no more than 19 mm in the high jump.

2. Obstruction (Rule 163.2)

Any competing runner or walker who jostles or obstructs another competitor, so as to impede his or her progress, shall be liable to disqualification. (Spike wounds or lower leg lacerations can occur whether there is outright interference or not. These will require wound care and tetanus prophylaxis.)

3. Assistance to Athletes (Rule 144)

In general, athletes may not be given assistance during competition. However, Rule 144.2 (b) states: “Physiotherapy and/or medical examination/treatment necessary to enable an athlete to participate or continue participation once on the competition area may be provided by members of the official medical staff appointed by the Organising Committee and clearly identified by arm-bands, vests or similar distinctive apparel. Accredited team medical personnel approved by the Medical or Technical Delegate specifically for the above purpose may be permitted in medical treatment areas outside the competition area. In neither case shall the intervention delay the competition or an athlete’s trial in the designated order. Such attendance or assistance by any other person whether during the competition or immediately before the competition once athletes have left the Call Room is assistance.”

C. Equipment and Facilities

1. Starting Blocks (Rule 161.1)

Starting blocks must be rigid, and capable of being fixed to the track by pins or spikes. The foot-plates on the frame may be adjustable, but must allow no movement during the start. They must be secured by clamps or a locking mechanism. (The locking mechanisms should be checked to assure that they will not slip, as a slipping block may cause a hamstring injury.)

2. Hurdles (Rule 168.2)

The top bar should be made of wood, the edge rounded, and firmly fixed at the extremities. The bar should be 7 cm wide and between 1.0 and 2.5 cm thick. A force of at least 3.6 kg and not more than 4.0 kg applied at the centre-top edge is required to overturn the hurdle. (A damaged, rough cross-bar may cause abrasions; a hurdle that is too heavy to tip when struck may cause the athlete to fall.)

The hurdles have five different height settings. In newer hurdles the weights are automatically adjusted when the height setting is changed; with older hurdles this must be done manually. If the weights are in the rearmost position with the lowest height of 76.2 cm, the force against a young competitor may be up to 6–7 kg.

3. Steeplechase (Rule 169)

The hurdles are 0.914 m high for men and 0.762 m high for women, and weigh 80–100 kg. The height and depth of the water jump are now the same for men and women. The water jump area should be covered with a synthetic surface, “or matting or synthetic padding, fixed in place, to allow spikes or shoes to grip satisfactorily.”

4. Vertical Jumps-Jumping and Landing Areas

a. High Jump (Rule 182.10)

The landing area shall measure not less than 5 m x 3 m, but it is recommended that it should not be less than 6 m x 4 m x 0.7 m in major international competitions

b. Pole Vault (Rule 183.11)

The landing area shall measure not less than 5 m x 5 m. For major international competition the landing area shall not be smaller than 6 m long (excluding the front pieces) x 6 m wide x 0.8 m high. The front pieces must be 2 m long.

The sides of the landing area nearest the box shall be placed 10 to 15 cm from the box and shall slope away from the box at an angle of approximately 30 degrees.

The stop board's upper edge shall be rounded with a radius of 5 up to 50 mm.

(Most injuries in the vertical jumps occur when the athlete strikes the ground outside the landing area, or falls from the landing pad onto the ground. Some older landing pads that are too small, as well as makeshift pads, have been responsible for severe injuries, including concussions, skull or cervical fractures, and quadriplegia.)

5. Horizontal Jumps—Takeoff and Landing Areas

a. Long Jump (Rule 185.5; 185.6)

The distance from the take-off board and the far end of the landing area shall be at least 10 m. The board shall be between 1 m and 3 m from the nearer end of the landing area.

b. Triple Jump (Rule 186.3; 186.4)

The distance from the take-off board to the far end of the landing area shall be at least 21 m. The distance from the take-off board to the nearer end of the landing area shall be: for men—13 m; for women—11 m for international competition. For other competition, the distance shall be appropriate for the level of competition.

(The landing area for both long and triple jumps should be filled with fine sand and should not contain any materials [rocks, gravel, debris] that could cause injury.)

6. Throwing Events (Rules 187–193)

a. Associated Risks

Most severe and fatal injuries in athletics are associated with the throwing events. Although the discus and hammer are thrown from protective cages, an implement may still strike officials, athletes, or spectators who venture imprudently into the field in or around the landing area. While landing areas are usually marked by flags and defined by chalk lines, these do not restrain errant implements from flying far beyond these boundaries. Everyone must be cautioned repeatedly to remain alert not only during the competition, but especially during the warm-up period, as well as during training.

b. Cage and Landing Sector

The landing sector for both discuss and hammer has been narrowed to 34.92°.

For the hammer cage, the height of the netting panels must be at least 7 m at the rear of the cage. Two movable netting panels 2 m wide and 10 m high are attached at the front of the cage and adjusted separately for left- and right-handed throwers. The netting shall be of sufficient strength to prevent the implement from passing through the panel. Note that hammer throwing is now also a women's event; the women's hammer weighs 4 kg.

c. Taping of the Hand

Tape on the hand may be used only to cover an open cut or wound. A physician may be called upon to verify the need for such taping.

7. Long Distance, Road Race, Cross-Country and Race Walking

a. Safety (Rules 230.8; 240.8; 250.8)

Roads used for the competition are closed in both directions, that is, not open to motorised traffic.

b. Medical (Rules 230.8 c, d; 240.8 b, c)

- i. A hands-on medical examination during the progress of an event by designated medical personnel clearly identified by the Organising Committee shall not be considered as assistance.
- ii. A competitor must retire at once from the race if ordered to do so by a member of the official medical staff appointed by the Organising Committee. Such staff shall be clearly identified by armbands, vests, or similar distinctive apparel.

c. Refreshments

i. Track events (Rule 144.4)

Events of 5000 metres or longer: may provide water and sponges if weather conditions warrant.

ii. Road Races and Cross-country (Rule 240.9 a–e; 250.8)

Water and suitable refreshments must be at the start and finish of all races.

- a) Races up to 10 km: drinking/sponging or refreshment stations at approximately 2–3 km intervals if weather conditions warrant.
- b) 10 km or longer: refreshments every 5 km; drinking/sponging stations (water only) midway between refreshment stations, or more frequently if weather conditions warrant.

iii. Race Walks (Rule 230.9 a–f)

Water and other suitable refreshments must be available at the start and finish of all races.

- a) All events on the track or road: drinking/sponging stations at suitable intervals.

- b) Over 20 km: refreshments at 5 km and thereafter every 5 km or every lap; drinking/sponging stations mid-way between refreshments.

(Drinking and refreshment stations are usually the responsibility of the road race/race walk management team. However, it is important to ensure that drinking water is available at all drinking/sponging stations, and not sponges alone. Sponging is ineffective in lowering core temperature, although it may produce transient skin sensations of coolness. Drinking fluids is critical.)

Current rules do not address the issue of scheduling races to minimise heat injury, other than to recommend that races be held in April–May or September–December. This is hardly adequate advice for tropical climates or the Southern hemisphere! Races should be held in the early morning or late afternoon–early evening, and not the heat of the day. Guidelines issued by the American College of Sports Medicine should be followed as closely as possible (see Appendix 4, *ACSM Position Stand on Heat and Cold Illnesses During Distance Running*). The team physician, medical committee, or IAAF Medical Delegate may need to become the athletes' advocate with the Organising Committee, insisting that distance races, road races, and race walks be held under the safest possible conditions, including scheduling, adequate drinking stations, and emergency personnel and facilities.

PRINCIPLES OF TRAINING

Training programmes are designed to improve performance by developing the appropriate energy sources, increasing muscular structures, and improving neuro-muscular skill patterns. Sports medicine professionals must be familiar with the basic principles and processes of training, so that they can evaluate training programmes and determine their adequacy in maintaining an athlete's health and preventing injury.

Training theory encompasses all aspects of fitness knowledge, including social, psychological, and scientific. The coach uses this information, along with knowledge about the athlete as an individual, to devise the most effective training programme. The scope of training theory is illustrated in Figure 3-1.

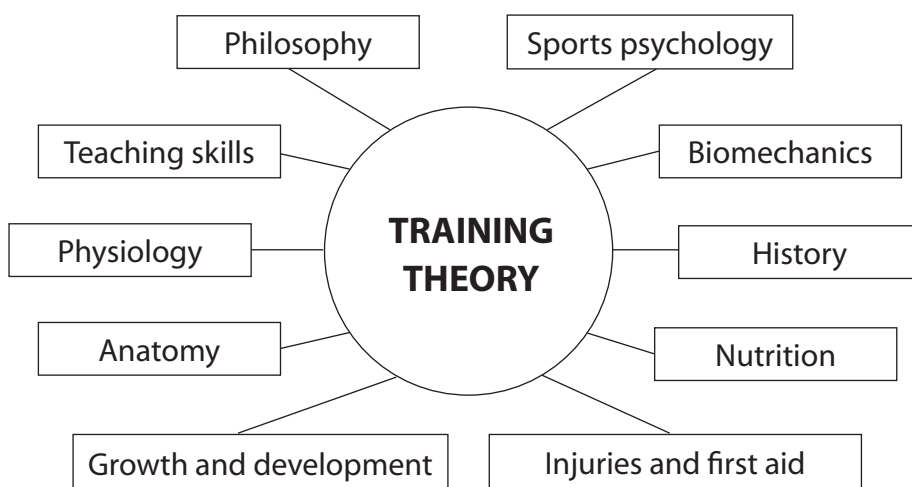


Figure 3-1. Scope of training theory.

A. Principles

1. Progressive Loading (“Overload”)

Biological systems can adapt to loads that are higher than the demands of normal daily activity. Training loads must be increased gradually, however, to allow the body to adapt and to avoid injury (system failure due to overloading). Varying the type, volume, and intensity of the training load allows the body an opportunity to recover, and to over-compensate (Figure 3-2). Loading must continue to increase incrementally as adaptation occurs, otherwise the training effect will plateau and further improvement will not occur (Figure 3-3).

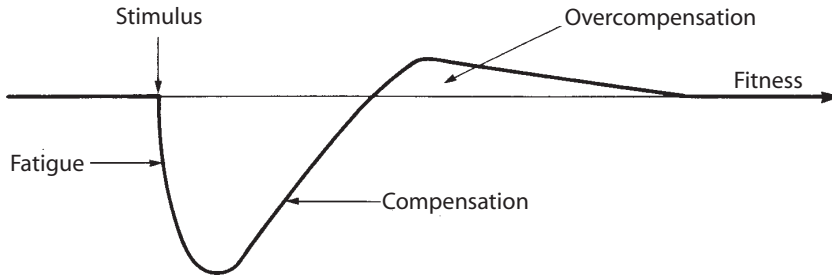


Figure 3-2. The law of overload.

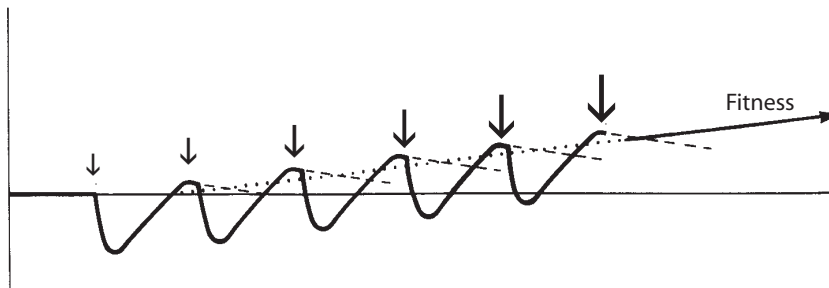


Figure 3-3. Principle of progressive overload—optimal improvement.

2. Adaptation

Adaptations to the demands of training occur gradually, over long periods of time. Efforts to accelerate the process may lead to injury, illness, or “overtraining” (see Part 2, of this chapter *Restoration, Recovery, and Overtraining*). Many adaptive changes reverse when training ceases. Conversely, an inadequate training load will not provide an adequate stimulus, and a compensatory response will not occur. Figure 3-4 illustrates the effects of various training loads.

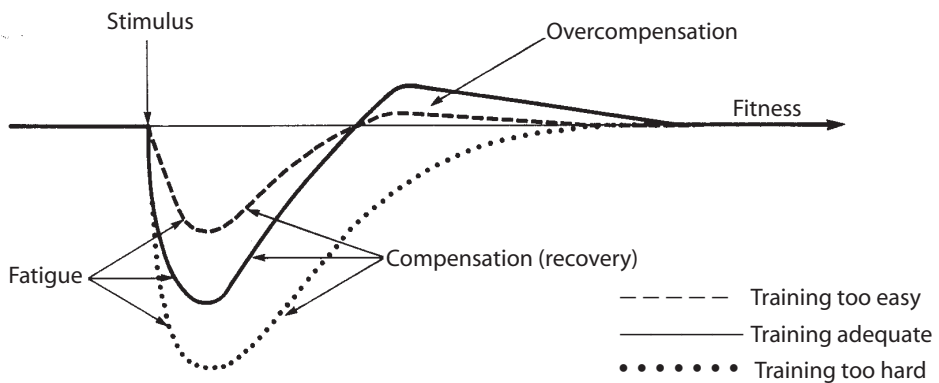


Figure 3-4. Different training loads have different effects on the athlete's recovery.

3. Specificity

Energy pathways, enzyme systems, muscle fiber types, and neuro-muscular responses adapt specifically to the type of training to which they are subjected. For example, strength training has little effect on endurance. Conversely, endurance training activates aerobic pathways, with little effect on speed or strength. Even so, a well-rounded training programme should contain a variety of elements (aerobic, anaerobic, speed, strength, flexibility), and involve all of the major muscle groups in order to prevent imbalances and avoid injuries.

4. Reversibility

A regular training stimulus is required in order for adaptation to occur and to be maintained. Without suitable, repeated bouts of training, fitness levels remain low or regress to their pre-training levels.

5. Variation and Recovery

Muscle groups adapt to a specific training stimulus in about three weeks and then plateau. Variations in training and periods of recovery are needed to continue progressive loading, without the risks of injury and/or overtraining. Training sessions should alternate between heavy, light, and moderate in order to permit recovery. The content of training programmes must also vary in order to prevent boredom and “staleness”.

6. Individual Response

Each athlete will respond differently to the same training stimulus. There are many factors that alter the training response: genetics, maturity, nutrition, prior training, environment, sleep, rest, stress, illness or injury, and motivation, to name a few.

7. Periodisation of the Training Cycle

The training programme must consist of a variety of elements, including cardio-respiratory (aerobic) fitness, general strength, anaerobic fitness (power), speed, neuro-muscular skills development, flexibility, and mental preparation. The emphasis placed upon each of these elements must vary during the training year, but will also depend on the athlete’s event and level of experience and maturity. Generally, basic preparation for all events should focus on general strength and aerobic fitness.

Training cycles usually last about 3 weeks, with a week of lower-intensity recovery before starting the next cycle. Skills acquisition should not be emphasised during a high-intensity training cycle, but should be reserved for periods of lower volume and intensity.

8. Maintenance

Gains achieved during high-intensity training periods can be maintained with a moderate level of work. Thus, by means of periodisation, some elements can be maintained with less work, while other elements are stressed.

B. Planning the Training Programme

1. Elements of Training and Fitness

A fitness training programme encompasses five basic biomotor abilities: strength, endurance, speed, flexibility, and coordination (Figure 3-5). Other elements that must be considered in a holistic programme include: specific skills acquisition, psychological training, and competition preparation.

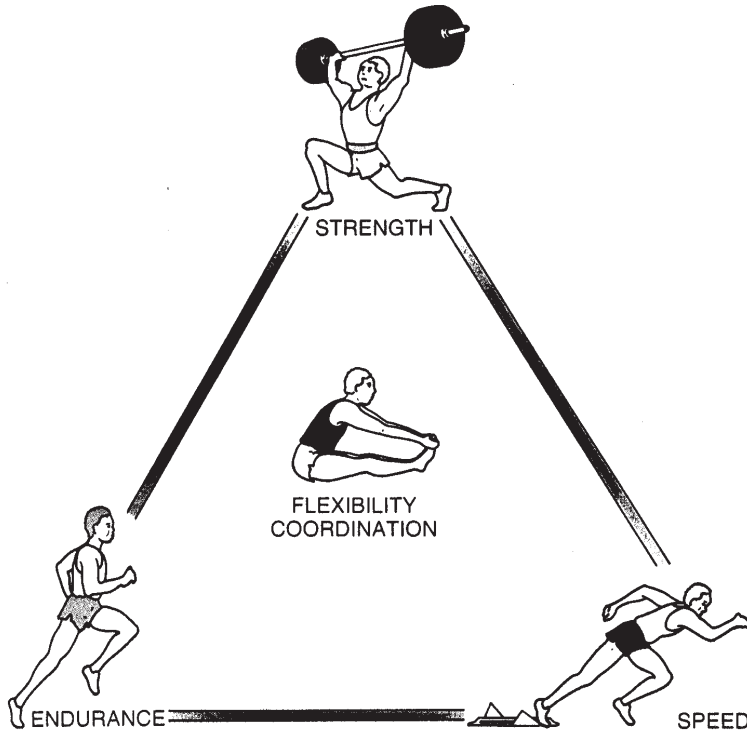


Figure 3-5. Relationship of the biomotor abilities.

A complete training programme must encompass all of the above elements. However, not all elements can receive equal emphasis throughout the training cycle. Many factors determine the type of training programme, and the stress placed upon each element. These include the age and sports maturity level of the athlete, his or her prior state of fitness, and the event(s) for which the athlete is preparing (Figure 3-6).

Although these biomotor elements are thought of as discrete entities, they are actually closely interrelated (Figure 3-7). The application of a training programme will impinge on a number of systems, and the coach must understand these relationships when devising a training programme.

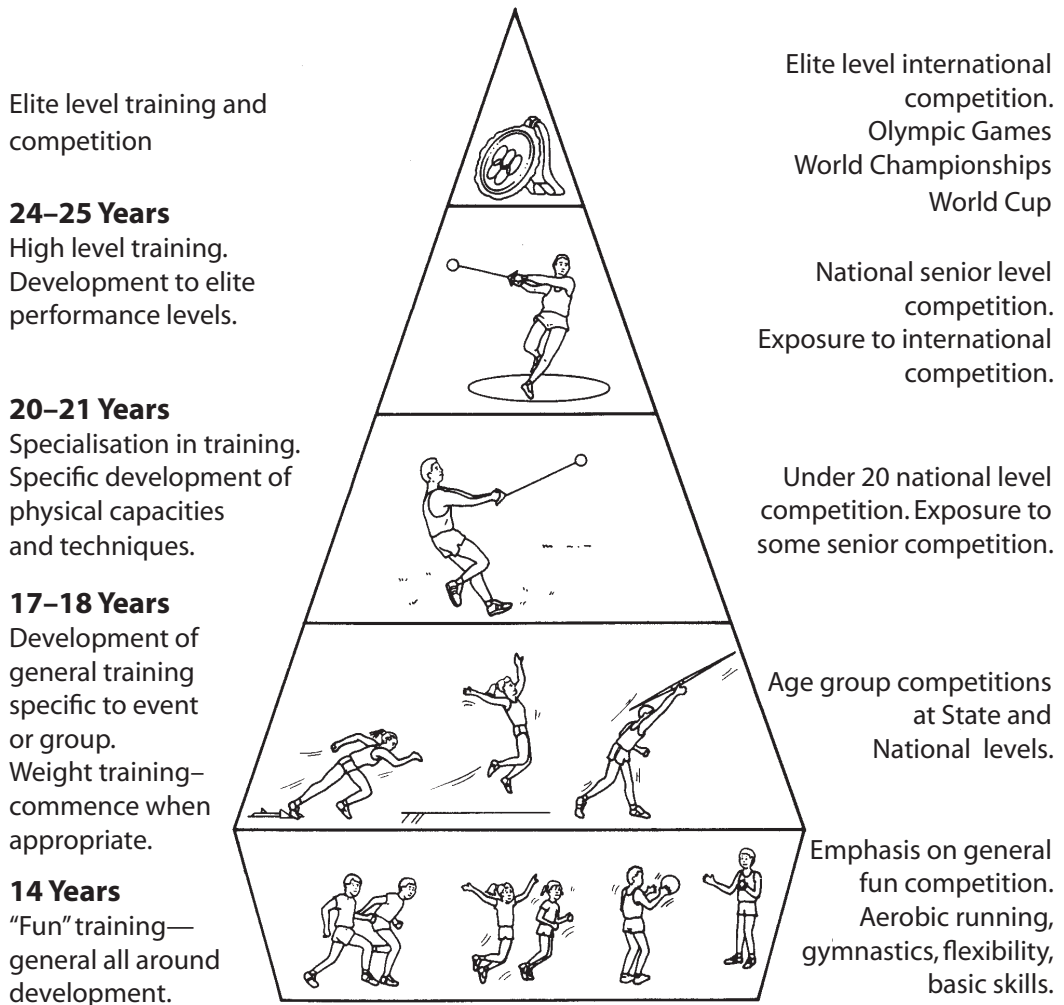


Figure 3-6. Training at any time must be seen as part of a long-term plan.

2. The Training Programme

The training process is divided into several periods (periodisation). These periods are usually termed as—

- General preparation
- Specific preparation
- Competition, and
- Transition (active rest)

The emphasis placed upon the various elements of training will vary both in *volume* and *intensity*. Volume refers to the total *quantity* of work, i.e., metres of running, or kilograms lifted during strength training. Intensity indicates the *quality* of training, usually in reference to the athlete’s maximal capability in that activity (percent of best performance).

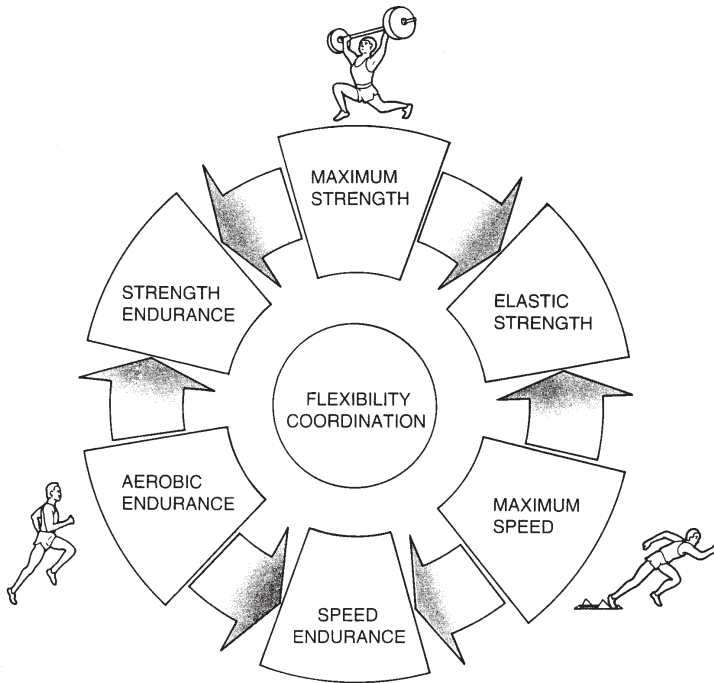


Figure 3-7. Inter-relationship of the various motor abilities.

During the preparatory periods, the volume of training is gradually increased, while the intensity is kept lower (Figure 3-8). As the competition phase is reached, the volume is slowly reduced as the intensity is raised. If both volume and intensity remain high, overtraining may occur (see Chapter 3, Part 2).

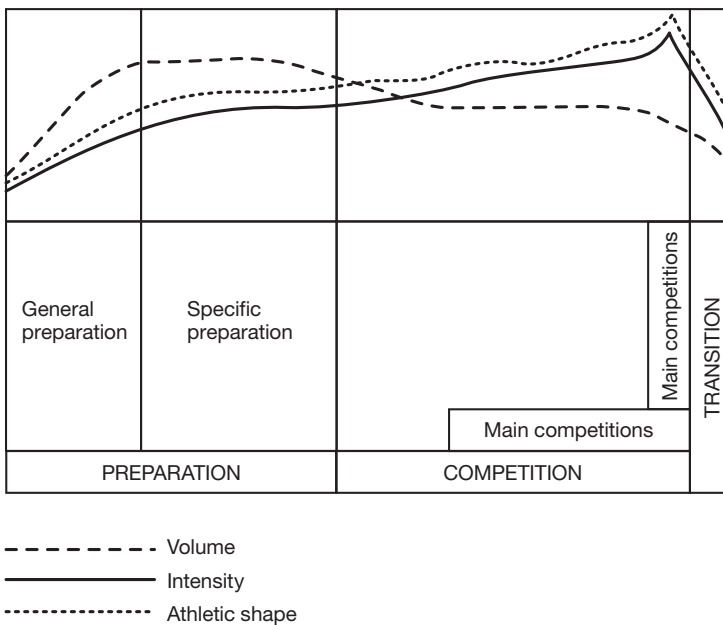


Figure 3-8. The general structure of periodised training.

RESTORATION, RECOVERY AND OVERTRAINING

Whenever a group of athletes is subjected to a training programme, some will respond optimally to the training stimulus, a few will not be adequately trained (undertrained), and others will experience an overly stressful response to the training load (overtraining).

A systematically applied, gradually progressive training load is required for an athlete to adapt and improve performance. However, for this adaptation to take place, adequate recovery strategies must be utilised. As the stressors associated with training gradually increase, so must the implementation of a broad range of recovery and restoration modalities be carefully included in the training process. While this concept may seem counter-intuitive to many highly-motivated athletes and coaches, it should be considered as integral to the training programme, i.e., “invisible training.”

It is the cumulative effect of training *and* non-training stressors, along with “under-recovery” that leads to performance decrements and the “overtraining syndrome.”

A. Restoration and Recovery

Restoration and recovery measures encompass a broad range of techniques, both active and passive. Recovery should include the physiological, psychological, and social realms.

1. Plan Training

The first step in preventing under-recovery is for the athlete and coach to develop a carefully-planned training strategy, with a periodisation system and allowances for “active rest.”

2. Self-monitoring

The athlete should keep a training diary and record not only the results of each workout, but also his or her subjective responses to it. Such an assessment might utilise such measures as the Rating of Perceived Exertion (RPE) and the Total Quality of Recovery (TQR) scales (see Kellmann 2002, p.16 table 1.2). Use of the weekly Recovery–Cue assessment (Kellmann 2002, appendix 12.A) may aid in following the athlete’s responses over time and allow for early intervention in cases of incipient over-stress.

Self-monitoring of the athlete’s responses to training and the athlete’s self-perceived mental state are the most sensitive cues to the total volume of stressors and his or her adaptation to them.

3. Psychological

Psychological strategies are important factors in reducing and managing stress. Relaxation training, imagery, and autogenic training are valuable tools in helping the athlete to maintain focus during competition, allaying excessive tension, and aiding

in recovery processes. Relaxation training is helpful for inducing sleep. Adequate sleep is essential for recovery, as many endocrine systems undergo optimal recovery during sleep.

4. Social

Social interactions can be a pleasant diversion from the rigours of training, especially if they are done in settings outside of the training milieu and with other friends who are not involved in sports.

5. Medical

Many medical modalities that have been found to be valuable restoration tools. Different massage techniques are useful in aiding warm-up, relaxing muscles after training, and in re-activation for subsequent exercise sessions. Hydrotherapy in several forms is used to flush out waste products and improve peripheral circulation, both locally and by nervous system activation. Ice baths, contrast baths, and hydro-massage stimulate venous circulation.

Active and passive stretching help muscles to lengthen, relieve tension within the muscle bundles, and enhance relaxation.

6. Nutrition

Maintaining adequate nutrition is essential for complete recovery (see Chapter 6, Part 1, *Nutrition and Athlete Health*). During and after exercise, it is important to remain hydrated. For short sessions water is adequate, but for longer periods glucose/electrolyte solutions replace losses and maintain caloric and salt stores. Glycogen repletion should begin immediately after exercise, using glucose /electrolyte solutions. Carbohydrate/protein mixtures may enhance glycogen repletion as well as restore muscle amino acids and rebuild muscle tissue. A high-carbohydrate meal should be eaten within 2–4 hours after exercise.

B. Planning a System for Recovery and Restoration

1. Before Training or Competition

- a. Develop a check-list of necessary preparations and form a consistent routine, but be flexible and prepare for unexpected changes (altered time schedule, order of competition, change of venue, weather conditions)—i.e., “stuff happens”!
- b. Carry out a gradual warm-up, stretch gently, and develop a sense of muscle “awareness.”
- c. Have a brief, activating massage.
- d. Utilise relaxation and imagery to focus on the competitive task and remove distractions.

2. During a Training Session

- a. Maintain hydration—consume water or a carbohydrate/ electrolyte drink.
- b. Rest between exercise bouts.

3. Between Multiple Daily Training Sessions or Rounds of Competition
 - a. Replace nutrients and fluids.
 - b. Stretch. Obtain a light massage. Take a brief nap.
4. During Competition
 - a. Maintain fluids and electrolytes.
 - b. Use psychological techniques to “psych-up” and “psych-down”.
 - c. Manage the environment: stay cool (or warm), use sun block if necessary.
5. After Training or Competition
 - a. Cool down and stretch.
 - b. Replace fluid losses—use a carbohydrate/electrolyte/protein drink. Consume 150% of fluid losses (weigh before and after event).
 - c. Massage: stroking, to stimulate waste removal and elicit relaxation.
 - d. Hydrotherapy: cold (or ice) bath, or hydro-massage.
 - e. Nutrition: eat a high-carbohydrate meal within 2–4 hours to restore glycogen.
 - f. Psychological: carry out a post-event evaluation with the coach, assessing both the good and bad aspects of the event (see questionnaire).
6. During Recovery Days (“Invisible Training”)
 - a. Use “active rest” by doing some form of light activity or cross-training, or play games (safely and not competitively!). Allow the muscles to work, while the mind and nervous system rest.
 - b. Do easy stretching or yoga.
 - c. Become involved socially and get away from the sports routine.
 - d. Use “nature therapy”—have a complete change of scene by walking in the park or forest, going to the beach, etc.

Remember that each athlete is unique, with individual ways of responding to stressors and to recovery techniques. What may be restorative to one may be stressful to another. Hence, each athlete must create his or her own armamentarium of restoration strategies, and use them diligently.

C. Overtraining vs. Under-Recovery

Overtraining is a poorly-defined complex of the body’s psycho-physiological responses to a wide variety of “stressors,” including: 1) an excessive or monotonous training load; 2) too-frequent competitions; 3) inadequate recovery time following an intensive work-load, or any combination of these; 4) other “stressors” in the athlete’s life, such as financial or work pressures, social issues, excessive travel (jet-lag), inadequate sleep and nutrition, or lack of recreational opportunities.

1. Definitions (See Ref. 7)

a. Overreaching (or Short-term Overtraining)

Overreaching (or short-term overtraining) can be defined as an accumulation of training and/or non-training stress resulting in short-term decrement in performance capacity with or without related physiological or psychological signs and symptoms of overtraining, in which restoration of performance capacity may take from several days to several weeks. Note that overreaching may be difficult to distinguish from the normal sense of fatigue that accompanies an intensive training program.

b. Overtraining (or Long-term Overtraining, “Staleness” or “Burnout”)

Overtraining can be defined as an accumulation of training and/or non-training stress resulting in long-term decrement in performance capacity with or without related physiological signs and symptoms of overtraining, in which restoration of performance capacity may take several weeks or months.

Overtraining may be looked upon as the OUTCOME of several systems' failures due to inadequate restoration and recovery strategies. Although the continuum between overreaching and overtraining syndromes appears logical, there is no scientific evidence to indicate that 1) overreaching precedes overtraining, or 2) symptoms of overtraining are worse than those of overreaching.

2. Incidence of Overtraining

The incidence of overtraining in high-level athletes has been reported from 5–15% over a 1-year period, according to different authors. However, it is difficult to interpret these data because many of these studies diagnosed overtraining without any performance measurement or performance decrease (a very important prerequisite). Moreover, terminology used is sometimes inconsistent and it is likely that some of the overtrained athletes only underwent transient overreaching syndrome.

3. Prevention of Overtraining

The highly-trained, strongly-motivated elite athlete constantly treads a fine line between optimal levels of training, and overtraining. Close communication between insightful coaches and athletes who are “tuned-in” to monitoring their own mental and physical responses to training is required to detect the “early warning signs” of overtraining (see below) and to react appropriately.

a. Guidelines for an Optimal Training Load

The optimal training load for an individual athlete depends on various factors, including genetic make-up, lifestyle, degree of physical and mental maturity, and state of initial fitness.

There are no hard and fast rules for determining how and when to adjust the training load, but empirical evidence suggests that an increase of no more than 5% each week during a training micro-cycle allows for adaptation and recovery. Furthermore, intensity and volume of training should not be increased simultaneously.

Not being able to devise a numerical index for training intensity and volume makes it difficult to quantify the training load. Therefore, training must be carefully documented in the athlete's diary. The athlete's subjective responses to and feelings about the training should be monitored and recorded, as should lifestyle factors such as hours and quality of sleep, nutrition, and other stressors. If signs of overtraining do become apparent, a careful record of activities should help pinpoint possible causes.

b. High-risk Activity Patterns

Certain activity patterns are especially likely to cause overtraining. These include:

- i. closely-spaced competitions without adequate recovery, or without an adequate recovery interval between a series of competitions
- ii. a sudden increase in training volume and/or intensity without a gradual build-up
- iii. use of a single, monotonous training format, such as interval training, which fatigues one muscle group or energy system
- iv. increase in other life stressors, such as inadequate sleep or nourishment, travel (especially across time zones), or adverse psychological encounters, etc.

Avoiding these patterns, especially by ensuring adequate recovery or diversion, is the best way to prevent overtraining.

4. "Early Warning Signs" of Overtraining

Athletes beginning to show evidence of overtraining exhibit several symptoms, often in combination. Sports medicine personnel and coaches should be alert for these early warning clues, and activate recovery efforts:

- a. An athlete feels that greater effort is needed to complete a training session, time trial, or competition. A longer recovery time is needed between exercise bouts.
- b. An athlete complains of persistent muscle stiffness and soreness, and requests frequent massage.
- c. An athlete feels a persistent sense of fatigue and inadequate recovery after a training session. A poor sleep pattern and elevated morning heart rate may accompany this feeling.
- d. An athlete exhibits irritability and moodiness in dealing with routine activities.
- e. An athlete loses the drive to train, and dreads the outcome of a poor training session.
- f. A female athlete experiences alterations of the menstrual cycle, especially amenorrhea (see Chapter 13, Part 1, *Endocrine/Menstrual Factors*).

These warning signals should indicate to the athlete, coach, and medical staff that a major adjustment in the training program is necessary. The team physician can

be of value in confirming this “diagnosis” and encouraging a programme of recovery rather than allowing the athlete to persist in a potentially harmful process.

5. Physiological and Laboratory Detection

Despite numerous efforts, the ability to detect incipient overtraining or overreaching has been elusive. Many athletes with overreaching or overtraining may show few or no measurable physiological changes, while others may have altered laboratory and psychological findings with no clinical evidence of being overtrained. Many different markers have been suggested as being useful, but none has been carefully validated in a research setting with adequate controls.

A number of indicators have been recommended, and if carried out on a regular basis to monitor the athlete, may be of some value:

Although it has not been agreed on by all, time to fatigue tests seem to be the most appropriate technique to diagnose states of overreaching or overtraining. This type of submaximal test allows different kinds of metabolic measurements that can be compared over the course of the season. In the field of maximal physiological parameters, reduction of maximal oxygen uptake and maximal heart rate, obtained during incremental tests, are frequently reported in overtrained or overreached athletes. However, whether the reduction of these parameters is the cause or the consequence of the premature fatigue associated with overtraining remains unknown.

Overtraining can disrupt the immune system, which makes the athlete more susceptible to infections. Upper-respiratory tract infections (URTI) have often been considered as a hallmark of the overtraining syndrome, but this has not been found by other researchers. In fact, it seems that a higher URTI incidence and the frequently associated low salivary IgA concentration more likely reflect an increase in the training load rather than a training dysadaptation syndrome (overreaching or overtraining). Some biochemical and immunological studies recently reported decreased serum glutamine, and glutamine/glutamate ratio, in overtrained subjects. This finding deserves further studies.

The endocrine system may show a stress response, with an initial rise in epinephrine and nor-epinephrine, ACTH and cortisol, and growth hormone (GH), followed by a decline in the level of these hormones (Selye’s “exhaustion stage”). For instance, the maximal blood cortisol response to exercise stress is often decreased in overreached athletes. Early hormonal studies on overtraining reported a decrease (more than 30%) in the testosterone/cortisol ratio due to a rise in cortisol and a reduction in testosterone (due to gonadotropin suppression). Unfortunately, the usefulness of this ratio as a diagnostic tool has not been supported in the literature.

Similarly, overtraining can profoundly affect the psychological status of the athlete. The widely-used Profile of Mood States (POMS) shows a characteristic “inverse iceberg profile,” with low levels of vigour, and high indices of fatigue, depression, and anger. This profile can be reversed with appropriate management of training, and time allowed for recovery. Others have shown that a simple seven-question profile administered daily or weekly may help to discriminate those who may become overtrained.

These psychological markers seem to be the most sensitive indicators of the athlete under stress, and should be administered on a regular basis, along with a review of the training diary and discussions between coach and athlete.

Changes in the levels of certain biochemical markers (CPK, ferritin, haptoglobin, etc.) are potentially suggestive of overtraining. However, these levels are also altered by intensive training, so unless there is frequent, regular testing of the athlete to establish an individual baseline and “normal range,” these tests cannot definitively diagnose overtraining.

Heart rate variability (HRV), which results from signal processing of the RR intervals (consecutive heart beats), has been recently investigated as a non-invasive diagnostic tool of overtraining. This hypothesis was supported by the fact that HRV can reliably attest for the sympathetic and parasympathetic tones. As overtraining is believed, by some authors, to be associated with autonomic nervous system disturbances, HRV monitoring was a good candidate. Unfortunately, most of the studies showed that the clinical value of HRV in the diagnosis of overtraining is not high enough for this method to be considered as a valuable tool.

Several other simple physiological indices may be useful in detecting overtraining IF they are recorded under carefully standardised conditions:

- a. Amount and quality of sleep. Sleep disturbances and insomnia may be an early indication of stress.
- b. Morning heart rate, if measured under standard conditions, may be a measure of recovery from training.
- c. Body weight. A stable morning (post-void) weight may indicate the adequacy of re-hydration and nutrition. Weight loss and a decline in appetite may be warning indicators of overtraining.

D. Summary

Clearly, overtraining remains a poorly-understood syndrome and much further research needs to be done. For now, coaches, athletes, and medical staffs must be alert to the many and varied possible early warning signs. Athletes should keep a detailed diary in which they record their training work, their subjective reactions to and feelings about it, and other pertinent life events such as sleep, nutrition, appetite, and personal stressors. A brief psychological profile test should be obtained at least weekly. Remind the athlete that overreaching or overtraining are always associated with an objective decrease in physical performance. Physiological factors such as resting morning heart rate, body weight, and amount and quality of sleep should be monitored. If possible, some hematologic and biochemical markers should be measured every two to three months to assess an athlete’s normal range of responses to training, in order to detect any deviations from the usual range.

The best method of avoiding the overreaching/overtraining syndrome is to monitor the athlete’s programme carefully, and adopt as an integral part of the athlete’s programme the restoration and recovery methods that have been described.

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SPECIFIC CONSIDERATIONS FOR THE CHILD AND ADOLESCENT ATHLETE

Sports medicine physicians must be familiar with the normal patterns of growth and development of the child and adolescent, in order to detect any abnormal patterns, and make appropriate judgments. This will allow the physician to detect any deviations during the pre-participation examination, guide children into appropriate activities, aid them in setting realistic goals concerning sports participation, and provide guidance to the community and coaches in the design of safe and effective training and sports programmes.

A. Developmental Levels (see Table 4-1)

1. Early Childhood (ages 3–5 years)

- a. Vision: not fully mature (difficulty in tracking moving objects and judging velocity)
- b. Balance: paradoxical decrease at 4–5 years due to overload in integrating visual and proprioceptive input.
- c. Motor Skills: Age 4—run, kick, hop, throw (20%), catch (30%)
- d. Learning Ability: short attention span; easily distracted; need instruction via auditory and visual input.
- e. Activity Recommendations: PLAY in a closed system (few variables, constant conditions); walk, run, swim, tumble. Organised sports and competition may INTERFERE with learning.

2. Childhood (ages 6–9)

- a. Vision: tracks speed and direction of moving objects with difficulty.
- b. Balance: automatic by age 7.
- c. Motor Skills: age 6—basic overhead throw developed; age 8—running skills matured.
- d. Learning Ability: short attention span; easily distracted; lacks rapid decision making; needs verbal and visual instruction; cooperation improved.
- e. Activity Recommendations: Skills acquisition in a closed system (run, swim, gymnastics); recreational play; low level organised sports.

3. Late Childhood (ages 10–12)

- a. Vision: adult pattern.
- b. Balance: improved; declines at puberty during peak height velocity (Tanner stage 3).
- c. Motor Skills: complex skills develop, but postural control may deteriorate (uncoordinated body segment growth patterns).

Table 4-1. Developmental characteristics of and sports participation guidelines for various age groups.

	Infancy (0–2 years)	Early Childhood (3–5 years)	Childhood (6–9 years)	Late Childhood (10–12 years)
Motor Skills	Skills primarily reflex; posture depends on visual input	Fundamental skills limited; balance skills limited	Fundamental skills improved; transitional skills begin; balance control becomes automatic	Transitional skills improved; balance control declines at puberty
Learning Skills	Response to training minimal; benefits of training not long-term	Attention span short; attention overexclusive; response to training limited	Attention span limited; attention overinclusive; cooperation improved	Attention selective; memory strategies used
Vision	Farsighted	Farsighted; eye movements imprecise; tracking of speed and direction of moving objects difficult	Tracking of speed and direction of moving objects improved, but still difficult	Patterns same as for adults
Guidelines for Sports Participation	Recognise that swimming programmes and exercise programmes of no advantage; encourage free play; provide safe, unstructured play environment	Avoid competition; provide limited instruction verbally and by demonstration; emphasise fun play	Keep competition minimal; keep rules of sport flexible; emphasise fundamental skills; keep instruction time short	Minimise competition; emphasise fundamental and transition skills; decrease intensity of sports involvement at puberty
Recommended Activities	Free play	Walking, running, swimming, tumbling, throwing, catching	Swimming, running, gymnastics, entry-level soccer and baseball; complex-skill sports such as football, hockey, basketball, and wrestling are difficult	Entry-level football, basketball, wrestling, and other contact/collision sports

- d. Learning Ability: integrate information from multiple sources; respond to verbal instruction.
- e. Sport Recommendations: continue fundamental and transitional skills; successful in skill and team sports, low level competitive sports.

B. Adolescent Growth and Maturation

1. Endocrinology

Prior to puberty, the hypothalamic-pituitary-gonadal feedback system is operative in a negative mode, and hormone levels remain low. With the onset of puberty, feedback sensitivity is diminished. This allows increased synthesis and secretion of hypothalamic gonadotrophin-releasing hormone (GnRH), which stimulates the anterior pituitary to produce gonadotrophins (LH and FSH) with subsequent rises in the gonadal sex steroids estrogen and testosterone.

2. Stages of Growth and Development

Growth and development at puberty occur in an orderly fashion, as described by Tanner. In general, the onset of pubertal maturation occurs earlier in girls than in boys. On average, the height spurt in girls occurs about two years earlier than in boys, with a peak height velocity at age 12 for girls, and age 14 for boys. This may vary, depending upon the nutritional status and geographic location of the subjects. (See Figures 4-1 and 4-2, and Table 4-2.)

Tanner's Sexual Maturation Ratings (Table 4-3) have been recommended as a means of determining a child's readiness to participate in certain sports, especially those involving complex skills, teamwork, and body contact.

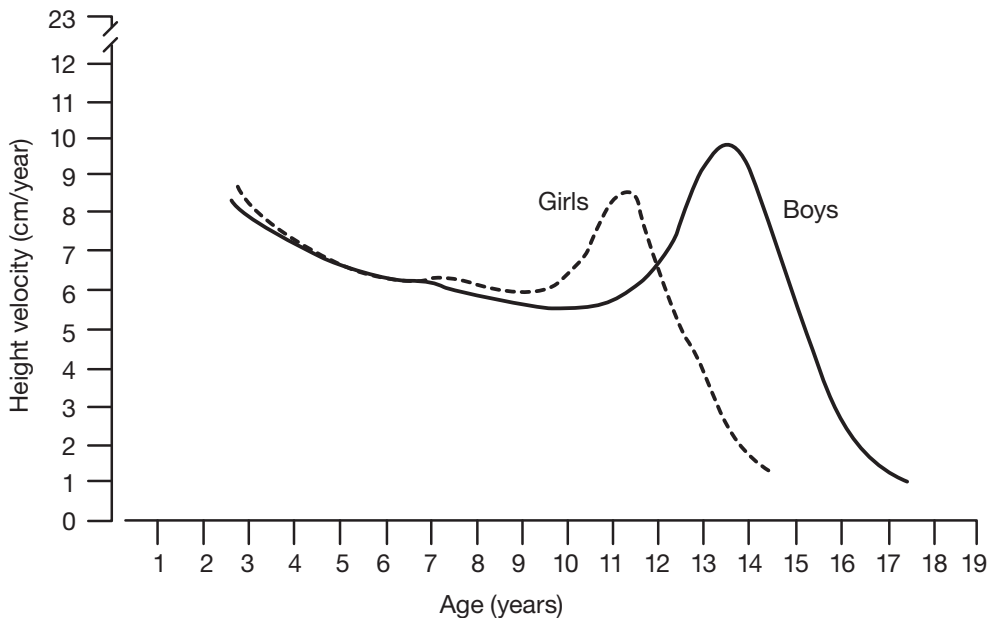


Figure 4-1. Fiftieth percentile height velocity curve for American boys and girls (Slap 1986).

Table 4-2. Typical timing of normal pubertal events in girls and boys (SMR=Sexual Maturity Rating—Tanner).

Event	Girls	Boys
onset of puberty	10 years (8–14 years)	12 years (9–15 years)
first sign	breast bud	testicular enlargement
PHV	12 years SMR = 2–3	14 years SMR = 3–4
peak weight gain		6 months after PHV
end of growth and maturation	16.5 years	15–18 months later than girls

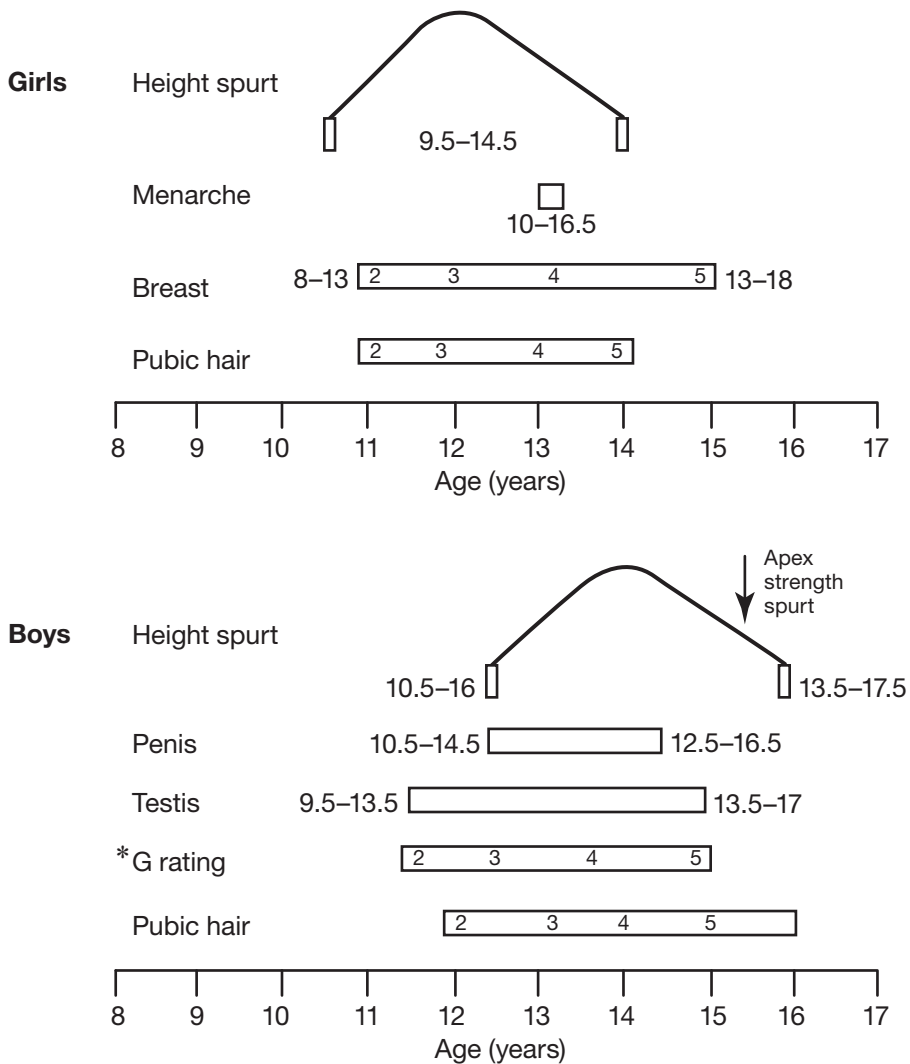


Figure 4-2. Sequence of pubertal events (Slap 1986). (*Genitalia)

Table 4-3. Tanner's stages of sexual maturation in girls (a) and boys (b).

a. Sexual Maturing Ratings (SMR) in girls (Tanner ratings)

Stage	Pubic Hair	Breasts
1	Preadolescent	Preadolescent
2	Sparse, slightly pigmented, straight, at medial border of labia	Breast and papilla elevated as small mound, areolar diameter increased
3	Darker, beginning to curl, increased amount	Breast and areola enlarged, without contour separation
4	Coarse, curly, abundant, but amount less than in adult	Areola and papilla form secondary mound
5	Adult feminine triangle, spread to medial surface of thighs	Mature, nipple projects, areola part of general breast contour

The illustrations for girls' Tanner stages are arranged in two columns. The left column shows the pubic hair development, and the right column shows the breast development. Each row corresponds to a stage from 1 to 5. Stage 1 shows minimal hair and undeveloped breasts. Stage 2 shows sparse hair and a small breast mound. Stage 3 shows darker, curling hair and enlarged breasts. Stage 4 shows coarse, curly hair and a secondary breast mound. Stage 5 shows a full adult hair triangle and mature breasts.

b. Sexual Maturing Ratings (SMR) in boys (Tanner ratings)

Stage	Pubic Hair	Penis	Testes
1	None	Preadolescent	Preadolescent
2	Scanty, long, slightly pigmented	Slight enlargement	Enlarged, scrotum pink, texture changed
3	Darker, begins to curl, small amount	Longer	Larger
4	Resembles adult type but less in quantity; coarse, curly	Larger, glans and breadth increased	Larger, scrotum darker
5	Adult distribution, spread to medial thighs	Adult	Adult

The illustrations for boys' Tanner stages are arranged in two columns. The left column shows the pubic hair development, and the right column shows the penis and testes development. Each row corresponds to a stage from 1 to 5. Stage 1 shows no hair and small genitalia. Stage 2 shows scanty hair and slight genital enlargement. Stage 3 shows darker, curling hair and longer penis. Stage 4 shows coarse, curly hair and further genital growth. Stage 5 shows a full adult hair triangle and mature genitalia.

3. Clinical Conditions and Maturation Levels

A number of clinical conditions are associated with certain stages of maturation (Table 4-4).

Table 4-4. Clinical factors and Sexual Maturity Ratings (Tanner stages).

Clinical Factor	Sexual Maturity Rating (SMR)
Hematocrit rise (male)	2-5
Male gynecomastia	2 or 3
Slipped capital femoral epiphysis	2 or 3
Worsening idiopathic scoliosis	2-4
Osgood-Schlatter's disease	3

Body composition changes markedly at puberty, as a result of rises in sex steroids secretion. Males exhibit a typical growth in muscle mass, while females show a deposition of fat in the estrogen-sensitive areas of the hips, thighs, and breasts (Figure 4-3).

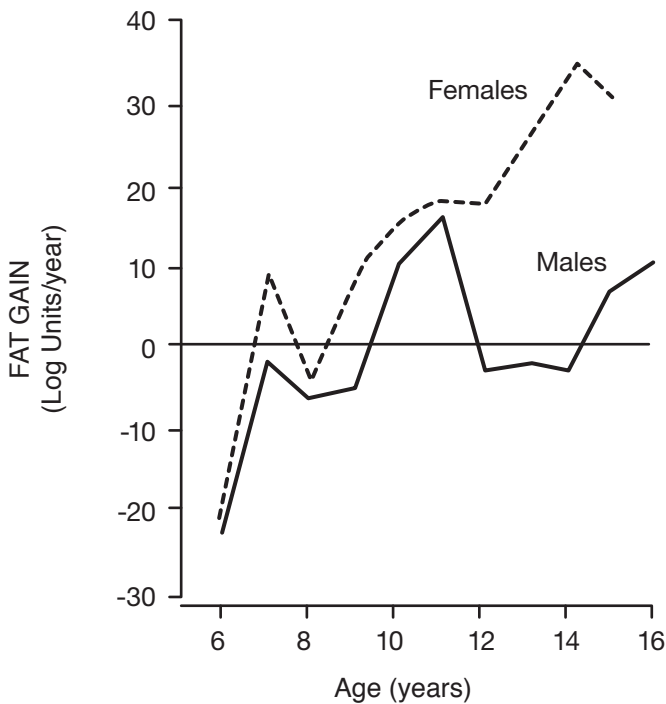


Figure 4-3. Mean increment in fat in boys and girls based on skinfold measurements at four sites (Tanner 1955).

C. Abnormal Patterns of Development

1. Precocious Puberty

Precocious puberty is defined as showing signs of the onset of puberty prior to age 8 in girls, and age 9 in boys.

2. Delayed Puberty

Delayed puberty is defined as:

- a. Female—lack of evidence of breast development by age 13.
- b. Male—no signs of testicular enlargement by age 14 1/2.
- c. Either sex—children who fail to progress normally through puberty.

3. Primary Amenorrhea

Primary amenorrhea is defined as the absence of menarche in a female of reproductive age. Signs include:

- a. Delay of menarche beyond age 16.
- b. Absence of menarche more than four years after thelarche (onset of breast development).

(See also Chapter 13, Part 1, *Endocrine/Menstrual Factors*.)

D. Musculoskeletal Growth and Development

1. Regulation of Musculoskeletal Growth

Bone responds to a wide variety of growth stimulators, including growth hormone, testosterone, sulfation factor, thyroxine, parathyroid hormone, and insulin-like growth factor. Muscle lengthens in response to stretch; growth occurs in the region of the musculo-tendinous junction. The maximum potential for growth is determined genetically. However, this potential can be attained only if there is adequate nutrition, physical activity, and good general health.

2. Bone Growth: Definitions (see Figure 4-4)

- a. Epiphysis—the end of the long bone that is bordered by the growth plate (physis) and by articular cartilage.
- b. Metaphysis—the flared portion of the bone that is between the epiphysis and the diaphysis.
- c. Diaphysis—the shaft of the long bone.
- d. Apophysis—the site of attachment of musculo-skeletal structures to the long bone; it is extra-articular.

3. Growth of Long Bones

a. Longitudinal Growth

Longitudinal growth occurs at the epiphyseal growth plate (physis) and at the articular cartilage. Chondrocytes divide, align longitudinally to form columns, and begin to enlarge and calcify. In this fashion, the epiphysis moves further away from the center of the bone.

b. Latitudinal Growth

The periosteum contributes to new bone formation from fibroblasts, to enlarge the circumference of the long bones. The ring of Ranvier enlarges the diameter of the physis.

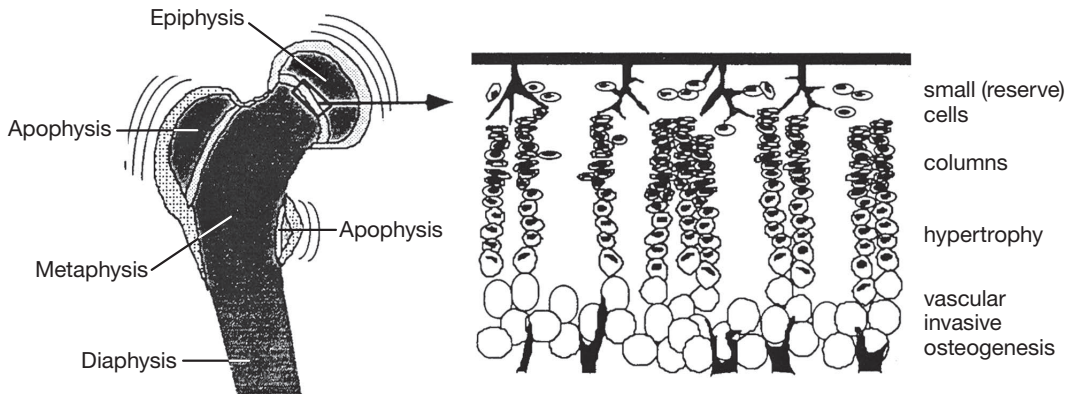


Figure 4-4. Bone growth.

c. Apophyseal Growth

- i. Physeal growth occurs as in a (see Figure 4-4), at the junction of the metaphysis and the apophysis.
- ii. Periosteal and fibro-cartilaginous formation of new bone occurs at the site of tendinous insertions.

d. Growth Rate

Growth is very rapid during the first two years of life, then slows to a relatively constant rate, except for brief, intermittent spurts. During pre-adolescence there is another major growth spurt (peak height velocity). Then the growth rate begins to plateau as maturity approaches.

E. Injuries to the Immature Skeleton—Musculo-skeletal Injuries Unique to Children and Adolescents

1. Unique characteristics of the immature musculo-skeletal system:

- a. Open growth plates afford both longitudinal and appositional growth.
- b. There is a relative disproportion between long bone length and adjacent musculature, as muscles lengthen in response to stretch.
- c. The periosteum is thicker. This stabilises the bone, both intact and fractured. The thicker periosteum requires more force to disrupt than in adults. Following a fracture, the vascular periosteum aids in rapid healing; however, it may act as a deforming force as it contracts over time.
- d. The long bones are more porous, so buckling (torus) fractures are more common.

- e. Long bones are more “flexible,” and may undergo plastic deformation as part of a fracture.
 - f. Thicker, growing articular cartilage leads to chondral or osteo-chondral fragmentation from over-use, especially at the distal femoral condyle, radial head, and humeral head.
 - g. There is greater vascularity of the knee menisci. This allows for healing more often than in the adult.
 - h. Different injury patterns occur at different ages, dependent upon the strength of the adjacent structures at the particular skeletal age.
2. Special Considerations in Injury Assessment

In addition to the usual diagnostic considerations, additional factors must be kept in mind when assessing injuries to the child and adolescent.

a. Acute Injuries

In evaluating all musculo-skeletal injuries, consider:

- i. Physeal injuries
- ii. Congenital anomalies
- iii. Neoplasms
- iv. Infections

in addition to the more common sprains and strains. During on-field examinations, DO NOT STRESS an injured joint, as it may cause further physeal damage. DO an X-ray first!

b. Over-use Injuries

Over-use injuries may affect all joints, and especially the physes. Consider:

- i. Inflammatory processes—apophysitis; osteochondritis dissecans
- ii. Metabolic diseases
- iii. Neoplasms
- iv. Infections

F. Physiological Characteristics of Children

1. Aerobic Capacity

Aerobic capacity (VO_2 maximum) increases with age. However, in terms of body mass, when aerobic capacity is expressed in terms of ml/kg, maximal aerobic power does not increase, and may even decrease in the second decade, with the increase in the child’s fat mass.

2. Anaerobic Capacity

Anaerobic performance in children is much lower than in adolescents and adults. This is likely due to the development of anaerobic enzyme systems, and also to poorer neuro-muscular control.

3. Energy Cost of Running

The metabolic costs of locomotion are higher in children, hence they have a lower metabolic energy reserve (the difference between maximal and sub-maximal energy cost). This is probably due to: 1) shorter stride length and faster leg turnover during running; and, 2) greater co-contraction of antagonist muscles during activity. Flexibility and relaxation training may help to reduce the energy costs of running, and improve economy.

G. Effects of Training on the Child and Pre-adolescent

1. Measurement Problems

A variety of physiological changes in children and pre-adolescents might be expected to accrue as a result of a training programme. However, it is difficult to assess the degree of trainability of various systems, due to a variety of confounding factors.

- a. The role of normal growth and development is difficult to differentiate from the effects of training. For example, relative changes in maximum oxygen uptake (ml/Kg) may be masked by changes in maximal heart rate, and increases in body fat mass.
- b. Children may lack the discipline required for training that is intensive enough to induce measurable physiologic changes.
- c. Intense training may not be employed for fear of causing injuries.
- d. Suitable control groups are difficult to select, as they should not be chosen solely on the basis of age and sex.
- e. Ethical and methodological problems limit the extent of studies, e.g. muscle biopsies, invasive procedures, etc.

2. Probable Physiologic Changes

Despite these limitations, a number of studies have suggested that the major physiological systems that respond to training in adults also respond in a similar fashion in children, though not always to the same degree.

- a. **Aerobic capacity**
Aerobic capacity may improve nearly as much as in adults. This capacity may be limited by the lower hemoglobin seen in children, with consequent lower oxygen-carrying capacity.
- b. **Anaerobic capacity**
Anaerobic systems may not be sufficiently mature to adapt well to training, and hence may limit the ability to respond well to anaerobic activities such as the 200m and 400m dashes.
- c. **Strength**
Strength may improve relative to body size compared to older children, though absolute gains may be small. Little muscle hypertrophy occurs, due to low hormonal (testosterone) levels. Strength gains are due to improvement

in a number of neurological factors, including increased neural recruitment, improved synchronisation of motor unit fibres, and better motor skills coordination. (See Table 4-5 for strength training guidelines.)

Table 4-5. Guidelines: strength training for children and adolescents.

<ol style="list-style-type: none"> 1. Pre-participation evaluation by a physician knowledgeable in sportsmedicine 2. Good quality equipment suitable to the size and age of the athletes 3. Strength training should be part of an overall conditioning and fitness programme 4. Supervision by a well-trained adult (National Strength and Conditioning Association) 5. Appropriate warm up and cool down period before and after strength training 6. Selection of sports-specific exercises appropriate to the level of physical and emotional maturity of the participant 7. Attention to proper technique: avoid Valsalva maneuver, hyperventilation, back hyperextension 8. Emphasis on dynamic concentric contractions as opposed to eccentric overload exercises 9. Emphasis on sets of high repetitions at low resistance 10. Each exercise should be taken through the full range of motion for maximum muscle development and to maintain flexibility 11. Competition (weight lifting, power lifting, body building) should be prohibited 12. Maximal lifts should not be performed until skeletal maturity (Tanner stage 5; see Table 4-3) 13. Programme design should be based on the principle of progressive resistance
<p><i>Sample Programme Prescription</i></p> <ul style="list-style-type: none"> • 1-3 sets of 6–10 exercises per session • Frequency of 2–3 sessions per week with rest day in between • Duration of 20–60 minutes per session • Progressive resistance: <ul style="list-style-type: none"> • Start at no resistance/weights until proper form is achieved • Then initiate resistance at the 6 repetition level, advance to 15 reps • Weight then added in 1–3 lb (500–1500 g) increments until child can do just 6 reps • Advance again to 15 reps before increasing weights

d. Heat Adaptation

Children adapt less well to exercise in the heat, especially at temperatures above skin temperature. Several factors are responsible for this discrepancy:

- i. At any exercise level, children produce more metabolic heat per kg of body weight (i.e., are less efficient).
- ii. Their larger surface area/body weight ratio permits greater heat absorption from the environment when air temperature exceeds skin temperature (approximately 32°–33° C). The smaller the child, the greater the potential for heat absorption.
- iii. Children produce less sweat. This is likely due to limitations in anaerobic energy production by sweat glands. Thus, the ability for evaporative cooling is lower. This is critical, as evaporation of sweat is the most important means of heat dissipation during exercise, especially under hot conditions.
- iv. Children require longer to acclimatise to a hot climate. Several days are needed for adjustment of the enzymatic and hormonal changes that affect the sweating mechanisms. Fourteen days may be required for full acclimatisation.
- v. Hypohydration (lower body water content) has more profound effects on children. During exercise, core temperature rises faster, affecting motor performance and cognitive skills. Children must be trained to drink frequently (every 15–20 minutes) even when not thirsty. A drink with electrolytes (especially sodium) and carbohydrate enhances palatability and fluid consumption.

H. Guidelines for Youth Training Programmes

Effective, safe training programmes for young athletes must take into account the age, sex, and maturation levels of each child. The duration, intensity, and frequency of training sessions will vary considerably, depending upon the above factors. Children should be encouraged to participate in a wide variety of activities and sports, and not be channeled into “specialisation” in one sport or event before reaching the mid- or late-teen years.

It is difficult to provide definite “dosages” of training for children who participate in the varied event disciplines of athletics. However, some general guidelines have been developed, based upon practical experience as well as a knowledge of the developmental levels of children (see Table 4-1).

All training sessions should be preceded by a suitable 20–30 minute warm-up period with emphasis on gradually increasing activity and stretching of all muscle groups, and followed by a gradual cooling down and further stretching.

1. Endurance Training (long distance running)

The recommended maximum competition distances for children of various ages are shown in Table 4-6. The weekly training distance should not be more than twice

Table 4-6. Recommended maximum running distance at different ages.

Age (Years)	Distance
Under 9	3 km
9–11	5 km
12–14	10 km
15–16	Half marathon (21.1 km)
17	30 km
18	Marathon (42.2 km)

the recommended maximum competition distance. Runs or races up to 10 km in length can be undertaken on a weekly basis for children aged 12–14; runs or races over 10 km in length require longer recovery periods. Training frequency for those up to 14 years old should not exceed 3 times per week. Those aged 15–18 can train up to 5 times per week.

2. Sprints

Training frequency for those up to 14 years old should not exceed 3 times per week. Those aged 15–18 can train up to 5 times per week. The duration of each session should not exceed 1.5 hours, including a warm-up and stretching component.

3. Throwing Events (shot put, discus, javelin, hammer)

Injuries can be avoided in throwing events if the correct technique is developed for each throwing discipline. Training frequency and duration should follow the following guidelines:

- a. No more than 3 training sessions per week.
- b. Each session should not exceed 1.5 hours (including warm-up).
- c. The total number of throws permitted for each session should not exceed 20 for athletes up to 14 years old and 40 for those between 15 and 18 years old.

4. Jumping Events (long jump, triple jump, high jump, pole vault)

Training frequency and duration should follow the following guidelines—

- a. Each session should be no longer than 1.5 hours (including warm-up).
- b. Athletes up to 14 years old should not perform more than 3 sessions per week with a maximum of 10 jumps per session.
- c. Athletes from 15 to 18 years old should not perform more than 5 sessions per week with a maximum of 20 jumps per session.

I. Roles of the Parents and Coach

The collaborative involvement and support of the parents and coach is essential if children are to achieve an enjoyable and successful experience in sports. Unreasonable demands and expectations from adults are a common cause for a child's dropping out of sport.

The child's reaction to this pressure may manifest itself in a wide variety of psychosomatic symptoms that may come to the attention of the sports physician. These may include headaches, gastro-intestinal disturbances, muscle aches out of proportion to the training load, and even proneness to injury in order to avoid participation. Guidelines for avoiding such situations are seen in Table 4-7.

Table 4-7. Guidelines for parents.

- Encourage children to participate if they are interested.
- Focus on the child's effort/performance, not the outcome of the events.
- Honest effort is as important as victory.
- Encourage children to participate according to the rules.
- Never ridicule.
- Children learn best from example. Applaud good plays by all teams.
- Do not question officials' judgement in public.
- Support efforts to remove verbal and physical abuse.
- Recognise the value and importance of volunteer coaches.

J. Special Considerations in Injury Rehabilitation

1. Goals of the Rehabilitation Programme

- a. Prevent recurrence of the injury or related injuries.
- b. Restore muscle strength to full power, strength, and endurance.
- c. Regain flexibility and coordination.
- d. Restore full performance capability.

2. Principles of Rehabilitation

- a. Special Problems
 - i. Short attention span.
 - ii. Unable to comprehend need for rehabilitation programme
 - iii. Teenagers feel invulnerable to injury.
 - iv. Feel set apart from peers.
- b. Application of Principles
 - i. Set a defined time limit for meeting goals—no more than two months.

- ii. Make the patient an active participant in the programme by using active exercises. Control pain and swelling so the rehabilitation programme can begin as soon as possible.
 - iii. Use short periods of activity.
 - iv. Utilise a small number of exercises, done correctly.
 - v. Make progress apparent by using many intermediate goals. Reward the patient for attaining each goal.
 - vi. Integrate the programme into the patient's regular schedule, i.e., while watching TV, talking on phone, etc.
- c. Special Rehabilitation Considerations for Children
- i. Flexibility and strengthening must be stressed. Muscles may be relatively short compared to bones, especially during periods of rapid growth. Non-ballistic stretching must be taught.
 - ii. Exercise programmes must be supervised carefully.
 - iii. Machines must be adjusted to the child's frame.
 - iv. Use caution with ultra-sound near growth plates.
- d. Athletic Activity During Rehabilitation
- i. Must be pain-free the morning after activity.
 - ii. Avoid activities that interfere with doing the rehabilitation exercises.
 - iii. No participation if the weakened area is likely to cause further injury.
 - iv. Do not use modalities (e.g. ice) or analgesics that could mask pain before an "at risk" activity.

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SPORTS PSYCHOLOGY

It has long been acknowledged that psychological skills are critical for athletes at the elite level. Athletes with the requisite “mental toughness” are more likely to be successful. In the past, it was assumed that these skills were genetically based, or acquired early in life. Now, it is commonly accepted that athletes and coaches are capable of learning a broad range of psychological skills that can play a critical role in learning and in performance.

A. Role of Sports Psychology

The specialised field of sports psychology has developed rapidly in recent years. The importance of a sports psychologist as an integral member of the coaching and health care teams is widely recognised.

Sports psychologists can teach skills to help athletes enhance their learning process and motor skills, cope with competitive pressures, fine-tune the level of awareness needed for optimal performance, and stay focused amid the many distractions of team travel and in the competitive environment. Psychological training should be an integral part of an athlete’s holistic training process, carried out in conjunction with other training elements. This is best accomplished by a collaborative effort among the coach, the sport psychologist, and the athlete; however, a knowledgeable and interested coach can learn *basic* psychological skills and impart them to the athlete, especially during actual practice.

B. The Medical Staff and Psychosomatic Disorders

The health professional often plays a major role in supporting the emotional health of athletes. An athlete’s psychological stresses may be manifested as somatic complaints, such as sleep disturbances, irritability, fatigue, gastrointestinal disturbances, muscle tension, or even injury. Athletes often turn to a therapist or physician for relief, either because they do not recognise the psychological basis of the physical complaint, or because they fear the services of a mental health practitioner due to the perceived stigma, or because no psychologist is available.

Therapists must be aware of the possibility of an underlying psychological basis for a complaint and inquire into the emotional status of the athlete as part of the medical history. Careful, non-judgmental questioning may reveal inter-personal problems with a coach, teammate, family member, or other individuals, or anxiety concerning an upcoming competition. In these situations, a sports psychologist is invaluable. If none is available, the physician or therapist may need to assume the role of sounding board, intermediary, or stress-management advisor. At times, being a patient listener and confidant may be all that is required. If mediation between parties is required, a neutral, non-judgmental stance must be maintained to help the parties air and resolve differences.

C. Preparing for Competition

Simple psychological skills to help the athlete manage the competitive performance environment include: 1) learning relaxation skills (e.g. progressive relaxation; slow, controlled, deep abdominal breathing; or autogenic training); 2) mastering all of the attentional styles (types of concentration); 3) imagery (both visualisation and kinesthetics); 4) appropriate self-talk; and 5) developing a pre-competition mental routine to be employed immediately prior to competition on game day (these routines are short [1–2 minutes] and use all of the mental skills just presented). (See also Part 2 of this chapter, *Competition Day Preparation*.)

D. The Injured Athlete

Athletes have a strong sense of body awareness, and take great pride in the capabilities of their bodies. Thus, injuries can be psychologically as well as physically devastating. The ability to train and compete well involves enormous ego. Athletes often identify themselves by who they are as an athlete. Thus, an injury places considerable stress on this self-identification. The more severe the injury, and the longer the recovery-rehabilitation period, the more prolonged and profound the mood disturbance may be.

Injured athletes commonly experience at least three emotional responses: isolation, frustration, and disturbances of mood:

1. The injury forces the athlete to become separated from teammates and coaches. Other team members may provide little support, and in fact they may shun their injured teammate to avoid reminders of their own potential frailty.
2. The athlete becomes frustrated because he or she perceives the loss of months of training and skills mastery, although there are many instances where athletes have used the recovery period to master mental and other physical skills to return successfully to competition.
3. Mood disturbances are common. The athlete may be temporarily depressed, or become upset by minor annoyances.

An injury can provide the athlete with an opportunity to work with a caring professional to re-assess his or her reasons for being in sport, and for redefining goals in sports participation.

The health care team must be aware and include psychological support as an integral part of the treatment and rehabilitation processes. At the outset, the athlete must be fully informed about the nature and severity of the injury, the prognosis for recovery, recommended course of therapy and rehabilitation, and an estimate of the time needed before training can be resumed. The athlete must be made a full partner in the treatment and recovery process, and given responsibility for therapeutic activities that can be carried out at home. The medical team must discuss openly the psychological changes that accompany an injury, and reassure the athlete that this is to be expected. Reassurance and supportive measures are generally adequate, but a visit from an athlete who has recovered from a similar injury may be of great value.

This entire process can be facilitated by a supportive and understanding medical staff. The formula:

Genuine Caring + Skills + Courage = Positive Outcome for the Injured Athlete must be kept in mind by the staff and the athlete, even though progress may be slow and uneven throughout the treatment and recovery process.

Referral to a sports psychologist may be necessary if the athlete is deeply disturbed, or if the injury is severe and a prolonged recovery is anticipated. All injuries involve a certain degree of fear and uncertainty, and the sports psychologist may be great value in helping to deal with this emotion (see Table 5-1 and Table 5-2).

Table 5-1. From common to clinical responses: gauging referrals to therapy.

Temporary Emotional Responses	Ongoing Emotional Patterns
Sadness	Depression
Feeling isolated	Withdrawal
Irritated	Explosive
Neutral	Numb
Unmotivated	Apathetic
Frustration	Frequent crying or emotional outbursts
Anger	Rage
Moderate change in appetite	Rapid weight loss or gain, or disordered eating pattern
Minor sleep disturbance	Insomnia

Table 5-2. Sports psychology consulting with injured athletes: when to make a referral.

Consider referring to a trained, experienced sport psychology consultant if injured athlete:

- Lacks confidence in his/her ability to recover, or to engage in the rehabilitation process.
- Lacks belief in the rehabilitation process.
- Has difficulty filtering out environmental distractions during rehab or training sessions.
- Is withholding effort out of fear (of re-injury, of failure, etc.).
- Loses focus easily when pain intensifies or when discouragement sets in.
- Is engaging in excessive cognitive thinking over simple tasks.
- Is unsure of how to set and attain meaningful goals.
- Has trouble controlling thoughts about the injury, or worries about re-injury.
- Is unable to control negative self-talk.
- Desires to maximise the utility of the rehab and wishes to work more intensely on developing his/her mental game (e.g. improving confidence, concentration, composure, trust).

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ATHLETE'S COMPETITION DAY PREPARATION

Many athletes use special psychological procedures to prepare themselves on competition day. The following exercises will help you develop your own competition-day routine and achieve that hard-to-define sense of “readiness”—it may be a sense of “tingling” or the simple subjective feeling that “this is my day.”

Too high a level of activation is experienced as “stress” or anxiety and leads to muscle tightness, poor efficiency, poor attention or concentration (chaotic thinking or too narrow a focus), and loss of smooth and responsive muscle coordination. Too low a level of activation is seen as low energy, a “flat” performance, little or no motivation, and wandering attention. Both profiles lead to performance errors. How one achieves that sense of readiness that precedes optimum performance varies with each person, so carefully review your best competition days and try to identify the cues (inside of you and in your environment) that seemed to help you prepare to compete well.

A. Identify Your Stress Profile

The next time you experience some type of stress (competition, tests, talking with someone you feel uncomfortable with, etc.), notice how stress affects your body and your mind. Be very specific.

1. Muscles that tighten: Jaw clenches, shoulders tighten, fists clench, stomach tightens, other:
2. Breathing pattern: Shorter and faster, rapid speech, other:
3. Gastro-intestinal responses: nausea or unsettled sensations in the stomach; more frequent bowel movements, other:
4. Other physical signs: Dry throat, upset stomach, cold hands and/or feet, rapid, pounding heart, sweaty palms, frequent urination, other:
5. Interpersonal responses: Rapidity of speech with different people, need to be around certain people (coach, teammate, family, friends, etc.), need to be alone, need to “show them” during warm-up, watching other athletes, other:
6. Personal cues: Mind goes blank (when?), forgetfulness, unable to focus attention well (easily distracted or too narrow a focus), things you say to yourself (I've got to do better this time, what am I doing here? I hope my coach/parents don't get mad if ..., I hope I don't goof ...), other:
7. Environmental cues: Air temperature, humidity, rain, crowd noises, officials, poor fit of clothes or shoes, equipment problems, other distractions:

Use this information to identify the early signs of stress

Individuals experience stress in consistent ways, and you need to find your own stress profile. Log your responses to stress as well as the cues that were present on your *best* competition days so that you can compare the two profiles.

B. Planning for Competition Day

By now you will have some idea of what your stress profile is: when too much or too little stress is activated, WHAT or WHO triggers the stress, and HOW it affects you (both physically and mentally). Once you know the cues that interfere with your performance, you can plan a programme of psychological and physical techniques to help reach a better performance level. Table 5-3 lists activities that may help you reduce tension, or help you “activate” yourself if you are feeling flat, unresponsive, or “down.”

Be sure to use psychological techniques in your daily training programme. Like any skill, these techniques require practice before you can use them effectively under pressure. Also, be sure to keep a log of techniques and routines that help you on competition day(s).

1. Plan for the night before competition:

You may wish to use mental rehearsal techniques, but don’t use them just before sleep—this is an activation activity, not a relaxation for sleep.

2. Day of competition:

- a. Know your competition schedule, and plan activities such as eating, reaching the competition site, and getting into the locker room so that there is no sense of rushing. Some athletes become more tense if they arrive too early—find the balance that’s right for you. List the time needed to reach the competition site and a schedule you plan to follow.
- b. Every 45 minutes–1 hour check yourself for signs of stress (from A, above) and take a minute to do a body check and use stress management/self-regulation techniques that work for you. List the signs of stress and the specific techniques you plan to use to reduce stress:

If tension is too great for self-control or self-regulation, who (teammate or coach) can help you? How?

Example: Help you check breathing; muscle check; quietly repeat relaxation phrases; place hands gently on your shoulders to help lower them to a more relaxed level; help move away from distracting noises or scenes to a quieter place, etc.

3. Psychological Strategies to Use Before Competition

Internal Muscle Check: Review each muscle group (standing, sitting, or lying down). Hands, arms and fists, forehead, eyes; cheeks and jaw; shoulders and upper back; stomach; hips and lower back, thighs; lower legs and feet.

Breathing Check: Inhale and feel slight tension; exhale and relax from top of head to knees and toes. Feel the relaxation roll down the body. Periodically inhale deeply, hold your breath and feel the tension throughout your body, then relax

Table 5-3. Relaxation and activation techniques.

Purpose	Imagery	Self-phrase	Muscle Relaxation	Brief On-Site Techniques
General relaxation (awake)	Pleasant scene such as favorite activity or place	I feel relaxed, warm, heavy	Try to feel relaxed and heavy by telling each muscle by name to become more and more relaxed while staying awake. Stretch and yawn. Alternate tension and relaxation of each muscle group	Internal muscle scan and check Breathing check and exercise
Sleep	Same as above. Use colour to “flood” awareness. Get rid of worries or distracting scenes	I feel relaxed, warm, serene, calm	Same as above (but move toward sleep)	
Warmth	Serene or active scene of pleasant warmth	I feel the blood flowing through my hands and feet, I feel very relaxed and warm	Couple with muscle relaxation	Small hand-held thermometer. Place hand on forehead; if hand is cooler, increase warmth.
Activation	VMBR*: imagine ideal performance in distance VMBR: experience self doing and rehearsing specific parts of performance VMBR: practice entering the competition with relaxed and alert confidence	Self-phrases tape I feel relaxed, alert, alive, energy is flowing through my body, my arms, my legs. I feel relaxed, yet ready to move quickly and alertly.	Muscle check for relaxation, yet with anticipation of movement	Same as above. Increase heart rate, if it is slow.

*Visual-Motor Behaviour Rehearsal

your jaw, exhale and feel the contrast of the relaxation as it rolls down your body. QUICKLY “scan” your muscles and release any tension you feel. Notice if your breathing is deep or shallow. Deepen it each time so that you can almost FEEL the air “tickle your belly button.” Relax each time you exhale.

Visual-Motor Behaviour Rehearsal (VMBR): Relax as much as possible. Now, as clearly and vividly as possible, imagine yourself in an ideal performance. If you see yourself “in the distance,” add the *feeling of actually experiencing yourself doing the activity*. The difference is feeling alertly relaxed with a very slight sense of muscle activity/tension vs. feeling heavily relaxed. This technique can be used to: 1) rehearse an entire performance; 2) review and correct a specific performance problem so that doing it correctly becomes second nature; 3) practice approaching the crowd or competition with confidence.

4. Four or five hours before the event:

- a. list your objective, e.g. you want to emphasise a fast start, confidence, aggressiveness, a particular strategic approach to the other competitors;
- b. determine how to achieve the objective, e.g. plan to take a moment to visualise a fast start to the gun immediately before getting into the blocks.

5. Immediately prior to the event (before stepping to the line, blocks, or into the ring):

- a. for a second or so, visualise your complete event as you would actually perform; see it happen, make this vivid visualising include the way the body is to feel as it performs;
- b. use an inner frame of reference—you are doing it IN the scene, not watching yourself do it;
- c. clear your mind after you have programmed your body by visualisation.

NOW, let your body take off and do its job automatically.

Reference

Partially adapted from:

1. Suinn, R. M. Body thinking for Olympic champs (appendix B). *In Psychology in Sports: Methods and Application*. Burgess, Minneapolis, 1980.

NUTRITION AND ATHLETE HEALTH

The sport of athletics includes a wide range of events whose requirements range from speed to endurance, from a light physique to explosive power, and from multiple events lasting less than a minute to a single race lasting more than 2–3 hours. Despite the extreme contrast in these characteristics, all athletes share some common nutrition goals (see Table 6-1). This chapter will briefly explore these goals.

Table 6-1. The athlete's nutrition goals.

Characteristic	The athlete's nutrition-related goals
Everyday eating or training diet	<ul style="list-style-type: none"> • Achieve and maintain a physique that is suited to the event • Eat to stay healthy and injury-free • Train hard and promote optimal adaptations and recovery from the training programme • Practice competition eating in training to fine tune strategies
Competition eating	<ul style="list-style-type: none"> • Prepare adequate fuel stores for the event • Eat and drink well on competition day to prepare for an event and to recover between multiple events • During prolonged events (> 1 hour), replace fluid and carbohydrate to enhance performance • Achieve competition strategies when traveling
Sports foods and dietary supplements	<ul style="list-style-type: none"> • Make use of specialised sports foods to meet nutritional goals when it is impractical to eat everyday foods • Make wise decisions about the use of nutritional ergogenic aids based on cost: benefit analysis

A. The Training Diet

1. Achieving Energy Needs

Energy needs vary according to body size, the energy cost of training (volume, frequency, and intensity of workouts) and requirements for growth or changes in body physique. As a result, energy needs vary not only amongst athletes, but vary between phases of the season and over the athlete's career. It is important for each athlete to achieve a suitable energy intake since this affects hormonal and metabolic function as well as the ability to provide adequate fuel for training, and to consume the range of nutrients and food components that promote good health.

There are often problems at the extremes of energy intake or requirement. Examples include the athlete who over-restricts their energy to reduce body

weight and body mass levels, the athlete who is unable to adjust to a new energy requirement while they are injured or in the off-season, and the athlete with very high energy requirements who is unable to consume adequate food in a busy day. A consultation with a sports dietitian can help such athletes to recognise their true energy requirements and adopt appropriate eating patterns and food choices.

A new concept that is being promoted is that of energy availability—that is, the energy that is left for body functions once the energy cost of training is taken into account (see Table 6-2). There is evidence that the body can tolerate a certain level of reduction in energy intake, but energy availability lower than 30 kcal (126 kJ) per kg of lean body mass is associated with impairments of metabolic, hormonal, and reproductive function. This is now identified as being important in the development of menstrual disorders in female athletes.

Table 6-2. Calculation of energy availability.

Definition of energy availability	Total energy intake—energy cost of training
<p><i>Example of low energy availability</i> Athlete = 50 kg distance runner, 10% body fat Training programme = 1000 kcal/day Energy intake = 2250 kcal</p>	<p>Calculations: Body fat = 10% or 5 kg Lean body mass (LBM) = 45 kg Energy availability = $2250 - 1000 = 1250$ kcal $= 1250/45$ kg $= 28$ kcal/kg LBM</p>
<p><i>Example of adequate energy availability</i> Athlete = 60 kg distance runner, 10% body fat Training program = 1000 kcal/day Energy intake = 3250 kcal</p>	<p>Calculations: Body fat = 10% or 6 kg Lean body mass (LBM) = 54 kg Energy availability = $3250 - 1000 = 2250$ kcal $= 2250/54$ kg $= 42$ kcal/kg LBM</p>

2. Maintaining the Ideal Physique

In many events, the athlete's physique plays an important role in promoting optimal performance. For example, a high level of muscularity is required to achieve explosive power in sprints or strength for throwing events. Furthermore, when the athlete is required to move their own body mass over long distances (distance running and walking) or against gravity (jumps and hilly running courses), a favorable “power to weight” ratio is achieved by being light and lean.

Some athletes automatically arrive at a desirable physique for their event, as a result of genetics and the conditioning effects of training and healthy eating. Other athletes need to manipulate their training and diet to produce an increase in muscle mass and/or a loss of body mass and body fat levels. In many cases, the expectations of such a program are unrealistic or the dietary strategies are unsound.

Loss of body fat/body mass should be gradually achieved by a program of mild energy deficit that still permits the achievement of the athlete's other dietary goals and allows the athlete to be reasonably free of food-related stress. The

athlete should not strive for minimal body fat levels per se, but rather a physique that is associated with good performances and health over both the long term and short term. An increase in muscle mass and strength is achieved by an appropriate resistance program with the support of adequate energy intake and strategic timing of food intake around training sessions.

3. Protein Needs

Athletes in events requiring strength and power (e.g. sprinters and throwers) often believe that protein intake is their most important nutritional concern, and that high protein diets and protein supplements are a required part of their preparation. The perceived link between a high protein intake and gain of muscle protein is understandable but is not supported by scientific evidence. By contrast, many athletes in endurance events (e.g. distance runners and walkers) pay little attention to dietary protein despite the importance of protein synthesis in the achievement of their desired training adaptations (e.g. repair of muscle damage and the synthesis of functional body proteins such as enzymes).

Whether heavy training increases protein requirements is still debated. Studies typically show that protein intakes needed to achieve nitrogen balance are not elevated in subjects habituated to an exercise load. However, this may not reflect the situation for athletes who practice progressive overload in their training. Nevertheless when the possible increases in protein requirements have been calculated for athletes in heavy training, they appear to be within the range of 1.2–1.6 g/kg body mass per day for both strength and endurance activities. Dietary surveys show that such intakes are achieved by most athletes, especially those consuming high-energy intakes. Therefore, an exaggerated focus on protein-rich foods or high protein supplements is unnecessary. Athletes who are most at risk of consuming protein intakes below this range are those who restrict total energy intake to lose weight. This is particularly the case for female athletes.

Recent studies suggest that the total *amount* of protein consumed by the athlete is not as important as the *timing* of intake in relation to training. The consumption of protein before and after a resistance workout has been shown to enhance protein synthesis and net protein balance in response to the training stimulus; this enhancement is still evident in the 24-hour picture of protein balance. This strategy should be integrated into the athlete's recovery eating program.

4. Meeting Fuel Requirements for Training

Although various substrates combine to provide the fuel for exercise, the body's carbohydrate stores are limited and are often less than the fuel cost of daily training. The athlete's everyday eating should provide adequate carbohydrate to fuel training and promote adequate recovery of muscle glycogen stores between workouts. Older dietary guidelines promoted a single recommendation for the carbohydrate content of the athlete's diet, expressing this as a percentage of total energy intake (e.g. 55–70% of daily energy).

However, it is now recognised that this terminology fails to recognise the varying fuel needs associated with different types of training, and falsely assumes that these fuel needs are always aligned with total energy requirements. Therefore, new guidelines express carbohydrate needs according to the type and volume of training, and the size of the athlete. It is suggested that daily carbohydrate needs vary from ~5 g/kg body mass for athletes undertaking a light training program or training that is not dependent on muscle glycogen, to intakes of 7–10 g/kg for prolonged and strenuous daily workouts or where optimal synthesis of glycogen is required. Of course, these guidelines are considered “ball park” figures and should be fine-tuned by the athlete according to their total energy budget and feedback from training performances.

5. Vitamins, Minerals, and Anti-oxidants

Food contains a variety of vitamins, minerals, anti-oxidants, and other food components that promote optimal function and health. Generally, we can be confident that a moderate to high energy intake (>2000 kcal or 8.4 MJ per day), chosen from a wide variety of nutrient-rich foods, is able to supply all needs for these micronutrients and food components. Athletes are at risk of inadequate intake of these compounds when they restrict energy intake, dietary variety, or both of these factors. This includes athletes following weight loss diets or other programs that limit food choices, fussy eaters, and athletes who are traveling to areas in which food availability is limited. These athletes should be directed to a sports dietitian for advice to improve the quantity and quality of their food intake. Where the athlete is unable or unwilling to implement such changes, a low-dose broad range multi-vitamin/mineral supplement should be considered.

The micronutrients that are at most likely to be consumed in inadequate amounts are iron and calcium. Iron deficiency occurs in athletes for the same reason that it occurs in the general population; intake of bio-available iron that is less than iron requirements or iron losses. Risk factors for low iron intake include low energy intake, vegetarian eating, and other eating patterns that restrict dietary variety and intake of red meats. While iron requirements are increased during growth and pregnancy, iron losses are increased by problems of gastrointestinal bleeding or malabsorption (e.g. ulcers, Crohn’s disease, or parasitic infections), excessive hemolysis associated with footstrike, and unusual blood loss (frequent blood donations, traumatic bleeding, and abnormal menstrual blood loss). It appears that the prevalence of iron deficiency anemia in athletes is similar to that of the sedentary population. However, there is a higher prevalence of iron deficiency without anemia (hemoglobin within normal ranges, but serum ferritin levels below 20–30 ng/ml) and recent studies show that this condition is associated with a reduced responsiveness to training. It is now recommended that interventions occur at this stage, even if it is only to prevent a further decline into anemia.

Prevention and treatment of reduced iron status should include attention to the factors that are causing iron drain, including dietary modifications to increase the intake of bio-available iron. Such dietary changes may include the frequent intake of

small servings of foods containing the heme form of iron (e.g. red meats, shellfish, liver) and increased consumption of good sources of non-heme or plant iron (e.g. fortified breakfast cereals, nuts and legumes, wholegrain cereals, and green leafy vegetables). The athlete should also plan meals to combine the plant sources of iron with factors that promote iron absorption (e.g. Vitamin C and “meat factor”), while reducing interaction with excessive amounts of food factors that reduce the bio-availability of this iron (e.g. phytate in bran or tannin in tea). Vegetarians should pay particular attention to such planning. Iron supplements may be part of the therapy required to achieve good iron status in athletes. However, athletes should be warned about self-prescribing iron supplements on the basis of perceptions of fatigue. Iron deficiency needs to be diagnosed and treated by appropriate experts in sports medicine and sports nutrition.

Some athletes also fail to consume adequate calcium in their diets; this is a factor but not a primary cause in the development of poor bone health and stress fractures. The athlete should receive early intervention in the treatment of such problems (see Female Athlete Triad, below). This may include dietary advice to correct low energy availability, and to increase dietary calcium intake, principally through the consumption of low-fat dairy products or calcium-fortified soy alternatives.

B. Special Issues

1. Eating for Recovery

The training and competition programs of high-level athletes often require several sessions of exercise each day. This has focused the attention on eating strategies that promote recovery after strenuous exercise and enhance the adaptations achieved by the training program. Nutrition-related aspects of recovery include refueling, rehydration, rebuilding and staying healthy. Rehydration requires the replacement of the fluids and electrolytes lost in sweat, and when the fluid deficit remaining after a workout or race is greater than 2% of body mass, it is sensible to implement a rehydration plan rather than relying on thirst or good luck. Strategies to remain as well-hydrated as practical during exercise are discussed in the section on competition eating (below). These strategies should also be used during training sessions, to enable the athlete to train optimally as well as to fine-tune the drinking practices that might occur during prolonged competitive events.

During the hours after exercise, the athlete should be guided to consume a volume of fluids equal to 125–150% of the remaining fluid losses. The replacement of electrolyte losses, particularly sodium, is also needed to maximise the retention of these drinks and the re-equilibration of body fluids. Sodium replacement can be achieved via the intake of electrolyte replacement products (e.g. oral rehydration solutions), foods with high sodium levels (e.g. bread, breakfast cereals, and other processed foods), or the addition of salt to meals.

The speedy resynthesis of muscle glycogen levels is assisted by the intake of carbohydrate-rich foods and drinks, and where there is less than 6–8 hours between workouts, it makes sense to maximise the time for efficient muscle refueling by

consuming a carbohydrate supply as soon as practical after the first session of exercise. The inclusion of protein to post-exercise recovery eating is valuable in promoting net protein synthesis after exercise, including gains in muscle mass and strength and the repair of muscle damage. Such recovery eating probably requires the intake of ~10–20 g of high quality protein and 1 g of carbohydrate per kg body mass in the hour following exercise. In the case of resistance training, there is some evidence that the consumption of protein *prior* to the session is particularly effective in promoting the net protein gain following a workout.

To achieve recovery eating goals for key sessions of training or competition, the athlete should organise a suitable supply of snacks that can be taken to their exercise venue, or re-organise their daily timetable so that meals can be eaten in proximity to the session. Examples of food combinations that provide protein and carbohydrate are found in Table 6-3.

Table 6-3. Examples of foods that can be used for rebuilding and refueling goals.

Foods providing 50 g carbohydrate for refueling goals	Foods providing 50 g carbohydrate and at least 10 g high quality protein for refueling and rebuilding
• 600–800 ml sports drink	• 1.5–2 cups breakfast cereal with 1/2 cup milk
• 450 ml soft drink or orange juice	• 200 g carton fruit-flavored yoghurt + 35 g cereal/granola bar
• 60–70 g packet of jelly beans	• 1 round sandwich with cheese/meat/chicken filling + piece of fruit
• 2 sports gels	• 500 ml flavored low-fat milk
• 3 medium pieces of fruit or 2 bananas	• 300 ml liquid meal supplement
• 2 thick slices of bread with jam or honey	• 2 slices of toast + 1 cup baked beans

2. Staying Healthy and Injury Free

To maximise the length and success of a sporting career, an athlete needs to stay healthy and injury free. Sound nutrition is one of the factors that can assist this goal. Conversely, poor eating practices can increase the risk of succumbing to sickness or injury, or lengthen recovery time. Heavy training is associated with a suppression of the immune system, especially during the hours following a bout of prolonged or strenuous exercise. Although products such as vitamin C, Echinacea, glutamine and bovine colostrum have been proposed as immune protectors, the available research fails to support overall benefits to the athlete's health. Instead the nutritional factors that are most likely to influence immune health are energy and carbohydrate status.

Inadequate energy intake is known to impair immune function and increase susceptibility to illness and infection. Carbohydrate depletion during exercise is associated with an increase in stress hormones and a suppression of immune

parameters. Strategies that enhance carbohydrate availability, such as carbohydrate intake during and after exercise, have been shown to reduce these effects. Studies that track the success of these strategies in reducing the incidence of illness and infection in athletes are required.

Energy restriction and carbohydrate depletion can also be factors in injury. Training while fatigued can increase the risk of both traumatic and overuse injuries. More importantly, there are clear links between restrained eating and poor bone health. New research shows that low energy availability affects rates of bone turnover; this is in addition to its indirect effects on bone via disturbances to reproductive hormones in females. This is discussed in more detail in the section on the Female Athlete Triad. Since athletes are usually concerned about illness and injury, emphasis on the benefits of well-chosen eating practices could provide a new education message in sports nutrition.

3. The Female Athlete Triad

The term Female Athlete Triad was coined over a decade ago to describe the inter-relatedness of eating disorders, amenorrhea, and osteopenia in female athletes. At this time the syndrome involved a clinical diagnosis of these three medical issues and was often described in female distance runners. The aim of drawing attention to this syndrome was to change perceptions about the problem of impaired menstrual status in female athletes.

Originally, the cessation of regular periods was considered a benign condition, and was sometimes prized by female runners as a convenience or reward for heavy training. However, the past decades have seen accumulating evidence of the negative effects of disturbances to reproductive hormones on bone accumulation and bone density. Immediate problems include an increased risk of stress fractures, while the long-term picture includes a premature onset of osteoporosis. (See Chapter 13, Part 1, *Endocrine/Menstrual Factors* for additional details.)

The updated version of the Female Athlete Triad now targets energy availability, menstrual health, or bone density. It considers that each of these issues enjoys a continuum between optimal health and frank disorder, and that the athlete should be alerted to any change in their status of any issue. In other words, athletes must be educated that negative outcomes occur at a much earlier stage than previously considered, and that there are benefits of an earlier diagnosis and treatment of problems. The detection, prevention, and management of the Female Athlete Triad, or individual elements within it, require expertise and, ideally, the teamwork of sports physicians, dietitians, psychologists, and the coach/fitness advisor.

C. Competition Eating

1. Fueling Up for Competition

In the days and hours prior to competition, the athlete should consume foods to prepare adequate fuel stores for their event. The normalised muscle glycogen levels of a trained athlete are considered adequate for the needs of events lasting

up to 60–90 minutes. Preparation for such events can be managed by 24 hours of high carbohydrate eating and tapered exercise. Races lasting longer than this—for example, a marathon or 50 km race walking—are associated with the depletion of muscle glycogen which causes fatigue known as “hitting the wall”.

Carbohydrate loading is a technique of maximising or super-compensating glycogen stores in the days leading up to the event. The original technique, described from research undertaken on relatively untrained subjects, required a period of low carbohydrate intake and hard training to deplete muscle glycogen levels (= 3 day depletion) followed by 3 days of high carbohydrate eating and exercise taper to supercompensate glycogen stores. More recent studies have shown that this depletion phase is unnecessary in trained individuals, and glycogen supercompensation can be achieved in as little as 36–48 hours of taper and high-carbohydrate eating (~10 g/kg/d). Optimising muscle glycogen stores will allow the endurance athlete to run or walk longer in their event at their optimal race pace. Carbohydrate consumed in the hours before, and during prolonged events can provide additional fuel for the muscle and central nervous system.

2. Eating On the Day of Competition: Pre-Event and Between Events

The goals of the pre-event meal are to provide a final top-up of fuel and fluid stores, and to leave the athlete feeling confident and prepared to compete at their best. In general, a carbohydrate-rich meal is promoted, and the athlete should experiment with the type, amount, and timing of food intake to find an individualised plan that suits the needs of their events. When high-intensity exercise or competition nerves increase the risk of gastrointestinal upsets during the event, the athlete should try to reduce the fibre intake, fat intake, or volume of their last meal. Some athletes find that a liquid meal (commercial liquid meal supplement or fruit smoothie) is a convenient option.

The competition requirements of sprinters, jumpers and multi-event athletes (heptathletes and decathletes) often include a series of events over a day or days. Food and fluid intake over these days should be organised to refuel and rehydrate according to the needs of the event and the environmental conditions. The athlete should organise a nutrition plan of meals and snacks that suits the timetable of warm-up, competition and recovery.

3. Fluid and Fuel Intake During Events Lasting More Than 1 Hour

In events of greater than one hour duration, there is both an opportunity and need to consume fluid and carbohydrate during exercise to promote optimal performance. The loss of fluid and electrolytes through sweating leads to a reduction in body water. Fluid deficits of as little as 2% of body mass can impair performance, particularly in hot weather, with the impairment increasing in ratio to the size of the fluid deficit. Fluid intake during a race should be undertaken in consideration of the athlete’s likely sweat rates balanced against the practicalities of the time lost in grasping and consuming the supplies at an aid station, and the risk of gastrointestinal problems. Monitoring weight changes before and after training sessions and events

can provide an estimate of sweat rates (1 kg loss ~1 liter of sweat), and help to develop a personalised fluid intake plan. (See Appendix 5, *IAAF Policy on Fluid Replacement*, for additional information.)

Ideally, the athlete should try to match fluid intake with sweat losses as well as is practical, or to keep the fluid deficit to less than 2% of body mass. Although this is sometimes difficult for elite athletes running at high speeds in hot-weather races, practicing drinking during training can help to improve the skills and tolerance of fluid intake “on the run”. Slower runners are better able to match fluid intake to their sweat losses, and in some cases, need to be warned about the dangers of overdrinking. Runners should not drink excessive amounts of fluid during an event so that they increase their body mass over the race. Overdrinking increases the risk of developing hyponatremia, which is a potentially fatal condition.

In events lasting greater than 60–90 minutes, race intake should also include carbohydrate to provide an additional fuel source. A strong literature shows that the intake of 30–60 g of carbohydrate per hour of a prolonged event can enhance performance by maintaining carbohydrate availability at a time when body carbohydrate stores are becoming depleted. This can be achieved by drinking commercial sports drinks that typically contain 4–8% carbohydrate and 10–20 mmol/l sodium. Such drinks have been manufactured to allow simultaneous replacement of fluid and fuel during exercise, and their pleasant taste has been shown to increase the voluntary intake of fluid compared to plain water. Recent studies have shown that the intake of carbohydrate may enhance performance of high-intensity events lasting about 1h (e.g. a half marathon). This is curious since muscle glycogen are not limiting in this event, at least if the athlete has undertaken a good nutritional preparation for competition. It is thought that benefits might be derived through the central nervous system, whereby carbohydrate supplementation causes enhancements of the perception of effort and pacing strategies. Further research is needed, and athletes in these shorter events should experiment with a race plan including carbohydrate intake from sources such as sports drinks, sports gels or confectionery items.

4. The Travelling Athlete

Most elite athletes are well-seasoned travelers, undertaking trips to training camps or specialised environments (e.g. altitude), and to compete. Athletes must be able to achieve their peak performance at important competitions such as Olympic Games or World Championships in an environment that is often both far away and different to their home-base. Many athletes compete in series like the Golden League or Grand Prix, which require them to travel almost weekly to compete against other athletes in this competition. Frequent travel can pose a number of challenges. These include disruptions to the normal training routine and lifestyle while the athlete is en route, jet lag, changes to food availability including absence of important and familiar foods, and a reliance on hotels, restaurants, and takeaways instead of home cooking. Even in an Athlete’s Village there are the temptations of an “all you can eat” dining hall. Depending on the destination, the athlete may be at

risk of gastrointestinal illnesses due to exposure to food and water with poor hygiene standards (see Chapter 12, *Infectious Diseases*).

There are many strategies that an athlete can put into place to overcome these challenges. These include preparation techniques such as investigating the likely food issues in the new environment, and organising a supply of important or missing foods to accompany the athlete. Items that are portable and practical for travel include powdered sports drinks and liquid meal supplements, sports bars, cereal/granola bars, breakfast cereal and skim milk powder, dried fruit and nut mixes, and rice cakes/crackers with spreads. Having an “emergency” supply of foods can allow the athlete to supplement inadequate meals, provide additional snacks, and be self-reliant in looking after nutritional needs at key times such as post-training or post-event.

D. Sports Foods and Supplements

Sports foods and supplements represent a multi-billion dollar industry, supported by aggressive marketing from manufacturers and word of mouth between athletes and coaches. Sports scientists believe that well-controlled research should underpin the promotion of any sports nutrition practice and are understandably frustrated that supplement manufacturers often make impressive claims about their products without adequate (or in some cases, any) proof. However, in most countries, legislation regarding supplements or sports foods is either minimal or not enforced, allowing unsupported claims to flourish and products to be manufactured with poor compliance to labeling and composition standards (see also Part 2 of this chapter, *A Rational Approach to Supplements*, for additional details). Athletes are usually unaware of these lapses.

Before making a decision to use a supplement or sports food, athletes and coaches should consider the likely benefits, balanced against the cost of the product and the risk of negative outcomes. Problems include the risk of side-effects or inadvertent intake of a substance that is banned in sport, leading to a “positive” doping outcome. The advice of a sports nutrition expert should be sought to provide such information.

The ever-growing range of sports nutrition products can be divided into two separate groups. Some supplements and sports foods address the special nutritional needs of athletes, and offer a simple or practical way to meet known nutritional goals. This group includes sports drinks, sports bars, liquid meal supplements and micronutrient supplements that are part of a prescribed dietary plan. Many of these products are specially designed to help an athlete meet specific needs for energy and nutrient, including fluid and carbohydrate, in situations where everyday foods are not practical to eat. The use of many of these products has been discussed in previous parts of this chapter. These sports foods and supplements can be shown to improve performance when they allow the athlete to achieve their sports nutrition goals. However, they are more expensive than normal food, and this consideration must be balanced against the convenience they provide.

Conversely, some products claim a direct ergogenic benefit to sports performance. Very few of these products have clear scientific evidence to support these claims, with a few exceptions including caffeine, creatine, and bicarbonate/citrate. Athletes should seek expert advice about such supplements to see if their sport/exercise warrants experimentation with these products to ensure that a correct protocol is tried. It should be noted, however, that supplements and sports foods are neither a short cut to optimal performance nor a replacement for the sound principles that underpin good training.

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A RATIONAL APPROACH TO SUPPLEMENTS

Nutrition scientists in recent years have identified an increasing number of elements that are now deemed essential for a complete diet. In addition to the well-known critical need for vitamins and macro-nutrients (energy fuels), they have noted the importance of the ‘essential’ amino acids, trace minerals, fatty acids, and phytochemicals, as well as many other organic compounds. With the increasing use of industrial food production processes, many of these elements have been found to be deficient in various segments of the population, including possibly athletes who are training intensively.

It is well known that the diets of many pre-adolescents and adolescents, including athletes, are often lacking in adequate calcium and iron. In this age group the consumption of “empty calories” (sugars and sweets) is well documented. This is especially true among young women. Further, those women athletes in whom weight control is a concern are often calorie-deficient and suffer from an “energy deficit” that may have long-term effects on skeletal and reproductive health.

A. Athletes and the Supplements Industry

In order to ensure that athletes are obtaining nutrients sufficient for their training needs, they should have a complete dietary analysis and counseling by a qualified sports nutritionist, including a blood analysis for macro- and micro-nutrients.

Instead, athletes and coaches have been convinced by the dietary supplement industry that all athletes’ diets are inadequate, and routinely need supplementation.

The “nutritional supplement” industry has now become a multi-billion dollar enterprise. This industry utilises modern advertising techniques and mass-marketing, which is often based upon pseudo-science, factual distortion, and nutritional half-truths, in order to promote its products.

The U.S. “Dietary Supplements and Health Education Act” of 1994 established the legal framework for the manufacture of supplements. A dietary/food or nutritional supplement is defined as:

1. A product (other than tobacco) that is intended to supplement the diet that bears or contains one or more of the following ingredients:
 - a. a vitamin
 - b. a mineral
 - c. an herb or botanical
 - d. an amino acid
 - e. a dietary substance for use by man to supplement the diet by increasing the total dietary intake
 - f. or a concentrate, metabolite, constituent, extract, or combination of any ingredient described above.
2. Is intended for ingestion in pill, capsule, tablet, or liquid form.

3. Is not represented for use as a conventional food or the sole item of a meal or diet
4. Is labeled as a “dietary supplement”

These products are sold primarily as “ergogenic aids”, “natural” or “legal” steroids, “fat burners”, energy or immune system “boosters”, etc. Many of these contain or are contaminated with androgenic pro-hormones that the body may convert to active anabolic steroids, resulting in a positive doping test.

The dietary supplement industry is unregulated, as these products are classified as “foods.” They are not required to demonstrate the efficacy of their claims, so long as they do not purport to treat diseases.

Numerous studies have shown that:

- The quantity of the listed products on the label may vary widely from the amount listed.
- Many products contain ingredients that are not listed.
- “Natural” products are not necessarily safe.
- Side-effects are seldom listed.
- They may interfere with other prescription medications.
- They may create deficiencies of other nutrients.
- Some products that are purported to contain “legal” or “natural” pro-hormones actually contain banned anabolic steroids.

Many studies have shown that a significant number of supposedly “legal” or “safe” supplements may contain anabolic steroid precursors or pro-hormones that will result in a positive doping test. This is especially true if the manufacturer also produces a line of products that contain these banned substances, as cross-contamination may occur during the manufacturing process. In addition, Geyer et al. (2004) have shown that a sizable percentage of supplements are contaminated with androgenic-anabolic steroids, even those manufactured by a company that does not produce a line of anabolic products. The majority of these products are manufactured in the U.S., but labels are not required to list the country of origin.

It has been suggested that athletes use only supplements that 1) have been approved by such agencies as the U.S. Pharmacopeia (USP) or similar, or 2) have been tested and certified by the manufacturer as being free of pro-hormones or other banned substances, and have had no contact with these substances during their production or transport. Such certification ensures the quantity and purity of the product, but does not ensure its being free of banned substances.

However, it is unlikely that this type of certification will occur to any extent, unless laws governing the manufacture of these supplements are altered radically. The number of elite athletes who are subject to doping control is miniscule compared to the total number of consumers. Hence, such testing and approval would not be cost-effective for the manufacturer. Further, even if the manufacturer were to test and certify its products, in the event of a positive doping test such verification would not likely render it immune from a legal liability suit.

B. IAAF Recommendations

In view of the foregoing, the IAAF must still recommend that:

1. Athletes must review their diet carefully, along with a qualified sports nutrition professional, to ensure its adequacy, and to determine whether any supplementation is necessary (see Part 1 of this chapter, *Nutrition and Athlete Health*).
2. In nearly all cases, the athlete's complete nutritional needs can best be met by a well-selected menu of properly-prepared foods. This menu is more likely to provide the essential micro- and macro-nutrients, vitamins, and essential amino acids in a well-balanced, readily absorbable combination than can be obtained by the use of a barrage of expensive, randomly-chosen "supplements." However, many health professionals do now recommend a simple multi-vitamin preparation daily for all adults, and specific supplementation may be required if a true deficiency is detected by reliable testing.
3. Athletes and coaches must realise that the consumption of supplements, even under the best of circumstances, risks the possibility of causing a positive test for a banned substance during doping control. This possibility may vary from manufacturer to manufacturer, and even from batch to batch by the same manufacturer. Hence, we cannot avoid the adage—CAVEAT EMPTOR! (LET THE BUYER BEWARE).

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PRINCIPLES OF INJURY PREVENTION

The sports medicine professional is concerned with the well being of the athlete and generally assumes the responsibility for overseeing the total health care for the athlete. Participation in sports places the athlete in a situation in which injury is likely to occur. Fortunately, most injuries are not serious and lend themselves to rapid rehabilitation, but the sports medicine professional must be capable of dealing with any type of trauma or catastrophic injury.

A. Physical Conditioning

Physical conditioning is a key principle of injury prevention. Appropriate conditioning programmes decrease the risk of injury, decrease the severity of an injury should it occur, and can help prevent re-injury. Maximising the chance for safe athletic performance requires adequate muscular strength and balance, power, endurance, neuromuscular coordination, joint flexibility, cardiovascular endurance, and good body composition for sport.

Improving specific components of fitness and conditioning reduces the risk of injuries. For example, strengthening the muscles of a joint helps reduce injuries to the area; regular exercise can significantly increase the strength of the ligaments surrounding the knee and prevent knee injuries; development provides increased strength that helps to stabilise joints; and improved movement skill is important in avoiding injury.

1. Strength

To improve muscle strength, stress must be progressive and gradually challenged or placed under additional loading. A conditioning programme's effects are specific to the type of stress applied. The SAID principle (Specific Adaptation to Imposed Demands) states that as the body is placed under stress of varying intensities and durations, it attempts to overcome the stress by adapting specifically to the imposed demands. For example, muscles around a joint can be developed and conditioned to provide optimal stabilisation of the joint. Likewise, when a muscle primarily produces motion of a joint, proper conditioning can prevent the muscle from undergoing an unwanted movement. The demands of a specific athletic event must be a progressive stress applied in that athlete's training.

Other components of strength conditioning that contribute to injury prevention are the ability of the muscle to contract or exert force at an accelerated speed, and muscular endurance, which allows the athlete to maintain an appropriate strength level over a period of time.

2. Balance

Proprioceptive or kinesthetic sense through balance training enhances motor control, which is needed to decrease the risk of injury or re-injury during practice or competition. When injury to a joint or musculo-tendinous structure occurs,

somato-sensory information is altered, adversely affecting motor control. Hence, rehabilitation should emphasise restoring the athlete's balance strategies. This will also decrease the risk of recurrent injury. The balance training tasks must be specific to the type of balance strategies required by the athlete's event.

3. Flexibility

Efficient performance requires a full range of motion, and adequate joint flexibility also decreases an athlete's susceptibility to injury. Normal muscular length-tension and adequate extensibility upon stretch aid in protecting the body from injury. The athlete's entire body is able to work more efficiently and safely after a period of warm-up, stretching, and skill-drills that are related to the athlete's event.

The warm-up period before practice or competition increases the body's tissue temperature prior to subjecting the musculo-tendinous structures to repeated stretch and contraction. Connective tissue has visco-elastic properties, which allow elongation of the tissue. Temperature has a significant influence on the mechanical behaviour of connective tissue under tensile stretch. Higher temperatures at low loads produce the greatest elongation with the least damage to connective tissue. Increased connective tissue temperature also increases extensibility.

Optimal stretching occurs only when voluntary and reflex muscle resistance is eliminated. Ballistic stretching is not a favorable method because as the muscles stretch rapidly, the intrafusal muscle spindles may be activated, causing a reflex protective muscle contraction. Forceful ballistic stretching can also cause micro-trauma of muscle fibres.

4. Endurance

Cardiovascular endurance is also a factor in injury prevention. The cardiovascular and respiratory systems must be adequately conditioned to delay the onset of fatigue. A fatigued athlete becomes vulnerable to injury when the nervous and muscular systems are unable to respond adequately to an injury-producing situation.

B. Appropriate Training Methods

Ensuring proper, efficient mechanics requires practice and effective coaching, including a systematic series of specific, repetitive, and progressive exercises and drills. Faulty mechanics must be corrected and good fundamentals ingrained. Exercises should include strength, relaxation, and flexibility specifically geared to the demands made on the body. (See Chapter 3, Part 1, *Principles of Training*.)

C. Rest and Recovery

Adequate sleep is important for general good mental and physical health, and becomes critical for recovery after intensive workouts. Chronic overexertion and fatigue can make the athlete susceptible to injury. (See Chapter 3, Part 2, *Restoration, Recovery, and Overtraining*.)

D. Muscle Soreness

Muscular over-exertion may present as muscle soreness, muscle stiffness, and muscle spasm. According to the *muscle spasm hypothesis* of muscle soreness, ischemia to the muscles releases pain substances from the muscle fibres and stimulates the pain receptors, resulting in reflex spastic contractions and a continued cycle of ischemia and pain. Stretching the muscles helps reduce the spasms and associated pain. According to the *tissue damage hypothesis*, micro-tears occur and pain/soreness results from the nerve-endings being stimulated by muscle tissue swelling. Proper massage may aid in reducing tissue oedema and decreasing accompanying muscle spasm. Ice applications or other forms of cryotherapy, and pool training, may facilitate the body's healing response. Appropriate rest will allow microscopic damage of the tissue to heal.

E. Appropriate Equipment

Shoes are the most critical piece of a track and field athlete's equipment and should be individually and carefully selected. Proper fitting shoes can mean the difference between a low and a high risk of injury for a track and field competitor. Training in improperly fitted shoes can result in chronic abnormal pressures to the foot and cause stress injuries or structural deformities. Minor skin irritations such as calluses and blisters can prove to be major hindrances to a runner. Improperly fitted or worn-out shoes can lead to mechanical disturbances and postural, muscular, and joint dysfunctions.

The recent revolution in shoe research, design, and production has created a plethora of shoes from which to choose. However, the athlete's shoes must meet the biomechanical requirements and adapt to the demands of the individual's event. Shoe surveys can be useful in analysing the specific qualities of shock absorbency, foot control, and flexibility, but athletes and coaches must be aware that new shoe models have produced new injury syndromes.

Field event implements must meet use and safety specifications. Every member of the sports team (coach, official, sports medicine personnel, athlete) must be aware of any hazardous field situation where the field event practices and competitions take place, and take action to assure the highest level of safety. (See Chapter 2, Part 4, *Sports Rules and Athlete Safety*.)

F. Psychological Factors

Athletes need to be psychologically prepared for practices and competition in order to reduce the risk of injury. Research has demonstrated a positive relationship between stressful life situations, especially those with high negative stress, and injury occurrence. In understanding the stress-injury relationship, Nideffer (1983) points out that muscle tension increases in response to stress. Increased tension in the antagonist and agonist muscle groups results in reduced flexibility and loss of motor coordination. Increased muscular tension also slows reaction time, which reduces the athlete's ability to respond.

Mental as well as physical fatigue can contribute to injury occurrence. The attention factor—the ability to maintain a high level of concentration—requires a large amount of energy; when combined with a rigorous training programme, reduced attention can result. This may lead to slowed reaction times and loss of neuromuscular coordination, thus increasing the potential for injury.

Athletes who have sustained an injury realise that they have to be mentally ready for return to sport to avoid risking re-injury. The role of attentional focus and muscular tension can be a major problem. Fear and/or worry about a second injury can cause stress and increased muscular tension. Preliminary studies have addressed hardiness (commitment, control, and challenge) of the athlete as a moderating factor in the stress-injury relationship. Athletes who exhibit greater qualities of this trait may be better able to control the attentional processing of information and in turn reduce the potential for occurrence of a second injury.

G. Training in Extreme Conditions

Athletes and coaches should take into account the temperature and humidity during training, and the need to acclimate after travel to a different, extreme climate or altitude. Extreme heat and humidity, cold, and altitude can adversely affect performance in many athletic events (see Chapter 11, *Environmental Factors Affecting Human Performance*). To avoid dehydration and the fatigue that can occur from inadequate fluid replenishment, athletes must drink extra water, juices, and other fluids (see Chapter 6, Part 1, *Nutrition and Athlete Health*). Athletes should learn to drink before they feel thirsty—by the time an individual is aware of thirst, they've lost 1% of their body weight; by 2% dehydration, the athlete may have reduced his or her work capacity by 10–15%. Assuring adequate water, and juices or sports drinks, helps keep the participant energised, focused, and better able to concentrate.

The health and safety of the athlete must be the number one priority in any practice or competitive situation. If unsafe climatic conditions occur, training should be curtailed, and practice or competition times re-scheduled to allow the safest environment for all participants (see Chapter 2, Part 4, *Sports Rules and Athlete Safety*).

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TRIAGE AND ACTION PLANS

Most events in track and field have a low risk of serious or critical injury, with the exceptions of events such as the pole vault, javelin, hammer, and discus. When providing medical care for an athletics event, both athletes and spectators should be considered. Therefore, planning should cover various athletic medical conditions as well as sudden illness (heart attacks, strokes, fainting, hypoglycemia, heat illness, asthma).

Sports medicine personnel must be acquainted with the principles of acute care, as much depends on the initial response. Personnel carrying out first aid must be familiar with the devices and medicines needed for primary care.

The following should be considered when providing medical support for an athletics event:

- Medical team and medical supplies
- Plan of Action (Emergency Action Plan)
- Triage and first aid measures

A. Medical Team

The medical team providing medical services for an event should include the following:

1. Medical physician with training in emergency medicine
2. Support staff (athletic trainers/physiotherapists, nurses, emergency medical technicians)
3. First Aiders (Red Cross)
4. Ambulance and driver on standby
5. Allied health care providers: masseurs, chiropractors, acupuncturists, etc.

As a guide, the number of First Aid staff required for an event would vary from 3–6 for a small event involving about 100 participants to 50–100 for a large event, e.g. a marathon involving more than 3000 participants.

B. Planning and Logistics

Planning and logistics for a major athletics competition include:

1. The need to have designated first aid stations, which could be in the form of tents or other cool, shady areas to treat injuries/illnesses.
2. Designated parking spaces for the ambulances.
3. Adequate first aid equipment and supplies including:
 - a. First aid kits
 - b. Portable, lightweight, reliable stretchers
 - c. Coolers of water, towels, cold packs (or ice cubes in plastic bags)
 - d. Communication sets (e.g. Walkie Talkie, cell phones)

4. Notification of all local medical facilities regarding the upcoming competition.
5. In addition, the team physician should coordinate:
 - a. Compliance with all local, state, and federal regulations regarding storing and dispensing pharmaceuticals.
 - b. Development of a chain of command that establishes and defines the responsibilities of all parties involved.
 - c. Regular rehearsal of the emergency response plan.
 - d. Establishment of a network with other health care providers, including medical specialists, athletic trainers/physiotherapists, and allied health professionals covering all competition sites (polyclinics, field events).
 - e. Establishment of a policy that includes the team physician and athletic trainers/physiotherapist in the dissemination of any information regarding the athlete's health status.

C. Competition-Day Planning

1. Optimised Medical Care

Competition-day planning optimises medical care for injured or ill athletes.

The team physician should coordinate:

- a. Medical operations and administrative medical policies.
- b. Preparation of the stadium/ field medical bags and station medical supplies.

2. Administrative Protocol

It is essential for the team physiotherapist/athletic trainer to coordinate:

- a. Assessment of environmental concerns and playing conditions.
- b. Presence of medical personnel at competition site with sufficient time for pre-competition preparations.
- c. Plan with the medical staff of the visiting teams for medical care of the athletes.
- d. Introductions of the medical team to competition officials.
- e. Review of the emergency medical response plan.
- f. Checking and confirmation of communication equipment.
- g. Identification of examination and treatment sites.
- h. Arrangements for the medical staff to have convenient access to the competition site.
- i. Post-meet review and necessary modifications to medical and administrative protocols.

3. On-Site Medical Supplies

The team physiotherapist/athletic trainer should have competition-day on-site medical bags and stadium medical supplies (see Appendix 6, *On-Site Medical Supplies for Injury Prevention and First Aid*).

D. Emergency Action Plan

Planning for any medical emergency is a must (“a failure to plan is the same as a plan for failure”). Points to note in planning include:

1. Specific instructions for specific personnel.
2. The route of evacuation by ambulance.
3. Medical supplies required.
4. Steps to take for likely serious problems e.g. collapse/unconsciousness, fracture, bleeding, medical transport for head and spine.

Once developed, the plan must be communicated to all concerned.

Some other important points to note about emergency planning:

1. Must include personnel trained (and certified) in CPR, athletic trainers/physiotherapists who are familiar with the first aid kit available (training/practice required).
2. Must include names and contact numbers of important persons, e.g. physician in charge.
3. Must include a map that shows site of sports event/training ground, evacuation route for ambulance (to nearest hospital), locations of emergency telephone(s) (and include emergency telephone numbers), first aid kits. For coin phones, the emergency plan should include having coins available (if necessary).
4. Get valuable input to the plan from physicians, facility managers, local emergency medical service personnel.
5. Plans need to be reviewed and improved regularly, e.g. yearly.

E. Triage and First Aid Measures

To ensure adequate and timely care of casualties, proper triage is important. The steps would include primary survey (ABCDE [airway, breathing, circulation, disability/neurologic status, expose athlete]), resuscitation, and secondary survey, as well as the other necessary first aid (for bleeding, fractures, head and spine injury, see Part 2 of this chapter, *First Aid Management of Acute Sports Injuries*). Common causes for sudden collapse include heart attack, stroke, heat exhaustion/stroke, and fainting.

1. Airway assessment is the first priority in any casualty. The airway must be kept open and patent (remove foreign debris, chin lift or jaw thrust).
2. Breathing is then assessed with “look, listen and feel” method. If the casualty is not breathing, then mouth-to-mouth resuscitation must be instituted. Medical coverage staff for sports events should be CPR- and defibrillator-trained (see Appendix 7, *CPR/Adult Basic Life Support*).
3. Palpating peripheral pulse, e.g., the carotid pulse at the angle of the jaw next assesses circulation. If there is no pulse, then external cardiac compressions are instituted, along with defibrillation where appropriate.

4. Disability is then determined with a very rapid and brief neurologic assessment using the mnemonic “AVPU” (alert, responding to vocal stimuli, responding to painful stimuli, unresponsive).
5. Exposure of the casualty must be adequate to allow for a careful examination and assessment.

Following the primary survey, the necessary resuscitative measures must be implemented, e.g. continued CPR, use of defibrillator when available/appropriate, fluid replacement (if necessary with an intravenous line), cooling of the athlete (if heat stroke/exhaustion suspected), stopping any bleeding (direct pressure is best and adequate for almost all types of bleeding), splinting of suspected fractures. Secondary survey may not always be feasible at the site of the event, and may need to be done only at the hospital (or in the ambulance while on the way to hospital). This involves surveying the whole body from head to toe to check for injuries. Procedures would include evaluating pupillary size, checking the fundi, assessing possible cervical spine injury, chest injury (e.g. pneumothorax), abdomen and extremities.

For athletes who are stable and conscious, management should focus on the injuries sustained or other medical conditions. The following section describes common problems and their first aid management.

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FIRST AID MANAGEMENT OF ACUTE SPORTS INJURIES

Sport is a potentially dangerous activity; fortunately, life-threatening injuries are rare. A sports physician should be acquainted with the principles of acute care, as much depends on first aid. It is very important to recognise injuries with serious outcome, and transport the injured to the adequate institution after proper first aid has been carried out. The physician carrying out first aid should be acquainted with the devices and medications of primary care. In this section we will deal with primary care during the pre-hospital phase of injury management.

A. Spinal Injuries

Injuries to the spine may occur when a faulty landing happens after jumping, as a result of being struck by an implement, or during conditioning training. Trauma to the spine may result in injuries to the bone, cartilage, tendon, or spinal cord. In severe vertebral or cordal injury, stability of the spine may be compromised and there may be evidence of neurological findings. Whenever the mechanism of injury or the clinical findings indicate bone or tendon involvement, spinal cord injury should be taken into consideration, unless proven otherwise.

If the patient is conscious, a fracture or cord injury is accompanied by severe neck muscle spasm and pain, which indicates the nature of the injury. However, the unconscious athlete is open to further cord injury unless the medical staff is not alert to this possibility. It is essential that no neck manipulation be carried out on the field.

During the initial evaluation, proper preventive steps must be utilised in order to prevent severe nervous system complications. These can occur, for example:

1. When nerve lesions remain unrecognised after bone-injury.
2. When first aid is delayed and irreversible nerve damage occurs.
3. When the unstable spine is improperly treated and deterioration develops.

The following indicates spinal or spinal cord injury:

1. Tenderness to the spine
2. Palpable spinal deformity or haematoma
3. Nervous system injury is suspected if there is:
 - a. Abnormality in chest breathing.
 - b. Abnormality of movements.
 - c. Segmental anaesthesia or numbness.
 - d. Pain that is referred to the limbs.

According to the above-mentioned principles, first aid should be carried out as follows:

1. Ensure breathing and circulation.

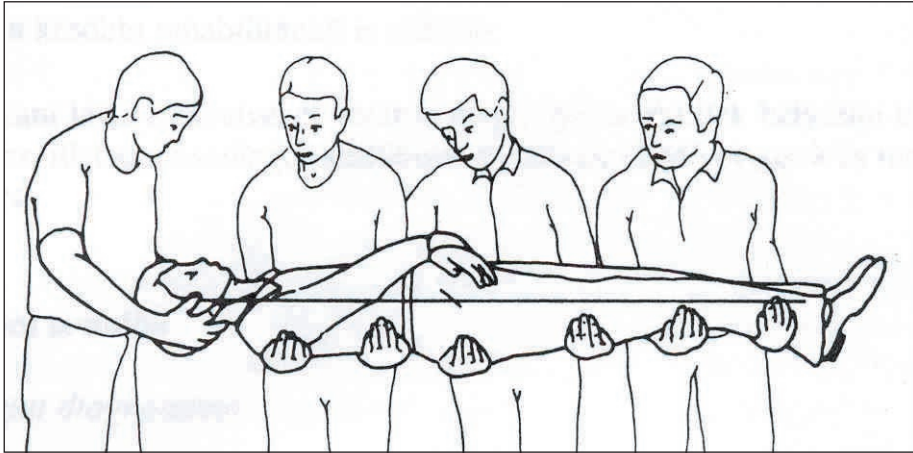


Figure 8-1. Proper technique for transferring a patient suspected of having sustained a neck injury, in the absence of a stretcher.

2. When spinal injury is suspected, a thorough neurological examination is to be performed as soon as possible, after breathing and circulation have been maintained.
3. If the mechanism of injury is unknown and the patient is unconscious, spinal injury must be assumed initially and treated accordingly. The same is necessary if the patient is conscious and spinal injury cannot be definitely ruled out.
4. If cervical injury cannot be ruled out, the neck should be stabilised routinely in a neutral position with a rigid brace or collar, sandbags, or with a Schantz-collar (but not a soft collar).
5. Having stabilised the neck, a vacuum mat is to be used as in spinal injuries. The injured should be laid on a scoop or shovel-stretcher, or, if these are not available, transfer should be carried out as shown in Figure 8-1.
6. The seriously injured should be transferred to a hospital where both modern diagnostic and therapeutic facilities are available. Transfer should be carried out by helicopter if possible.
7. In case of a definite cord injury, high dose methyl-prednisolone should be administered during the pre-hospital phase (30mg/kg within 15 minutes, 5 mg/kg 45 minutes later, 4 mg/kg for 23 hours). Smaller doses of steroids are inadequate, as only with high doses is oedema, which is responsible for later neurological symptoms, prevented.
8. Volume substitution may be necessary, because cross sectional lesions of the spine may lead to vasodilation and shock. Therefore infusion and alpha-adrenergic medication (epinephrine or dopamine) may be necessary.
9. The injured should be protected from being over-heated or cooled.
10. Narcotic analgesics are contraindicated in case of spinal injury.

Table 8-1. Glasgow Coma Scale used to assess degree of head injury.

Reaction	Points
Eye opening	
<input type="checkbox"/> Spontaneous	4
<input type="checkbox"/> To speech	3
<input type="checkbox"/> To pain	2
<input type="checkbox"/> No response	1
Motor response	
<input type="checkbox"/> Follows commands	6
<input type="checkbox"/> Localises pain	5
<input type="checkbox"/> Movement or withdrawal to pain	4
<input type="checkbox"/> Decorticate flexion	3
<input type="checkbox"/> Decerebrate extension	2
<input type="checkbox"/> No response	1
Verbal response	
<input type="checkbox"/> Alert and oriented	5
<input type="checkbox"/> Disoriented conversation	4
<input type="checkbox"/> Speaking but nonsensical	3
<input type="checkbox"/> Moans or unintelligible sounds	2
<input type="checkbox"/> No response	1

B. Head Injuries

Head injuries usually occur as a direct trauma to the head. Often concomitant injury to the spine or spinal cord must be considered. Injuries to the soft tissues of the head are treated as in other soft tissue injuries (disinfection of the wound and surroundings, sterile gauze, and tetanus anti-toxin). If the athlete is unconscious, we must proceed under the assumption that there is a fracture of the cervical spine.

In cases of head injury, attention must be focused on potential intracranial lesions (cerebral concussion, cerebral contusion, subdural, or epidural haematomas). Treatment should be carried out as follows:

1. A thorough neurological assessment should be made. During the examination, signs and symptoms of possible neurological deficiencies are sought. Symptoms of increased intracerebral pressure should be looked for. The seriousness of head injury can be estimated by the Glasgow Coma Scale as seen in Table 8-1, which is also useful as an indicator for intratracheal intubation or for later assessment.¹
2. Complete CPR if necessary (see Appendix 7, *CPR/Adult Basic Life Support*).
3. In case of increased intracranial pressure, the following should be initiated:
 - a. The patient's head should be placed at 30°.
 - b. Intravenous diuretics should be administered (40 mg of furosemide).
 - c. Mannitol 0.25–1 g/kg IV.

The signs and symptoms of intracranial pressure should be sought for carefully: (headache, vomiting, tachycardia, hyperventilation, hypertension, meningeal signs (Brudzinki, Kernig-sign), stiffness of the neck, drowsiness, stupor, wide-, non-reacting pupils, anisocoria, coma, Kussmaul-breathing, convulsions, increase in muscular tone, bradycardia, hypotension, flaccid muscles, hyperpyrexia, slow breathing, brady-arrhythmias, no circulation, no breathing).

4. If inadequate circulation or breathing are noticed, oxygen should be administered.
5. An IV line should be ensured and intravenous crystalloids should be given, so that mean pressure of 90mm Hg is maintained. In the later stage, reduced cerebral perfusion pressure is necessary in order to prevent cerebral oedema.
6. The patient should be transferred to a hospital where neurosurgery is available.

Please note: If the examiner notices symptoms of brain concussion (transient unconsciousness, dizziness, nausea, vomiting, paleness), the patient should be admitted to the hospital, because an intracranial lesion should be suspected. It is possible that, in case of head injury, the patient will regain consciousness and be symptomless. However, small veins around the dura may rupture causing a haematoma, which will present with late neurological symptoms. This is called the lucid state (status lucidum).

C. Fractures

Fractures may result from several causes, including direct trauma, such as a blow; from twisting; or as the end result of an unrecognised incomplete stress fracture.

The diagnosis can often be made from the history, plus physical findings. The physical examination will reveal the classical signs and symptoms of fractures (local oedema, pain, deformity, restriction of movement).

First aid in case of fractures:

1. Do not move the patient until the injury is totally immobilised. Do not attempt to straighten a misshapen bone or joint to change its position.
2. Fractures should be stabilised (most commonly pneumatic devices are used). If the ends of these fractures are not stabilised, secondary injuries to the nerves and vessels may occur. A compartment syndrome may also develop.
3. If a broken bone pierces the skin, take steps to prevent infection.
4. Take steps to prevent shock. Lay the patient flat, elevate the feet 20–25 cm and cover with a coat or blanket. Keep flat if a head, neck, or back injury is suspected.
5. Analgesics may be administered if there is severe pain (see Soft Tissue Injuries, below).
6. The patient may need to be admitted to the hospital for final care.

D. Joint Dislocation and Subluxations

Always keep in mind that dislocations or subluxations of the joint may injure the surrounding area (vessels, nerves, tendons), as well as the capsule of the joint.

In case of joint injury the following is recommended:

1. The joint should be examined thoroughly. An unstable joint and haematoma indicate capsule injury. Every joint has its own method of examination, which will not be discussed in this text.
2. The joint should be put at rest and stabilised with a splint or bandage.
3. In case of pain, local or general anaesthetics are necessary.
4. Ice packs should be administered to prevent eventual later consequences; they also lessen pain.
5. The patient should be referred to a clinic or hospital, where diagnostic procedures including X-ray, ultrasound, or MRI are available to diagnose fractures or intra-articular lesions and cartilage and soft tissue involvement.

E. Ligament Strains

Strains of the ligaments usually take place on the proximal and distal endings, rarely along the whole ligament.

In case of strains, the following is recommended:

1. A thorough physical examination, which will reveal the degree of the injury of the ligaments as follows:
 - a. *First degree*: swelling and tenderness is possible, the joint is stable.
 - b. *Second degree*: under loading the ligament is loose, but there is a stable end-point.
 - c. *Third degree*: the whole ligament is disrupted, movement is lax (no endpoint).
2. In case of first degree injury, treatment is as for soft tissue injuries (see below).
3. Second and third degree injuries should be treated in hospital after exact diagnostics. Third degree injury is usually treated surgically.

F. Muscle Strains

Muscle strain is the most frequent injury in sports. It usually occurs due to bad technique: while strengthening the agonist muscular group, the antagonist group is not relaxed accordingly, due to ionic imbalance or fatigue. Strains usually occur where the muscle attaches to the bone, or in the muscle itself at the musculo-tendinous junction. In children, the muscle may avulse the apophysis; for example, the hamstring attachment at the ischial tuberosity

In case of strains, the following is recommended:

1. For muscle strains, determine the degree of the injury according to its anatomic and functional status.

- a. *First degree*: the muscle fibres are partially injured. There is local swelling and tenderness if the patient contracts the muscle against resistance.
 - b. *Second degree*: more fibres are injured. Minor contraction will cause pain or the muscle cannot contract; the injury is palpable.
 - c. *Third degree*: the tendon of the muscle is detached from its adhesion point or a large amount of the muscle is damaged. The muscle is functionless, the lesion can be palpated, haematoma results within a short time.
2. Principles of first aid for muscle strains are the same as for soft tissue injuries (see below).

G. Soft Tissue Injuries (see also Chapter 9, *Soft Tissue Damage and Healing*)

There are many mechanisms that may cause soft tissue injury in athletics, from overload of soft tissues (e.g. overextension of the muscle) to direct trauma. Soft tissue injury is accompanied by surrounding oedema, haematoma, and tissue necrosis. The accumulated blood sets off an inflammatory cascade that results in further swelling. This leads to increased pressure on the surrounding tissues causing hypoxia, which will increase the degree of the injury. In case of inadequate first aid, the injured area and its surrounding develop scarring and muscle atrophy due to haematoma, tissue necrosis and oedema.

Soft tissue treatment is performed according to the mnemonic **PRICES**:

1. P = protection: protect the injured area from further damage.
2. R = rest: the limb should be put at rest (this should be done before examination), the degree of injury should be quickly assessed because the least load may result in deterioration.
3. I = ice: icing of the injury has many benefits:
 - a. Lessens the pain, so the surrounding muscle tone will decrease.
 - b. Increases vasoconstriction, which reduces bleeding.
 - c. Hypothermia reduces the oxygen and nutrition demands of the injured tissues.
 - d. Local inflammatory reaction decreases.

Please note: In case of minor injuries the athlete should be instructed to cool the injury. In case of extensive injury, an ice bath should be used.

After training, it is recommended that ice be applied to the muscles, because loading leads to microtraumas. If chemical ice packs are used, they should be applied through a layer of clothes or wrap in order to prevent freezing.

Usually cold packs are used for 10–20 minutes every 2 hours, during the first 2 days.

4. C = compression: compression raises the tissue pressure, which reduces bleeding and swelling. Compression should be used during and after ice therapy. The bandage should be applied firmly, from distally to proximally,

with an overlap of one-half the bandage's width. An ice pack can be placed over a layer of compression wrap.

5. E = elevation: the injured area should be elevated above the level of the heart. This will decrease swelling by enhancing drainage via lymphatic channels, and reducing venous stasis.
6. S = support: use braces, splints, etc. to support the injured area.

H. Medical Treatment

Effective medication should be administered throughout the entire treatment process, taking into account banned drugs. If in spite of local measures the patient still has pain, the first drug of choice is paracetamol (500 mg–1g). If paracetamol is not enough, more effective drugs should be administered. NSAIDs are useful and popular: diclofenac 50 mg given parenterally (maximum daily dose 150 mg), ibuprofen 800 mg (maximum daily dose 2400 mg), indomethacin 50 mg (maximum daily dose 100 mg); in addition to their analgesic effect they are also good anti-inflammatory drugs. NSAIDs can cause stomach irritation for some individuals. In this case, an H₂-blocker may be used to reduce the amount of gastric acid secretion. If the injury is accompanied by extreme bleeding, the administration of an NSAID should be considered carefully, as they reduce platelet aggregation and increase bleeding. Corticosteroids have no place in first aid treatment.

Please note: Pain is a good indicator of the status of the injury. Analgesics and anti-inflammatory drugs should never mask pain in an effort to allow an athlete to continue to compete or train, which may lead to severe consequences.

I. Follow-Up Management

Depending upon the site of injury and its extent, additional treatment may be utilised as a part of the treatment/rehabilitation process (see Chapter 9, *Soft Tissue Damage and Healing*, for additional details).

1. Protected Mobilisation

Protective taping and bracing permits the injured area to be mobilised actively and passively while damaged tissue is protected. This prevents excessive stress on muscles, joints, and ligaments during the healing process.

2. Electro-Therapeutic Modalities

Electrical therapies are an additional means of providing heat energy to deep tissues, mobilising lymphatic and capillary circulation, and promoting healing. These modalities include: interferential current, ultrasound, and magnetic field therapy. Ultrasound should be used with caution around children's physes.

3. Manual Therapy

Manual techniques are useful in the healing process and in reducing the sequelae of injury. Stretching is valuable in reducing tissue contraction and muscle

spasm, and preserving muscle and ligament length. Friction massage is helpful in decreasing scar tissue contraction that follows the inflammatory reaction. Mobility exercises, both passive and active, are essential for maintaining joint range of motion and muscle length.

4. Fitness Maintenance

During the acute phases of the healing process, as well as during rehabilitation, cardiovascular fitness must be maintained. This can be accomplished in a variety of ways, depending upon the site of the injury. An exercise bicycle can be used if the lower extremity can bear weight. Otherwise, swimming, or running in a swimming pool with a flotation jacket, can be used. At first, the athlete can “run” in deep water, and then progress to shallower water with partial weight bearing. Strength and range of motion of all the uninjured parts must be maintained with appropriate stretching and resistive exercises.

J. Evaluation of Treatment

The clinician should constantly assess each treatment’s effectiveness by comparing symptoms and signs prior to and after treatment. Continual evaluation leads to the most appropriate treatment course for the specific injury and allows the programme to be adapted to the individual’s needs.

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Footnote

¹The Glasgow Coma Scale is the most widely used scoring system for quantifying the level of consciousness following traumatic brain injury. It is used primarily because it is simple, has a relatively high degree of interobserver reliability, and correlates well with outcome following brain injury.

The Scale is easy to use, particularly with a table such as shown in Table 8-1 (page 3 of this section). One determines the best eye opening response, the best verbal response, and the best motor response. The score represents the sum of the numeric scores of each category. A Coma Score of 13 or higher correlates with a mild brain injury, 9–12 is a moderate injury, and 8 or less a severe brain injury.

However, there are limitations to a simple application of the table. If there is an endotracheal tube in place, the patient cannot talk. Hence, it is important to document the score by its individual components. For example, a Glasgow Coma Score of 15 would be detailed as follows: E-4, V-5, M-6. An intubated patient would be documented as E-4, V intubated, M-6. Of these factors, the best motor response is probably the most significant.

Other factors that alter the patient's level of consciousness will interfere with the Scale's ability to reflect the severity of the brain injury. Hence, shock, hypoxemia, drug or alcohol use, or metabolic disturbances may alter the Scale independently of brain injury. A spinal cord injury will invalidate the motor score, and an orbital injury may impair the ability to open the eye.

For more information on the Glasgow Coma Scale, see www.trauma.org/scores/gcs.html.

SOFT TISSUE DAMAGE AND HEALING: THEORY AND TECHNIQUES

A. Mechanisms of Injury

Many factors produce mechanical injuries or trauma in sports. Soft tissue damage occurs through direct or indirect trauma to muscles, ligaments, and joint capsules. Usually, direct trauma refers to an injury occurring from blunt trauma or a sudden overload, and is known as macrotrauma, i.e., true muscle tear or ligament sprain. In contrast, indirect trauma results from repeated submaximal loading, leading to clinical signs and symptoms. Injury presents itself in three stages: acute, subacute/overuse, and acute/chronic.

The first, or acute, stage of direct trauma stems from sudden overloading, or macrotrauma (e.g. a 100 meter runner exploding out of the starting blocks). The subacute/overuse stage occurs when increased loads degenerate body tissues due to excessive cumulative loading, leading to microtrauma and an accompanying inflammatory response (e.g. achilles tendinitis in the endurance athlete or runner, Figure 9-1). The last type, acute/chronic stage, integrates both cumulative loading and sudden overloading (e.g. chronic achilles tendinosis that ruptures in a long jumper). Chronic tendinosis is a degenerative condition without inflammation.

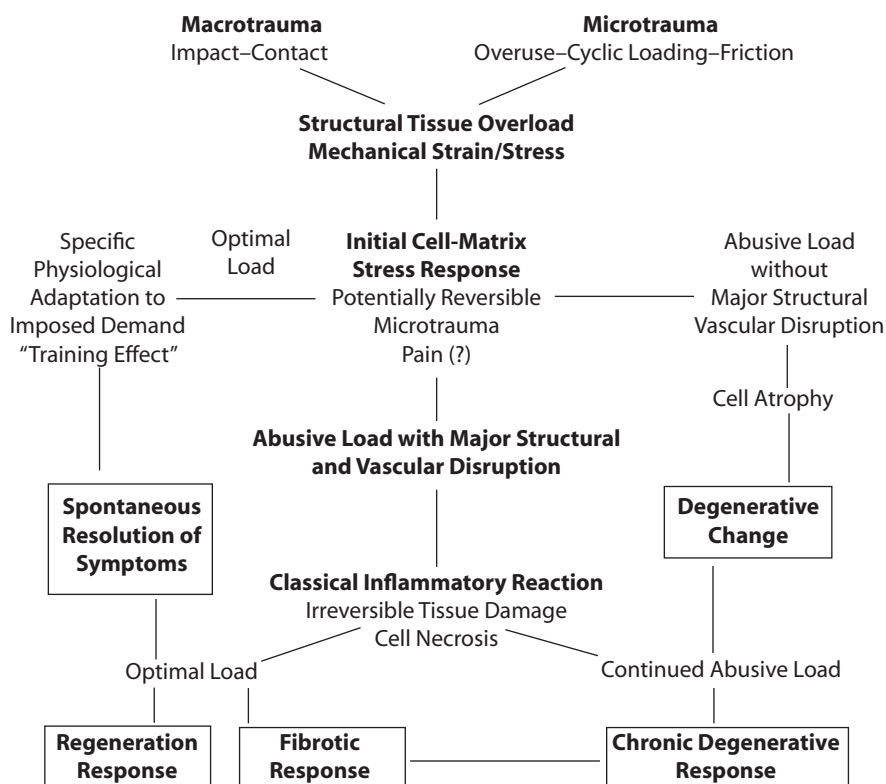


Figure 9-1. Schema demonstrating theoretical pathways of sports-induced tissue damage.

Whether muscle injury is caused by direct or indirect trauma, the end result is tissue dysfunction characterised by pain, inflammation, and altered internal tissue stress. The injury often results in functional disability, whereby an athlete may be able to carry on daily living routines, but is limited in his or her capacity to train and compete.

Any activity loads and deforms tissue, an effect known as a stress/strain, and described through a load and tissue elongation curve. As connective tissue is deformed it either stretches or tears, depending on the magnitude, rate, and intensity at which the loading occurs. Collagen deforms under low loading and fails at high loads. When the load is removed from normal tissue during the elastic phase, the material returns to its pre-stretch length. Injury occurs when the tissue is stretched into the plastic phase, causing tissue failure.

Of all the tissues involved, tendon is the least elastic. The most frequent site of injury in muscle strains is the myotendinous junction, because of increased collagen content at the transition zone of muscle sheath to tendon. This area has decreased local extensibility, as does scar tissue, and is frequently termed a stress riser. This transition in biologic tissues, which also appears at the tendoperiosteal junction, is a point that is more susceptible to stress and injury.

The relatively new term “tendinopathy” has been adopted as a general clinical descriptor of tendon injuries in sports. In overuse clinical conditions in and around tendons, frank inflammation is infrequent and if seen, is associated mostly with tendon ruptures. Tendinosis implies tendon degeneration without clinical or histological signs of intratendinous inflammation, and is not necessarily symptomatic. The term “tendonitis” is used in a clinical context and does not refer to specific histopathological entity. Tendonitis is commonly used for conditions that are truly tendinosis, however, and leads athletes and coaches to underestimate the proven chronicity of the condition.

Paratendonitis is characterised by acute oedema and hyperemia of the paratendon with infiltration and inflammatory cells, and possibly the production of a fibrinous exudate with the tendon sheath causing a typical crepitus, which can be felt on clinical examination.

The term “partial tear of the tendon” should be used to describe the macroscopically evident partial tear of a tendon. This is an uncommon acute lesion. Most articles describing the surgical management of partial tears of a given tendon in reality deal with degenerative tendinopathies. The combination of pain, swelling, and impaired performance should be labeled tendinopathy. According to the tissues affected, the terms tendinopathy, paratendinopathy, or pantendinopathy (from both the tendon and the surrounding tissues involved) should be used.

B. Examining Soft Tissue Injuries

Examination for injury in soft tissues such as muscle involves initial palpation with minimal force or compression (in the case of acute injuries), and progresses to firmer compression or higher loads if increased density has not been distinguished

or pain has not been provoked at the site of the suspected lesion (see Table 9-1 for examination steps). One can also have the athlete contract the muscle to increase the tension or passively stretch the myotendinal unit while palpating the area. The pain associated with palpation is secondary to the stimulation of free nerve endings with inflammation, decreased extensibility of tissue, or tissue insufficiency.

While palpating muscle tissue, one should search carefully through various layers of tissue to find remnants of injuries and healing. Subtle tissue texture abnormalities may exist, and might be missed if the tissue were examined erratically. These abnormalities must be considered in formulating an assessment. However, the clinician must avoid going too deep or hard with palpation, using pain as a guideline.

The clinician needs to apply pressure and to sense the reactivity of the tissue. Since scar tissue heals three dimensionally, it does not fall into place like a brick. Instead, scar tissue reaches in the direction of the fascia and the neighbouring muscle sheaths, binding these tissues together. For example, when a runner strains a hamstring, the sheath tear heals and binds to neighbouring muscle sheath. The hamstring muscle group still functions to flex the knee, yet the athlete complains of dull ache or pain in the posterior thigh. The reason may be that independent movement has been lost and the area of scar tissue has limited the extensibility of the myotendinal unit. Muscles do function and limbs do move, but the normal gliding that occurs between neighbouring tissues is lost. As a result, there is a constant low-grade inflammatory process at the site of the decreased mobility. Scar tissue has a poor blood supply and is not as strong or resilient as the primary tissue it replaces. This area will likely be a site of re-injury secondary to the transition zone of normal tissue to scar tissue.

Table 9-1. Examination of soft tissue injury.

<ol style="list-style-type: none"> 1. History <ul style="list-style-type: none"> • onset • pain location • mechanism of injury • prior treatment and rehabilitation • goals of athlete 2. Physical exam <ul style="list-style-type: none"> • inspection • AROM/PROM • palpation • neurological; myotome, dermatome, peripheral nerve tests, deep tendon reflexes • strength and motor control • special tests • functional exam • gait analysis 3. Assessment 4. Treatment goals 5. Treatment plan 6. Treatment procedures

C. The Wound Healing Process

1. Reaction: The Inflammatory Phase

This first phase can last up to 72 hours, and involves a number of inflammatory responses, manifested by pain, swelling, redness, and increased local temperature. Accumulation of exudate and oedema begins the process of tissue repair following injury when a blood clot forms and seals the area. In musculotendinous injuries, there is myofilament reaction and peripheral muscle fiber contraction within the first two hours. Oedema and anoxia result in cell damage and death within the first 24 hours, and the release of protein breakdown products from damaged cells leads to further oedema, tissue hypoxia, and cell death. Oedema and joint swelling, with or without pain, is associated with a reflex inhibition of spinal activation of skeletal muscle. Phagocytosis then begins to rid the area of cell debris and oedema.

2. Regeneration and Repair: The Fibro-elastic/Collagen-forming Phase

This phase lasts from 48 hours up to 6 weeks. During this time structures are rebuilt and regeneration occurs. Fibroblasts begin to synthesise scar tissue. These cells produce Type III collagen, which appears in about four days, and is random and immature in its fiber organisation. Capillary budding occurs, bringing nutrition to the area, and collagen cross-linking begins. As the process proceeds, the number of fibroblasts decreases as more collagen is laid down. This phase ends with the beginning of wound contracture and shortening of the margins of the injured area.

3. Remodelling Phase

This phase lasts from 3 weeks to 12 months. Gradually, cross-linking and shortening of the collagen fibers promote formation of a tight, strong scar. It is characterised by remodelling of collagen so as to increase the functional capabilities of the muscle, tendon, or other tissues. Final aggregation, orientation, and arrangement of collagen fibers occur during this phase.

Regeneration of the injured muscle does not fully restore muscle tissue to its prior levels, as fibrous scar tissue slows muscle healing. The two processes of healing and fibrosis compete with each other and impair complete regeneration. Transforming Growth Factor–Beta 1 (TGF- β 1) is an ubiquitous substance that initiates a cascade of events that activate both myogenesis and fibrosis.

Measures that may block fibrosis have been shown experimentally to alter the effects of TGF- β 1 on the fibrotic process. Decorin is a proteo-glycan that impedes fibrosis by combining with TGF- β 1. Suramin is an anti-parasitic drug that competes with TGF- β 1 for its binding sites to the growth factor receptor. Interferon gamma disrupts the pathways involved in TGF- β 1 signal transduction, and when given i-m 1–2 weeks after an injury improved muscle function in animal models. All of these agents are under active study, and have undergone clinical trials.

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PRINCIPLES OF REHABILITATION OF THE INJURED ATHLETE

A. Rehabilitation: A Definition

A rehabilitation programme should be designed with individual short-term and long-term goals in mind. The overall programme and individual exercises should progress safely and effectively. Rehabilitation clinicians should know how to assess the patient's status, incorporate therapeutic modalities and exercises, and evaluate the program's outcomes. In general, the process of rehabilitation can be defined as the restoration of normal anatomical and physiological function (see Table 9-2 for a summary of steps involved in rehabilitation).

The rehabilitation process of an injured athlete may be divided into stages. This is convenient, but it should be noted that the boundaries between these stages are not clearly defined. Biological systems are more than just assemblages of discrete units: variability, overlap, and interaction are the rule and not the exception. From a pathophysiologic standpoint, tissue injuries can be conveniently divided into three stages or phases. For each phase, occurrences at the cellular level and their consequences at the system level determine the scientific rationale on which the rehabilitation plan is based. It is very important to understand the basic pathophysiology of each stage since our therapeutic interventions depend on it (see Part 1 of this chapter, *Soft Tissue Damage and Healing: Theory and Techniques*, for additional details).

Understanding the pathomechanics of injury through background knowledge in anatomy and biomechanics is critical in defining a rehabilitation programme. Using therapeutic modalities will enhance the athlete's chance of a safe and rapid return to competition. Use of medications to facilitate healing may aid the healing process. Understanding the concept of the kinetic chain as an integrated functional unit involving muscles, tendons, bones, ligaments, fascia, articular, and neural system will be essential for the evaluation and rehabilitation of a particular biomechanical movement or task.

Injury/illness produces a variety of emotional responses, demanding the clinician's understanding of the psychological aspect of rehabilitation. Therapeutic exercise is essential in athletic conditioning, injury prevention, and rehabilitation. Implementing rehabilitation tools will avoid the "cookbook" approach to rehabilitation.

B. Stages of a Sports-Related Injury: The Bases for Rehabilitation

1. Stage 1: Acute Inflammatory Phase

The first phase of most sports-related injuries is characterised by an inflammatory reaction that involves pain, redness, swelling, and an increased local temperature that can last up to 72 hours. Initial treatment of this phase usually includes some form of immobilisation or restriction of motion. It should be remembered, however, that immobilisation can cause early and significant negative effects on various organs and physiological systems. For example, metabolic processes leading to muscle catabolism, atrophy, and weakness of the quadriceps muscle start as early as 6 hours

Table 9-2. Summary of rehabilitation goals and treatment plan.

Goals		
<i>Physiological</i>		
Importance of Controlling Swelling		
• Initial injury management and swelling control is critical		
Reestablishing Neuromuscular Control		
Restoring Range of Motion		
Restoring Muscular Strength, Endurance and Power		
• Isometric Exercise		
• Isotonic Exercise		
• Isokinetic Exercise		
• Progressive Resistive Exercise		
• Open versus Closed Kinetic Chain Exercises		
Maintaining Cardiorespiratory Fitness		
<i>Functional</i>		
Restoring Postural Control and Stability		
• Plyometric Exercise		
• Core Stabilisation		
Functional Progression		
• Restore endurance and activity tolerance		
• Regain ability to return to athletics with or without modifications and/or appliances		
Treatment Plan		
<i>Therapeutic Exercises</i>		
• Strength training; isometrics, dumbbell, cuff weights, 'Theraband', surgical tubing, concentric/eccentric contractions, isotonic and isokinetic equipment, Swiss ball and plyometrics, and PNF (proprioceptive neuromuscular facilitation) exercises		
• Stretching; active, passive, and PNF techniques		
• Joint mobilisation		
• EMG Biofeedback		
<i>Procedures</i>	<i>Modalities</i>	
• Functional activities	• Hot pack	• Ultrasound
• Neuromuscular re-education	• Cold pack	• Electrical stimulation
• Gait training	• Laser/Light Therapy	• Iontophoresis
	• Aquatherapy	• Phonophoresis
<i>Traction</i>	<i>Massage</i>	
• Manual or mechanical traction	• Soft tissue mobilisation	

after immobilisation of the knee joint. Any rehabilitation modality—such as heat or deep massage—that may enhance or potentiate the inflammatory reaction, and thus slow the healing process, should be avoided. In addition, we should consider the psychological effects of injury. The post-injury period is very difficult for the competitive athlete whose goal is to return to the sport as soon as possible. It is important to provide the athlete with the details of the injury, and explain possible consequences, the importance of the rehabilitation process, and the prognosis and time frame regarding the return to competition.

2. Management and Rehabilitation of Stage 1

Rehabilitation goals during the first phase are as follows: 1) protect the athlete from further injury; 2) control pain; 3) limit swelling; and 4) promote normal healing. Therapeutic and rehabilitative strategies appropriate for this stage include pharmacologic intervention, use of physical modalities, immobilisation, and therapeutic exercise.

a. Pharmacological Intervention

The most frequently used drugs include non-steroidal anti-inflammatory medications, analgesics, local anesthetics, and in some cases injectable corticosteroids. The rationale, indications, and contraindications for use of these drugs are discussed elsewhere in this manual.

b. Physical Modalities

The most important physical therapy modality used in this stage is cryotherapy (cold therapy), usually accompanied by protection, rest, ice, compression, elevation, and support; this combination is commonly called the P.R.I.C.E.S. therapy (see also Chapter 8, Part 2, *First Aid Management of Acute Sports Injuries*, and Part 3 of this chapter, *Therapeutic Modalities*). Cold helps reduce tissue temperature, decrease blood flow and swelling, produce vasoconstriction, and alleviate pain and muscle spasm. In general, crushed ice should be used. Compression using an elastic bandage and elevation of the extremity above the level of the heart may help control swelling. Another modality used to alleviate pain at this stage is transcutaneous electrical nerve stimulation (TENS), which is sometimes applied in combination with ice (see Part 3 of this chapter, *Therapeutic Modalities*).

c. Immobilisation

As mentioned above, this stage may require the immobilisation of a joint or an extremity. Immobilisation accelerates formation of granulation tissue, limits scar size, and improves penetration of fibers through connective tissue, but as mentioned previously it also has negative effects. Early mobilisation increases tensile tissue strength, improves orientation of regenerating muscle fibers, stimulates resorption of connective tissue scar, improves recapillarisation, and decreases muscle atrophy and weakness.

d. Therapeutic Exercise

Therapeutic exercise may be beneficial during this early stage to minimise deconditioning and to promote rapid transition to the second stage. If symptoms permit, exercises to increase range of motion and static (isometric) exercises to minimise strength loss may be started in the injured part and related muscles. Conditioning of the uninjured body parts should be instituted. Transition to this second stage varies with the type and severity of injury. In general, it is desirable to start the second phase as soon as possible to promote faster recovery and return to training and competition.

3. Stage 2: Regeneration and Repair—The Fibro-Elastic/Collagen-Forming Phase

The second stage of an athletic injury is called the repair or fibroblastic phase. This phase lasts from 48 hours up to 6 weeks. During this time structures are rebuilt and regeneration occurs. Fibroblasts begin to synthesise scar tissue. The nature of the functional losses will determine the selection of therapeutic modalities and exercises needed for this phase. This is a risky period because the absence of pain may tempt the athlete (or the coach) to return to training and competition before the injured tissues are fully rehabilitated.

4. Management and Rehabilitation of Stage 2

The rehabilitation goals in the second phase are to: 1) allow normal healing (similar to the first phase); 2) maintain function of uninjured parts; 3) minimise deconditioning of the athlete; 4) increase joint range of motion or flexibility; 5) improve muscle strength, local muscular endurance, and power; 6) increase aerobic capacity and power; and 7) improve proprioception, balance, and coordination. These goals can be achieved by physical therapy and therapeutic exercise.

a. Physical Modalities

Physical modalities can be of great benefit during this stage (see Part 3 of this chapter, *Therapeutic Modalities*). Heat has been shown to increase temperature, blood flow, and extensibility of soft tissue. Therefore, heating modalities are useful at the start of the rehabilitation session and before stretching exercises are done. Hydrocollator packs, laser lamps, hydrotherapy with warm water, fluidotherapy and paraffin baths are used to increase the temperature of superficial tissues.

Ultrasound and short wave diathermy are examples of deep heat modalities. Ultrasound has been shown to enhance the tensile strength of healing tendons.

Electrical stimulation is another useful modality during this stage. Since the patient may still have pain and joint swelling, activation of motor units may be less than optimal. Electrical stimulation may enhance motor unit recruitment during exercise and facilitate muscle training.

b. Therapeutic Exercise

The most important component of rehabilitation during this stage (and perhaps all stages) is exercise training. The type of exercise used depends on the objective. For example, recovery of normal range of motion requires

stretching or flexibility exercises. Restoring flexibility should be a priority because later strength and aerobic conditioning may depend on first achieving full joint range of motion. As mentioned previously, stretching is more effective when tissues are warmed beforehand, which may require assistance from a therapist. A thorough stretching routine including all major parts of the body should be completed daily.

Muscle strength can be developed using different types of muscle actions and equipment. Muscle actions can be classified as static (isometric) or dynamic (isotonic and isokinetic). Both have been shown to induce adaptations in skeletal muscle function and can be useful in different clinical situations. Dynamic muscle actions can be further divided into concentric and eccentric groups, both useful for conditioning. Recent evidence suggests that eccentric actions may be more effective, but must be used with caution due to the common effect of muscle soreness.

Methods or equipment used for strength conditioning include maximal voluntary contractions at different joint angles with no joint movement (static training), electrical stimulation during voluntary contractions (see Part 3 of this chapter, *Therapeutic Modalities*), gravity, resistance of the therapist, free weights, “isotonic” equipment such as pulleys and benches, surgical tubing, isokinetic equipment, and variable resistance equipment.

Rehabilitation strength conditioning requires a prescribed training plan outlining the type, intensity, duration, and frequency of exercise. Appropriate action and equipment depends on the clinical condition of the athlete (for example, if there is joint swelling, use static exercises with electrical stimulation). To induce significant gains in strength, exercise intensity should be above 60–80% of the one repetition maximum of the athlete. Usually, three sets of 8–10 repetitions are performed with each repetition including concentric and eccentric muscle actions. Free weight and machine weight lifting contain both concentric and eccentric muscle contractions. Each muscle group is usually trained three times per week.

Early strength gains are due to neurological factors, while muscle hypertrophy occurs only after several weeks of training. Restoration of optimal strength may require 3–6 months while maintenance training at a lower frequency (twice per week) should be a permanent component of the programme (see Stage 3: Remodelling Phase, below).

Local muscular endurance can be developed using exercises and equipment similar to those used to develop strength. The classical approach to developing tolerance to fatigue is to use lighter loads (less than 60% of the athlete’s one repetition maximum) and higher repetitions (20 or more). Actually, strength conditioning contributes to muscular endurance. A stronger athlete can tolerate a higher absolute load for a longer period of time since that load represents a smaller percentage of his/her maximum strength. The relevance of local muscular endurance training depends on the particular demands of the event. It is more important to the sprinter and middle-distance runner than the long-distance runner or discus thrower.

Aerobic conditioning should be part of the rehabilitation programme for all athletes at this stage. Cycling (stationary), swimming, and rowing improve aerobic capacity and promote recovery of full joint range of motion. The exercise

programme should consider the type of exercise, intensity (60–85% of maximal heart rate), duration (20–60 minutes as tolerated), and frequency (3–5 times per week).

Rehabilitation during this stage should include exercises to develop proprioception, coordination, and balance. In particular, injuries to the ankle and knee joints can affect proprioception. Although the majority of this training occurs during the third phase, it can be started here.

c. Tissue Mobilisation in the Healing Process

The goal of mobilising soft tissue in the healing process is to reintroduce a controlled stress as the scar matures in an attempt to influence its final form and function. Non-mobilised scar tissue heals in an irregular formation, whereas tissues mobilised with modified stress heal with parallel fiber arrangement. This parallel fiber arrangement demonstrates more elastic qualities, whose redundant folds allow mobility without irritation or pain. Examples of good healing are gliding tendons and lengthy, elongated adhesions. Conversely, examples of poor healing are restricted tendons and short, dense adhesions.

Soft tissue mobilisation will not remove scar tissue, yet it will help restore more normal properties to that tissue. Soft tissue mobilisation is performed like massage, but is much more specific. The clinician uses his or her fingertips to identify the lesion, to monitor tissue changes, and to perform the treatment. When applying soft tissue mobilisation as a treatment choice, the clinician needs to identify the area of increased density, then distinguish the borders and document the feel and density of the tissue. The ability of the tissue to tolerate loading will give the clinician an idea of the level of reactivity and state of healing.

Two of the approaches to load application are:

- “Beach Erosion”: Application of a low load to gradually change the tissue density and promote remodelling.
- “Tidal Storm”: A high, forceful load to break adhesions; this is more applicable to old, dense scar.

When performing either of these techniques, the clinician should use an oil-based cream or lubricant to decrease dermal irritation. The clinician should make sure to clean the skin with alcohol after treatment to prevent possibility of dermal irritation.

Exercise in the form of controlled early mobilisation has been effective in minimising adhesions during healing. Exercise may cause injury; controlled exercise, however, will contribute to resolution and help prevent further injury.

5. Stage 3: Remodelling Phase

The third stage in the rehabilitation of a sports-related injury is called the remodelling phase. This phase lasts from 3 weeks to 12 months. It is characterised by remodelling of collagen so as to increase the functional capabilities of the muscle, tendon, or other tissues.

Critical issues to be considered during this phase are residual strength deficits in individual muscles, imbalance between agonist and antagonist groups of muscles, side to side asymmetry, loss of sports specific skills, and the need for a gradual return to training and competition as determined by the severity of the injury and the duration of the two preceding phases.

6. Management and Rehabilitation of Stage 3

The third stage of the rehabilitation process is characterised by the return of the athlete to training and competition. The rehabilitation goals of this stage are continued conditioning, development of sports-specific skills, and prevention of further injury. During this stage, the athlete returns to the training programme—physical conditioning, technical and tactical training, and psychological preparation—designed by his or her coach. Communication between health professionals and coaches is critical at this stage. Sports-specific exercises, drills, and technical skills must be re-introduced gradually depending upon the severity of the injury and the duration of the first two phases of rehabilitation. If possible, the therapist should attend a training session and evaluate the functional capacity of the athlete.

The return to competition is the final goal of rehabilitation, but several criteria must be considered before allowing the athlete to compete. These include the absence of symptoms, normal flexibility, adequate strength (90% of the uninjured side), and the ability to perform. Finally, to prevent recurrence of the injury, general conditioning should be maintained.

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THERAPEUTIC MODALITIES

The basic rule of therapeutic modality use is, “There is no cookbook protocol!”. Although the proceedings make general statements, it is still the practitioner’s knowledge that guides his or her clinical decision-making. The injuries that athletes incur are just as individual as the athletes themselves. Approach each injury as a separate entity and utilise the knowledge and clinical decision-making abilities to formulate a treatment protocol (Figure 9-2).

It is imperative to address safety issues in regards to therapeutic modality use. As healthcare practitioners, it is understood and expected to “cause no harm.” Precautions must be taken in all instances so that a situation of healing does not become a situation of harm. Each therapeutic modality introduced in this chapter will be accompanied by safety measures and possible side effects.

As every inventor and invention offers a new therapeutic modality, manufacturers attempt to package the device as a convenient option for the busy healthcare practitioner. While this chapter will review basic tenets of therapeutic modality usage, remember that today’s market offers a plethora of various models and types of therapeutic modalities, all with different characteristics. This chapter does not take the place of the operator’s manual published for each specific modality.

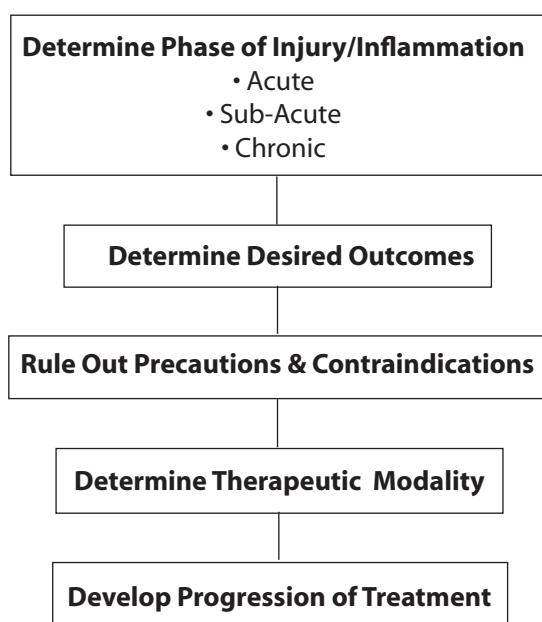


Figure 9-2. Treatment protocol.

A. Patient Preparation

1. Conduct a Thorough Evaluation

Along with the basic injury evaluation process, the clinician should review basic history of previous therapeutic modality treatments.

- a. Has the athlete ever had _____?
- b. If so, what was their reaction?
- c. How many treatment sessions were needed?
- d. Were there any negative outcomes?
- e. Did the treatment work?
- f. What medications are they presently taking?
- g. Does the athlete have a history of any medical illness?

2. Observe

Inspect the area to be treated for any obvious precautions or contraindications such as:

- a. Open wounds
- b. Skin abrasions
- c. Rash of unknown origin
- d. Desensitised areas
- e. Surgical implants (i.e., fixation devices)

B. Therapeutic Modality Set-up

After selecting the therapeutic modality, prepare the treatment area and modality equipment so that the athlete is safe, comfortable, and the area being treated is not compromised. If the modality chosen requires assistance, the clinician situates oneself so that they are comfortable as well (this becomes important when conducting an ultrasound treatment, for instance). The clinician should be able to access the therapeutic modality at any point during the treatment session to change parameters or cease the treatment.

C. Patient / Athlete Preparation

The area to be treated should be exposed and observable by the clinician at all times. In areas of discretion, sheets, towels or privacy curtains may be used.

D. Modalities

1. Hydrocollator

One of the most common forms of thermotherapy, the hydrocollator pack is typically used as a superficial heating therapeutic modality.

Indications: Chronic pain, muscular spasm, tissue extensibility, increasing blood flow.

Precautions: Thermal hypersensitivity.

Contraindications: Acute inflammation, malignancy, desensitised skin.

Preparation: Position the athlete so that the area to be treated is exposed. Do not allow athlete to position him or herself on top of Hydrocollator pack. Body weight decreases the tissue interface (area between modality and tissue) and increases heat conduction, thereby increasing risk for burns. The clinician should prepare the heating pack by applying six layers (i.e., towels) between the pack and the athlete.

Treatment Duration: 15–20 minutes or until heat has dissipated.

After Application: The clinician should monitor the athlete's reaction to treatment and manually check area underneath the hydrocollator pack at five minute intervals to prevent risk of burn. A post-treatment skin check should also be done.

2. Hydrotherapy (Warm Whirlpool)

Whirlpool units vary in size according to possible body parts able to be treated. Whirlpool units should be maintained at a temperature of 98°–108°F (37°–43°C) and checked periodically for tolerance. Care and appropriate measures must be taken when treating a body part that presents with open lesions or abrasions. The whirlpool should be thoroughly cleansed and disinfected prior to and post-treatment.

Indications: Pain, muscular spasm, tissue extensibility.

Precautions: Thermal hypersensitivity.

Contraindications: Malignancy, desensitised skin.

Treatment Duration: 15–20 minutes.

Preparation: Position the athlete on an elevated stool or seat depending on area to be treated. Allow athlete to acclimate him or herself to temperature.

After Immersion: The clinician should monitor the athlete's reaction to treatment and manually check area to prevent risk of burn. A post-treatment skin check should also be done.

3. Paraffin Bath

Paraffin wax can be used as a thermal agent for the extremities such as the hands and feet. Paraffin wax has a low melting point and specific heat that allows the athlete to gradually heat the extremity with low risk of burn. Paraffin wax should be mixed with mineral oil in order to ensure proper heating. The ratio of paraffin wax to mineral oil is 7:1 and temperature of the basin should be maintained at 48°–51°C (120°–125°F).

Indications: Chronic pain, muscular spasm, tissue extensibility, increasing blood flow.

Precautions: Thermal hypersensitivity, allergy to mineral oil or wax.

Contraindications: Acute inflammation, malignancy, desensitised skin.

Preparation: Athlete must thoroughly wash extremities to be treated and

remove all jewelry and accessories from the area. A towel and plastic bag should be prepared next to the athlete. The two most commonly used techniques include the multiple dip method and the dip and wrap method.

After Application: The clinician should monitor the athlete's reaction to treatment and observe the area for adverse reaction after the wax has been removed. The wax may be placed back in the basin and used again if uncontaminated.

4. Ultrasound

Ultrasound is a deep heating agent that utilises acoustic sound waves to generate mechanical disruption of tissues. Possible effects of ultrasound include deep tissue heating and non-thermal tissue manipulation. The parameters of common ultrasound units found in the athletic healthcare setting may be changed in order to achieve desired outcomes. The physical nature of acoustic energy generated from an ultrasound transducer has many clinical considerations.

The size of the transducer head should be chosen in regard to the structure being treated. If the area to be treated is larger than 2 to 3 times the transducer, the clinician should re-evaluate the choice of modality or protocol. Many ultrasound units' transducer size varies from 1 cm² to 10 cm².

Effective Radiating Area (ERA) determines the actual area where radiating energy is transmitted. The ERA is less than the actual size of the transducer; therefore the clinician should incorporate this ratio when determining appropriate transducer size.

Frequency is commonly referred to as the number of wave cycles completed per second. Ultrasound frequencies found on most units range from 1 MHz to 3 MHz (2 MHz is offered on some units). The lower the frequency, the deeper the ultrasound beam penetrates. Therefore, 1 MHz is used as a deep heating frequency (2–5 cm) while 3 MHz is used to heat superficial structures (1–2 cm).

Duty Cycle is the ratio of “on” to “off” periods of time while the ultrasound unit is actively transmitting energy. Selecting a continuous setting indicates that the transducer is transmitting acoustic energy 100% of the time and producing thermal effects. Common “Pulsed” intervals ranging from 20–50% can also be selected to produce non-thermal effects of ultrasound. Intensity is the amount of acoustic energy that the ultrasound unit is producing over a given area. It is proportional to the area being treated as seen with Spatial Average Intensity (referring to the amount of energy watts) the ultrasound unit is producing over the spatial area of the transducer head. This parameter is measured in watts per squared centimeter, or w/cm². As one can see, this is a combination of both intensity and area and can be affected by choosing different intensities or transducer sizes.

Beam Non-uniformity Ratio (BNR) is an indication of the ultrasound beam's depth and homogeneity of energy transmitted across the transducer. One might imagine an uneven jagged mountain range as the area of the transducer head and the largest peak occurring in the range as the peak intensity of the beam. A low ratio,

such as 2:1, indicates that when using an intensity of 1 w/cm^2 , the peak intensity of the ultrasound beam will reach as high as 2 w/cm^2 . When using a unit with a larger BNR, the risk for developing “hot spots” or areas of intense heat increases. Clinicians must be aware of these factors to avoid burning the athlete and potentially causing more tissue damage.

Modality Preparation–Ultrasound

The clinician should position the ultrasound unit proximal to and on the athlete’s affected side. Generous amount of ultrasound transmitting media, such as an aqueous gel, should be applied to the area and to the surface of the transducer to assure proper coverage. The gel increases the energy transmission of the ultrasound transducer and prevents reflection of acoustic energy. Other forms of liquid media have been identified to effectively transmit ultrasound waves. Alternative forms of media include a fluid filled bladder or underwater immersion. The parameters for the specific treatment session should then be selected (see Table 9-3 and Figure 9-3).

Athlete Preparation: The athlete should be seated or positioned comfortably so that the area to be treated can be accessed with the transducer head. The clinician should educate the athlete on the ultrasound treatment and instruct them to offer feedback as to how the treatment is progressing.

Indications (Thermal effects): Tissue extensibility, chronic pain, muscular spasm, increasing blood flow, inducing inflammatory response to resolve chronic injury.

Indications (Non-thermal Effects): Increase local metabolism, increase tissue-healing factors, bone healing.

Precautions: Units possessing a high BNR, thermal hypersensitivity, allergy to gel or transmission media.

Contraindications: Acute inflammation (thermal), malignancy, desensitized skin, eyes, reproductive organs and fluid filled cavity organs, epiphyseal plates, infection.

Treatment Method: The clinician should place the transducer head over the transmission gel and apply approximately 5–10 foot/lb of pressure and begin moving the transducer head in concentric circles at a slow steady pace making sure to stay within the imaginary 2–3 transducer sizes of the effective treatment area. The clinician should gather continual feedback from the athlete as the treatment progresses. Any indication of uneven heating or areas of intense heat should be assessed by the clinician.

5. Phonophoresis

A clinician might wish to add an anti-inflammatory medication to the coupling medium to solicit its effects combined with therapeutic ultrasound. Phonophoresis is the process by which ultrasound waves promote transdermal tissue absorption of the medication. It is believed that increasing local tissue metabolism and increasing vascular permeability allow for effective transmission of the medication into the

Table 9-3. Range of temperature increases at various intensities.

Intensity	1 MHz	3 MHz
0.5	0.04 C/min	0.3 C/min
1.0	0.2 C/min	0.6 C/min
1.5	0.3 C/min	0.9 C/min
2.0	0.4 C/min	1.4 C/min

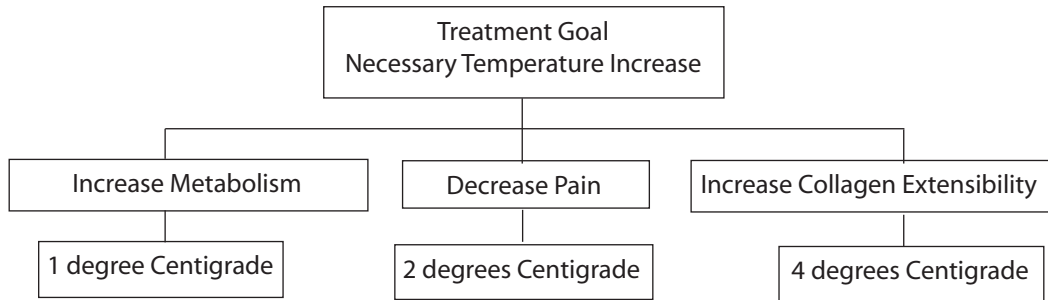


Figure 9-3. Ultrasound protocol.

blood stream. Phonophoresis has been shown to be effective when conducting both pulsed and continuous (100% duty cycle) ultrasound treatments, depending on whether heating the tissue is indicated. The procedures to conduct the ultrasound are the same, although duration might have to be increased to allow proper absorption. The clinician must be aware of the legal ramifications of using a prescription medication and act accordingly to laws and regulations. Some commonly used medications are 10% Hydrocortisone, salicylates and lidocaine.

6. Cryotherapy

Ice is a highly effective first aid agent. The application of ice to an acute (rapid onset) injury will decrease blood flow to the injured area resulting in reduced pain and decreased swelling. The body’s natural response to a traumatic injury is to rush blood to the site of injury. Application of ice causes capillary constriction and reduces blood flow, and decreases the release of inflammatory cytokines. This means less blood is released into the injured area, resulting in less swelling and a quicker healing time.

Remember, heat has the opposite effect and will increase blood flow to the injured area, increasing swelling and delaying the healing time. Heat should only be applied after the swelling and discoloration have been resolved, which is usually 2 days from the time of the initial injury. Guidelines for cryotherapy include the following:

- The application of ice to the affected area should begin as soon as possible.
- Apply the ice for 20 minutes at a time. Ice for no longer than 20 minutes at one time. Icing beyond 20 minutes offers no additional benefit. In fact, due

to a reflex vasodilation, the body will start to produce heat to the affected area, reversing the benefits of icing, resulting in increasing swelling and delaying the healing process. The application of ice can be repeated at least 3 times a day or more, but a 30 minute rest period of no icing needs to follow every ice treatment.

- Ice can be applied via ice bag, ice cups, frozen gel packs or cold whirlpool treatment. In the cases of gel packs and whirlpool treatments the affected area needs to be covered. In the use of ice bags or ice cups, no barrier is required.
- Elevate the area above the heart during the cold application treatment when possible.
- During the application of an ice treatment it is normal to experience the sensations of cold, burning, pain, and numbness. Redness will be present, but will subside in 15 minutes following the treatment. However, some individuals have an adverse reaction to cold and develop severe pallor (Raynaud's Syndrome) or even blisters.
- Never constrict blood flow when icing. Remove or loosen tight articles of clothing or tight tape surrounding the affected area. Also, ice bags should be applied on top of the affected area, verse lying on the ice. This can constrict blood flow.

a. Ice Pack

One of the most conventional therapeutic modalities, an ice pack can provide analgesia and prevent further swelling incurred from trauma. Both commercially made synthetic ice packs and natural ice-filled bags can be used in instances of inflammation.

Indications: Acute inflammation, chronic inflammation, hyperemia, excessive swelling.

Precautions: Hypersensitivity to cold.

Contraindications: Desensitised skin, allergic reaction to cold.

Athlete Preparation: Athlete should be lying down on treatment table in comfortable position so that injured area may be elevated. The clinician should apply the ice pack over the injured area and secure in place with an elastic bandage adding compression to the affected area. After application of the elastic bandage, the clinician should initially check for uninhibited circulatory flow distal to the injury site.

During Treatment: The clinician should monitor the athlete's feedback while the ice pack is applied and manually check for any adverse skin reactions to treatment.

Treatment Duration: Approximately 15–20 minutes.

Post Treatment: Remove ice pack and bandage and visually inspect area for adverse reactions brought on by exposure to cold. When applying commercial ice packs, it is important to check the integrity of the pack as many are made with harsh chemicals that might affect the skin.

b. Cold Whirlpool

Use of cold-water immersion has shown benefits of pain modulation and decreases in swelling by allowing active range of motion. Whirlpool units should be maintained at a temperature of 50°–60°F (10°–15.5°C) and checked periodically for tolerance. The athlete should not rest an extremity on the whirlpool in such a way that normal blood flow could be occluded. Care and appropriate measures must be taken when treating a body part that presents with open lesions or abrasions. The whirlpool should be thoroughly cleansed and disinfected prior to and post-treatment.

Indications: Pain, muscular spasm, prevent or decrease swelling.

Precautions: Hypersensitivity to cold.

Contraindications: Infection, desensitised skin.

Preparation: Position the athlete on an elevated stool or seat depending on area to be treated. Allow athlete to acclimate him or herself to temperature.

Treatment Duration: 15–20 minutes.

After Immersion: The clinician should monitor the athlete's reaction to treatment and manually check area to prevent adverse skin reaction. A visual inspection of the skin should also be performed following the treatment session.

c. Ice Cup

An ice cup decreases the tissue interface of the modality between the skin, has an equivalent temperature of ice, and provides for a mild massaging effect of the traumatised tissue. The effective area of an ice cup is small in comparison to other cryo-therapeutic devices.

Indications: Acute inflammation, chronic inflammation, hyperemia.

Precautions: Hypersensitivity to cold, large injured area.

Contraindications: Desensitised skin, allergic reaction to cold.

Athlete Preparation: Athlete should be lying down on the treatment table in a comfortable position so that injured area may be elevated. The clinician should apply the ice cup to the skin and begin stroking the ice cup over the injured area.

During Treatment: The clinician should monitor the athlete's feedback while the ice cup is moved over the area of trauma and manually check for adverse skin reactions to treatment.

Duration of Treatment: Approximately 10–15 minutes. Ice cup treatments can be considerably shorter due to the temperature of the modality.

Post Treatment: Remove ice cup and visually inspect area for adverse reactions brought on by exposure to cold. A general erythema or hyperemia over the treated area is normal in this case.

7. Electrical Stimulation

The use of electricity to treat pathology is a complex concept and should be further examined from the standpoint of physics and physiology. This section deals with the practical skills clinicians might use in the athletic healthcare setting when using electrical stimulation. As with all therapeutic modalities, extreme care and prudence must be priorities when treating an injury with electrical currents. It is wise for the clinician to experience the various types of electrical currents used in their facility to gain an appreciation of the specific device's capabilities and sense the athlete's perception of the treatment.

Choosing parameters is usually dictated by the feedback garnered from the athlete or the effects observed by the clinician (i.e., muscle contraction). The ranges associated with the following electrical currents should be used as starting points. The treatment goals determined by the clinician must be the deciding factor when selecting parameters on the modality (Figures 9-5–9-8). Parameters are specific to the device and manufacturer, but common elements of many electrical modalities include:

Intensity: amplitude of current

Pulse frequency: number of pulses per interval of time (minute)

Pulse duration: length of time of an individual pulse

Polarity: dominant net charge of a current

Indications: Pain modulation, wound healing, muscle re-education, oedema reduction, muscle recruitment, peripheral nerve injury.

Contraindications: Pacemakers, cancerous lesions, hemorrhage, pregnancy, metal implants in treatment area.

Athlete Preparation: The athlete's skin should be clean and excess hair should be cut short, but not shaved, to allow proper contact between the electrodes and the skin. Athlete should be positioned so that electrodes can be placed comfortably surrounding injury. The athlete should be educated about the effects of electrical stimulation and possible sensations they might experience.

a. Interferential Current (IFC)

Interferential current generates two high frequency waves from two separate channels in a quadpolar arrangement (four electrodes) to penetrate the initial impedance of the skin. The two carrier waves (varying in range from 4000–5000 Hz) eventually cross each other within the tissue producing a resultant wave or frequency (commonly 1–100 Hz). The resultant wave is a lesser frequency and effective in neuromuscular stimulation.

Indications: Pain, increase venous flow, muscle re-education.

Precautions: Skin irritation, hypersensitivity to electricity.

Contraindications: Dermatological diseases, idiopathic pain, cardiac pacemakers, thrombophlebitis, electrical current crossing the spine.

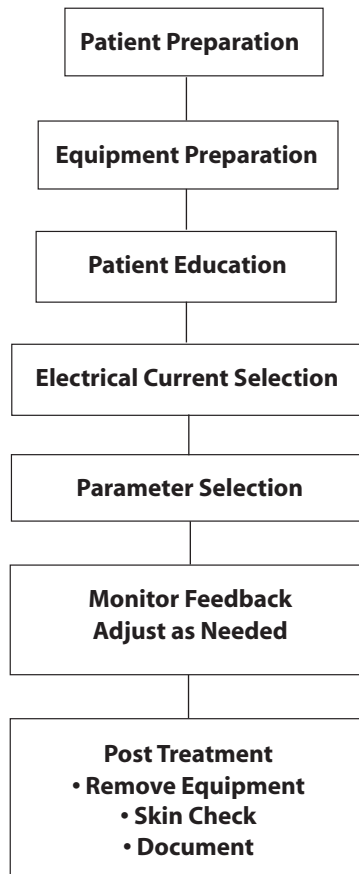


Figure 9-4. Electrical stimulation protocol.

Electrode set up: When conducting a quadpolar IFC treatment, pairs of electrodes should be placed on opposite ends of the injury site so that a crossing “X” pattern is formed.

During Treatment: The clinician should monitor the athlete’s feedback and be sure that the sensations the athlete is experiencing are in line with the intended treatment goals. Any deviation should be noted and adjusted appropriately.

Post Treatment: Remove equipment, conduct a skin check and document treatment.

b. Premodulated Current

The premodulated current is a form of interferential current albeit, using a bipolar (two-electrode) set-up. The resultant wave is created within the generator and transmitted through the electrodes into the tissue. The same indications, contraindications, and parameters apply for premodulated current. The clinician must incorporate body part and size when deciding between IFC and premodulated modalities.

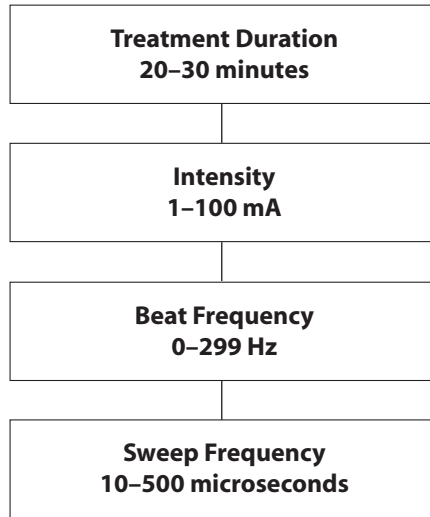


Figure 9-5. Premodulated current protocol.

c. Transcutaneous Electrical Nervous Stimulation (TENS)

To dispel any confusion, TENS is a “non-specific” term that refers to all forms of electrical stimulation. However, there are small, portable, battery powered units referred to as TENS units.

Indications: Pain.

Precautions: Same as for electrical currents.

Contraindications: Same as for electrical currents.

Electrode set up: TENS units consist of two channels, both able to supply two leads (electrodes). The clinician must be aware of the injury location and size in order to choose electrode placement.

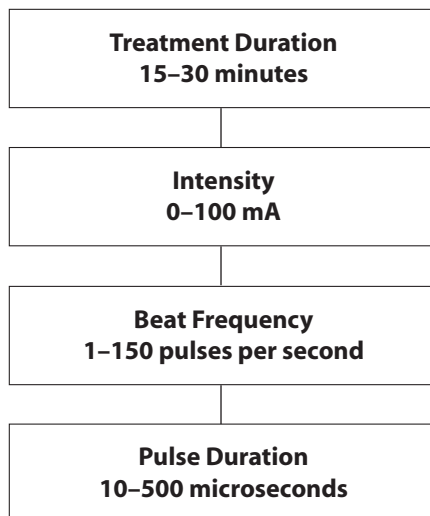


Figure 9-6. TENS protocol.

During Treatment: The clinician should slowly increase intensity until the athlete can feel the current. The clinician should then monitor the athlete's feedback and be sure that the sensations the athlete is experiencing are in line with the intended treatment goals (i.e., pain control). Any deviation should be noted and adjusted appropriately.

Post Treatment: Remove equipment, conduct a skin check and document treatment.

d. High Voltage Pulsed Current (HVPC)

A form of functional electrical stimulation, HVPC is commonly used to induce a motor response (i.e. muscle contraction) but can also be used for sensory stimulation (i.e., pain modulation) and oedema control. HVPC is a monophasic current that requires the use of a dispersive pad.

Indications: Muscle re-education, muscle strengthening, increased blood flow, pain, and wound healing.

Precautions: Same as for electrical currents.

Contraindications: Movement would worsen injury, same as for electrical currents.

Electrode set up: The clinician must decide what polarity the active electrode will be according to the treatment goal. A dispersive electrode, typically 2–3 times the size of the active electrode, should be placed on the ipsilateral side of the injury.

During Treatment: The clinician should slowly increase intensity until the athlete can feel the current and observable contractions are noted. The clinician should then monitor the athlete's feedback and be sure that the sensations the athlete is experiencing are in line with the intended treatment goals (i.e., muscle contraction). Any deviation should be noted and adjusted appropriately.

Post Treatment: Remove equipment, conduct a skin check and document treatment.

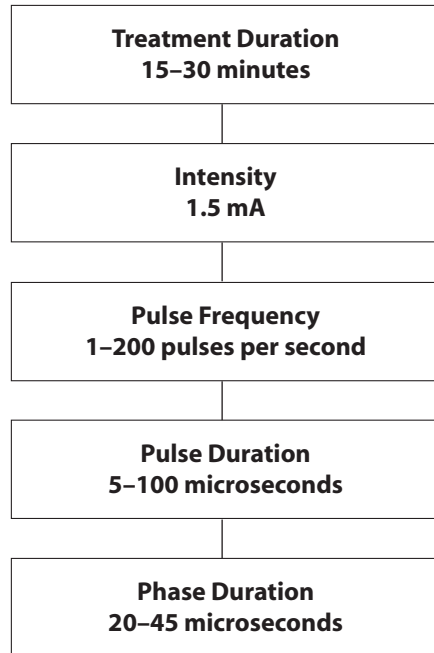


Figure 9-7. HVPC protocol.

e. Russian Current

Another form of functional electrical stimulation, Russian stimulation has been recorded to have effective results in motor muscle contraction and re-education. Peripheral nerve injury, disuse atrophy and post-surgical cases have shown improvements in muscle recruitment. The pulses of Russian current are grouped into bursts and delivered to the tissue in 5:1 intervals. This process allows for a smooth, pain-free muscle contraction.

Indications: Muscle re-education, muscle strengthening.

Precautions: Same as for electrical currents.

Contraindications: Movement could worsen injury, same as for electrical currents.

Electrode set up: The clinician should become acquainted with neuromuscular motor points of the affected muscle(s) and apply the electrodes accordingly.

During Treatment: The clinician should slowly increase intensity until the athlete can feel the current and an observable contraction is noted. The clinician should then monitor the athlete's feedback and be sure that the sensations the athlete is experiencing are in line with the intended treatment goals (i.e., muscle contraction). Any deviation should be noted and adjusted appropriately.

Post Treatment: Remove equipment, conduct a skin check and document treatment.

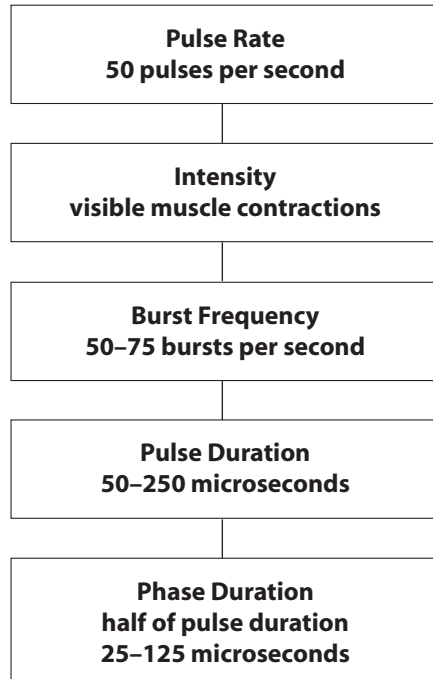


Figure 9-8. Russian current protocol.

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ANKLE AND FOOT INJURIES

Sprain of the lateral ankle ligaments is a very common injury. Approximately 25000 people experience it each day in the U.S., and 6000 people a day in France. A sprained ankle can happen to athletes and non-athletes, children and adults. It can happen when people take part in sports and physical fitness activities, or when they simply step on an uneven surface, or step down at an angle. In 2/3 of cases, the degree of sprain is mild or moderate, grade 1 or 2. Ankle injuries constitute 25% of all sports-related injuries, including 21% to 53% of basketball injuries and 17% to 29% of all soccer injuries.

The evaluation of ankle injuries can be simplified by understanding how anatomic factors dictate specific injury patterns. The high number of recurrent sprains and the frequency of long-term complications from instability and arthritis suggest that the current management protocols may not be always optimal. Athletes often return too quickly to the sports arena before their rehabilitation is complete. Athletes and coaches, as well as some physiotherapists and physicians, often fail to appreciate the risk of recurrent injury or chronic disability. The pressure exerted on the practitioner by athletes and coaches to return athletes to play as soon as possible must be balanced with the need to ensure complete recovery.

A. Anatomy and Biomechanics

The ankle joint is a simple hinge joint between the leg and the foot. The bones of the leg (tibia and fibula) form a sort of slot and the curved top bone of the foot (talus) fits between them. The talus is held to the tibia and fibula by ligaments. Each ligament is a semi-elastic structure and is made of many strands of collagen fibres. The ligaments of the ankle hold the ankle bones and joint in position. They protect the ankle joint from abnormal movements—especially twisting, turning, and rolling of the foot. Ligaments usually stretch within their limits, and then go back to their normal positions. When a ligament is forced to stretch beyond its normal range, a sprain occurs. A severe sprain causes actual tearing of the elastic fibres.

The ligament on the inside of the ankle (superficial and deep deltoid ligaments) has two layers; the deepest one is most important. The lateral ligament is made up of three separate bands: one at the front (anterior talo-fibular ligament: ATFL), one in the middle (calcaneo-fibular ligament: CFL) and one at the back (posterior talo-fibular ligament: PTFL). The front band is the ligament usually injured in sprains or tears of the ankle ligaments, and the middle band is sometimes affected.

The stability of the talo-crural joint depends on both joint congruency and the supporting ligamentous structures. The lateral ankle ligaments (Figure 10-1a), responsible for resistance against inversion and internal rotation stress, are the ATFL, the CFL, and PTFL. The deltoid ligaments, which are responsible for resistance to eversion and external rotation stress, are less commonly injured. However, an injury to these ligaments indicates severe trauma.

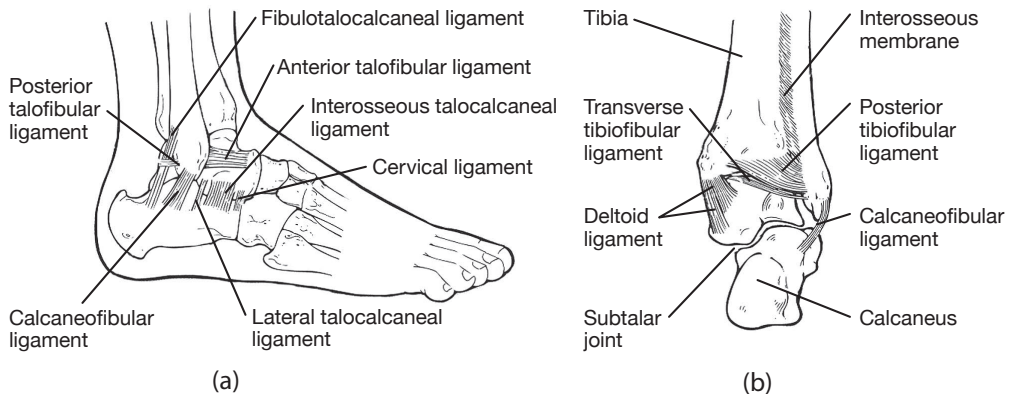


Figure 10-1. Lateral (a) and syndesmotic (b) ligaments of the ankle.

The ATFL resists ankle inversion in plantar flexion, and the CFL resists ankle inversion during dorsiflexion. The CFL spans both the lateral ankle joint and lateral subtalar joint, thus contributing to both ankle and subtalar joint stability. The PTFL is under greatest strain in ankle dorsiflexion and acts to limit posterior talar displacement within the mortise as well as talar external rotation.

The calcaneus articulates with the talus above it by three facets, to form the subtalar joint. The subtalar joint controls foot supination and pronation in close conjunction with the transverse tarsal joints of the midfoot. The CFL provides stability to inversion and torsional stresses to both the ankle and subtalar joints. Up to 50% of apparent ankle inversion observed actually comes from the subtalar joint. The CFL, the cervical ligament, the interosseous ligament, the lateral talocalcaneal ligament, the fibulotalocalcaneal ligament (ligament of Rouviere), and the extensor retinaculum contribute to stability of the subtalar joint.

The tibia and fibula have a small joint between themselves just above the ankle (tibio-fibular ligaments). The syndesmotic ligaments, responsible for maintaining stability between the distal fibula and tibia, consist of the anterior tibiofibular ligament, the posterior tibiofibular ligament, the transverse tibiofibular ligament, the interosseous ligament, and the interosseous membrane (Figure 10-1b). Injuries to the ankle syndesmosis occur as a result of forced external rotation of the foot or during internal rotation of the tibia on a planted foot.

The ligament at the front is involved in 10–20% of ankle sprains; the ligament at the back, like the deltoid ligament, is mainly damaged in association with severe fractures of the ankle bones.

Clinically, the most commonly sprained ankle ligament is the ATFL, followed by the CFL.

B. Mechanisms of Injuries

Lateral ankle sprains occur as a result of landing on a plantar flexed and inverted foot. These injuries occur while running on uneven terrain, stepping in a hole, stepping on another athlete's foot during play, or landing from a jump in an

unbalanced position. When this happens, the full force of the body's movement is placed on the anterior talo-fibular ligament. This may stretch, with tearing of some of its fibres (sprain) or it may tear completely. If there is a major injury of the anterior talo-fibular ligament, the forces transfer to the calcaneo-fibular ligament and the tibio-fibular ligaments, which may also be sprained or torn. Occasionally small pieces of bone may be torn off with the ligaments.

In a few cases, a twisting force on the ankle may cause other damage. The bones around the ankle may be broken, a piece of the joint surface inside the ankle may be chipped off, ligaments connecting other bones in the foot may be sprained or torn, or the tendons around the ankle may be damaged.

C. Patient History

Given the strong correlation between the mechanism of injury and diagnosis, identifying the joint position at the time of injury is a useful first step in the clinical evaluation. Therefore, it may be clearer if the examiner shows the patient what is meant by the various terms or has the patient demonstrate the mechanism of injury with the uninjured ankle.

Revisiting the precipitating activity may help determine if the injury was unavoidable or resulted from inherent weaknesses. Jumping and landing on another athlete's foot or stepping in a rut on the field is likely to injure a previously normal ankle. Sprains that are unprovoked or occur in situations that wouldn't injure a normal ankle raise concerns for other diagnoses, such as tarsal coalition, osteochondritis, or peroneal tendon dislocation.

The history should include the location of pain, presence of swelling, and functional capacity, including the ability to bear weight, walk, run, and jump. The history should also include whether the patient heard a "pop" and a review of prior injuries, previous diagnostic studies, treatments, and any residual impairments. The patient's current sports participation history and training plan help to gauge conditioning needs during recovery and requirements for return to play.

D. Physical Examination

The exam of the injured ankle starts with an assessment of the degree and location of swelling and ecchymosis. Palpation should include bony landmarks such as the lateral malleolus, the medial malleolus, the fibula, the fifth metatarsal, and, in skeletally immature patients, the physis. Achilles tendon, peroneal tendons, and posterior tibial tendon should also be palpated, because injuries to these structures may mimic ankle sprains. Soft-tissue palpation includes the ATFL, CFL, PTFL, deltoid ligament, and peroneal tendon. Tenderness over the anterior joint line or syndesmosis may indicate a sprain of the interosseous membrane.

A careful neurologic examination is essential to rule out loss of sensation or motor weakness, as peroneal nerve and tibial nerve injuries are sometimes seen with severe lateral ankle sprains.

Provocative tests for lateral ankle instability include the anterior drawer test, inversion stress test, and the suction sign. The anterior drawer test is specific for the ATFL and can be done with minimal pain or guarding. Two provocative tests for syndesmotic ligament injury are the squeeze test and the external rotation stress test.

Tests for range of motion, strength, and proprioception are likely to be abnormal in the acute setting but may help assess deficits in patients who have chronic or recurrent sprains.

1. Grading

Various systems are used for grading the severity of ankle sprains. It is cumbersome to assign a grade 1 to 3 rating to each of the three lateral ligaments that may be injured. Some clinicians prefer to use the number of injured lateral ligaments to assess severity. An isolated sprain to the ATFL is considered a grade 1 (mild) sprain. A two-ligament injury involving the ATFL and CFL is a grade 2 (moderate) sprain. A grade 3 (severe) sprain indicates all three lateral ligaments have been injured.

Alternatively, grading is more commonly determined by the extent of functional disability. Grading of ankle sprains guides treatment, rehabilitation, and prognosis:

- a. **Grade 1 sprain:** Slight stretching and some damage to the fibres (fibrils) of the ligament.
- b. **Grade 2 sprain:** Partial tearing of the ligament. If the ankle joint is examined and moved in certain ways, abnormal looseness (laxity) of the ankle joint occurs.
- c. **Grade 3 sprain:** Complete tear of the ligament. If the examiner pulls or pushes on the ankle joint in certain movements, gross instability occurs.

E. Radiologic Evaluation

The decision to order radiographic studies should be based on the probability of finding bony abnormalities. When radiographs are indicated, the standard views should include anteroposterior, lateral, oblique and mortise. The Ottawa clinical decision rules (for patient from age 15 to 60 years old) were proposed as a means to reduce the number of unnecessary radiographic studies without sacrificing sensitivity for detecting fractures. These guidelines state that an ankle radiographic series should be obtained if bone tenderness is present over the lateral or medial malleolus, or if the patient is unable to bear weight for four steps both immediately post-injury and in the emergency department. Exclusions for use of the Ottawa ankle rules are age younger than 15 years, older than 60 years, intoxication, multiple painful injuries, pregnancy, head injury, or diminished sensation due to neurologic deficit. X-rays are least likely to be warranted for patients who exhibit laxity of the ATFL without other clinical findings. Bone scans, magnetic resonance images (MRIs), computed tomography (CT) scans, and arthrograms all have diagnostic utility for specific injuries (fractures; avulsions; talar dome fracture) but have little role in the initial evaluation of ankle sprains.

Foot radiographs should also be obtained if the physical examination demonstrates tenderness in the hindfoot, midfoot, or forefoot.

1. Stress Radiographs

Stress radiographs help document lateral ligamentous chronic ankle injury—especially chronic instability but are not required to make the diagnosis of an acute ankle sprain.

F. Early Treatment

Ligamentous injuries undergo a series of phases during the healing process: hemorrhage and inflammation, fibroblastic proliferation, collagen protein formation, and collagen maturation. The more severe the ligament injury, the greater the time required to progress through the stages of healing. Early mobilisation of joints following ligamentous injury actually stimulates collagen bundle orientation and promotes healing, although full ligamentous strength is not re-established for several months. Therefore, early treatment focuses on regaining range of motion while protecting the injured ligaments against re-injury. Limiting soft-tissue effusion speeds healing.

The standard early treatment following an acute ankle sprain is PRICE (protection, rest, ice, compression, and elevation). Cryotherapy, compression, and elevation are essential to limit initial swelling from hematoma and oedema around the ankle and speed ligamentous healing. Early use of cryotherapy, applied in the form of ice bags, a cold whirlpool, or a commercially available compressive cuff filled with circulating coolant, has been shown to enable patients to return to full activity more quickly. Compression can be applied by means of an elastic bandage, felt doughnut, neoprene or elastic orthosis, or pneumatic device.

1. Early Mobilisation

Protected weight bearing with an orthosis is allowed, with weight bearing to tolerance as soon as possible following injury.

2. Bracing

Protection of the ankle during initial healing is essential. This may be accomplished with taping, a lace-up splint, a thermoplastic ankle stirrup splint, a functional walking orthosis, or a short leg cast. Flexible and semi-flexible braces have been shown to effectively limit ankle inversion and to resist passive torque. More severe injuries usually require longer immobilisation. Early protected range of motion in a flexible or semi-rigid orthosis is superior to rigid cast immobilisation in terms of patient satisfaction, return of motion and strength, and earlier return to function.

3. Rehabilitation (see also Chapter 9, Part 2, *Principles of Rehabilitation of the Injured Athlete*)

Physical therapy of the injured ankle is divided into 5 phases by some authors: acute, subacute, rehabilitative, functional, and prophylactic; or into 3 phases

by others: **Phase 1** is rest, ice, compression, and elevation (RICE) and protected weight bearing as needed. **Phase 2** consists of restoring ankle motion, strength, and proprioception and can begin when the patient can place some weight on the ankle. **Phase 3** includes activity-specific drills before return to full activity.

With the 5 phases protocol, the exact timing of each phase varies with the severity of the sprain. The acute phase is based on PRICE with goals to limit effusion, reduce pain, and protect from further injury. The subacute phase focuses on decreasing and eliminating pain, increasing pain-free range of motion, continuing protection against re-injury with bracing, limiting loss of strength with isometric exercises, and continuing modalities to decrease effusion. The rehabilitative phase emphasises regaining full pain-free motion with joint mobilisation and stretching, increasing strength with isotonic and isokinetic exercises, and employing proprioceptive training. The functional phase focuses on sports-specific exercises with a goal of returning the patient to sports participation. The prophylactic phase seeks to prevent recurrence of injury through preventive strengthening, functional proprioceptive drills, and prophylactic support as needed.

With the 3 phases protocol to mobilise and rehabilitate grades 1 and 2 sprains:

a. Phase 1

Phase 1 is directed toward reducing swelling, protecting the injured ligaments, and beginning weight-bearing activity. Ice, compression, and elevation may be used to control swelling. The ankle can be protected in a figure-eight brace, tape, ankle corset, or cast boot, depending on the severity of injury. The level of protection should allow the patient to begin weight bearing as soon as possible. Crutches may be necessary for pain-free ambulation in some patients, but prolonged immobilisation or non-weight bearing (NWB) have little benefit and, arguably, may have adverse effects on the patient's recovery.

b. Phase 2

Phase 2 begins when the swelling has subsided and the patient is ambulating without discomfort. The goal of phase 2 is to restore ankle range of motion and build strength in the surrounding muscles—particularly the peroneals. Active range-of-motion exercises include drawing the letters of the alphabet with the toes. Restoring full dorsiflexion is critical for regaining speed, explosiveness, and jumping ability. Dorsiflexion can be tested by having the patient do a one-legged squat with the heel touching the ground. Dorsiflexion of the uninjured ankle can be used for comparison.

Strengthening can be done with isometric exercises, manual resistance, or elastic tubing exercises. The peroneals compensate for laxity in the lateral ligaments and should be emphasised in the strengthening programme. Pain or swelling associated with the exercises indicates that the patient is not ready for this phase of rehabilitation. When the resistance and number of repetitions performed with the injured ankle is equal to the uninjured side, the patient can progress to phase 3 of rehabilitation.

In the latter parts of phase 2 and early parts of phase 3, most athletes are able to tolerate low-impact exercise.

c. Phase 3

Phase 3 exercises commence when joint motion and strength are back to normal. The goal of the early part of phase 3 is to restore the proprioception that is predictably lost with ankle sprains. Proprioceptive deficits may be increased by prolonged NWB or immobilisation and may lead to further injury if not corrected. Proprioception can be measured by a modified Romberg test. The patient's ability to maintain balance on one foot is compared with the uninjured side. Proprioception can be restored by use of a balance board or exercises such as playing catch or brushing teeth while balancing on one foot. Braces or tape may be helpful, in part because of their proprioceptive input.

Late Phase 3: at the end of the third phase of rehabilitation comes the re-adaptation period to prepare the athlete to return to the field. This period consists of functional progression from rehabilitation exercises to sport-specific skills. When all of the earlier phases have been completed, the patient may begin a return-to-running program that starts with jogging and progresses to running, sprinting, circles, figure eights, cutting, pivoting, and jumping. When all of these activities can be done without pain or limitation, the patient may be cleared to return to practice and, eventually, full participation.

Protection with taping or bracing during daily activity is recommended until strength returns to normal. When the patient is ready to start the functional progression, protective devices are recommended only during exercise and sports participation.

Compliance with and efficacy of the rehabilitation programme may be enhanced if the patient is able to work with a certified athletic trainer or physical therapist. When a course of rehabilitation is prescribed during the initial evaluation of the injury, many important details of later care may be lost. Contact with a physician, physical therapist, or trainer during each of the three phases of rehabilitation can help ensure that patients are progressing at a reasonable rate and correctly performing exercises appropriate for their level of recovery. A well-designed rehabilitation programme includes a clear plan for implementation, monitoring, and follow-up care.

G. Non-surgical Treatment Results

Primary surgical repair of the torn lateral ankle ligaments has been advocated by some as treatment for elite athletes and young adults; however, it has not been supported in comparative studies that recommend early non-operative functional treatment of ankle ligament injuries.

It has been documented that satisfactory subjective and clinical stability have been restored with non-operative treatments such as casting, taping, bracing, and early physical therapy. A prospective study of 146 patients with grade 3 ankle sprains who were randomised into operative or non-operative groups found that

the group treated with an ankle orthosis for 6 weeks returned to work faster. No difference in joint laxity between the groups was found on stress radiographs performed 2 years post-injury.

Syndesmotic ligamentous injuries without fracture or gross widening of the ankle mortise are treated non-operatively with a short leg cast or brace, followed by physical therapy. The patient should be advised that these injuries result in longer periods of disability than injuries to the lateral collateral ligaments. If diastasis of the syndesmosis is evident on plain radiographs, operative stabilisation of the ankle mortise is accomplished with a syndesmotic screw.

H. Evaluating Chronic Symptoms

Chronic pain following ankle injury is common. In a retrospective study of 457 patients treated with immobilisation or bracing, 72.6% reported residual symptoms at 6 to 18 months.

In the evaluation process, the workup should center on whether the patient's chief chronic ankle complaint is pain or instability (Figure 10-2). If the primary problem is ankle pain, a concentrated effort should be made to rule out occult fracture of the foot or ankle. A technetium bone scan is an excellent screening test to rule out occult fractures and to guide further treatment. If the bone scan reveals increased uptake in a discrete area, a spot radiograph or computed tomography scan is useful to further identify the exact location of fracture. Occult or associated injuries to the tendons of the foot and ankle should also be considered, and MRI is the most useful exam to identify and confirm them. Table 10-1 lists some commonly missed occult fractures and tendon pathologies.

Table 10-1. Commonly missed diagnoses in patients with chronic ankle pain.

<p>Fractures</p> <ul style="list-style-type: none"> • Talar dome osteochondral • Lateral talar process • Anterior process calcaneal • Lateral malleolar • Posterolateral distal fibular flake • Fifth metatarsal base • Navicular <p>Tendon Injuries</p> <ul style="list-style-type: none"> • Achilles rupture • Peroneal tendon rupture • Peroneal tendon subluxation/dislocation • Posterior tibial tendon rupture • Anterior tibial tendon rupture • Flexor hallucis longus tendon rupture
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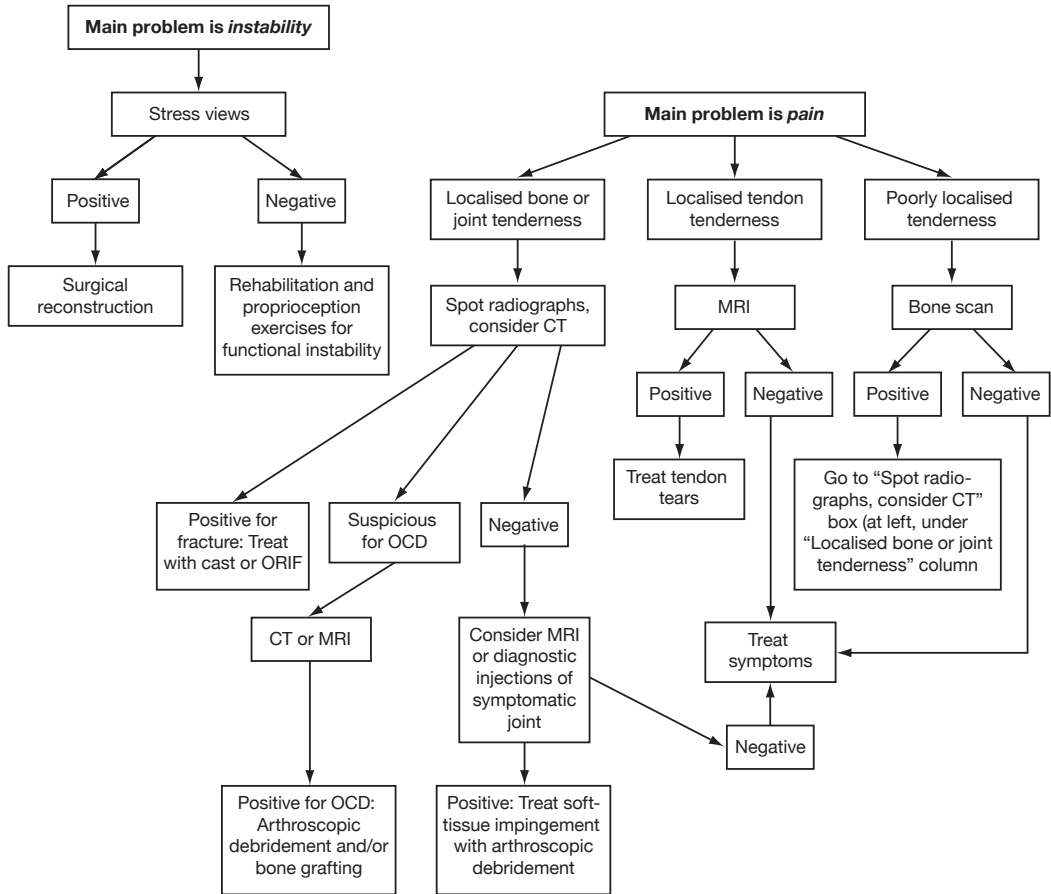


Figure 10-2. Algorithm for chronic ankle pain and instability.
OCD = osteochondritis dissecans; ORIF = open reduction and internal fixation

1. Other Soft Tissue Causes

Other soft-tissue causes of chronic ankle pain include anterolateral ankle impingement (meniscoid lesion), anteroinferior tibiofibular ligament impingement (Basset's ligament), and anomalous peroneal pathology. Injury to the lateral ankle ligaments may produce scarring of the ATFL and joint capsule, leading to the formation of "meniscoid tissue" in the anterolateral ankle. Anterolateral impingement can develop when inflamed tissue is pinched between the talus, fibula, and tibia. The distal fascicle of the anteroinferior tibiofibular ligament may abrade the anterolateral surface of the talus when the ankle is dorsiflexed during abnormal anterior translation of the talus. An anomalous or accessory peroneal tendon may also cause chronic posterolateral ankle pain.

2. Osteochondral Fractures

Fractures of the talar dome, which occur in association with ankle sprains, are commonly overlooked. These occur when there is a compressive component to the inversion injury, especially when landing from a jump. Usually the fracture is

not detected initially and the patient presents some time later complaining of an unremitting ache in the ankle, despite appropriate treatment for an ankle sprain.

A radioisotopic bone scan will confirm the presence of an osteochondral fracture. Grade II, III and IV will be evident on a CT scan, but only MRI will pick up a grade I lesion. Grade I and II should be treated with a NWB cast for 6 to 8 weeks. Grade IIa, III and IV fractures require arthroscopic removal of the fragment. A comprehensive rehabilitation programme with a graduated return to weight bearing is then required.

3. Tibialis Posterior Tendonitis

Tibial posterior tendonitis (Figure 10-3) is the most common cause of medial ankle pain. This condition may occur as a result of prolonged stretching into eversion and is often associated with excessive subtalar pronation. Treatment with physiotherapy, NSAIDs, and orthotics may be required to control excessive pronation.

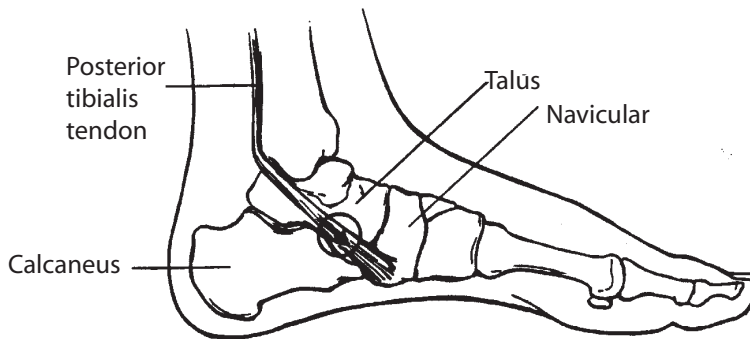


Figure 10-3. Tibialis posterior tendonitis—excess pronation puts additional stress (arrow) on this tendon.

4. Flexor Hallucis Longus Tendonitis

Flexor hallucis longus tendonitis presents with pain on toe-off or forefoot weight bearing. It is aggravated by resisted flexion of the first toe or stretch into full dorsiflexion of the hallux. This condition is often associated with posterior impingement syndrome as the FHL tendon lies in a fibro-osseous tunnel between the lateral and medial tubercles of the posterior process of the talus. Treatment consists of physiotherapy, NSAIDs, and stretches.

5. Tarsal Tunnel Syndrome

This syndrome occurs as a result of entrapment of the posterior tibial nerve in the tarsal tunnel where the nerve winds around the medial malleolus. This syndrome often occurs as a result of trauma (inversion injury to the ankle) or overuse associated with excessive pronation. Features of this condition are pain radiating into the arch of the foot, heel and toes, and pins and needles and numbness on the sole of the foot aggravated by prolonged standing, walking or running. Treatment may consist of corticosteroid injection and control of excessive pronation by orthotics.

6. Medial Malleolus

Stress fracture of the medial malleolus should also be considered in the running athlete with persistent medial ankle pain.

7. Lateral Pain

Lateral pain is generally associated with a biomechanical abnormality, and can have a variety of causes:

a. Peroneal Tendonitis

Peroneal tendonitis (Figure 10-4) is the most common overuse injury causing lateral ankle pain. Inflammation of the peroneal tendons or sheaths may be due to excessive eversion (running on slopes, etc.) and is commonly associated with excessive pronation. Localised tenderness over the peroneal tendons is occasionally associated with swelling and crepitus. Treatment consists of physiotherapy, assessment of biomechanical abnormalities and correction.

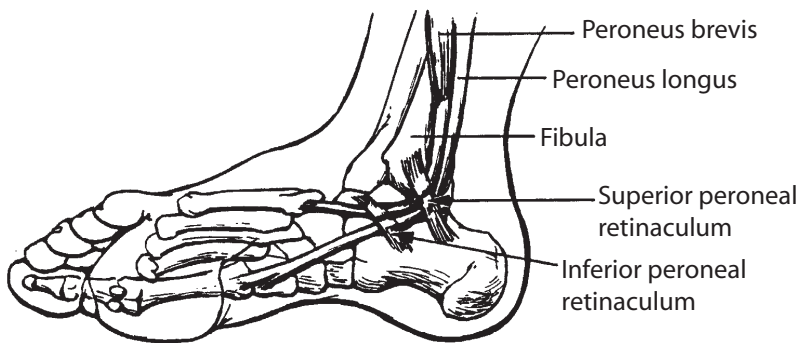


Figure 10-4. Lateral view of peroneal tendons and support structures.

b. Sinus Tarsi Syndrome

The calcaneus and the talus articulate via three facet joints and are supported by several surrounding ligaments to form the subtalar joint (see A. Anatomy b. Sinus Tarsi Syndrome

The calcaneus and the talus articulate via three facet joints and are supported by several surrounding ligaments to form the subtalar joint (see A. Anatomy and Biomechanics). Injuries to this complex may result in the “sinus tarsi syndrome.” This syndrome is often due to poor biomechanics and chronic overuse, or results from an acute ankle sprain. It often occurs after repeated forced eversion (e.g. high jump take-off). Forced passive eversion of the subtalar joint elicits pain and the subtalar joint is often stiff. Treatment includes mobilising the subtalar joint, NSAID, and biomechanical correction. Local anesthetic injections may also be required.

8. Anterior Ankle Pain

Anterior ankle pain related to overuse is usually due to:

a. Tibialis Anterior Tendonitis

Tibialis anterior tendonitis presents as localised tenderness, crepitus and pain on resisted dorsiflexion. It is usually due to restriction in joint ROM or downhill running. Treatment requires NSAID, physiotherapy and mobilisation of the ankle joint.

b. Anterior Impingement

Anterior impingement of the ankle may be the cause of chronic ankle pain or may follow an ankle sprain. As a result of persistent forced dorsiflexion (kicking), exostoses develop on the anterior margins of the ankle joint. As they become larger they impinge on overlying soft tissue and cause pain. Pain is reproduced by standing and lunging forwards (positive anterior impingement test). X-ray with a “hinge” view will identify the bony spurs. Treatment of mild cases involves AP glides of the talo-crural joint at the end range of dorsiflexion. Corticosteroid infiltration can be an effective treatment in more severe cases. Surgery to excise larger exostoses may be required.

9. Instability

If the primary problem is ankle instability, the patient will experience feelings of “giving way” of the ankle on uneven ground, inability to play cutting or jumping sports, loss of confidence in ankle support, reliance on braces, and a history of multiple ankle sprains. If, on further evaluation, stress radiographs are positive for mechanical lateral ligamentous laxity, surgery is indicated to reconstruct the loose ligaments.

If stress radiographs are nondiagnostic for mechanical laxity, the patient may have functional ankle instability due to deficient neuromuscular control of the ankle, impaired proprioception, and peroneal weakness. Treatment in this case should be directed toward restoring peroneal tendon strength and ankle motion and improving ankle proprioception with physical therapy. Other causes of instability, not demonstrated by stress radiographs, include rotational instability of the talus, subtalar instability, distal syndesmotom (tibiofibular) instability, and hindfoot varus malalignment.

I. Treatment Options: Surgical

Surgical treatment for ankle sprains is rare. Surgery is reserved for injuries that fail to respond to non-surgical treatment, and for persistent instability after months of rehabilitation and non-surgical treatment.

The patient continues to experience multiple episodes of lateral ankle instability, and mechanical problems are documented by stress radiographs. Most procedures are designed to tighten or reconstruct the ATFL and CFL.

Surgical options include:

- **Arthroscopy:** A surgeon looks inside the joint to see if there are any loose fragments of bone or cartilage, or part of the ligament caught in the joint.

- **Reconstruction:** A surgeon repairs the torn ligament with stitches or sutures, or uses other ligaments and/or tendons found in the foot and around the ankle to repair the damaged ligaments

Following lateral ankle ligamentous reconstruction, most postoperative regimens immobilise the ankle in a cast for 4 weeks followed by an orthosis for an additional 4 weeks. Physical therapy with an emphasis on peroneal strengthening and proprioceptive training is instituted 6 to 8 weeks after surgery. Return to sports occurs at about 3 months postsurgery.

1. Rehabilitation After Surgery

Rehabilitation after surgery involves time and attention to restore strength and range of motion so the athlete can return to pre-injury function. The length of time one can expect to spend recovering depends upon the extent of injury and the amount of surgery that was done. Rehabilitation may take from weeks to months.

2. Rehabilitation Exercises

Rehabilitation is used to help to decrease pain and swelling and to prevent chronic ankle problems. At first, rehabilitation exercises may involve active range of motion or controlled movements of the ankle joint without resistance. Water exercises may be used if land-based strengthening exercises, such as toe-raising, are too painful. Lower extremity exercises and endurance activities are added as tolerated. Proprioception training is very important, as poor proprioception is a major cause of repeat sprain and an unstable ankle joint. Once the patient is pain-free, other exercises may be added, such as agility drills. The goal is to increase strength and range of motion as balance improves over time.

Specific exercises for competitive athletes would probably use more intensive strengthening and proprioceptive exercises. Exercise bands are available from supply houses. Grade 1 ankle sprain don't need a rehabilitation programme.

The exercise protocol for grade 2: 3 times per week, for 9 to 15 sessions depending on the progression of symptoms. Physiotherapy methods are useful but no one is preferable. Ultra-sound is not useful. The most important thing is to recover an active mobility in the sagittal plane in a painless range of motion. The movements should be done slowly and controlled to reap the full benefits. Patients are encouraged to discontinue using crutches or canes as soon as pain will allow. Walking is permitted to the limits of pain. Proprioception exercises begin between 10 and 15 days after the disappearance of pain and with a good mobility of the ankle.

The next phase of rehabilitation is agility drills and sport-specific drills that should be guided by a healthcare professional.

J. Risk Factors/Prevention

The best way to prevent ankle sprains is to maintain good strength, muscle balance, and flexibility.

- Warm-up before doing exercises and vigorous activities.
- Pay attention to the surfaces on which one runs, walks, or works.
- Wear good shoes.
- Pay attention to the body's warning signs to slow down when feeling pain or fatigue.

K. Foot Injuries

1. Rear-foot

The most common causes of rear foot pain are:

a. Plantar Fasciitis

The plantar fascia is a dense fibrous membrane that extends the entire length of the foot, from the calcaneal tubercle to the proximal phalanges. It protects the underside of the foot and helps support the arches (Figure 10-5).

Plantar fasciitis is a degenerative condition of the plantar aponeurosis. It is caused by repetitive microtrauma as part of an overuse syndrome.

Predisposing factors may be anatomic, such as pes cavus or pes planus, leg length discrepancy, or excessive pronation; or biomechanical, such as poor foot gear, muscle tightness, nerve entrapment, or over-training.

Pain occurs on initial standing, as the plantar fascia contracts during sleep. On examination, there is usually point tenderness at the medial calcaneal tuberosity.

Treatment is primarily non-surgical. Analgesics such as NSAIDs help to control pain. Properly fitted and cushioned foot gear is essential, along with orthoses when needed.

Dorsiflexion night splints prevent contraction of the plantar fascia during sleep, and are an effective adjunct. Stretching the gastrocnemius and soleus, as well as the toes, is an important part of the treatment regimen. Wall stretches and the use of slant and rocker boards aid in dynamic stretching.

Corticosteroid injections may relieve pain rapidly, but increase the risk of tendon rupture and fat pad atrophy. Delivering corticosteroids by iontophoresis is safer, but the effects may be short-lived. The use of extra-corporeal shock wave therapy (ESWT) has been espoused in recent years as treatment for a wide variety of tendinoses, including plantar fasciitis. However, results remain controversial due to a variety of factors. For recalcitrant cases, endoscopic

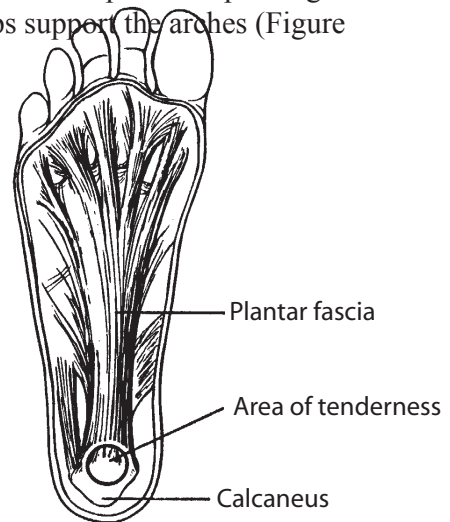


Figure 10-5. Plantar fasciitis.

fascial release has been proposed as a safer approach than open surgery, although not all patients become pain-free.

b. Fat Pad Contusion

This type of contusion occurs as an acute injury after a fall onto the heel or chronically as a result of excessive heel strike, such as long jumping. Treatment consists of avoiding aggravating activities, and strapping. A padded heel cup is helpful for jumpers.

c. Calcaneal Stress Fractures

These stress fractures can be shown with a radioisotopic bone scan and need NWB for 6 weeks.

2. Midfoot

The most important causes of midfoot pain are:

a. Navicular Stress Fracture

It is important to diagnose this condition, as significant morbidity is associated with non-union. Dorsal foot pain and pain and tenderness over the navicular are clinically suggestive. Isotopic bone scan and follow-up CT scan are required for complete diagnosis. Treatment requires a molded cast with NWB for 6 to 8 weeks. Tenderness over the "N-spot" must have subsided for a clinical clearance, and mobilisation of the stiff ankle and foot is essential after the cast is removed.

b. Extensor Tendonitis

Extensor tendonitis will cause an ache over the dorsal aspect of the mid foot and insertion of tibialis anterior. The extensor tendons may be weakened and strengthening is essential.

c. Midtarsal Joint Sprains

These joint pains happen occasionally, especially when instability of the foot is present. In particular, the calcaneonavicular ligament may be injured.

3. Forefoot

Forefoot pain can be caused by:

a. Metatarsal Stress Fractures

The athlete complains of fore-foot pain, aggravated by running or weight-bearing activities. The neck of the second metatarsal is the most common site of pain. A bone scan may be needed to confirm the diagnosis.

The most difficult fractures to manage are those at the base of the second metatarsal, the proximal shaft of the fifth metatarsal, and the sesamoid bones. The base of the second metatarsal can be treated with a NWB cast for 4 to 6 weeks, but occasionally may require internal fixation if non-union occurs. Most sesamoid fractures heal if rested, but may go on to avascular necrosis if neglected.

Acute inversion injury of the foot may cause avulsion of the peroneal brevis tendon, or fracture of the proximal shaft of the fifth metatarsal (Jones fracture).

This fracture often results in non-union. It requires a NWB cast for 4 to 6 weeks, or internal fixation if casting fails.

b. First Metatarsophalangeal Joint Sprain

This sprain occurs as a result of excessive forced dorsiflexion of the first MTP joint, and is referred to as “turf toe”. There is a history of vigorous “bending” at the first MTP joint, with pain on movement. The injury involves a sprain of the plantar capsule and ligament. Physiotherapy and orthotic correction may be required.

c. Sesamoid Injuries

Sesamoid injuries can include traumatic fracture, stress fracture, and sprain of a bipartite sesamoid. These are usually associated with marked tenderness and swelling in the sesamoid region. The patient will often walk with their weight borne laterally to compensate. Physiotherapy, padding to distribute the weight, and corticosteroid injections can all be effective.

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LOWER EXTREMITY INJURIES

Lower extremity injuries are the most common in athletics. Running, jumping and throwing produce tremendous ground reactive forces that must be absorbed by the body's kinetic chain. Although the foot and ankle are the first links in the system, the forces are transmitted upward.

Normally, muscles absorb approximately 80% of the impact of running, with the remainder of impact forces taken up by bone and adjacent tissues. The repetitive nature of running creates multiple musculoskeletal injuries and dysfunction. Muscles respond in characteristic patterns, which often result in muscle imbalance leading to injuries.

A. Proximal Extremity Disorders

1. Iliopsoas Strain and Hip Dysfunction

Runners typically spend more time in hip flexion (iliopsoas) and little of the running stride in hip extension (gluteus maximus). The iliopsoas becomes facilitated, hypertonic, and shortened. The antagonist muscle, the gluteus maximus, responds with inhibition, hypotonicity, and weakness. This can alter the arthrokinetics of the lumbopelvic region and lower extremities.

2. Hernias

Different types of hernias may occur, including inguinal, abdominal, or femoral. Any of these may be caused by a sudden rupture of the fascia and muscle or they may develop progressively over time. Inguinal hernias are the most common and the most easily diagnosed. Observe for any obvious bulge and palpate over the deep and superficial rings for a thrill (Figure 10-6). Occult (sports hernias) are more difficult to diagnose and often treated as only a "strain." Early diagnosis is very important and may require a pelvic CT scan, MRI, or surgical referral. Occasionally, the diagnosis is made via laparoscopy. Surgical repair is the only definitive treatment.

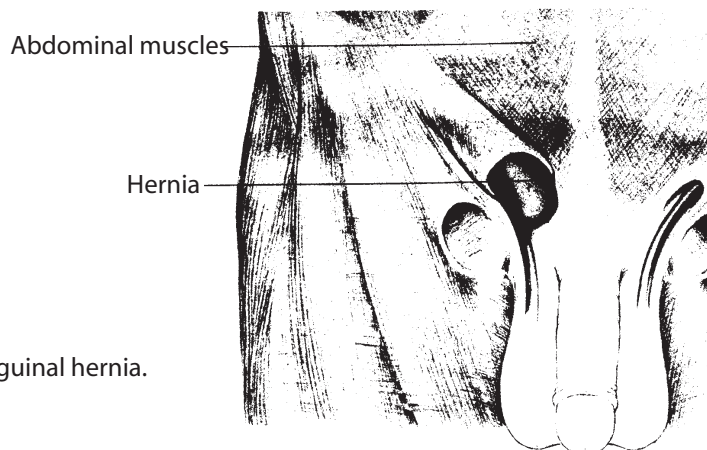


Figure 10-6. Inguinal hernia.

3. Traction Apophysitis and Avulsion (Figure 10-7)

The rectus femoris takes origin from the anterior *inferior* iliac spine, and the sartorius takes origin from the anterior *superior* iliac spine. Both of these muscles can cause traction at these sites and lead to apophysitis in the young athlete, and also to avulsion. These should be treated as a 3rd degree strain and managed accordingly. They seldom, if ever, need surgery.

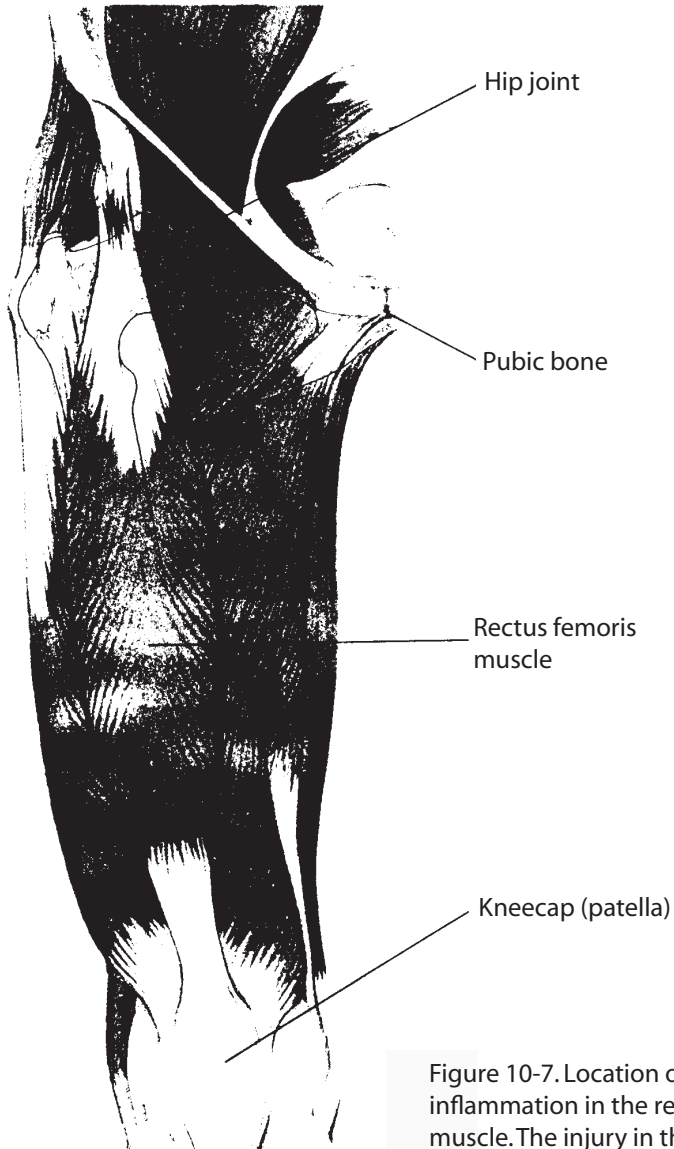


Figure 10-7. Location of a rupture and inflammation in the rectus femoris muscle. The injury in this example is located in the origin of the muscle.

4. Hamstring Pain

Hamstring pain is common in athletes and can result from acute tears, chronic scarring, referred pain from the lumbar spine, or from dural structures.

Hamstring strains are one of the commonest and most disabling injuries to sprinters, jumpers and hurdlers, and accounts for one-third to one-half of the injuries in this group, and up to three-fourths of the rehabilitation time. *Acute strains* occur during rapid acceleration or deceleration, and present as acute pain and a tearing sensation in the hamstring area. The tear occurs at the musculo-tendinous junction, and can be mild or severe, depending on the number of fibres torn. The athlete will have pain on stretching the muscle, local pain that may be high, mid-muscle, or low, and pain on resisted contraction.

The hamstring muscle group consists of the three muscles: the biceps femoris, the semi-membranosus, and the semi-tendinosus. The hamstring muscles are predominantly Type II (fast-twitch) fibres that allow the muscle to respond rapidly in actions such as sprinting. However, Type II fibres are also more subject to fatigue with repeated rapid contractions, and hence more likely to sustain an injury.

The hamstring muscle group crosses two joints, the hip and the knee, and is responsible for four different actions:

- a. Extension of the hip joint in conjunction with the gluteus muscles
- b. Deceleration of knee extension at the end of lower leg forward swing phase, at approximately 30° short of full extension
- c. Stabilise the knee during the stance phase of gait
- d. Assist the gastrocnemius in extending the knee during push-off

Predisposing factors to injury include:

- a. Anatomic-lumbar lordosis
- b. Lack of flexibility
- c. Inadequate warm-up
- d. Inadequate strength and strength imbalances, esp. quadriceps vs. hamstring
- e. Muscle fatigue
- f. Inadequate recovery and rehabilitation post-injury. (“The commonest cause of an injury is a previous injury.”)

Prevention strategies include:

- a. Postural evaluation
- b. Flexibility evaluation
- c. Strength testing
- d. Corrective measures for the above
- e. Proper warm-up and stretching. Keep muscles warm during activity.
- f. Proper conditioning and strength-endurance development

Treatment is through initial PRICES therapy, then good physiotherapy with massage, ultrasound, a good stretching programme, muscle strengthening, and

graduated return to full activity. Mild tears usually require about 3 weeks for recovery, but severe tears may delay return to full activity indefinitely.

Chronic scarring may result from repeated hamstring tears, and can cause pain in the hamstring with running due to entrapment of nerve tissue in the scarred area. An aggressive stretching programme and local deep massage are needed to break down the scar tissue.

High hamstring tendinopathy is a special condition that occurs as an over-use injury among middle and long-distance runners. Patients note a deep aching pain in the buttocks or posterior thigh with high-intensity running. This tendinopathy occurs at the attachment of the hamstrings to the ischial tuberosity. There is fibrosis and hyaline degeneration of the attachment. The fibrosis may entrap part of the sciatic nerve, leading to radicular neuropathic pain in the extremity, and mimic pain of lumbo-sacral origin,

Diagnosis may be difficult, although there is usually tenderness at the hamstring attachment at the ischial tuberosity. An MRI may be needed to demonstrate oedema in the ischium or the tendinous attachment. Treatment involves physical therapy modalities and a stretching and strengthening programme.

Referred pain from the lumbar spine can cause hamstring pain, and can be related to facet joint injury or dysfunction, as well as to spondylolysis, spondylolisthesis, spinal or foraminal stenosis, or disc herniation. Always remember to check the lumbar spine when someone presents with hamstring pain. Treatment of the lumbar spine with manual therapy sometimes relieves the hamstring pain.

Dural adhesion between the nerve roots of the lumbar spine and the dura can cause hamstring pain, and can be due to chronic inflammation around the lumbar facet joints or the associated soft tissue. Tightness of the piriformis muscle can also cause hamstring pain by irritating the sciatic nerve as it passes beside this muscle or through it. Stretches (including “slump stretches”) have been devised to free the adhered dura and help relieve pain.

5. Adductor Strains

Adductor strains are common in hurdlers and most field event disciplines. The adductor muscles can be strained at their origin at the pubic symphysis or further distally in the muscle belly. Other conditions may lead to groin pain, and adductor strains should be differentiated from osteitis pubis, pelvic, or high femoral stress fractures, or sacroiliac joint referral. Adductor muscle strains are tender when palpated at the area of the strain, and cause pain both on stretching and on resisted adduction. They can be treated by PRICES and local physiotherapy as mentioned above. A good stretching programme and appropriate warm up and warm down will help prevent adductor tears.

6. Referred Pain

Referred pain from the lumbar spine should be managed as appropriate for the signs found on examination.

7. Stress Fractures

It is important to accurately diagnose stress fractures around the hip—if they are missed they can cause permanent hip disability.

a. Femoral Neck

Stress fractures of the neck of the femur need immediate non-weight bearing (NWB) rest, either in bed or on crutches, depending on whether there is evidence of fracture on plain X-ray. If the fracture is evident on plain X-ray, then referral to an orthopedic surgeon should be considered for pinning of the neck of the femur to avoid complete fracture and avascular necrosis of the femoral head. If the cortical defect is on the traction side, surgery is most likely required, however those on the compression side often heal with rest.

If the fracture is diagnosed only on scintigraphy or MRI, management should consist of NWB for at least 3 weeks, then only day-to-day activity, without training, for several weeks. Alternative non-weight bearing training, such as water-running, cycling, and a stretching programme can be followed. After the end of 12 weeks the athlete may be able to make a graduated return to full activity.

b. Femoral Shaft

Stress fractures of the upper femur do not put the head of the femur at risk and a more symptomatic programme can be followed. Usually 8 weeks is sufficient relative rest using alternative exercise as listed above, followed by a graduated return to full activity.

c. Pubic Rami

Six weeks is often long enough for pubic rami stress fractures to heal, and basic rehabilitation principles should be followed.

B. Lower Leg Disorders

Exercise-related leg pain in runners accounts for 10–15% of running injuries, and may be responsible for up to 60% of leg pain syndromes. The most common causes of leg pain include:

1. Periostitis (“shin splints,” medial tibial stress syndrome [MTSS])
2. Exertional compartment syndrome
4. Posterior leg disorders: gastrocnemius-soleus strain; achilles tendon syndromes
3. Stress or fatigue fractures

Shin pain in athletes is difficult to diagnose, and the history of pain plays an important part in elucidating these problems.

1. Periostitis

Periostitis is the most common shin injury that runners experience. There is onset of pain early during activity, which may subside with continued activity, although this is variable. Pain may continue following activity. The pain is diffuse

along the anterior, but more likely, the posterior border of the tibia. It may be distinguished from a stress fracture of the tibia by its diffuse nature, as opposed to the localised pain that occurs with a stress fracture.

The major causes of periostitis are biomechanical problems (usually excessive pronation), training errors, inappropriate footwear, or major changes in running surface or terrain. Biomechanical factors need to be corrected, and proper shoes and appropriate orthoses obtained before therapy can be effective. Local massage of the tender areas, combined with ice massage and ultrasound, is the most effective treatment. Rehabilitation should include relative rest during treatment and a graduated return to exercise.

2. Exertional Compartment Syndrome

Exertional compartment syndrome is another cause of calf or shin pain. This condition, in which the sheath around the anterior or posterior compartments is too tight, presents with increasing pain with the onset of running. Pain is not present initially, but over 15–20 minutes the calf or anterior shin pain increases. Pain comes on earlier if the running pace is harder or if the athlete runs hills. The athlete experiences a cramping pain, and cannot usually “run through” it. Diagnosis is by history and by measurement of compartment pressures in the exercising muscles. Surgery is usually required to correct the syndrome. The pain will also subside if the level of training is reduced, but this approach is not appropriate for an elite athlete.

3. Posterior Leg Disorders

a. Gastrocnemius/Soleus Complex

Calf pain can be caused by small or large tears of the gastrocnemius/soleus complex. The medial head of the gastrocnemius is a common area to injure, as is the junction of the Achilles tendon with the calf complex. This can be treated with local physiotherapy and an appropriate stretching regime.

b. Achilles Tendon

Achilles tendinosis is a common problem in athletes. Causes often include training errors or biomechanical factors, but the condition can come on for no apparent reason. There can be inflammation of the paratenon around the tendon with associated thickening (diffuse), crepitus and pain on movement, or on getting out of bed in the morning. The achilles tendon itself can be inflamed with a great deal of local pain on palpation of the tendon. Localised thickening of the tendon can occur, and is often associated with cystic changes in the tendon.

Treatment should include the following: correct poor biomechanics, institute a stretching programme, massage the tendon (as well as other modalities), and oversee a graduated return to full activity. Surgery to strip the tendon sheath, remove the cystic area within the tendon, or repair a partial tendon rupture may be required.

4. Stress Fractures—Tibia

Stress fractures of the tibia, ankle, and foot are a major cause of injury (see Part 1 of this chapter, *Ankle and Foot Injuries*, for further discussion of stress fractures). Bone is being remodeled constantly. Excessive stresses increase bone turnover, but repetitive stresses outstrip the bone's reparative capacity and stress fractures occur. Precipitating factors include rapid increases in training load, poor or worn footgear, training on hard surfaces, and failure to heed the symptoms of an impending fracture. Additional risk factors include low body weight, eating disorders, including the Female Athlete Triad (disordered eating, amenorrhea, and osteopenia), and other poor nutritional practices.

Stress fractures of the tibia are usually located proximally at the tibial flare, and the junctions of the upper and middle thirds, or the middle and lower thirds. Clinical assessment is usually by local palpation and percussion of the injured site and by stressing the tibia by a valgus maneuver or by getting the athlete to jump on the injured leg. These fractures are sometimes seen on plain X-ray, especially if the X-ray is taken after about 2 to 3 weeks post-onset of pain. However, at best there is a 60% discovery rate. Scintigraphy or MRI is the investigation of choice and should always be ordered if there is clinical suspicion but a negative X-ray.

Tibial stress fractures require relative rest and alternative non-weight bearing exercise such as previously described. They typically take between six and eight weeks to heal, and the physician or physiotherapist should work with the coach to plan the athlete's gradual reintroduction to training. Stress fractures of the anterior mid-shaft of the tibia are renowned for delayed union and should be treated very conservatively with rest. They sometimes require bone stimulation and occasionally, bone grafting or drilling.

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KNEE INJURIES

A. External Knee Disorders

1. Iliotibial Band Syndrome

Iliotibial band (ITB) syndrome is an overuse injury that occurs when the iliotibial band repeatedly rubs over the lateral femoral epicondyle (Figure 10-8). The soft tissues in that area become swollen and painful; symptoms are aggravated by further knee motion. Etiology may be due to rapid increases in training, by overtraining, by running on a slanted surface such as a roadside or by running downhill. It can be associated with biomechanical abnormalities such as genu varum, supinated feet and, in some cases, excessive pronation. On palpation there is tenderness over the lateral femoral condyle as the iliotibial band rubs backward and forward over the condyle through about 30° of knee flexion, as is common in running.

Treatment includes local physiotherapy to the area, stretching exercises for the ITB, and attention to biomechanics. Sometimes an adventitial bursa forms below the ITB; this may respond to local injection with corticosteroid. There is rarely any need for surgical intervention, but there are some operations described that may help if conservative measures fail.

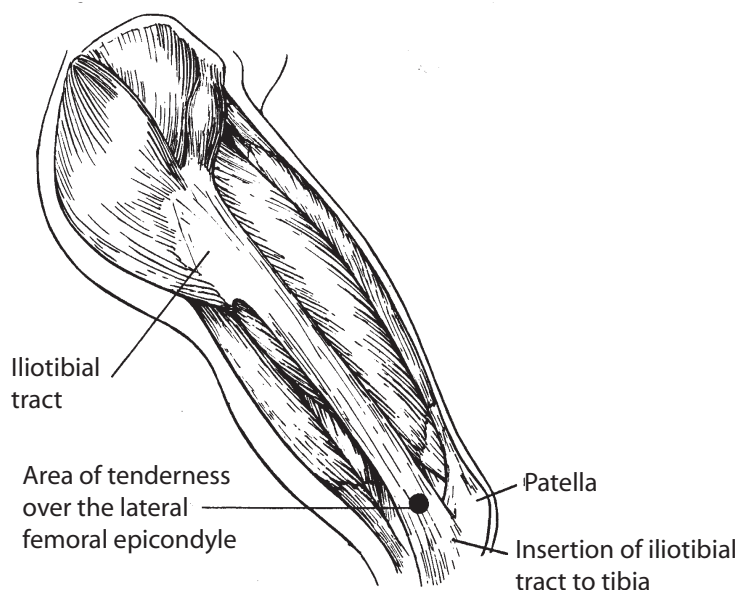


Figure 10-8. Iliotibial band syndrome.

2. Patellar Tendinopathy

Jumpers are especially prone to patellar tendinopathies (Figure 10-9). The injury occurs at the inferior pole of the patella and can be palpated locally with the knee in extension or at 30° of flexion. Associated thickening and crepitus of the tendon and sheath may also occur. Stretching is important in both prevention and therapy, as

quadriceps tightness is an important cause of the injury. In advanced stages, lesions such as cystic degeneration or partial tear within the tendon can occur. These can be seen on ultrasound or MRI, and often require surgical intervention. However, the recovery from surgery takes a long time, although results are usually satisfactory.

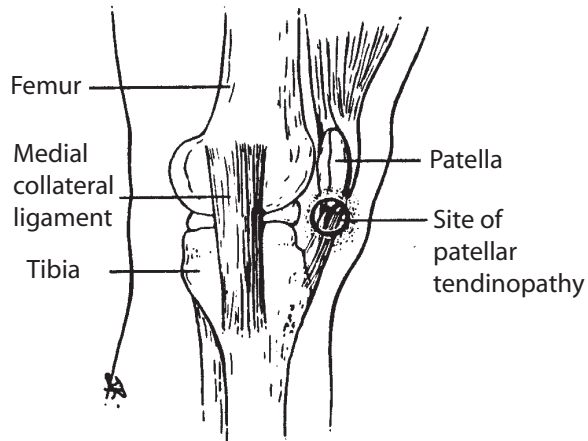


Figure 10-9. Patellar tendinopathy.

3. Patellar Tendon Insertion Inflammation

Inflammation of the patellar tendon insertion into the tibial tubercle can occur from overuse, or be one of the adolescent apophysities. In 12- to 16-year-olds, inflammation of the apophysis called Osgood-Schlatter's disease can occur. It is secondary to traction of the patellar tendon on the immature growth plate. Treatment consists of relative rest, stretching, and gradual increase in athletic activity as the pain subsides.

This injury can also be seen in mature athletes, and is an overuse syndrome similar to proximal patellar tendonitis. Local physiotherapy is required, but surgery or local injection is rarely, if ever, needed.

4. Retropatellar Pain

Retropatellar pain or "runner's knee" is the most common knee problem seen in runners, and is usually caused by training error or poor biomechanics. Pain is often felt deep within the knee and into the posterior knee. It is often worse with climbing stairs or running hills, and after standing up from a sitting position with the knee bent for any length of time ("theatre sign"). Excessive foot pronation is often associated with the condition, and should be looked for and treated.

This condition responds best to treatment regimes described by Australian physiotherapist Jenny McConnell. They consist of stretching the lateral muscles of the quadriceps inserting into the knee and strengthening the vastus medialis obliquus (V.M.O.) muscle to help pull the patella medially. Tape is also used to pull the patella medially or correct rotational abnormalities. Patellar tracking is corrected by

using an exercise programme of limited knee flexion (15° – 20° of flexion), making sure that the patella is aligned over the second toe. Appropriate footwear, with or without orthoses, can correct excessive pronation and therefore tibial torsion. Surgery is rarely indicated for retropatellar pain, unless there is an anatomic defect or cartilage degeneration.

5. Quadriceps Insertional Pain

Quadriceps insertional pain occurs at the superior border of the patella. It is an acutely tender area, and the athlete usually will point to the painful spot. It should be treated with the usual physiotherapy modalities.

6. Pes Anserinus Bursitis

Pes anserinus bursitis presents as pain over the medial tibial flare, caused by inflammation from rubbing of the hamstring tendons over the bursa separating them from the medial tibial flare. This condition can be secondary to excessive pronation, which should be assessed. It will usually respond to local physiotherapy, but may require corticosteroid injection.

B. Internal Knee Disorders

1. Meniscal Lesions (Figure 10-10)

Meniscal tears are much less common than previously thought, and many are associated with an unstable knee in which the anterior cruciate ligament is torn. Meniscal lesions that are associated with a twisting injury are more obvious than insidious cleavage tears of the meniscus. Acute injuries usually involve horn tears or bucket handle tears associated with a specific twisting injury. Horn tears are almost always posterior and are associated with a mild-moderate effusion in the knee joint. Cleavage tears are often degenerative in nature, but can be acute. They are horizontal tears as opposed to the posterior horn tears, which are vertical tears.

Pain may be reproduced in meniscal injury by local palpation over the joint line or by rotational testing of the tibia on the femur. Patients can “duck walk” in the clinic to elicit pain. Both horizontal and vertical tears are best treated with arthroscopic repair or excision, but cleavage tears do not recover from surgery as quickly as horn tears, as a large area of exposed raw meniscus remains after operation.

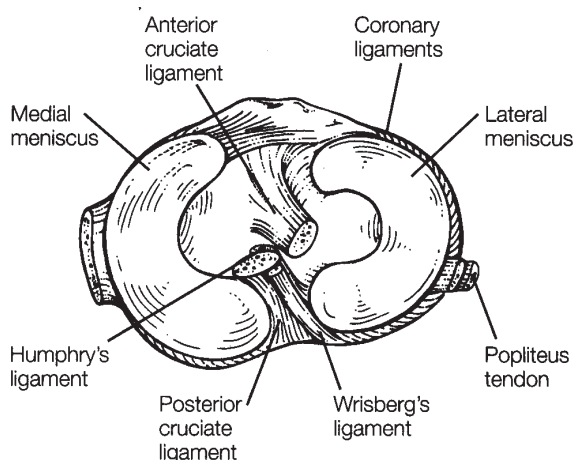


Figure 10-10. Locations of the medial and lateral menisci.

2. Anterior Cruciate Ligament Tears

Acute knee injuries are rare in runners, but not unusual in athletes in the jumping or throwing events. Tears of the anterior cruciate ligament (Figure 10-11) are the most devastating types of injury, and are best diagnosed initially on history of rotational deceleration injury or hyperextension. About 70% of athletes who tear an ACL report feeling or hearing a “pop” in the knee. Swelling may begin immediately or within a few hours. If the knee is tensely swollen with blood, aspiration under sterile conditions will help relieve the athlete’s pain and make examination easier. A meniscus tear may produce a slower effusion—perhaps noticed the next morning.

The knee should be examined for laxity at 90°, but this is not as sensitive in diagnosing isolated anterior cruciate tears as the Lachman test. This test is performed with 10°–20° of flexion and feeling for increased anterior draw.

The pivot shift test is used to detect anterior lateral rotary instability. The patient is placed in a supine position and relaxed. The knee is examined in full extension. The tibia is internally rotated, with one hand grasping the foot and the other hand applying mild valgus or abduction stress at the level of the joint. Then, with flexion in the knee at approximately 20°–30°, a jerk is suddenly experienced at the anterior lateral corner of the proximal tibia. This shift is the anterior lateral subluxation of the lateral tibial condyle. A positive test is indicative of ACL injury.

Most athletes will require ligament reconstruction using either a hamstring tendon or the patella tendon as a graft after the initial swelling has subsided.

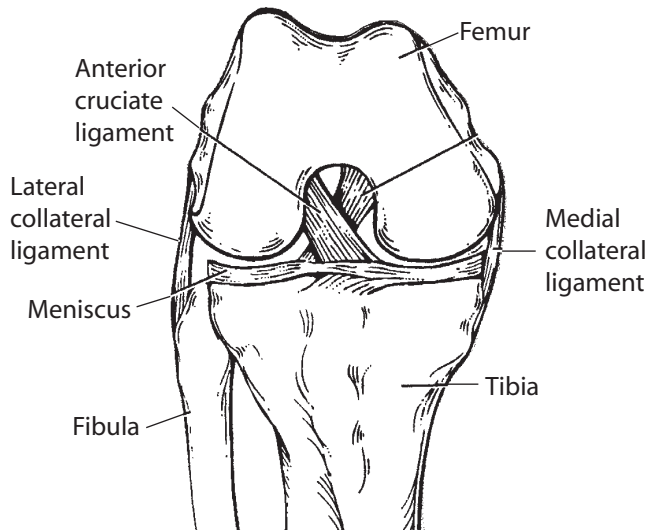


Figure 10-11. Anterior view of the right knee in flexion, showing the location of the anterior cruciate ligament.

Rehabilitation involves regaining the range of motion initially and reducing the fluid in the knee. As movement becomes pain-free, the athlete will be able to run in a straight line and cycle. The next aim is to increase proprioception in the knee; this is done by balancing on a tilt or wobble board. Retraining the hamstrings is paramount in rehabilitation, as they can compensate for the disability since they attach to the upper tibia. A planned resistance training programme is needed, primarily to strengthen the hamstrings but also, to a lesser extent, the quadriceps. Along with this, the athlete should be taught “catch kicks” to train the hamstring to contract quickly.

3. Posterior Cruciate Injuries

These injuries occur when the athlete falls directly onto the knee or hyperextends it, and are usually managed conservatively using basic strength rehabilitation principles. There are problems with retropatellar pain following this injury, and quadriceps strengthening is important. Be aware of associated fractures of the tibial plateau and refer these for orthopedic management.

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SPINAL INJURIES

A. Causes of Spinal Injuries

Track and field training and competition create many chances for extreme and possibly injurious spinal stresses. Postural stress can cause general and specific aches and pains, and through accommodation of joint and soft tissue structures, result in dysfunction. Lifting in weight training, throwing weighted implements, and spinal torsion and compression caused by pole vaulting, jumping, hurdling, and running can all cause acute or chronic back syndromes. Precipitating factors include:

1. Sitting Posture

A good sitting posture maintains the spinal curves normally present in erect standing posture. Poor sitting posture reduces or accentuates the normal curves enough to stress the ligamentous structures and induce pain. A poor sitting posture can produce pain to the back itself without any additional stress or injury. An athlete suffering from low back pain can experience increased pain from sitting or rising from sitting. When an individual sits in a chair for a few minutes, the lumbar spine assumes the fully flexed position, in which the muscles are relaxed and the weight bearing stress is absorbed by the ligamentous structures. An increase in intradiscal pressure occurs as the spine moves toward the flexed position in sitting, and decreases as the spine moves into extension.

2. Lack of Postural Extension

Another predisposing factor to low back pain is the loss of lumbar extension. A loss of spinal extension influences the athlete's posture in sitting, standing, walking, and running. From faulty postural loading, the spine undergoes adaptive changes.

3. Frequency of Flexed Position

The majority of activities that an individual performs occur in the flexed position. Theoretically, this produces stress on the annular wall and causes the fluid nucleus to move posteriorly.

4. Unexpected and Unguarded Movements

Unexpected and unguarded movements in track and field may cause an acute episode of low back pain. Throwers and jumpers often experience muscular strains or ligamentous sprains. In attempts to reduce low back pain episodes, it is necessary to examine and advise each athlete regarding the precipitating factors involved.

5. Lifting

Lumbar intradiscal pressure has been shown to increase with lifting movements from a forward bent position. Maintaining a functional neutral position (an individual's functional range between flexion and extension) and lifting with bent knees aids in symptom-free lifting. Correct lifting and throwing techniques are vital in preventing back injuries.

B. Evaluating Back Injuries

Assessment of back pain should involve a thorough history and evaluation. One should understand the athlete's subjective complaints and comments, and determine the area of symptoms, as well as the severity and nature of the symptoms. The evaluator should also determine whether the symptoms are constant or intermittent, and what positions or movements provoke the pain. Objective evaluation of movement testing to reproduce the symptoms, as well as a neurologic evaluation (if indicated) should be performed. Back pain of mechanical origin can be classified as one of three syndromes:

1. Postural Syndrome

Pain of postural origin is intermittent and appears when soft tissues surrounding the lumbar joints are placed under prolonged stress. Upon evaluation, inspection and lumbar range of motion is normal. Postural assessment generally indicates poor sitting and standing posture; treatment should work to correct posture, strengthen muscles if any weakness is found, and stretch tight structures.

2. Dysfunction Syndrome

Dysfunction syndrome occurs when adaptive shortening and resultant loss of mobility cause pain before gaining a full range of motion. Adaptive shortening and loss of mobility can result from poor postural mechanics, spondylosis, trauma, or disc derangement. Treatment should emphasise lengthening of the shortened tissues and improving range of motion.

3. Derangement

Disturbance of the intervertebral disc mechanism is responsible for the most disabling cause of mechanical low back pain. The actions of the disc have been described and documented by various authorities to explain the relationship of the disc and increased pain upon movements. Minor disc bulging may cause deformity and limitation of movement, and certain movements of the spinal column increase the bulge while others may reduce it. Shifting the fluid nucleus of the disc may also disturb annular material. A herniated nucleus pulposus may cause nerve root compression, radicular symptoms, and altered neurological findings.

C. Treatment and Rehabilitation

After the potential stresses and the structures are identified, a plan of treatment may include back education with a review of proper back mechanics, and assessment of any faulty mechanics present while executing the athlete's specific skill; modality intervention; and mobilisation and exercises to achieve pain relief and regain function. A plan of back care may be a progression of self treatment and management for each individual, depending on the spinal injury.

The primary treatment aim is restoration of normal painless joint range by:

1. relief of pain and reduction of muscle spasm
2. restoration of normal tissue-fluid exchange, soft tissue extensibility, and normal joint relationship and mobility
3. correction of muscle weakness or imbalance
4. restoration of adequate control of movement and stabilisation
5. relief from chronic postural stress
6. functional return for the athlete
7. prevention principles to avoid recurrence
8. restoration of the athlete's confidence

The order of importance for goals will differ with each individual. The philosophy of treatment and rehabilitation of specific back injuries may differ depending on the health care deliverer's educational and clinical background and experiences, as well as the treatment and rehabilitation techniques which have proven successful for that individual. Self treatment should emphasise the principles of postural correction, repeated extension or flexion movements, use of lumbar aids and supports, and use of various local treatment modalities such as cryotherapy, or heat application. Other treatment may utilise electrical stimulation, traction, acupressure/acupuncture, medical intervention of local injection or oral analgesic/or anti-inflammatory drugs, techniques of joint mobilisation and manipulation, muscle energy techniques for regaining muscle balance, PNF (proprioceptive neuromuscular facilitation), mobilisation techniques for soft tissue and nerves (see Chapter 8, Part 3, *Principles of Rehabilitation of the Injured Athlete*, for more detail). Functional lumbar stabilisation progression and functional training may be integrated as one or in combination depending on the athlete's deficits and the goals of treatment and rehabilitation.

The lumbar spine has optimal positions in which it functions most efficiently and these positions vary depending on the stresses it must withstand. There is no one best position for all functional tasks and activities, and it will vary from athlete to athlete. A good functional position is generally near the mid-range of all available movement of the lumbar spine, and the athlete must learn good lumbopelvic control. For the athlete to learn to maintain the low back within a functional range, he or she must develop a kinesthetic sense in order to feel and control back movements and positions so that it becomes a habit during all activities, and the athlete must maintain this coordination, strength, mobility, and endurance to perform well.

Management in preventing recurrences of back injury for the track and field athlete begins with an understanding of what they do, how they do it, and the cause of injury. The basic principle is to avoid extreme positions or extreme stress for long periods, and continue with preventative/maintenance exercises for range of motion, muscle flexibility, strength, and power.

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UPPER EXTREMITY INJURIES

Upper extremity injuries usually occur in the shoulder or elbow of javelin, discus, or hammer throwers or shot putters. Overuse (too much too soon), biomechanical imbalance due to improper technique, or failure to completely rehabilitate a prior injury are the most common causes. Runners may injure an upper extremity due to a fall, collision, or other accident. More injuries occur in training than in competition.

The entire body is important in maximising optimal performance and in preventing injuries of the upper extremity. Hip-shoulder orientation and trunk position change throughout the throw. A shot putter must support the heavy shot with his or her fingers while the large scapular anchoring muscles must slow the arm after the shot has been released. Discus and hammer throws are centrifugal motions that produce fewer shoulder problems than do overhead events.

A. Shoulder Injuries

Throwing actions place a significant demand on the shoulder complex, requiring a precise, coordinated effort to create velocity and accuracy. The most common injuries in throwers include rotator cuff tendinitis due to overuse and eccentric overload, subtle instabilities, labral degenerative changes and tears, and secondary subacromial and parascapular pathology.

1. Rotator Cuff Tendinopathy and Impingement Syndrome

Rotator cuff tendinopathy is the most common cause of shoulder pain in throwers. The rotator cuff muscles are the supraspinatus, infraspinatus, teres minor and subscapularis (Figure 10-12). Also making up the functional unit of the rotator cuff are the long head of the biceps brachii tendon, the acromion process, the coracoacromial ligament, and the acromioclavicular joint.

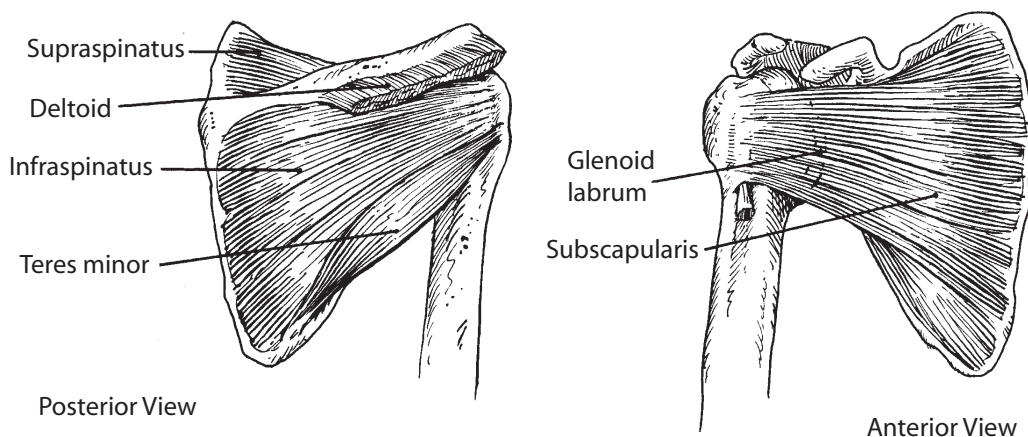


Figure 10-12. Rotator cuff muscles and insertions.

The rotator cuff's three primary functions are humeral head depression, active external rotation of the shoulder, and dynamic stability of the shoulder. The rotator cuff maintains the articulation of the humeral head within the glenoid, thereby supplying an effective fulcrum across which the power muscles of the shoulder (deltoid, pectoralis major, and latissimus dorsi) act to elevate the arm and permit active use at and above the shoulder level (Figure 10-13). The posterior rotator cuff muscles, the infraspinatus and teres minor, are the principal external rotators of the shoulder. The surface of the humeral head is three or four times larger than the glenoid. The rotator cuff muscles provide dynamic stability of the glenohumeral joint while the glenohumeral ligaments provide static stability.

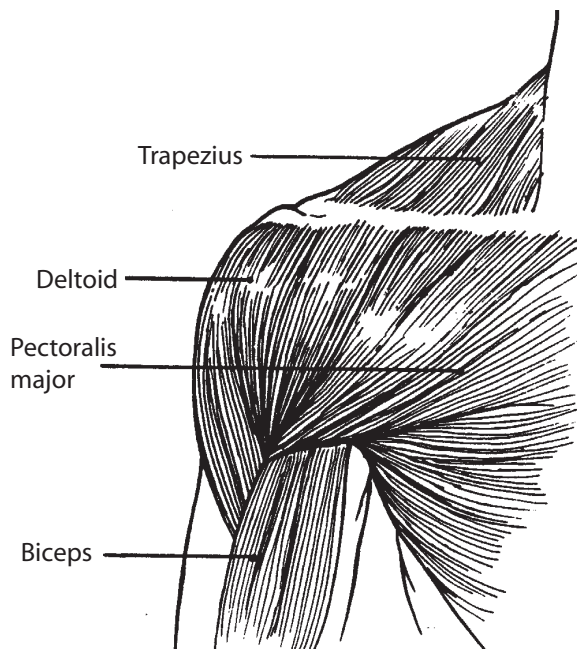


Figure 10-13. Major shoulder muscles.

Rotator cuff tendinitis can occur in several ways. If the glenohumeral joint static stabilisers fail to contain the humeral head, the rotator cuff must substitute by eccentrically contracting. Increased muscle load leads to early fatigue, eccentric overload, and inflammation. The supraspinatus muscle and tendon play the largest role in head depression during shoulder abduction and therefore are the site of most common overuse injuries. When fatigued, the shoulder no longer resists superior head translation, leading to impingement in the subacromial space. The infraspinatus, teres minor and posterior deltoid externally rotate the shoulder, while the pectoralis major and latissimus dorsi assist the subscapularis with internal rotation. Inequality in strength of internal and external rotation may lead to tendinitis. Weakness of the external rotators while the shoulder is decelerating in the follow-through may lead to fatigue and then tissue damage, especially of the infraspinatus tendon.

Impingement syndrome is a symptom complex initially described and classified by Neer (1972). The primary symptom is anterior shoulder pain which sometimes radiates laterally to the deltoid insertion and is exacerbated by activities at shoulder level or above (Figure 10-14). The functional arc of elevation of the shoulder is in the frontal, not lateral plane. Mechanical impingement of the rotator cuff against the anterior aspect of the acromion and the coracoacromial ligament may lead to inflammation and subsequent tears of the rotator cuff tendons. Neer classified three stages of the impingement syndrome as: 1) oedema and hemorrhage in the subacromial space; 2) thickening and fibrosis of the subacromial bursa; and 3) full thickness rotator cuff tears. Instability of the glenohumeral joint may lead to impingement due to abnormal anterior humeral head translation secondary to the laxity.

When the arm is lifted upward and outward (abduction) at an angle of 70°–80° to the body, the bursa and the injured tendon may be trapped against the lower edge of the shoulder blade and against the coraco-acromial ligament, causing pain. The condition is called trapping or impingement syndrome.



When the arm is further lifted upward and outward at an angle greater than 120°, the bursa and the injured tendon slide under the acromion process of the shoulder blade. This releases the pressure, and therefore the pain, on the tendon.

Figure 10-14. Impingement syndrome.

Examination of a patient with shoulder impingement usually reveals a painful arc from 80° to 140° abduction in forward flexion. Common impingement tests are: 1) forced passive stretching and extreme forward flexion of the shoulder with the forearm pronated; 2) forced internal rotation at 90° of forward flexion; 3) extreme horizontal adduction (cross-over test). Injecting the subacromial bursa with Lidocaine should significantly decrease or eliminate the pain with the impingement test.

2. Shoulder Instability

Shoulder instability is defined as excessive symptomatic displacement of the humeral head in its relationship to the glenoid fossa. Subluxation is the partial loss of joint congruency. Instability may result from acute trauma or chronic repetitive stresses, which slowly elongate capsular restraints. Acute dislocations are usually the result of a fall, while throwers may acquire shoulder instability over a period of time.

An athlete with a current dislocation frequently presents holding his or her arm by the opposite hand in slight adduction and external rotation. The acromion may be prominent and there is a sharp contour of the affected shoulder compared to the smooth deltoid outline of the uninjured shoulder. The shoulder should be reduced and immobilised.

When the instability is due to chronic repetitive stresses, the history will be less obvious. The symptoms may often be vague and the diagnosis difficult to make. The pain is sometimes poorly defined but may be worse with horizontal adduction and external rotation. There may be a vague feeling of instability or apprehension with overhead movement.

Examination of the patient with subtle shoulder instability may reveal that the range of motion is grossly equal in both shoulders. Most likely there will be some apprehension with the shoulder in 90° of abduction and maximal external rotation. The “relocation test” described by Jobe and Kvitne (1990) may be positive. This is performed with the patient lying supine and the shoulder abducted 90° and maximally externally rotated. Anteriorly directed forces on the proximal humerus should cause pain and a sensation of anterior subluxation which is relieved by posterior pressure on the anterior aspect of the humerus. Testing for posterior instability can be done with the arm at shoulder level and adducted approximately 30° while applying posterior-directed force to the humeral head. X-rays may reveal a Hill-Sach’s lesion, which is a defect of the humeral head due to compression of the head against the glenoid while the humerus subluxes. A CT/arthrogram may be needed to rule out a rotator cuff tear. Complex multi-dimensional instabilities may be difficult to diagnose.

3. Stress Fracture

A stress fracture of the proximal humeral physis or osteochondritis is common in the athlete with an immature skeleton. Repetitive stress caused by torque during the acceleration phase of throwing while the arm accelerates forward and rotates internally may lead to tendinitis in adults and stress fractures in youths. The strong young athlete’s shoulder flexors, internal rotators, and adductors can exert tremendous force on the proximal humeral physis.

Young athletes with stress fractures usually present with pain produced by throwing. There may be focal pain over the deltoid insertion and perhaps the general rotator cuff without any instability or impingement signs. The pathognomonic radiographic finding is widening of the proximal humeral physis compared to the normal shoulder.

Treatment should consist of possibly limited immobilisation, ice, and physical therapy, with a strength programme beginning at 4 weeks and perhaps return to throwing in 8 weeks.

B. Evaluation Guidelines

1. Differential Diagnosis

It is important, although sometimes difficult, to make a definitive diagnosis when a shoulder injury occurs. History and examination of shoulder injuries should rule out other pathology as a referred source of shoulder pain. Cervical injuries, including degenerative joint disease and radiculopathies, may refer pain into the upper extremities. Pain may also be referred from the thoracic spine and the viscera. Other possible causes are thoracic outlet syndrome, brachial plexus injury, subclavian artery occlusion and peripheral nerve entrapment, or myofascial pain syndrome.

2. Examination

The shoulder examination should begin with observation of both shoulders relaxed and contracted. Any asymmetry, muscular atrophy, effusions, erythema, ecchymosis, winging of the scapula or lateral glide or obvious dislocation or separation should be noted. Any crepitus or popping during the examination should be noted. A screening examination of the cervical and thoracic spine should be performed and a more detailed examination of these areas done if history or examination warrants.

Range of motion testing should include active, passive, resistive, and functional. The examination should include the impingement instability test.

Imaging, which may be indicated, may include x-ray, CT/arthrogram, magnetic resonance or bone scan.

3. Treatment

Treatment should be diagnosis specific but may include relative rest, immobilisation, physical therapy, ice, anti-inflammatory medication, spontaneous reduction, surgery, and correction of biomechanical imbalances.

C. Elbow Injuries

1. Valgus Overload Syndrome

Valgus overload syndrome is one of the most frequent and significant elbow injuries in javelin throwers. This syndrome is caused by a combination of *medial tension overload* and *lateral compression overload*. The javelin should be thrown overhead with elbow extension; incorrect “round arm” throws lead to valgus overload.

a. Medial Tension Overload

A great deal of force is exerted on the flexor and pronator attachments at the medial epicondyle when the arm is abducted and externally rotated in the

throwing motion. Repetitive traction stress results in micro-tearing of the tendon or of the muscle fibres near the epicondyle. The bony attachment is stronger than the tendon or muscle fibres, and an ulnar traction spur with focal calcification in the tendon substance may develop. In an immature athlete, repetitive stress may cause separation of the epiphysis.

b. Lateral Compression Syndrome

Strong compressive forces to the lateral joint of the elbow associated with valgus stress may lead to damage of the radial head, capitellum, or both (osteochondral fracture and even loose body may result). Symptoms include lateral elbow pain with activity, and catching or locking. Signs would include a tender radiocapitellar joint, lateral swelling, or crepitus with forearm pronation-supination. X-rays may reveal a loss of the radiocapitellar joint space with marginal osteophytes or loose bodies. If symptoms are not resolved with rest and anti-inflammatory medication, treatment may include joint debridement with removal of marginal osteophytes and loose bodies.

2. Ulnar Nerve Damage

Chronic over-stress at the medial elbow can cause ulnar nerve damage due to chronic inflammation or chronic stretching. The ulnar nerve may become inelastic or mobile and be compromised due to formation of fibrous scar tissue.

3. Joint Degeneration

The articulator surfaces of the radius and capitellum are subjected to compressive and rotational loading. Throwers can develop loose bodies in the lateral compartment, as well as osteocartilaginous fragments or marginal spurs. Osteochondritis dissecans of the capitellum may occur in the immature athlete. Extension overload can strain the triceps at the musculotendinous junction. The combined effects of excessive medial traction, lateral compression, and extension overload can result in excessive joint degeneration.

4. Bicipital Tendinitis and Rupture

Another frequent elbow overuse syndrome is bicipital tendinitis due to excessive elbow flexion and supination. Symptoms include anterior elbow pain with flexion and supination, and weakness secondary to pain. One must rule out a partial or complete biceps tendon rupture, a brachialis muscle tear, an anterior capsule tear, or lateral antebrachial cutaneous nerve compression syndrome. Approximately 97% of biceps tendon ruptures occur at the proximal aspect; only 3% occur distally. Male athletes over 30 years of age who have been treated with corticosteroid injections are most likely to sustain a bicipital tendon rupture.

5. Median Nerve Compression Syndrome

Median nerve compression syndrome (pronator syndrome) may occur due to mechanical compression by hypertrophied muscle or aponeurotic fascia. This may initially be misdiagnosed as lateral epicondylitis, because there is significant lateral

supracondylar pain with pain in the anterior proximal forearm. Cramping may occur. There may be numbness in the volar forearm or radial 3 1/2 digits and thumb. There should be a positive Tinel's sign at the proximal forearm, but negative Tinel's sign at the wrist and a negative Phalen's sign at the wrist. Resisted palmar flexion of the middle finger may produce pain at the medial elbow rather than the lateral. Surgical decompression may be necessary if rest, modified training, and physical therapy do not resolve it.

6. Triceps Tendinitis and Fracture

Posterior elbow pain may be a sign of triceps tendinitis due to overload of the triceps by repetitive extension. X-rays may be normal but may demonstrate degenerative calcification, hypertrophy of the ulna, or triceps traction spur. Differential diagnosis should include bursitis or a stress fracture of the olecranon. A triceps fracture may occur with or without olecranon avulsion.

7. Lateral Epicondylitis

Throwers often experience lateral epicondylitis due to overload of throwing or weight training. The athlete will have pain over the lateral epicondyle, and frequently over the radial head and extensor tendons of the forearm. Resisted dorsiflexion of the wrist should increase the pain.

D. Wrist Injuries

1. Tendinitis

Tendinitis of the wrist and fingers occurs in athletes who must repetitively flex and extend their wrist. Tenosynovitis and ganglion cyst may occur. Overuse injuries of the fingers are relatively rare but may occur.

2. Wrist Fracture

A carpal navicular or scaphoid fracture is one of the most frequently missed fractures in the athlete. Any athlete who has tenderness to palpation of the anatomic snuff box between the extensor tendons of the thumb just distal to the radius should be treated as if the scaphoid is fractured even if the initial X-rays are negative. Repeat films 10–14 days later may show the fracture line as bone resorption progresses. Appropriate immobilisation for this fracture is a thumb spica cast. Surgery may be needed if there is a non-union.

3. Carpal Tunnel Syndrome

Carpal tunnel syndrome may occur due to repeated flexion and extension of the wrist. The median nerve may be entrapped. The athlete may complain of numbness, tingling, and pain, primarily in the thumb and three radial fingers. Retrograde pain and paresthesia may occur. Tinel's sign and Phalen's sign should be positive. (Tinel's sign is tapping the carpal tunnel to reproduce the tingling sensation. Phalen's sign is forced volar flexion of the wrist for 90 seconds or more to reproduce the tingling.) Significant weakness of the adductor pollicis brevis and opponens pollicis

muscles may occur. Electromyography and nerve conduction velocity tests may be needed for positive diagnosis. Treatment should include splinting, nonsteroidal anti-inflammatory medication, and physical therapy. In some cases, surgical decompression may be needed.

4. de Quervain's Syndrome

Stenosing tenosynovitis of the first dorsal compartment of the wrist is called de Quervain's syndrome. There may be pain and swelling of the abductor pollicis and extensor pollicis brevis. Finkelstein's test is positive. (The patient should tuck his or her thumb inside the other fingers while the physician moves the fist into ulnar deviation; a positive test is pain in the tendons where they cross the distal radius.) Treatment should include rest with a thumb spica splint, physical therapy, followed in some cases by a corticosteroid injection.

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HEAT AND COLD

A. Thermal Regulation and the Human Body

1. Human Body Temperature

The human body's core temperature varies from day to day, and from time to time, but these fluctuations are small, usually no more than 1.0°C. Humans are homeothermic and body temperature is regulated at about 37°C +/- 1°C. The thermoregulatory center in the hypothalamus plays a very active role in keeping body temperature in the normal range.

External (*climatic*) and internal (*metabolic*) heat sources influence body temperature. Heavy exercise, illness, and not only hot and humid but also cold and windy environments alter body temperature outside the normal range. Ambient temperature, humidity, air movement, and radiant heat from the sun, as well as warm and cold surfaces, contribute to climatic heat stress. Metabolic heat is produced by exercise.

Body temperature reflects a careful balance between heat production and heat loss. There is a continuous heat exchange between the body and the environment. Bi-directional routes for heat exchange are: convection (Cv), conduction (Cd), and radiation (R). There are also two uni-directional routes: metabolic heat (M) increases the thermal load; evaporation (E) decreases the load. The net heat storage (S) formula is:

$$S = M +/- Cv +/- Cd +/- R - E$$

When the net heat storage (S) is positive, body temperature will rise and when S is negative, it will go down.

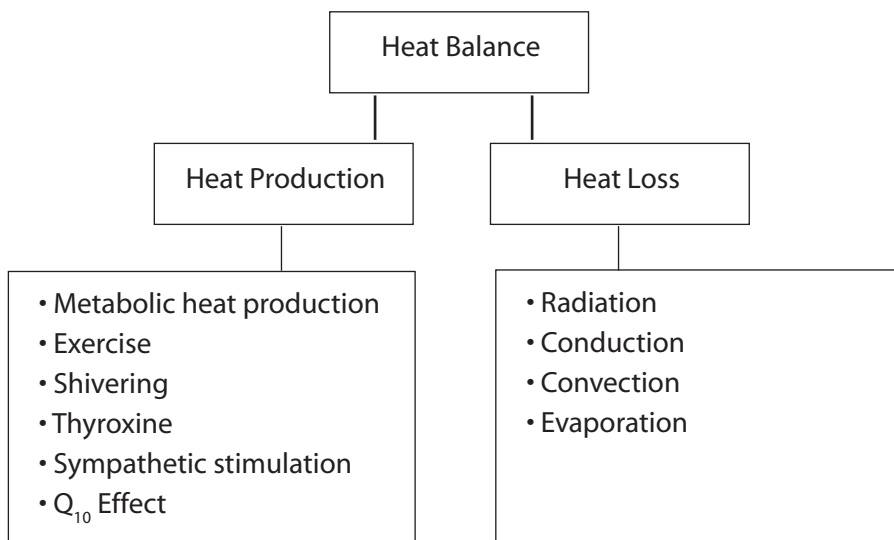


Figure 11-1. Heat exchange between the body and the environment.

2. Climatic Heat Stress

Air temperature alone is not an accurate index of climatic heat stress. Temperature and other important factors, including radiant energy, wind velocity and humidity, contribute to climatic heat stress. To determine the overall effect of these factors, a combination of measurements must be made. The Wet Bulb Globe Temperature (WBGT) index has been introduced to assess climatic heat stress. It consists of three thermometers: dry bulb (Tdb), which measures air temperature; wet bulb (Twb), which measures relative humidity; and black globe (Tg), a measure of solar radiation. The difference between the wet and dry bulb temperatures indicates the environment's capacity for cooling by evaporation. A very modern, simple and handy apparatus to measure the WBGT is available.

The WBGT provides a single temperature reading to estimate climatic heat stress and cooling capacity of the surrounding environment.

The Wet Bulb Globe Temperature (WBGT) =

$$(0.7 \times Twb) + (0.1 \times Tdb) + (0.2 \times Tg) \quad (\text{outdoors})$$

$$(0.7 \times Twb) + (0.3 \times Tdb) \quad (\text{indoors})$$

The importance of relative humidity is indicated by its 70% contribution to the WBGT.

The WBGT is the basis for recommendations by the American College of Sports Medicine (see Appendix 4, *ACSM Position Statement: Heat and Cold Illnesses During Distance Running* and Appendix 5, *IAAF Policy on Fluid Replacement*) for exercise under various environmental conditions for the fit general population. Highly trained elite athletes often are able to exceed these limits, but with appropriate cautions.

3. Metabolic Heat Stress

A large part of the energy that the body generates is degraded to heat. All body tissues produce heat that can be used to maintain body core temperature. When heat production exceeds the body's heat loss, body temperature rises. Intense exercise may increase the metabolic energy expenditure 20–25 times over resting levels. No more than 25% of this energy is utilised for muscular movement—the remainder is heat, which the body must dissipate. Metabolic heat is transferred by convection from working muscles to the blood stream, and thence to the body's core. Without adaptive mechanisms, even moderate exercise would elevate body temperature by 1°C every 5–6 minutes; thus, exercise would be limited to no more than 20–30 minutes before heat stress fatigue or life-threatening hyperthermia intervened.

In fact, the body is able to respond to a heat load through a variety of physiologic mechanisms: sweat rate, body and skin blood flow shifts, cardiac output, respiratory rate, and a sensation of heat intensity. Well-trained endurance athletes can sustain a core temperature of 39°–41°C for prolonged periods. The muscles' energy systems become more chemically effective with a small rise in muscle temperature. However, a critical thermal maximum is reached at 42°C (108°F), so there is a limited safety margin at intense levels of exercise. Heavier athletes run a higher risk of overheating than lighter athletes when exercising at the same rate.

a. Physiologic responses

Core temperature is sensed by the hypothalamic thermoregulatory center. This center then sets off a number of circulatory adjustments in an effort to dissipate heat. Primarily this involves increasing cardiac output and redistributing blood from the visceral organs to the working muscles and skin.

During heat stress, skin blood flow may be up to 20-fold higher than at rest level. Sweat glands become more active for increasing evaporative heat loss.

Four mechanisms are responsible for heat exchange at the skin surface:

- i. **Conduction** has a minimal effect on body heat transfer, as it depends upon direct contact between skin and a cooler object. Conduction could be utilised by immersion in water to cool or warm the body.
- ii. **Convection** is responsible for transferring heat from working muscles and the skin surface. The circulatory system transports the heat generated in the active muscles to the surface of the body. The air around the body is in constant motion and it sweeps away the warmed air molecules on the skin surface. It is dependent upon (a) the temperature differential between skin and the environment; and (b) the heat transfer coefficient, which varies with available body surface area and wind velocity. Minimal body fat and loose-fitting clothing enhance an athlete's convection potential.

Conduction and convection constantly remove body heat when air temperature is lower than skin temperature. Inversely, convection and conduction cause heat gain in a very hot environment.

- iii. **Radiation**, including solar radiation and radiant heat from tracks, roads, and surrounding structures, can be a major contributor to an athlete's heat load. At rest, radiation is the primary method for discharging the body's excess heat. The heat is given off in the form of infrared rays. Radiation heat loss or gain depends upon the temperature gradient between the skin and the environment.
- iv. **Evaporation** is the most important heat dissipation mechanism in warm environments. Though it accounts for only 20% of body heat loss at rest, more than 80% of body heat loss is achieved by evaporation when environmental temperature exceeds 20°C (68°F) (Table 11-1). Fit athletes can produce up to 30 ml of sweat per minute, but not all of this is available for heat elimination. Evaporative rate is determined by air velocity and the water vapor pressure gradient between the skin and the environment. This latter is determined by the **relative humidity** of the air. High humidity limits sweat evaporation and therefore, heat loss. Each liter of effective evaporated sweat removes 580 Kcal from the body. Evaporation plays a very important role in dissipating heat during exercise.

Table 11-1. Estimated heat loss at rest and during exercise at 70% VO_2 max.

Mechanism of heat loss	Rest		Exercise	
	% total	Kcal/min	% total	Kcal/min
Conduction and convection	20	0.3	15	2.2
Radiation	60	0.9	5	0.8
Evaporation	20	0.3	80	12.0

4. Acclimatisation to Heat and Maximising Heat Loss

Partial adaptation to heat stress occurs even with training at moderate temperatures, but full acclimatisation can be achieved only with repeated bouts of exercise in the heat. At least five to ten days of training in the heat are required for full acclimatisation. This should begin by training at a reduced intensity (60–70% of the usual load), so as to avoid heat-related disorders.

There are numerous adaptations that occur as a result of acclimatisation to exercise in the heat:

- a. Sweat rate in the skin areas exposed to heat is higher, thus enhancing evaporative capacity. An increased production of aldosterone can strongly stimulate the sweat glands and cause them to reabsorb more sodium and chloride.
- b. Earlier onset of sweating leads to a lower skin temperature, improved core-skin temperature gradient, and less demand for blood flow to the skin. This latter provides improved muscle blood flow and oxygen supply.
- c. Plasma volume is increased, due to an increased production of aldosterone and antidiuretic hormone (ADH). Aldosterone causes sodium and chloride retention by the renal and sweat tubules, and ADH increases renal water retention. Hence, there is a decrease in sweat sodium and chloride, but not potassium. More sodium is retained, which promotes water retention. Plasma and interstitial fluid volumes can increase 10–20%.
- d. Body core temperature can be kept lower, as heat dissipation is more efficient.
- e. Heart rate is lower at any given work load, as the core temperature is lower, plasma volume is higher, skin blood flow distribution is decreased, and there is improved stroke volume.
- f. The perception of heat stress is reduced.
- g. Onset of fatigue is delayed, as the rate of muscle glycogen utilisation is decreased.

Men and women acclimatise equally well. Training in a hot, humid environment is more stressful than training in hot, dry conditions.

5. Cold Exposure Thermoregulation and Minimising Heat Loss

The body's hypothalamic set-point for temperature regulation is about 37°C +/- 1°C. A decrease in skin or core temperature signals the thermoregulatory center in the posterior hypothalamus to set off a number of mechanisms to increase heat production. These include:

- a. Shivering—involuntary muscular contractions in response to cold. This can cause a 4–5 fold increase in heat production. Shivering results in decreased muscular coordination and impairs performance.
- b. Non-shivering thermogenesis. The sympathetic nervous system releases epinephrine and nor-epinephrine in response to cold exposure, causing anaerobic glycolysis and a release of free fatty acids from fat stores. This mechanism occurs in young children because of their rich brown fat.
- c. Increased thyroxin production. Hypothalamic thyrotropin-releasing hormone (TRH) rises, stimulating TSH release and ultimately elevated thyroxin production to increase general metabolic rate.
- d. Peripheral vaso-constriction. The sympathetic nervous system stimulates skin's smooth muscle contraction, thus shunting blood away from the skin and into deeper tissues.

The balance between heat loss and heat production is controlled by a number of factors. Generally, the greater the gradient between skin and environmental temperature, the greater the heat loss. However, a great number of anatomic and environmental factors affect the rate and degree of heat loss. For example, body size and body composition influence heat loss. Subcutaneous fat acts as an insulator. Smaller athletes such as children have a higher surface area/mass ratio, and may sustain greater heat loss. Clothing helps reduce heat loss.

The degree of heat loss also depends on air movement (convection), humidity, evaporation (sweating), and ambient temperature. Wind velocity exacerbates heat losses from convection, radiation, and evaporation. This is known as the Wind Chill Effect, and is expressed as the Wind Chill Factor (Table 11-2).

Exercise in the cold can affect muscle function. Muscle functions best at a temperature of 40°C. Cooling alters the nervous system and the muscle fiber's recruitment pattern. Muscle shortening velocity and power decrease when the temperature is lowered. Muscle glycogen utilisation is higher during exercise in the cold. Epinephrine and nor-epinephrine secretion increases markedly. However, FFA may not rise as much as in a normal environment, as blood flow to subcutaneous fat areas is lower, and less FFA are mobilised.

So long as clothing is adequate and metabolic rate remains high, the body temperature and function can be maintained. However, as fatigue develops and exercise intensity (i.e., race pace) slows, heat production declines and hypothermia may develop.

Table 11-2. Wind chill index.

		Thermometer Reading °F (°C)										
		50 (10)	40 (4.4)	30 (-1.1)	20 (-6.7)	10 (-12.2)	0 (-17.8)	-10 (-23.3)	-20 (-28.9)	-30 (-33.3)	-40 (-40)	-50 (-45.6)
Wind speed (mph)	Equivalent Temperature (°F)											
5		48	37	27	16	6	-5	-15	-26	-36	-47	-57
10		40	28	16	4	-9	-24	-33	-46	-58	-70	-83
15		36	22	9	-5	-18	-32	-45	-58	-72	-85	-99
20		32	18	4	-10	-25	-39	-53	-67	-82	-96	-110
25		30	16	0	-15	-29	-44	-59	-74	-88	-104	-118
30		28	13	-2	-18	-33	-48	-63	-79	-94	-109	-125
35		27	11	-4	-20	-35	-51	-67	-82	-98	-113	-129
40		26	10	-6	-21	-37	-53	-69	-85	-100	-115	-132
		Minimal Risk				Increasing Risk			Great Risk			

c.f. °C = (°F - 32) / 1.8

B. Thermal Regulation and Disorders

1. Heat Related Disorders

Heat related disorders occur when thermoregulatory mechanisms fail to compensate for elevations in core temperature caused by environmental or metabolic heat load. Heat related disorders might encompass a wide spectrum of symptoms of varying severity, ranging from heat cramps and dehydration to heat exhaustion and life-threatening heat stroke.

- a. **Heat cramps**, the least serious type of heat related disorders, are caused by loss of sodium and potassium associated with heavy sweating in unacclimatised individuals.
 - i. Signs/symptoms: painful skeletal muscle spasms, primarily of the muscles most heavily used during exercise (e.g. calves, abdomen).
 - ii. Treatment: move individual to a cooler location and administer fluid/electrolyte solutions or a saline solution, generally taken orally.
 - iii. Prevention: adequate fluid/electrolyte replacement, added salt to food; eat a balanced, high K⁺ diet.
- b. **Dehydration** commonly accompanies exercise in warm, humid conditions, when fluid replacement is inadequate. It complicates heat exhaustion and heat stroke.
 - i. Signs/symptoms: fatigue, lethargy, irritability, loss of coordination, faintness, altered consciousness.
 - ii. Treatment: cool fluids; dilute electrolyte solutions.
 - iii. Prevention: pre-hydrate; adequate fluid replacement during activities.
- c. **Heat exhaustion** is a serious heat illness caused by increased exercise heat load plus dehydration. The pathophysiology of heat exhaustion is the inability of the cardiovascular system to adequately supply blood to organs, especially to the brain.
 - i. Signs/Symptoms: “core” (rectal) temperature elevated, usually not above 39.5°C (103°F); “goose flesh”, headache, lethargy, fatigue, dizziness, fainting, hypotension, rapid pulse, altered consciousness, nausea, vomiting, incoordination.
 - ii. Treatment: (See also Appendices 8 and 9, *Differential Diagnosis and Treatment of Exertional Heat Stroke and Heat Exhaustion on Site and in a Clinic Setting*).
 - Move to a cool, shaded area.
 - Remove excess clothing.
 - Elevate feet to avoid shock
 - Begin immediate cooling with cold or iced cloths, sponges, etc. to torso, axillae, groin, other exposed areas.
 - Begin hydrations with cool fluids, orally if possible, otherwise start IV fluids (Dextrose/0.5N saline).
 - Monitor vital signs, rectal temperature if possible.

- Transfer to hospital, when unconscious or incomplete response to therapy.
- iii. Prevention:
- Avoid competition under adverse conditions, or adjust pace to existing conditions.
 - Utilise acclimatisation measures prior to competition.
 - Pre-hydrate; emphasise hydration during the competition.
 - Wear appropriate clothing, which will “breathe” and allow sweat to evaporate.
- d. **Heat stroke**, a life-threatening heat-related disorder and a medical emergency, is difficult to distinguish from heat exhaustion because sweating may continue. Heat stroke represents thermoregulatory failure, with reduction in skin blood flow in order to maintain the central circulation. Core temperature is more elevated, usually 40°C or higher.
- i. Signs/symptoms: core temperature exceeding 40°C, cessation of sweating, rapid pulse, rapid respiration, hypotension, CNS symptoms predominate: unsteady gait, confusion, combative behaviour, reduced consciousness, convulsion, and coma. These signs and symptoms represent a medical emergency.
- ii. Treatment: (see also Appendix 8)
- Move to a cool, shaded area. Lay down with feet elevated.
 - Loosen or remove clothing.
 - Begin cooling at once. In the field, it may be necessary to assume that temperature is elevated, as taking a rectal temperature may not be feasible. Oral or axillary temperature is quite unreliable. Apply cool water, and fan to increase evaporation. Apply ice packs over major vessels in neck, axillae, groin. Cool to a rectal temperature of 39°C (102°F).
 - Start IV fluids (Normal saline)
 - Evacuate to a medical facility as early as possible. Manage as a medical emergency, with monitoring of cardiac, neurologic, and renal function, and electrolyte balance.

(For prevention and cautions related to thermal injuries, see Appendix 4, *ACSM Position Stand on Heat and Cold Illnesses During Distance Running* and Appendix 5, *IAAF Policy on Fluid Replacement*.)

2. Cold Injuries

Activities in water and in alpine locales, and prolonged activity can cause cold injuries. The cold stress conditions of concern are hypothermia and frostbite. Hypothermia affects cardiovascular, respiratory, CNS and neuromuscular systems. Hypothermia occurs when core body temperature decreases enough to affect body functions—usually below 35°C (95°F). It may be classified clinically as mild, moderate, or severe (Table 11-3).

Table 11-3. Categories of hypothermia.

Category	Rectal Temperature	Symptoms/Signs
Mild Hypothermia	33°–35°C	Symptoms/Signs: shivering, very cold, hunger, lethargy, confusion, muscle spasm and difficulty with motor tasks, decreased race pace, slurred speech, ataxic gait, slow reflexes.
Moderate Hypothermia	30°–33°C	Symptoms/Signs: may not be shivering, semi-conscious with confused actions and irrational behaviour, extremely tired, irritable and depressed, poor judgment, loss of memory, disoriented, poor coordination, muscle stiffness, slurred speech, slow and/or irregular pulse.
Severe Hypothermia	less than 30°C	Symptoms/Signs: loss of consciousness, pupils dilated, heartbeat faint or undetectable.

- a. **Hypothermia: Mild.** Rectal temperature 33°–35°C
 - i. Signs/symptoms: shivering, hungry, lethargic, confused, poor coordination, slurred speech, ataxia.
 - ii. Treatment: insulate the athlete with dry clothing, continue mild exercise, and administer warm liquids.
- b. **Hypothermia: Moderate.** Rectal temperature 30°–33°C
 - i. Signs/symptoms: semi-conscious, confused, irrational, disoriented, muscle stiffness, slow, irregular pulse.
 - ii. Treatment: exogenous heat via a warm shower, warm water bottles, body contact, inhale warm, moist air; warm liquids; the athletes should be handled with care due to myocardial irritability.
- c. **Hypothermia: Severe.** Rectal temperature below 30°C
 - i. Signs/symptoms: unconscious, pupils dilated, faint or absent heartbeat.
 - ii. Treatment: transported to a medical facility by emergency vehicle. The trunk must be rewarmed and continuous CPR may be necessary; handle the patient with care to avoid arrhythmias.
- d. **Frostbite** occurs when there is cold-induced peripheral vasoconstriction, leading to tissue freezing.
 - i. Prevention: appropriate clothing—mittens, footgear with dry wool or polypropylene socks, etc.

- ii. Treatment: leave parts frozen until thawing can be done without the possibility of re-freezing. Keep entire body warm. Avoid physical contact between injured tissue and surroundings. Thaw slowly and gently in warm water, 37°–40°C.
- e. Prevention of cold injuries
 - i. Administrative
 - Plan adequately to avoid wind-exposed race courses.
 - Caution athletes concerning wind-chill factor, adequate clothing, etc.
 - ii. Athlete preparation
 - Acclimatise to cold conditions—10 days is an ideal acclimatisation period.
 - Ensure adequate nutrition and use muscle glycogen loading to maximise heat production. Avoid caffeine and alcohol. Hydrate before and during a race.
 - Wear layers of material that will draw sweat from the skin and allow evaporation. Wear head cover.
 - Control pace to avoid late slowing and reduced heat production.

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ALTITUDE

Altitude adversely affects performance in aerobic events (i.e., those lasting more than two minutes), because the partial pressure of oxygen decreases as barometric pressure falls. This leads to a decline in pulmonary diffusion of oxygen into the blood. A measurable effect on maximal oxygen uptake ($\dot{V}O_{2\max}$) can be seen at elevations as low as 1524 m (5000 ft).

A. Environmental Conditions at Altitude

1. Atmospheric Pressure

Atmospheric pressure decreases as altitude increases, but the percentage of gases in the air remains constant. Air always contains 20.93% oxygen, 0.03% carbon dioxide, and 79.04% nitrogen. The pressure that oxygen molecules exert (PO_2) is directly related to the atmospheric pressure (P_b). This change in the partial pressure of oxygen directly affects the transfer of oxygen between the lungs and the blood, and between the blood and the tissues (Table 11-4).

Table 11-4. Changes in the partial pressure of oxygen with increasing altitude.

Altitude	P_b (mm)	PO_2 (mm)
0 (sea level)	760	159.2
1000	674	141.2
2000	596	124.9
3000	526	110.2

2. Air Temperature

Air temperature decreases about 1°C for every 150 m of ascent. This lower temperature also reduces the relative humidity of the air, and increases water losses by evaporation from the skin and the lungs. The increased respiratory rate and loss of respiratory water in dry air can lead rapidly to dehydration, especially during exercise.

3. Solar Radiation

Solar radiation increases at altitude, as less ultraviolet light is blocked by the reduced atmosphere and the lower water vapour pressure.

B. Physiological Responses to Altitude

1. Respiratory Responses

a. Ventilation

As the partial pressure (PO_2) of oxygen decreases, more air must be taken in to provide adequate tissue oxygenation. This increase in ventilation reduces the amount of alveolar and blood carbon dioxide, leading to respiratory alkalosis. The kidney compensates by excreting more bicarbonate ion, decreasing the blood's buffering capacity and reducing the alkalosis (compensated respiratory alkalosis).

b. Pulmonary Oxygen Diffusion

Oxygen diffusion across the alveolar-capillary membrane is dependent upon alveolar PO_2 . This decreases as altitude increases, leading to a decrease in oxy-hemoglobin saturation. At sea level hemoglobin is 98% saturated, but this falls to 92% at 2400 m (8000 ft).

c. Muscle Oxygen Gas Exchange

The pressure gradient between blood and muscle oxygen concentration is 74mm Hg at sea level (94mm–20mm). This gradient is the major factor responsible for driving tissue oxygenation. At 2400 m the arterial PO_2 is about 60mm Hg, while tissue PO_2 remains at 20mm Hg, a gradient of only 40mm Hg, or a decrease of nearly 50%.

2. Cardiovascular Responses

a. Blood Volume

Plasma volume decreases soon after altitude exposure, and levels off after a few weeks. This leads to an increased red cell concentration (hematocrit), aiding oxygen delivery to tissues. Plasma volume is gradually restored, and erythropoietin from the kidney stimulates red cell production.

b. Cardiac Output

Cardiac output must increase at altitude to compensate for the reduced PO_2 and decreased oxygen delivery to tissues. Initially this is accomplished by an increase in heart rate, as stroke volume is lower due to the decreased plasma volume. After a few days, tissue oxygen extraction improves (increased a-v O_2 difference) and this reduces the cardiac demands. At maximal workloads, maximal stroke volume, heart rate, and tissue oxygen diffusion are reduced, thus total maximal VO_2 and aerobic work are diminished.

3. Metabolic Adaptations

As oxidative pathways are limited at altitude, there is a shift toward anaerobic energy sources. At any given work level, lactic acid production is higher than at sea-level. However, at maximal workloads lactic acid is lower, possibly because work levels are too low to maximally activate all energy systems.

4. Maximal Oxygen Uptake

Maximal oxygen uptake measures the ability of the body to take in, transport, and utilise oxygen. It decreases as altitude increases, but does not begin to fall until atmospheric PO_2 drops below 125 mm Hg. This occurs at an altitude of about 1600 m (5250 ft). $\dot{V}O_2$ max. is related to the decline in the barometric pressure and the partial pressure of oxygen (PO_2). $\dot{V}O_2$ max. decreases 11% for each 1000 metres above the 1600 metre level.

C. Adaptations to Chronic Altitude Exposure

1. Blood

Erythropoietin stimulates red cell production, and eventually a higher hemoglobin and hematocrit. Adequate dietary iron is essential to meet this demand for increased erythropoiesis.

2. Muscle

Muscle cross-sectional area may decrease with chronic altitude exposure, though capillary density increases to deliver blood to the tissues. At very high altitudes (over 2500 m), levels of muscle enzymes decline, so that muscles are less able to generate ATP aerobically or anaerobically.

3. Cardiorespiratory

Ventilation is stimulated by the hypoxia of altitude. This causes carbon dioxide removal and respiratory alkalosis. Bicarbonate is excreted and remains low, decreasing buffering capacity.

Muscle oxygen uptake decreases at altitude, and improves little with prolonged exposure. This may be due to the profound hypoxia that occurs during exercise at altitude and the consequent inability to train at an adequate intensity and volume.

D. Effects on Training

Many athletes live at altitude, and others elect to train there in hopes of enhancing their performance at lower elevations. However, maximal aerobic capacity and submaximal aerobic training pace decrease at altitude, especially above 2500–3000 m. Prolonged stays at altitude may be detrimental to high-intensity endurance performance, so altitude training should be interspersed with periods near sea level.

A number of studies have shown that a programme of altitude exposure (hypoxia) interspersed with periods of near-sea-level exposure to permit intense training, when carried out for 3–4 weeks, enhances sea-level performance. This is most likely due to an erythropoietin-induced increase in red cell mass and aerobic capacity, although improved running economy may be a factor.

E. Preparation for Competition at Altitude

Many major competitions, including the Olympic Games (1968) and the World Championships (1997) are conducted at altitude. While this provides an advantage

for sprinters, hurdlers, and jumpers, endurance events of 800 metres and longer are adversely affected. Therefore, a period of adaptation to altitude is essential in preparation for competition, if one lives at sea level. There is still much to be learned about optimal preparation, but a few principles seem to be well accepted:

1. The adaptation period at altitude should be at least 3 or 4 weeks prior to the start of competition.
2. Athletes should be in good general health, free of medical and orthopedic problems, and not iron deficient prior to beginning altitude training. Iron supplementation should be used if ferritin levels are low.
3. Training volume, intensity, nutrition, and health should be carefully monitored during the training and competition processes.
 - a. During the first week, aerobic adaptation should be stressed. Training volume should be moderate, about 75–80% of sea-level loads.
 - b. Intensity should increase in the second week, with mixed aerobic and anaerobic sessions.
 - c. During week three, emphasis is on maintaining speed, with aerobic work at the highest levels possible. Intensity of runs may be maintained by increasing rest time between interval runs.
 - d. Week four is for tapering and recovery prior to the start of competition.

F. Altitude Illness

Rapid ascent to elevations of 1500m or more, especially above 2400m (8000 ft), may result in acute “mountain sickness” or, rarely, a severe altitude illness syndrome, such as high altitude pulmonary oedema (HAPE), or high altitude cerebral oedema (HACE). *Caution:* Athletes with sickle cell trait or G-6-P deficiency may sustain a life-threatening crisis with rhabdomyolysis when exposed to high altitude, especially with the added stress of exercise without prior adaptation.

Symptoms of altitude illness include headache, nausea, lethargy, anorexia, vomiting, and disturbed sleep patterns. Symptoms may begin within hours of ascent, peak in 1–2 days, and generally resolve in 3–4 days.

High altitude pulmonary oedema (HAPE) is a medical emergency. Symptoms and signs include those of altitude sickness plus cough, dyspnea, frothy sputum, chest pain, tachypnea, respiratory distress, and pulmonary rales. HAPE occurs more often in young, active people following heavy exercise in cold air.

High altitude cerebral oedema usually occurs after rapid ascent to 4000 m or more. Signs and symptoms include severe headache, ataxia, confusion, blurred vision, and altered consciousness. Rapid removal to a lower altitude is essential, plus oxygen and IV glucocorticoids.

The following may help prevent altitude illness:

1. A gradual ascent of no more than 300 m–600 m per day over 2400 m should be made.
2. Acetazolamide (Diamox) may be helpful prophylactically.

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AIR POLLUTION

A. Air Pollution

Athletes exercising in urban environments may be exposed to a variety of pollutants that may affect performance. The most common atmospheric pollutants include carbon monoxide, ozone, particulate matter, sulfur oxides (SO_x), nitrogen oxides (NO_x), and peroxy-acetylnitrate (PAN). The “pollution index,” calculated as pollutant concentration \times ventilation volume \times exposure time, is the best way to monitor potential threats to athletes.

- **Carbon monoxide (CO)** readily combines with hemoglobin to reduce tissue oxygenation, increase cardiac work, and impair psychomotor function. It is insidious, as it causes no local irritant symptoms to the airways.
- **Oxidants (ozone)** cause tracheal and bronchial irritation, chest tightness, and induce bronchospasm and reduced lung function (decreased FEV₁). They affect endurance performance at levels of 0.1-0.3 ppm. Asthmatics may be more adversely affected and will need more thorough management.
- **Particulate matter** is composed of solid and liquid droplets, and may contain acids (nitrates and sulfate), organic chemicals, allergens (pollens and molds), metals, soil, and dust. Particles are less than 10 microns in size: “fine particles are defined as less than 2.5 microns, while “course” particles are 2.5–10 microns. High levels of particulate matter may affect the health of older people, increasing morbidity and mortality of those with pulmonary and cardiac disease, as well as affect young people with asthma. Long-term exposure may lead to a permanent decrease in lung function and chronic bronchitis.
- **Sulfur oxides (SO_x)** irritate the upper airway, causing transient bronchospasm. They are a significant problem for asthmatics, even at low concentrations.
- **Peroxyacetylnitrate (PAN)** and **Nitrogen oxides (NO_x)** cause symptoms similar to those triggered by ozone.

1. Prevention/Management

a. Administrative

Schedule events at locations and at times when pollutants are at the lowest levels—away from traffic, near parks and oceans. To minimise ozone exposure, race early in the AM or late PM.

b. Athlete Adaptation

Adaptation to ozone and sulfur oxide may occur, with less bronchospasm and irritative symptoms. Asthmatics may be treated with cromolyn sodium, and aerosol adrenal corticoids and/or beta-2 agonists. “Normal”

athletes may develop exercise-induced asthma (EIA) and require beta-agonist therapy.

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Circadian rhythmic oscillations affect numerous physiologic functions that contribute to athletic performance. Circadian rhythms are expressed as oscillations in physiologic systems (body temperature, heart rate, hormone secretory rates), and responsiveness to internal stimuli (neuro-transmitters, electrolytes, metabolic substrates) or external stimuli (environmental factors, food, drugs, other stressors). Circadian rhythms are synchronised by periodic environmental changes (primarily the light-dark cycle), social interactions, and other environmental factors.

A. Effects on Performance

Athletic performance can be affected by: 1) the time-dependent changes in circadian peak to trough, and amplitude of physiological variations, and by 2) disruption of circadian rhythms due to trans-meridian travel and subsequent changes in the sleep-wake cycle (i.e., “jet lag”). Circadian dysrhythmias (“jet lag”) can result in a wide variety of symptoms, including fatigue, insomnia, sleep disturbances, headaches, irritability, altered gastro-intestinal motility (constipation), and impaired athletic performance.

Factors that may affect the degree of circadian dysrhythmicity include the rapidity of travel and number of times zones crossed, direction of flight, rate of adaptation of various systems, synchroniser intensity, diet, and individual factors (personality, age, gender, chronotype).

1. Distance and Speed

Crossing as few as two or three time zones may affect performance, but in general the more time zones crossed, the greater the disruption in circadian rhythms. Obviously, rapid travel by jet plane is more disruptive than intermittent or slower means of travel.

2. Direction of Travel

East-bound travel is more disruptive than west-bound travel and recovery takes longer. This is because human physiological systems prefer a circadian cycle longer than 24 hours, and thus adapt better to a phase delay (lengthening) than to a phase advance (shortening). Physiological systems adapt 30–50% faster to westward travel than to eastward travel.

3. Rate of Adaptation

Different physiological systems adapt to time-zone changes at different rates. Those that respond to environmental cues, such a heart rate, adjust more rapidly than internally controlled systems such as core temperature. The specific systems responsible for decreased athletic performance have not been identified, but are probably related to alterations in the sleep-wake cycle and the body temperature circadian cycle.

4. Synchroniser Intensity

Appropriate exposure to external synchronisers, such as daylight, regular meals, social interactions, moderate physical activity, and the establishment of regular sleep-wake cycles can help shift circadian rhythms so that they are in phase with the local environment.

5. Diet

Different foods cause different bodily reactions and can affect whether we feel sleepy or awake. Starchy and sweet foods provide quick but transient energy. They stimulate the indoleamine metabolic pathway and encourage higher concentrations of tryptophan, an essential amino acid, to enter the brain and be converted to sleep-inducing serotonin. As serotonin levels rise, sleep comes more readily. Thus, starchy and sweet foods ultimately tend to make one feel lethargic and can aid in sleep.

In contrast, high protein foods help stimulate the “get-up-and-go” catecholamine pathway (adrenaline pathway, starting from amino acid tyrosine and resulting in the synthesis of the neurotransmitters dopamine, norepinephrine, and epinephrine) and tend to make us more alert.

Thus, what and when one eats helps the biological clock function. Adjustments triggered by diet help the body adapt to travel across time zones by contributing, along with other “zeitgeber” (see below), to a shift of the biological from home time to destination time.

6. Individual Differences

Individuals differ considerably in their ability to adapt to changes in circadian rhythms. About 20–30% have little difficulty, while a similar percentage never adjusts well. In general, the “lark” or morning type adapts poorly to westward travel, while the “owl” (extrovert) adapts less well to eastward flight.

Introverted and highly neurotic persons have more difficulty adjusting to jet-lag. Highly motivated individuals (such as athletes) usually can overcome any tendency to performance decrement through extra effort.

7. Minimising Jet Lag’s Effects

Everyone has an internal “biological clock” guiding daily life. This clock prepares the digestive system to receive food at normal times and causes production of digestive juices even before we smell food. It also helps regulate energy, strength, and alertness, and causes one to become tired about the same time each evening.

Important steps to reduce jet lag involve adjustment of time cues, or “zeitgeber” (pronounced “tseitgaybur”). Zeitgeber are activities and inputs that help the body interpret its environment, determine whether it is day or night, and decide what activities (including conscious, unconscious, and biochemical activities) are appropriate for the time. Major zeitgebers are diet, light, exercise, drugs, and social interaction. Adjustment of these cues before and during travel help the body reset its internal clock more quickly and easily.

The body's sense of time is affected by both internal and external factors. The biological clock is coordinated by hormones, which are produced by endocrine glands in varying amounts and kinds throughout the day. Hormones help determine alertness, muscle proficiency, and mood, and their interplay with external time cues determines how strong the body clock's signals will be.

There are a number of steps that can help minimise the effects of jet lag (see Appendix 10, *Recommendations for Minimising Jet Lag*, for a summary of recommendations):

- a. Arrive at the competition site as early as possible. Allow one day for each time zone of eastward flight, and 0.6 days for each zone of westward travel.
- b. Adjust eating, sleeping, work, and training schedules in the direction of the new time zone, by 1–2 hours each day over a 4–5 day period. This may not be practical in a family or work situation.
- c. Arrange flights so as to arrive close to local bed-time.
- d. Eat high protein /low carbohydrate meals for 3 days prior to flight, and during flight. Try to follow a meal schedule that will be used at your destination. After arrival, starting with breakfast and lunch, eat high protein/low carbohydrate meals, and use tea or coffee to enhance synchronisation.
- e. Avoid alcohol as a sleep-inducer, as it interferes with REM sleep and delays sleep-wake cycle adaptation. Do not use coffee, tea, or caffeine-containing drinks during flight. Drink large amounts of caffeine-free drinks (juice, water) to prevent dehydration.
- f. At departure, set watch to destination time, and adjust sleep, meals, and other activities accordingly. East-bound travellers should avoid bright lights, movies, and socialising until breakfast of the day after arrival.
- g. Upon arrival, immediately adjust schedule (social contacts, bright sunlight, meals, training, etc.) to local time. Maintain regular sleep-wake schedules.

Stressors, such as competition anxiety, coping with climate and food changes, and other environmental and social factors can exacerbate the effects of “jet-lag.” Dealing successfully with these elements may minimise the effects of circadian dysrhythmias.

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INFECTIONS ASSOCIATED WITH SPORTS

This chapter identifies many of the diseases that the sport physician/coach/athlete is likely to encounter, along with their appropriate therapies. Keep in mind that this is not a comprehensive list of all possible diseases or symptoms. Part 1 presents details of infectious organisms and antimicrobials; in Part 2 the focus is on specific diseases.

Infection in a specific organ can be caused by organisms such as bacteria, viruses, protozoas, fungi, prions, rickettsia, chlamydia and infestation by helminths (worms). There is a wide spectrum of severity including cases that are asymptomatic. Some of these are self limiting and require no treatment

The physician/coach may encounter a wide variety of infectious conditions. Some are quite symptomatic, such as various forms of food poisoning or respiratory illness, but may not require antibiotics. Others are more insidious, for example urethral symptoms may indicate potentially serious sexually transmitted disease requiring detailed diagnosis and complicated therapy.

A. Overview

Infections in athletes may be classified into several categories that vary considerably in their specific relation to sports participation. These include: 1) sports-associated; 2) lifestyle-associated; 3) travel-associated; and 4) general population-related.

1. Sports-Associated Infections or Complications

These infections or complications result directly from participation in sports.

- a. Skin infections are especially common. These include abrasions, cellulitis, and furuncles due to skin damage. Fungal infections, such as tinea and other dermatophytes, affect the inter-triginous areas between the toes (“athlete’s foot”) and the crural areas. Blisters are subject to secondary infections.
- b. Wound infections may occur from puncture wounds (such as from spikes), lacerations, or other deep tissue injuries.
- c. The danger of tetanus must always be considered when there is a deep puncture wound.
- d. Decreased immunity has been associated with intensive training. This may render the athlete more susceptible to a wide variety of infections, especially viral.
- e. Worsening or complications of infections may occur if physical activity persists in the face of an infectious process, particularly viral ones. Examples include myocarditis, pericarditis, and toxic shock with sepsis.

2. Infections Associated with Lifestyle

Infections associated with lifestyle are those to which the athlete becomes predisposed because of participation in sports.

- a. Group living situations such as those associated with team membership, travel and housing, or living in an athletes' village with athletes from many regions, facilitate the spread of infections by droplets, contact, and common-source outbreaks (e.g. food).
- b. Promiscuous behaviour is facilitated by travel opportunities and group living, and peer pressure increases the risk of exposure to many sexually-transmitted diseases.

3. Travel-Associated Infections

Travel markedly increases the possibility of contact with different infectious agents that are more prevalent in the new geographic locale. In addition, contact with other athletes and individuals from throughout the world at major athletic meetings exposes the athlete to many unfamiliar organisms. In either case, the athlete likely lacks adequate immunity to most of these “new” pathogens.

4. General Population Related Infections

The athlete is at least as susceptible, if not more so, to any pathogen that is present in the community at any given time. However, even a “minor” infection can impair the effectiveness of an athlete who is expected to function at peak efficiency.

5. Management

The extent to which an infectious disease can be evaluated and treated will vary considerably depending upon the level of diagnostic technology available, and the range of therapeutic agents that can be obtained.

The circumstances that exist at the time as well as the potential seriousness of the illness will determine the urgency with which diagnosis and treatment will be undertaken. If a critical competition is upcoming, there is usually a tendency to undertake a much more aggressive approach to diagnosis, and to initiate empiric therapy sooner.

B. Sport Hygiene, General Health, and Preventive Measures

1. Healthy Lifestyle

Infectious disease cannot always be prevented. Furthermore, the outcome of an infection depends not only on the specific therapy used but also on the immunologic condition of the individual. Thus, attention to proper diet and nutrition status is important. Athletes should have a sensible attitude about sufficient rest and sleep, and the compatibility of a normal sex life with intense physical activity. Athletes should also be aware of the ill effects of tobacco, alcohol, and drug abuse. (See Appendix 11, *General Health and Hygiene: Recommendations for Athletes.*)

2. Basic Personal Hygiene

High level physical activity necessitates meticulous care of the skin in order to prevent viral (warts), bacterial, and fungal infections. Physical workouts lead to excessive sweating, so regular washing with soap is mandatory. Athletic clothing should be suitable for prevailing weather conditions and fit properly to avoid chafing and abrasions. Soiled clothing should be laundered between workouts. Athletes should be aware, however, of possible allergic reactions and contact dermatitis due to materials or dyes used in apparel, soaps, detergents, and deodorants. Improperly fitted footwear can cause blisters that may become secondarily infected.

The effect of poor dental health on performance should be pointed out and the need for proper dental care, hygiene and examination stressed.

Traveller's diarrhea is a common problem during international travel. Athletes should be aware of the usual causes and basic preventive measures (see Part 2 of this chapter, *Infections at Large*). In general, travellers should avoid tap water and drink only bottled water and beverages. Where necessary, tap water should be boiled or purified with iodine or chlorine. Raw vegetables, undercooked meat or fish, unpasteurised milk and unpackaged foods sold by street vendors should be avoided.

C. Infectious Agents

Infection can be caused by a variety of organisms, including bacteria, viruses, protozoa, fungi, and helminths (worms).

1. Bacteria

Bacteria are in most instances unicellular organisms with well defined nucleus (with the exception of the actinomycetes). They are usually classified according to their mechanism of movement and type of cell walls. A more practical method for sports medicine practitioners is to classify bacteria by their shape (e.g. cocci or bacilli) and staining characteristics (gram-positive or gram-negative). Dependent on the structure of the cell wall, staining characteristics indicate very different biological activity and thus different sensitivity to antibiotics. Refer to Table 12-1 for characteristics and sensitivities of disease-causing bacteria.

Bacteria may affect the human body directly, causing local inflammation and cell destruction, or by means of toxins formed within the body or preformed in ingested food. Virulence may be affected by the size of the inoculum.

Antibiotics are widely used to inhibit biological activities of bacteria (for details, see antibiotics section); however, bacteria often develop resistance to antibiotics by producing enzymes that destroy the drug, changing their permeability to the drug, or altering metabolic pathways. Resistance to antibiotics usually involves genetic changes that can be transferred between diverse bacteria. This means that resistance to new drugs can develop rapidly.

Table 12-1. Characteristics and antibiotic sensitivities of disease-causing bacteria.

Gram-Positive Bacteria**• *Staphylococci***

Found on the skin or noses of most humans. Commonly form a small superficial abscess but have the potential to spread and form large abscesses anywhere in the body. Staphylococci form toxins. 90% of strains are penicillin resistant and 5% methicillin resistant.

• *Streptococci*

A very heterogeneous group of common pathogens. They cause diverse infections such as pharyngitis and pyoderma. Infections may later lead to rheumatic fever and glomerulonephritis. Sensitive to penicillin.

• *Pneumococci*

Cause pneumonia. Sensitive to penicillin.

• *Clostridium perfringens*

Ubiquitous anaerobic bacteria found both in the colon and soil. Frequent cause of food poisoning through the toxin.

• *Clostridium difficile*

Produces both cytotoxin and endotoxin. Cause of antibiotic-associated colitis, most often due to clindamycin but also ampicillin and cephalosporin. Oral vancomycin is the most effective treatment.

• *Clostridium tetani*

Found worldwide in soil. Produces a powerful neurotoxin that causes tetanus. There is a very effective vaccine to protect against it.

Gram-Negative Bacteria**• *Escherichia coli***

A natural inhabitant of the gastrointestinal tract; contributes to normal function and causes no problem in the gut. However, if introduced to other sites in the body it can cause infections. *E. coli* accounts for the majority of urinary tract infection. However, there are specific strains of *E. coli* that can cause diarrhea. They are enterotoxigenic (ETEC), which produces a toxin similar to that of *Vibrio cholerae*, characterised by voluminous watery diarrhea. Enterohemorrhagic (EHEC), enteroinvasive (EIEC), enteroaggregative (EAEC) and enteropathogenic (EPEC) cause bloody diarrhea. No drug is uniformly active against all strains, but ampicillins, cephalosporins, tetracyclines, trimethoprim-sulfamethoxazole and nitrofurantoin may all be useful.

• *Salmonella*

Classification is complex. Over 2000 different serotypes are known. It is important to differentiate between enteric fever caused by *S. typhi* and *S. paratyphi*, and salmonellosis caused by the other serotypes. *S. typhi* is the cause of typhoid fever, usually acquired from contaminated water or food from human carrier. Usually sensitive to chloramphenicol. Other types are called nontyphoidal or *S. enteritidis*. They are commonly found in farm animals and may get into various food products, frequently causing food poisoning. They are usually sensitive to ampicillin, chloramphenicol, third generation cephalosporins, and fluoroquinolones. However, they should not be treated with antibiotics when there are no systemic symptoms

Table 12-1 (cont'd). Characteristics and antibiotic sensitivities of disease-causing bacteria.

- ***Shigella***

A highly communicable human pathogen when orally ingested. It is a major cause of dysentery (blood diarrhea), because of extensive ulceration of the colonic mucosa. They are sensitive to fluoroquinolones and certain cephalosporins, but gaining resistance to trimethoprim/sulfamethoxazole. They are usually resistant to sulfonamides, tetracycline, and chloramphenicol.

- ***Neisseria gonorrhoeae* (gonococci)**

A sexually transmitted disease, causes mainly local infection of mucous membranes but may cause systemic infections. Numerous penicillin resistant strains have appeared.

- ***Neisseria meningitidis* (meningococci)**

Cause meningitis. They are sensitive to penicillin. Outbreaks have occurred among military recruits in camps, but have not been reported among athletes.

- ***Legionella***

Small ubiquitous bacteria found in aquatic environment, first discovered in 1976. Cause pneumonia through inhalation, especially in elderly or debilitated patients. Sensitive to erythromycin.

- ***Vibrio cholerae***

Two biotypes, classic and El Tor, pathogenic only to humans. *V. cholerae* produces enterotoxin that may cause diarrhea up to 20–30L/d. Hydration is the most important aspect of therapy, but antibiotics—particularly tetracycline—may shorten the course.

- ***Vibrio parahaemolyticus***

Present in coastal waters. May cause diarrhea after ingestion of raw or incompletely cooked seafood.

- ***Campylobacter jejuni***

Belong to the intestinal flora of animals. Can contaminate drinking water and cause diarrheal disease in humans.

- ***Yersinia enterocolitica***

Toxin-producing, causing diarrhea. Sensitive to most antibiotics but therapy is rarely needed.

- ***Plesimonas* and *Aeromonas***

Both found in fresh water and have both been associated with diarrhea after ingestion of contaminated fish.

Spirochetes

- ***Trepanoma pallidum***

Causes syphilis. Universally sensitive to penicillin

Rickettsia

Intracellular parasites; numerous types are found throughout the world. They are transmitted through various vectors such as ticks, fleas, or lice. The most fearsome was typhus, now rare. Other types cause Rocky Mountain spotted fever and related diseases. They are sensitive to chloramphenicol and tetracycline.

Mycoplasma

Small, wall-less, fastidious bacteria. *M. pneumoniae* is a common cause of respiratory infections, while *M. hominis* and *Ureaplasma urealyticum* cause non-gonococcal urethritis. They are sensitive to erythromycin and tetracycline, but not penicillin.

Table 12-1 (cont'd). Characteristics and antibiotic sensitivities of disease-causing bacteria.

Chlamydia

Small intracellular parasites, classified as bacteria. *C. trachomatis* was originally known as the cause of trachoma but is now one of the major causes of sexually transmitted diseases. *C. pneumoniae* are recently recognised pathogens causing pneumonia, and are sensitive to erythromycin and tetracycline.

Mycobacteria

Rod-shaped bacteria that have special staining characteristics described as "acid fast". Several species, including *M. tuberculosis*, *M. bovi*, and *M. kansasii* are examples of so-called atypical mycobacteria that are pathogenic for humans. Resistant to chemical agents and can survive for a long time in dried sputum. They infect the host either through the respiratory or GI tract, or the skin. Special groups of antimycobacterial antibiotics in combination therapy are needed for treatment.

2. Viruses

Although classified as microorganisms, viruses differ from all other cellular forms of life. Only 20–300nm in size, they consist of a core of nucleic acid (either DNA or RNA) enclosed in a protein coat, with or without an outer coat.

Inert in the extracellular environment, viruses replicate only in living cells. Host cells usually suffer injury. Many viruses are host specific, pathogenic only to one particular species of animal or plant; other viruses are broadly pathogenic, infecting humans and other animals.

Most infectious viruses enter their hosts through the mucosa of the respiratory or gastrointestinal tract; some enter through the genital tract or directly into the bloodstream during injections.

Viruses vary considerably in size, biologic activity, and virulence. Some viruses can cause tumours or leukemia. Some cause only transient subclinical infections, others chronic persistent infections, and others fulminant infections that quickly lead to death.

Immunologic responses to viruses also differ considerably. Some immune responses result in permanent immunity. Some viruses can be immunised against, others cannot. A few types are sensitive to antiviral agents, but many others are totally resistant.

Viruses are classified in many different ways, including nucleic acid type, size and morphology, immunologic properties, methods of transmission, pathology and symptomatology. Table 12-2 lists some characteristics of the primary disease-causing viruses, the diseases associated with them, drug sensitivities, and immune responses.

3. Protozoa

Protozoa are classified as unicellular animals up to 30 μm in size. Table 12-3 lists the primary disease-causing protozoa.

Table 12-2. Viruses associated with human diseases.

DNA containing viruses**• Parvoviruses**

Cause erythema infection.

• Papovaviruses

Papilloma (wart) viruses, progressive multifocal leucoencephalopathy.

• Adenoviruses

Exist worldwide and are present year-round. May cause diseases in the eye, respiratory, GI and urinary tract. They induce immunity.

• Herpesviruses

Establish lifelong persistent infection in their host and reactivate periodically. They include herpes simplex, varicella-zoster, Epstein Barr (mononucleosis), cytomegalovirus. Herpes virus is sensitive to acyclovir, idoxuridine and related drugs.

• Poxviruses

Cause smallpox, vaccinia, cowpox

• Hepadnaviruses

Cause Hepatitis B.

RNA containing viruses**• Picornaviruses**

Rhinoviruses cause the common cold, enteroviruses (polio, coxsackie), hepatitis A.

• Reoviruses

Rotavirus causes infantile gastroenteritis, orbiviruses cause Colorado tick fever.

• Arboviruses

Cause encephalitis, yellow fever, dengue.

• Togavirus

Causes rubella.

• Arenaviruses

Cause Lassa fever.

• Coronaviruses

Cause acute upper respiratory fever (cold).

• Retroviruses

Cause of sarcoma and leukemia. Human immunodeficiency virus (HIV) is a nononcogenic retrovirus of the lentivirus subfamily. There are 2 types, HIV-1 and HIV-2. The virus has a selective affinity for the CD4 molecule receptor on T helper-inducer lymphocytes. This leads to infection and destruction of the lymphocytes, but the T4 cells have critical role in the human immune response.

• Bunyaviruses

Hantaviruses cause hemorrhagic fevers and nephropathy.

• Orthomyxoviruses

Influenza viruses. Frequent cause of epidemics of respiratory illnesses throughout the world. Three main immunologic types are known: A, B and C. Type A is highly variable antigenically and is the cause of most cases of epidemic influenza. Type B exhibit antigenic changes and cause epidemics. Type C is antigenically stable and causes only mild illness.

• Paramyxoviruses

Viruses of measles, mumps, parainfluenza and respiratory syncytial virus.

• Prions

Cause Creutzfeldt-Jacob disease.

Table 12-3. Protozoa associated with human diseases.

<p>Amoebas Parasites of the large intestine. May cause severe ulceration of the colon leading to "colitis."</p> <p>Plasmodium (malaria) Four types, <i>P. vivax</i>, <i>P. ovale</i>, <i>P. malariae</i>, and <i>P. falciparum</i>. Infection through bite of infected female mosquito. Multiply in the liver of the host and later infect red blood cells. (See Part 2 of this chapter, <i>Infections at Large</i>, for more information on malaria.)</p> <p>Giardia lamblia Usually transmitted via contaminated water. Infests the duodenum and jejunum, causes diarrhea.</p> <p>Trichomonas Several types occur; <i>T. vaginalis</i> is the only significant human pathogen. Causes infection of vulva, vagina, and cervix in women and prostate, seminal vesicles and urethra in men.</p> <p>Others <i>Cryptosporidium parvum</i>, <i>Isospora belli</i>, <i>Sarcocystis</i></p>
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4. Fungi

Fungi are classified as a lower form of plants, and some cause superficial, subcutaneous, or systemic infections. Table 12-4 lists the primary fungi associated with human infections.

Table 12-4. Fungi associated with human diseases.

<p>Dermatophytes <i>Tinea pedis</i> (athlete's foot), <i>T. cruris</i> (jock itch).</p> <p>Candida Normal habitant of the genital and gastrointestinal tract where it may gain dominance and cause infection (vaginitis, oesophagitis).</p> <p>Other <i>Coccidioides immitis</i> <i>Histoplasma capsulatum</i> <i>Aspergillus fumigatus</i> <i>Pneumocystis carinii</i></p>
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5. Helminthes

A variety of parasitic nematodes, cestodes, and trematodes are associated with human diseases. Table 12-5 lists the helminthes most likely to cause human infections.

Table 12-5. Helminthes associated with human diseases.

Nematodes (roundworm)		
Nematodes have a variety of complex life cycles. A dozen are significant human parasites; another dozen occasionally cause infection. Hosts may become infected via food, water, soil (skin penetration), or mosquito bites. Nematodes primarily associated with diseases in humans include:		
<i>Anisakis</i>	<i>Strongyloides</i>	<i>Ascaris lumbricoides</i>
<i>Trichinella</i>	<i>Enterobius vermicularis</i>	<i>Trichuris trichiura</i> hookworms
Cestodes (tapeworms)		
Tapeworms have ribbon-like segments, each with a complete male and female system. All except <i>Hymenolypis nana</i> use an intermediate host. Tapeworms primarily associated with diseases in humans include:		
<i>Diphyllobothrium latum</i> , copepod-fish-human pathway		
<i>Echinococcus</i> , dog, infected herbivore		
<i>Taenia saginata</i> , beef tapeworm		
<i>Taenia solium</i> , pork tapeworm		
Trematodes (flukes)		
Most flukes are hermaphroditic, use an intermediate host, and cause infection by ingestion. Schistosomes are separate-sexed and cause infection by penetrating the skin. Trematodes primarily associated with diseases in humans include:		
<i>Clonorchis sinensis</i>		
<i>Schistosomes</i>		

D. Antimicrobials

Antibiotics are classified either as bactericidal (killing) or bacteriostatic (inhibiting bacterial multiplication). They act on bacteria by four separate mechanisms:

- Inhibition of cell wall synthesis
- Inhibition of cell membrane function
- Inhibition of protein synthesis
- Inhibition of nucleic acid synthesis

In practice, the physician has to select antibiotics based on a clinical diagnosis of which organ is infected and what is the most likely causative organism. Final selection of appropriate antibiotics may depend on sensitivity tests and even serum assays of bactericidal activity. Table 12-6 lists the major classes of penicillins; Table 12-7 lists additional antimicrobial agents and their applications; Table 12-8 lists the relative costs of oral antibacterials.

Table 12-6. Penicillins and cephalosporins.

 β -Lactam Antibiotics (penicillins and cephalosporins)

This class of antibiotics is named for the β -Lactam ring, which interferes with the synthesis of the bacterial wall by binding to specific proteins called penicillin-binding-proteins (PBP). Bacteria can become resistant to these antibiotics by producing β -Lactamases, enzymes that destroy the antibiotics. Penicillins may cause allergic reactions in many patients.

Penicillins

Penicillin G is mainly used parenterally; penicillin V is used orally. Penicillins are active against *S. pneumoniae* and most types of streptococci, *Treponema pallidum* (syphilis) and meningococci. Gonococci and staphylococci are highly resistant to penicillin.

Aminopenicillins

The best known aminopenicillins are ampicillin and amoxicillins (available in oral and parenteral forms), which are active against many gram-negative bacterias such as *E. coli*, in addition to the Gram-positive types. Both penicillins and aminopenicillins can be destroyed by the β -Lactamase produced by gram-positive and gram-negative bacterias. Clavulanate is a β -Lactamase inhibitor that can be combined, for example, with amoxicillin (e.g. Augmentin®) for use against some lactamase-producing bacterias.

Penicillinase-resistant penicillins

Produced for treatment against staphylococcal infection. Methicillin (injectable) was the first produced, but staphylococci are becoming increasingly resistant. The best known oral versions are cloxacillin and dicloxacillin.

Cephalosporins

Cephalosporins also contain a β -Lactam ring, but with a different structural attachment. They are classified into first-, second- and third-generation cephalosporins, depending on activity. Among them are compounds that are active against both gram-positive and negative bacterias, and are available both in oral and parenteral forms.

D. Antiviral Chemotherapy

As viruses are intracellular parasites, antiviral agents must be capable of selectively inhibiting viral function without damaging the host cell.

Nucleoside analogues are the most common antiviral agents, active mainly against herpes viruses. They inhibit nucleic acid replication by inhibiting enzymes of the metabolic pathways for purines or pyrimidines. They include acyclovir, ganciclovir, zidovudine, AZT (which inhibits replication of HIV), and Iioxuridine.

Other antiviral agents include foscarnet and interferon, and amantadine, which blocks viral penetration of the host cell and is effective prophylactically against influenza.

Table 12-7. Additional antimicrobial agents and their applications.

Vancomycin

A glycopeptide active only against gram-positive bacteria, and used mainly parenterally.

Aminoglycosides

E.g. gentamycin, tobramycin and amikacin are active against various gram-negative bacteria and are used mainly parenterally. Streptomycin is used mainly against mycobacteria.

Tetracyclines

Bacteriostatic agents, active against many gram-positive and gram-negative bacteria and used mainly as oral compounds.

Erythromycin

Active against many gram-positive and gram-negative bacteria and is used mainly in oral form. It is particularly useful for patients with penicillin allergy.

Metronidazole

Originally used mainly orally against trichomonas infections, but is also useful for amoebiasis and giardiasis. It is active against various gram-negative bacteria and is particularly effective in intravenous form against anaerobic bacteria.

Sulfonamides and Trimethoprim

Among the earliest antibiotics, their usage has been renewed in combination with trimethoprim (TMP-SMX). This combination has activity against many gram-positive and negative bacteria. Some patients may show severe form of allergic reaction.

Quinolones

E.g. ciprofloxacin and norfloxacin are chemically synthesised antibiotics and have activity both against gram-positive and negative bacteria.

Ethambutol

Active against *M. tuberculosis*.

INH

Active against *M. tuberculosis*.

Rifampicin

Active against *M. tuberculosis*, some gram-positive and gram-negative cocci, some enteric bacteria, chlamydiae, and poxviruses.

Table 12-8. Relative costs of oral antibacterials.

Name of drug	Dosage	Ratio
Doxycycline	100 mg x 1	1
TMP/SMX	160/800 mg x 2	1.2
Phenoxym. penicillin	1000 mg x 3	1.5
Ampicillin	500 mg x 4	2
Dicloxacillin	250 mg x 4	2
Erythromycin	500 mg x 4	3–5
Cephalexin	500 mg x 4	5–10
Cefuroximum	250 mg x 2	5–25
Augmentin	125/500 mg x 3	6–25
Cefpodoximum	200 mg x 2	10–15
Clarithromycin	500 mg x 2	12
Ciprofloxacinum	500 mg x2	13–15

E. Antifungal and Antiparasitic Agents

Table 12-9 lists the major antifungal and antiparasitic agents.

Table 12-9. Major antifungal and antiparasitic agents.

Antifungal Agents	
Amphotericin	Fluconazole
Flucytocine	Clotrimazole
Ketoconazole	Miconazole
Antiparasitic Drugs	
Iodoquinol	Pentamidine
Mebendazole	Chloroquine phosphate
Thiabendazole	Quinidine gluconate,
Albendazole	Q. dihydrochloride, Q. sulfate
Praziquantel	Pyrimethaminesulfadoxine
Bithionol	Mefloquine
Quinacrine	Halofantrine

F. Immunisation

Prevention is better than a cure, and immunisation has been a major advancement in improving human health.

1. Childhood

In most countries, young children are routinely immunised against diphtheria, tetanus and pertussis (DTP), and polio (OPV). In some countries pediatric immunisation has been expanded to also include measles, mumps and rubella (MMR), and Hemophilus and hepatitis B.

2. Adults

Adults should receive tetanus and diphtheria toxoids every 10 years. Combined measles-mumps vaccine should be given to males who have not been immunised or infected, and seronegative females of reproductive age should receive rubella vaccine.

3. Target Groups

New vaccines are continually being developed, but these are currently targeted at specific groups, few of which include active athletes (but perhaps rather the elderly athletic officials!).

Influenza and pneumococcal vaccines are given mainly to the elderly, although circumstances may require administering them to athletes. Hepatitis B immunisation may be considered for sexually active individuals, and immunoglobulin for non-immunised individuals exposed to infections.

4. Travellers

Prior to travel, the physician should ensure that athletes have received routine vaccinations such as tetanus and polio. Requirements for additional immunisation may vary depending on the travel destination. The team physician should refer to guidelines issued by local health authorities.

Hepatitis A is prevalent in many countries. Routine prevention in the past has utilised passive immunisation with Immune Globulin. A Hepatitis A vaccine (Havrix[®]) is available, although it is expensive. Similarly, new, effective typhoid vaccines are now available, though still relatively costly. These include the oral vaccine Ty21A (Vivitif[®]) and typhoid Vi polysaccharide vaccine (Typhim[®] Vi).

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2. Wilson, M. E. A World Guide to Infections. Oxford: Oxford University Press, 1991.
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INFECTIONS AT LARGE

A. Acute Infectious Diarrheal Diseases

Traveller's diarrhea has been defined as the passage of more than 3 unformed stools in 24 hours with discomfort, occurring in a person visiting another country where food contamination may occur. Food or water contamination by bacterial, viral, or protozoal pathogens is the most common cause of traveller's diarrhea (Figure 12-1). In less than 50% of cases can the infectious organism be found in the stool. Of course where the poisoning is due to preformed toxin such as staphylococci, the organism is hardly likely to be found.

Diarrhea usually starts suddenly; it is often watery and, in approximately 20 percent of cases, bloody. Cramps, nausea, vomiting, and fever may also occur. Symptoms commonly start on the third day after arrival and may last for 3–5 days, with a median of 2 days. Diarrhea may last longer than 7 days in 10% of patients and, rarely, longer than 1 month.

Enterotoxin-producing E. coli, a variant of the most common large bowel bacteria, is the most common cause of this condition. The clinical course may vary considerably, from mild troublesome symptoms to fulminant cholera-type disease.

Staphylococcus aureus is the cause of the classic "food poisoning." Symptoms occur 2 to 6 hours after eating contaminated food, but often last less than 10 hours. Infection is caused by ingesting preformed enterotoxin and may occur in more than 75% of those who have eaten the same food. Vomiting is a very prominent feature of this condition.

Clostridium perfringens is also a significant cause of food poisoning, and infections of this type often appear in a microepidemic pattern following ingestion of contaminated meat or poultry. Typically, two or more individuals who have eaten the same food become sick simultaneously 6–12 hours after eating. Cramps are often more prominent in this condition than in *E. coli* infections.

Campylobacter jejuni causes approximately 10% of food poisoning cases. This bacteria is associated with contaminated water and raw milk from domestic animals. The incubation period may be from 2 to 6 days.

Other organisms that can trigger traveller's diarrhea/food poisoning symptoms include:

- *Shigella*, a frequent cause of bloody diarrhea or dysentery.
- *Salmonella* (nontyphoidal). Salmonella infections occur more frequently in the industrialised world than in developing countries, and cases are increasing among young people. Infection is usually from food, such as poultry and eggs.
- *Viruses* of various types, such as rotavirus and Norwalk agent. These have been associated with epidemics in group situations (e.g. camps) and may account for up to a third of the food-poisoning epidemics that have occurred in the U.S. Incubation period is 18–72 hours.

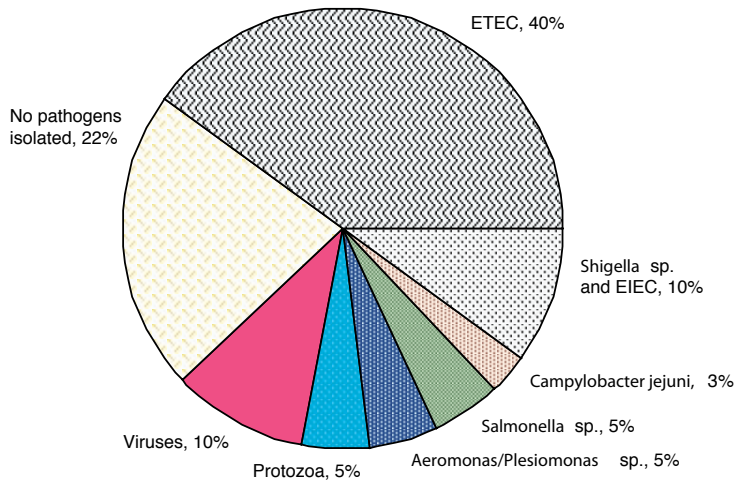


Figure 12-1. Spectrum and approximate prevalence of enteropathogens in traveller's diarrhea. ETEC = enterotoxigenic *E. coli*, EIEC = enteroinvasive *E. coli*.

- *Giardia*, a protozoa that has been associated with waterborne epidemics in the U.S., Russia (former Soviet Union), and developing countries. The incubation period is long (1–3 weeks), and resultant diarrhea may be voluminous, greasy, and floating.
- *Cryptosporidium* and *Entamoeba histolytica* can also cause food poisoning.

1. Prevention

To prevent traveller's diarrhea, athletes should avoid contaminated food, including raw, peeled fruits, vegetables, and other uncooked foods. Water should be bottled or boiled. Bacterial pathogens may survive in food up to 50°C and can be found in ice cubes in drinks.

Many antibiotics as well as bismuth taken prophylactically may decrease the frequency of attacks, but not altogether prevent them. However, antibiotics may at times cause serious adverse effects, and prophylaxis is not recommended except for persons in impaired health or when individuals must undertake a short trip to a high risk area (Figure 12-2).

2. Treatment

Most of these illnesses are clinically mild and self-limiting, and no specific therapy is needed. If the patient has a high fever and frequent bloody diarrhea of any duration, specific diagnosis by stool cultures may be advisable.

Patients should be kept hydrated. In most instances carbonated drinks and salted crackers are sufficient; intravenous fluids are rarely needed. Specific solutions containing electrolytes and glucose should be encouraged. If taken early, bismuth subsalicylate can ease symptoms.

In an athlete with "traveller's diarrhea" and facing an important competition, many antibiotics can shorten the course of illness. These include tetracycline

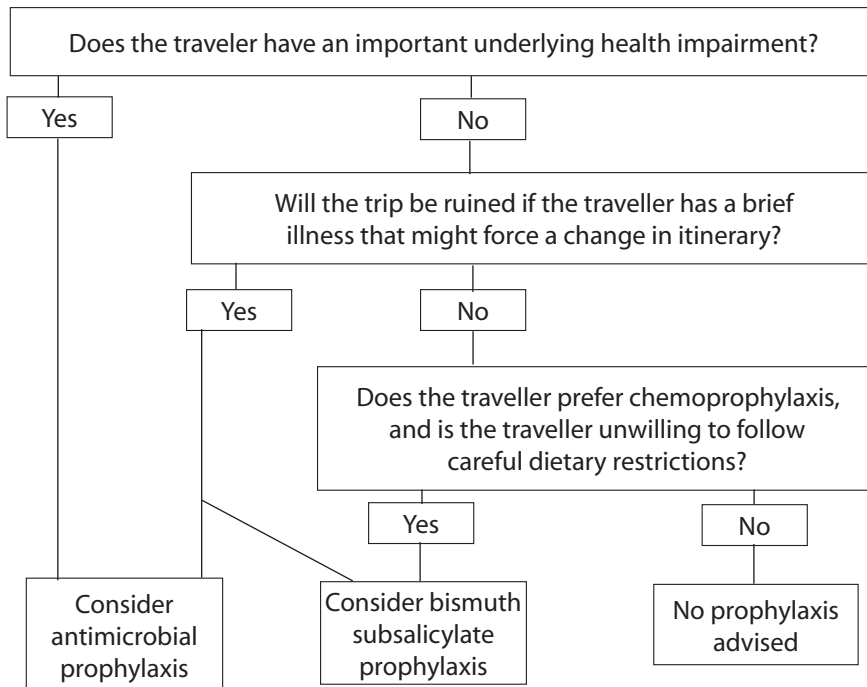


Figure 12-2. Determining the advisability of prophylaxis against traveller's diarrhea during a stay of less than 3 weeks in a high-risk area of Latin America, Africa, or Southern Asia.

products, sulphamethoxazole/trimethoprim, or the quinolone drugs such as ciprofloxacin or norfloxacin. Loperamide or diphenoxylate may relieve abdominal cramps. Antibiotics may be contraindicated in salmonellosis; antiperistaltic agents may be contraindicated in shigellosis.

B. Respiratory Diseases

Acute viral respiratory illness (upper respiratory illness, URI)

These illnesses, which usually involve the upper respiratory tract but can at times also involve the lower respiratory tract, are among the most common afflicting humans. They occur worldwide, with peaks in the fall and spring, and are contagious, spreading by droplets and direct contact. Respiratory illnesses may present with different syndromes, including the common cold, pharyngitis, croup (laryngotracheobronchitis), tracheitis, bronchiolitis, bronchitis, and pneumonia.

Sixty percent to seventy-five percent of respiratory illnesses are caused by one or more of the approximately 200 types of viruses associated with these diseases. The most frequent causative agents are rhinovirus, coronavirus, respiratory syncytial virus, parainfluenza virus, and adenovirus. Clinical symptoms are similar and exact diagnostic methods are rarely necessary.

1. Treatment and Prevention

Respiratory illnesses are usually mild and self-limited. Patients should be kept hydrated, and some may benefit from analgesics and decongestants. Be aware that some formulations contain epinephrine, which is on the list of banned substances. For the athlete facing an important competition, early antibiotic use is a prudent course. Interferon spray applied intranasally may prevent some rhinovirus infections but may also cause irritation. See Appendix 12, *Respiratory Tract Infections*, for additional guidelines on preventing infections, exercise during an episode of respiratory tract infection, and return to exercise following infection.

2. Complications

Physicians should watch for superimposed infections, especially of the sinuses or of the bronchi and lungs, including pneumonia.

Pneumonia

Pneumonia is defined as inflammation of the parenchyma of the lung involving the alveolar units, usually caused by bacteria. Inflammation leads to consolidation of the lung tissue and impairment of gas exchange.

Typically, a pyogenic bacterial pneumonia follows a viral illness and presents with the abrupt onset of chills and fever, a cough with purulent sputum, chest pain, and dyspnea. Symptoms caused by viral or mycoplasma pneumonia may develop more slowly, and pain and respiratory distress may be less prominent.

Pneumococcus is the most common bacteria to be expected in a community-acquired pneumonia in a previously healthy individual. *Mycoplasma pneumoniae* and other types are less frequent and no definite agent is defined in 20–40% of cases.

Examination may reveal a sick individual with high fever, chest pain, dyspnea and tachypnea and abnormal breath sounds on auscultation. Examination of the sputum with Gram stain and cultures and chest radiograph (X-ray) are required for accurate diagnosis.

Treatment

The coach, and even the physician, travelling abroad with an athlete who develops the above-mentioned condition should seek expert local help.

Pneumococcus used to be universally sensitive to penicillin, but in recent years strains have appeared that are resistant to various antibiotics. Sputum culture is therefore very important for specific therapy but high doses of penicillin would still be regarded as the initial drug of choice. If the patient is allergic to penicillin, erythromycin might be considered.

C. Sexually Transmitted Diseases

Athletes must be made aware that any sexual contact without adequate protection might lead to an incapacitating illness. Condom use must be stressed, and condoms should be included in the medical supplies. The most common sexually transmitted pathogens include the following:

- **Bacteria:** *Neisseria gonorrhoea* (gonococcus), *Chlamydia trachomatis*, *Treponema pallidum* (syphilis), *Calymmatobacterium granulomatis* (granuloma inguinale), *Hemophilus ducreyi* (chancroid).
- **Viruses:** Herpes simplex viruses (HSV), Human immunodeficiency viruses (HIV), Hepatitis viruses (HBV).
- **Others:** *Trichomonas vaginalis*, Pubic lice, Scabies, Candida.

The most frequently encountered symptoms of sexually transmitted disease include the following:

- *Urethritis in men.* Classified as gonococcal or nongonococcal (NGU), urethritis presents with burning on urination. Discharge is more prominent with gonococcal infection. NGU is most often caused by chlamydia, but also by HSV. This disease may progress to epididymitis.
- *Lower genitourinary tract infection in women.* Symptoms are mainly burning on urination, vaginal discharge, vulvar irritation, and dyspareunia. Infection may involve the urethra and bladder, vulva, vagina, and cervix and may lead to infections of the uterus and fallopian tubes.
- *Genital ulcers.* Herpes simplex virus is the most frequent cause of genital ulcers, but syphilis and chancroid should also be considered.

1. Treatment

The physician must obtain a careful sexual history and perform a detailed physical examination to assess the possible extent of the infection. STDs are reportable. Whenever a patient presents with symptoms that are thought to be those of STD, multiple coinfections must be suspected. Gram stain and cultures should be done, as well as immunologic studies for chlamydia, syphilis, hepatitis, and HIV.

Assuming that *N. gonorrhoea* and/or *C. trachomatis* are the cause of the STD, the patient should be given a single dose of ceftriaxone 250 mg intramuscularly and doxycycline 100 mg orally twice daily for 10 days.

AIDS

AIDS is caused by the human immunodeficiency virus (HIV). Transmission occurs mainly through sexual contact, infected blood transfusions, and needle sharing among IV drug users (including anabolic steroid users). Infection with the virus affects lymphocytes, causing immunosuppression.

Development of the clinical disease may take 8–10 years from the initial infection. The most common manifestation is infections with unusual opportunistic organisms such as *Pneumocystis carinii* and *Toxoplasma gondii* (protozoa), *Candida albicans* and *Cryptococcus neoformans* (fungi), *Mycobacterium avium* and *Mycobacterium tuberculosis* (bacteria), as well as the better-known bacteria such as Salmonella, Hemophilus, Streptococcus, and Staphylococcus. Kaposi sarcoma is a frequent complication of AIDS, as are various lymphoid neoplasms. Numerous immunologic laboratory tests are now available to diagnose the disease.

Education and behaviour modification remain the cornerstones of prevention, and must be continually stressed to sexually active men and women. Travelling athletes are particularly at risk, especially in areas of high HIV-prevalence. Athletes who may have been exposed to HIV should be tested. Significant advances have been made in the treatment of patients with HIV infection. The cornerstone is a combination of antiretroviral therapy and appropriate therapy of opportunistic infections.

D. Hepatitis

Viral hepatitis is a systemic infection that mainly affects the liver. Five categories of viral agents are presently known; all cause similar clinical illness. The viruses can be distinguished from one another by their antigenic properties. They include A (HAV); B (HBV); Delta agent associated with HBV (HDV); and 2 types of non-A, non-B, one bloodborne (called C); and the other enterically transmitted (called E):

- **Hepatitis A.** HAV is spread almost exclusively by the fecal-oral route, enhanced by poor personal hygiene and overcrowding. Outbreaks have been traced to food, water and shellfish. Incubation lasts from 15–45 days and the virus can then be found in liver, blood, and stool samples. The infection causes the formation of antibodies to the virus (anti-HAV), initially of the IgM class but later IgG. These remain indefinitely and cause lasting immunity to HAV.
- **Hepatitis B.** The major route of HBV infection is through inoculation (via skin or mucous membranes) of infected serum or blood products, but as most body fluids (particularly semen and saliva) contain virus, any intimate contact—especially sexual—may cause infection. Incubation period is from 30–180 days. With HBV, the concentration of antigens and viral particles in blood may reach a very high concentration. Three types of antigens have been identified: hepatitis B surface antigen HBsAg, hepatitis B core antigen HBcAg, and hepatitis Be antigen HBeAg. Of these the HBsAg is the most important. All of these lead to formation of antibodies: anti-HBs, anti-HBc and anti-HBe. By measuring the different antigens and antibodies it is possible to assess the stage of infection and infectivity of the body fluids.
- **Hepatitis D.** The Delta agent or virus is a defective RNA virus that requires the helper function of the HBV virus. In some areas of the world it is endemic among those with hepatitis B and spread by nonpercutaneous means. In other areas it is mainly associated with blood transfusions.
- **Non-A, non-B hepatitis.** Two different types of non-A, non-B have been identified, called C and E:
 - HCV is associated with blood donations. Infection is spread via pooled donor products such as concentrates of blood factors. Products such as albumin and immune globulin constitute no risk because of prior treatment.
 - HEV causes waterborne non-A, non-B hepatitis.

1. Clinical Features

Symptoms of hepatitis are systemic and variable. Patients may experience anorexia, nausea, vomiting, fever, fatigue, arthralgias, myalgias, headache, and cough for 1–2 weeks prior to the onset of jaundice and passing of dark urine for several days. These symptoms may subside with the onset of jaundice, when the patients might have hepatosplenomegaly and adenopathy.

Increase in serum aminotransferases AST and ALT (formerly SGPT and SGOT) may precede rise in bilirubin. The enzymes may reach 400–4000 IU but do not necessarily correlate with the liver injury. Jaundice is visible when the bilirubin rises to 43 $\mu\text{mol/L}$ (2.5 mg/dL). Typical range is 85–340 $\mu\text{mol/L}$ (5–20 mg/dL), usually equally divided between conjugated and unconjugated fractions.

Serologic tests can be used to diagnose the various types of hepatitis. HAV-hepatitis is based on detection of IgM anti-HAV and HBV infection on HBsAg. Diagnosis of non-A, non-B can be made if IgM anti-HAV, HBsAg, and IgM anti-HBc are not present.

2. Course

Almost all previously healthy patients with hepatitis A recover completely, as well as approximately 90% of hepatitis B patients; 10% of patients with hepatitis B experience a more severe course leading to early death, or chronic hepatitis and cirrhosis. Superinfection of delta agent in Hepatitis B may increase the severity. Long-term carriers of HBsAg have an increased risk of hepatocellular carcinoma.

After transfusion-associated hepatitis C a significant number of patients continue to have biochemical abnormalities and histology consistent with chronic hepatitis. Approximately 10% of those who contract hepatitis C may develop cirrhosis after 10 years.

HEV infection often leads to chronic hepatitis.

3. Treatment

No specific therapy is needed for typical acute viral hepatitis. In fulminant hepatitis, complicated supportive measures are necessary, leading even to liver transplant.

4. Prevention

- a. **Hepatitis A.** Active immunisation with vaccine and passive immunisation with IG are available. All preparations of IG contain anti-HAV. Athletes travelling to areas of high risk should strongly consider use of immunisation. When given before exposure or early in the incubation period, immunisation may prevent or attenuate clinically apparent hepatitis A and cause long-lasting passive immunity.
- b. **Hepatitis B.** Vaccine for active immunisation has now been prepared by recombinant DNA technology or from healthy HBsAg carriers. Prior to potential exposure, immunoprophylaxis individuals in high risk groups are given injection at 0, 1, and 6 months. Post-exposure immunisation is given by both HBIG and vaccine.

- c. **Delta hepatitis** can be prevented by giving hepatitis B vaccine.
- d. **Hepatitis C and E.** The efficacy of IG prophylaxis has not been proven.

E. Malaria

Malaria is caused by the protozoon *Plasmodium*, which is transmitted by the bite of the *Anopheles* mosquito, and occasionally by blood transfusion or needle sticks. It is the most serious human parasitic disease, affecting approximately 200 million people and causing over 1 million deaths each year.

1. Epidemiology

Malaria occurs throughout most of the tropics. Four species of the genus *Plasmodium* infect humans: *P. falciparum*, *P. vivax*, *P. ovale*, and *P. malariae*. *Plasmodium falciparum* is predominant in Africa, New Guinea, and Haiti. *Plasmodium vivax* is more common in Central Africa and the Indian subcontinent. The prevalence of both species is approximately equal in South America, east Asia, and Oceania. *Plasmodium malariae* is found in most areas, but is much less common. *Plasmodium ovale* is relatively unusual outside Africa.

Some areas have experienced a resurgence of the disease due to increasing drug resistance. Most cases experienced by U.S. and European travellers are acquired in sub-Saharan Africa. Malaria symptoms may begin as early as 8 days after initial exposure and as late as several months after departure from a malarious area, after chemotherapy has been discontinued. Almost all deaths are caused by *P. falciparum*.

2. Etiology

Human infection begins when a female anopheline mosquito inoculates plasmodium sporozoites from its salivary glands during a blood meal. They are carried via the bloodstream to the liver where they infect hepatocytes, reproduce asexually, and thus create thousands of merozoites. The liver cell eventually bursts and after entering the blood stream the merozoites invade the erythrocytes, beginning the symptomatic infection.

The disease is caused by the direct effects of red cell invasion and destruction and the host reaction to this process. The merozoite gradually grows to occupy most of the red cell, divides, and when the cell ruptures, numerous merozoites are released, which again can infect red cells and repeat the cycle. These cycles occur at 48 or 72 hour (*P. malariae*) intervals.

After invading the red cell, the parasite progressively consumes and degrades intracellular proteins. This leads to sequestration of mature forms of the parasite in vital organs, such as the heart and brain, where they interfere with microcirculatory flow and metabolism and continue to develop away from the principal host defense, i.e., splenic processing and filtration.

The specific immune response to malaria limits the rising parasitemia and, with exposure to sufficient strains, eventually confers protection from disease, but not from infection. The complexity of the immune response in malaria, and the parasite's evasive mechanism, have caused slow progress towards a vaccine.

3. Symptoms

The first symptoms of malaria are nonspecific, and may resemble those of a viral illness. They can include headache, fatigue, and muscle pains followed by fever. Diagnosis rests on identifying the parasite in peripheral blood smears.

The most severe form is seen in *P. falciparum* infection. The patient may have multifactorial anemia, with accelerated red cell destruction, dysfunction of the spleen, and bone marrow suppression. Renal failure may occur, as well as lactic acidosis, hypoglycemia, and coma with convulsions.

Chronic complications (hyperreactive malarial splenomegaly) lead to hypergammaglobulinemia, normochromic anemia and splenomegaly with increased vulnerability to infections.

4. Prevention and Prophylaxis

The Anopheles mosquito feeds primarily between dusk and dawn. Exposure can be minimised by wearing protective clothing, and using repellent (“DEET”) on exposed skin and in impregnated bed nets.

Chemoprophylaxis depends on which area the traveller intends to visit, and the risk of encountering chloroquine-resistant *P. falciparum* (Table 12-10). Up-to-date information should be sought from the appropriate health authority and/or the World Health Organisation.

- a. Chloroquine is recommended for travel to areas of risk where chloroquine-resistant *P. falciparum* has NOT been reported. The drug should be taken for 1–2 weeks before and continued for 4 weeks after travel. Daily doxycycline is also effective against *P. falciparum* but should be used only for areas of mefloquine resistance. It should be taken 1–2 weeks before travel and then for 4 weeks after leaving the area.
- b. Mefloquine is recommended for areas of risk where chloroquine-resistant *P. falciparum* has been reported. The drug should be taken for 1–2 weeks before and continued for 4 weeks after travel. Resistance has been reported in both Asia and South America. A combination of proguanil and dapsone has also been used in areas with chloroquine-resistant strains of *P. falciparum*.
- c. Pyrimethamine-sulfadoxine Fansidar® and proguanil have been used, but resistance has limited their use.

5. Self-treatment

Travellers taking chloroquine alone have been advised to take 3 tablets of Fansidar® if they suspect that they have developed malaria.

6. Treatment

If the team physician suspects the diagnosis of malaria, he or she should at once consult with an infectious disease expert who is familiar with effective drug therapy for the region.

Table 12-10. Malaria chemoprophylaxis according to geographic area.

Regimen	Drug of choice	Alternative Drug
<i>Chloroquine sensitive</i> Central America Caribbean	Chloroquine	proguanil mefloquine
<i>Chloroquine resistant</i> 1. South America Amazon (Brazil) 2. Asia 3. Africa (sub Saharan) 4. SE Asia and Oceania	mefloquine mefloquine mefloquine or mefloquine mefloquine	chloroquine + pyrimethamine sulfadoxine ^a doxycycline ^b chloroquine + pyr/sulfadoxine chloroquine + proguanil + pyr/sulfadoxine ^a doxycycline

^a(Fansidar) Single-dose presumptive therapy when prompt medical attention is not available.

^bDoxycycline is recommended for those staying overnight along the Thai-Cambodian or Thai-Myanmar (Burma) borders.

F. Helminthic Infections

Diverse helminthic pathogens can inhabit the small and large bowel of humans. Helminths are multicellular, multisystem organisms, with complex life cycles. They consist of roundworms (nematodes) and flatworms (platyhelminths), which include flukes (trematodes) and tapeworms (cestodes). Most of them develop in intermediate hosts or soil before they infect humans; some are capable of autoinfections.

Warm temperatures help ova survive for a long time and facilitate the maturation of ova and larvae into infectious forms. Poor sanitation favours the spread of helminths. Infection may be caused by ingestion of contaminated food or water, or invasion of larvae through exposed skin.

The life cycle and sensitivity to therapeutic agents are often similar. In some instances ova are ingested; they develop in the small intestine, where adults reside for the remainder of their lives. Other helminths follow a complex path through the body before coming to reside in the small intestine. Eosinophilia occurs when larvae migrate through tissue, but may not be seen when adult worms are present only in the gastrointestinal tract.

The clinical signs of infection depend on the number of parasites, pathogenicity, and the immune response of the host. Tissue damage can be caused by direct toxic effects or immune responses. The signs and symptoms of infections may differ, and include abdominal pain, diarrhea, and weight loss. The individual can be infected with multiple species of parasites as well as other organisms, which may complicate the picture. In general the diagnosis is made by identifying larvae, ova, or cysts in the feces. In some instances assays have been developed to detect antigens in the stool. Chemotherapy is available for the most of the helminthic pathogens. Successful vaccines have not yet been developed.

Nematodes (roundworm)

Among the most prevalent parasites of the humans are *Ascaris lumbricoides*, *Necator americanus*, and *Ancylostoma duodenale*, which reside in the small bowel and *Trichuris trichiura*, which resides mainly in the colon. In many areas of the tropics residents can be infected with more than one of these parasites. *Ascaris* and *Trichuris* infections occur when ova are ingested in fecally contaminated food and water. Larvae of the hookworm invade the skin. An eruption may be seen at the site of entry. After a series of developmental stages, they pass through the lung, resulting occasionally in symptoms, infiltrates, and eosinophilia. These nematodes then settle down in the intestinal tract. They produce prodigious number of ova, which are excreted.

1. Signs, Symptoms, and Treatments

Infected individuals may complain of abdominal discomfort and diarrhea. As they may also be infected with other enteropathogens, it can be difficult to decide which of the parasites is responsible for their symptoms.

Clumps of *Ascaris* can cause obstruction of the small bowel. Individual ascaris, which can reach more than 30 cm in length, may migrate into the common bile duct or pancreatic duct and cause inflammation. Hookworms can produce abdominal pain, bloody diarrhea, and anemia.

Trichuris trichiura is associated with colonic lesions and chronic bloody diarrhea. These three parasites can produce malnutrition in children. The diagnosis of roundworm infection is made by finding ova in the stool. These parasites are susceptible to a number of agents such as mebendazole and albendazole.

Strongyloides stercoralis is endemic in many tropical areas. Like hookworm, larvae invade through the skin and elicit pulmonary infiltrates with eosinophilia as they migrate through the lungs. *Strongyloides* in the intestine can produce abdominal discomfort, diarrhea, and eosinophilia. Autoinfection can occur. Disseminated hyperinfection may occur in immunocompromised individuals. The diagnosis is made by finding larvae in the stool. Thiabendazole and Ivermectin are effective as treatment.

Trichinella species are prevalent worldwide. The intestinal phase occurs in the first week, after ingestion of meat infected with cysts. The larvae penetrate the small bowel epithelium and develop into the adult stage. This phase is associated with nausea, abdominal pain, and diarrhea, followed by periorbital oedema and muscle aches when larvae penetrate into muscle cells.

A number of additional roundworms should be kept in mind, such as: *Trichostrongylus* species in cattle raising areas; *Capillaria philippinensis* in areas of South East Asia and the Philippines; *Angiostrongylus costaricensis* in scattered areas of Latin America; and *Anisakiasis* in coastal areas.

Trematodes (flukes)

Schistosomiasis is the most prevalent and important of the helminthic infections. Their life cycle is complex, involving snails. They infect their human hosts by

entering the skin, or when they are consumed in uncooked food. *Schistosomiasis mansoni* is endemic throughout Africa and is found in many areas of Latin America and the Middle East. *Schistosomiasis japonicum* is found in Asia.

1. Signs, Symptoms, and Treatments

Adults reside in venules in the mesenteric plexus where they release their ova, causing mucosal inflammation in the intestines with hypertrophy and ulceration. Patients complain of abdominal pain and bloody diarrhea. Eggs reaching the liver produce granulomas, leading to fibrosis, portal hypertension, and hepatosplenomegaly.

The diagnosis is made by finding ova in the stool or in biopsy specimens, or suggested by anti-schistosomal antibodies. Mucosal friability may be seen at colonoscopy. Praziquantel is effective against all forms of schistosomiasis and Oxamniquine against *S. mansoni*.

Several flukes live within the gastrointestinal tract. *Fasciolopsis buski* is acquired when people ingest contaminated raw water plants, resulting in inflammation of the small intestine and epigastric pain, nausea, and diarrhea of varying severity. *Heterophyes heterophyes* and *Metagonimus yokogawai* cause similar symptoms.

Tapeworms

Tapeworms have complex life cycles. Adults live in the intestinal tract of their final host while larvae are found encysted in the tissue of intermediate hosts such as cattle or pigs.

1. Signs, Symptoms, and Treatments

Taenia saginata and *Taenia solium* tapeworms reach great lengths in humans but cause minimal disease. The ova of *Taenia solium* can produce cysts in the brain and other tissues, a condition known as neurocysticercosis, which is an important cause of morbidity in Latin America and in some areas of Asia and Africa.

Humans serve as both the intermediate and definitive host of the dwarf tapeworm *Hymenolepis nana*, and autoinfection is common. Light infections are often asymptomatic, but heavy infections can produce loss of appetite, abdominal discomfort, diarrhea, and anorexia. Niclosamide and praziquantel are active against adult tapeworms in the intestinal tract; praziquantel or albendazole can be used in the treatment of neurocysticercosis.

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ENDOCRINE/MENSTRUAL FACTORS

The female athlete responds to a programme of regular exercise in a similar fashion to the male. Women show improvements in aerobic capacity, strength, and “speed” to the same qualitative degree as men. However, due to differences associated with in-utero hormonal effects on myocyte stem cell number, as well as those induced by estrogen vs. androgen, women have a smaller skeleton, less muscle mass, lower hemoglobin levels, and a higher proportion of body fat. Thus, women’s world records are 7–10% lower than those of men. Female athletes generally show training-induced structural changes of lower body fat and a higher percent of muscle than untrained women.

A. Endocrine Function

The changes in body composition and energy metabolism associated with intense exercise may be responsible for a number of changes in endocrine function, particularly those related to the reproductive cycle.

1. Menarche

A number of studies have suggested that an exercise programme begun early in life may delay the onset of menarche. This has not been confirmed, but women who have not begun menarche by age 16 in the northern hemisphere (and perhaps earlier in other populations) should have an endocrine evaluation.

2. Exercise-Related Changes in the Menstrual Cycle

Exercise and its energy demands, if not compensated by adequate nutrition, may affect several cerebral neuro-transmitters and, subsequently, the hormones of the hypothalamic-pituitary-ovarian axis. These hormonal changes may be reflected in various ways: luteal phase deficiency, anovulatory cycles, and exercise-associated amenorrhea (EAA). About 2–5% of the untrained female population has one of these abnormalities; among distance runners and some athletes in other events the incidence ranges from 5–65%.

a. Luteal Phase Deficiency

In this condition, the menstrual cycle length is unchanged, but the luteal phase is shortened. Progesterone secretion is deficient, probably associated with a defective mid-cycle LH surge. The subject usually does not notice any changes, and therefore does not seek evaluation unless complications occur. These include infertility, endometrial hyperplasia, and a reduced bone mass (with stress fractures). Studies show an absence of the basal body temperature (BBT) rise (due to the lack of the LH surge), low plasma progesterone, and an abnormal endometrial biopsy. This may represent a precursor to the development of anovulation or amenorrhea. Although menstrual cycles may be within normal limits, it is uncertain whether estrogen therapy may be necessary to prevent bone mineral loss.

b. Anovulation

Anovulatory cycles may be short (less than 21 days between menses) or very long (35 to 150 days). Affected women may produce adequate estrogen, but do not have an LH pulse, and have low progesterone levels. The unopposed estrogen causes proliferative endometrial growth and may lead to irregular, heavy bleeding. This can cause iron deficiency and anemia. Management may include monthly progestin therapy during days 14–25, or oral contraceptives for sexually active women. Clomiphene may be used to induce ovulation if pregnancy is desired. However, in these cases there may not be adequate estrogen to protect bone mineral competence, and estrogen replacement or an oral contraceptive should be considered. Athletes and physicians should always be aware that clomiphene is included in the list of prohibited substances.

c. Exercise-Associated Amenorrhea (EAA)

This is the commonest type of menstrual change noted in athletes, and occurs in one of two forms:

- i. Primary amenorrhea. Primary amenorrhea is the absence of menses by age 16. This is probably due to multiple factors, including intense training from an early age, plus dietary inadequacy leading to an energy drain. Risks include a low bone density, scoliosis, and stress fractures. Amenorrhea beyond age 16 should be fully evaluated.
- ii. Secondary amenorrhea. This is defined as the absence of 3 to 12 consecutive menses. The lack of a uniform definition makes the incidence difficult to determine. About 2% to 5% of “normal” women are amenorrheic at some time. The incidence in athletes ranges from 5% to 65%, depending upon the sport and event. This condition is most common among distance runners.

The causes of amenorrhea are not well-defined, but are probably multi-factorial. Intensive training demands in the face of inadequate caloric and nutritional replacement leads to an “energy drain” that affects cerebral neuro-transmitters and the hypothalamic-pituitary-ovarian hormone system.

EAA is a type of hypothalamic amenorrhea. The gonadotrophin-releasing hormone (GnRH) pulse generator is suppressed. Many hormones that affect the GnRH pulse generator are altered by exercise. These include the endorphins, prostaglandins, catecholestrogens, serotonin, catecholamines, dopamine, cortisol, etc. These hormones in turn affect the release of LH and FSH, and thus estrogen and progesterone. A combination of the above factors, which results in an energy drain, is likely responsible for this reversible suppression of the GnRH pulse generator. The long-term consequences include infertility, a reduced bone density, stress fractures, and increased injuries.

3. Medical Assessment

Changes in the menstrual cycle associated with exercise are probably caused by a variety of complex, interrelated factors that are still under study. Menstrual

Table 13-1. Differential diagnosis of secondary amenorrhea.

Source	Abnormal Findings
<i>Uterine</i> Pregnancy Asherman's Syndrome	Positive pregnancy test Uterine scarring
<i>Pituitary</i> Prolactin-secreting adenoma Tumours Pituitary failure	Elevated prolactin Abnormal sella X-ray/CT Low FSH, LH, TSH
<i>Ovary</i> Ovarian failure Ovarian tumours Polycystic ovary syndrome (PCO)	Elevated LH, FSH Palpable mass LH/FSH = 3/1, elevated testosterone, DHEA-S
<i>Hypothalamus</i> Anorexia nervosa Tumour Exercise-induced amenorrhea	Distorted body image Abnormal sella X-ray/CT History
<i>Adrenal</i> Cushing's disease/syndrome Congenital adrenal hyperplasia	Elevated cortisol Elevated androgens, 17-OH progesterone
<i>Other Endocrine</i> Hyperthyroid Hypothyroid	Suppressed TSH Elevated TSH
<i>Medications</i> Oral contraceptives Anabolic steroids/testosterone	

changes should not automatically be attributed to the exercise programme, and other causes must be considered (Table 13-1).

EAA should be considered part of the Female Athlete Triad of disordered eating, amenorrhea, and a low estrogen state leading to decreased bone mineral content and eventually osteoporosis. Evaluation requires a physical examination and careful medical history, including the timing of menarche and changes in the menstrual cycle in relation to the initiation and intensity of training. Weight fluctuations and nutrition habits should also be correlated with menstrual changes. Concerns about body image, ideal weight, and methods of weight control must be noted. Many distance runners have adopted the "be thin to win" myth and develop a distorted perception of their body image. They acquire disordered eating patterns, including bulimia and anorexia. These behaviors may be difficult to elicit initially, so the examiner must be persistent. Athletes at risk of disordered eating should be evaluated and appropriate referrals made (Figure 13-1).



Figure 13-1. Disordered eating decision tree.

Team physicians should also be alert to other gynecologic disorders, sexual activity, sexually transmitted diseases, contraceptive practices, thyroid disorders, and other medical illnesses. Symptoms and signs of androgen excess, such as male pattern hair growth and acne (endogenous or exogenous androgens), visual changes (pituitary mass), and galactorrhea (prolactin-secreting adenoma) should also be evaluated.

4. Testing

A step-wise protocol should begin with a pregnancy test, thyroid stimulating hormone (TSH) and a prolactin level (see Figure 13-2). Androgens (testosterone, DHEA-S and androstenedione) can be measured if virilisation or hirsutism are noted. If the pregnancy test is negative, a progesterone challenge test may be started. Progesterone can be administered orally in doses of 5–10 mg daily for 7–10 days, or in a single intramuscular dose of 100–200 mg of progesterone in oil. Progesterone converts an estrogen-stimulated proliferative endometrium into a secretory one, which is shed when the progestin is stopped. Bleeding suggests anovulation rather than hypothalamic amenorrhea. The pregnancy test should precede the progesterone challenge because this hormone is teratogenic.

If the progesterone test is positive, search for causes of unopposed estrogen, including androgen excess syndromes. If there is no response, causes of inadequate estrogen must be sought, including pituitary or ovarian failure. Measure LH and FSH, which are elevated in primary ovarian failure and low in hypothalamic-pituitary disorders such as anorexia nervosa and EAA.

5. Risks of Altered Menstrual Function

Abnormal menstruation or amenorrhea are often regarded by athletes as favorable, because they believe that performance is affected by the menstrual cycle and that amenorrhea is indicative of “leanness” and fitness. Recent studies have shown, however, that serious consequences result from prolonged amenorrhea, or even a short luteal phase. This hypoestrogenic state results in an uncoupling of bone formation and resorption, with increased resorption resulting in an approximate 4% loss of trabecular bone for each year of amenorrhea. This results in a higher incidence of stress fractures, and also eventual osteoporosis. Mineralisation is only partially restored with estrogen therapy and cannot be restored with calcium administration alone. Therefore, intervention should take place within 3 to 6 months of the onset of amenorrhea.

Further, the low estrogen state also affects the vascular endothelium and may be a factor in the development of premature atherosclerosis. Also, athletes must be cautioned that EAA should not be considered a form of contraception, as ovulation may occur before menses resume.

6. Management

EAA should be regarded as a form of “energy drain,” symptomatic of several possible stresses upon the athlete. Adequate nutrition must be emphasised, with stress placed upon a balanced, high-carbohydrate diet (see Chapter 6, *Nutrition*, section C.3). There should be a calcium intake of 1500–2000 mg daily, to provide a substrate for bone mineralisation. Physical and psychological stresses should be minimised. It will be difficult if not impossible for the athlete to accept a reduced training load unless there is clear evidence of overtraining. A bone mineral density examination (DEXA scan) may be needed to demonstrate to the athlete the presence of osteopenia and the need for nutritional changes and further therapy.

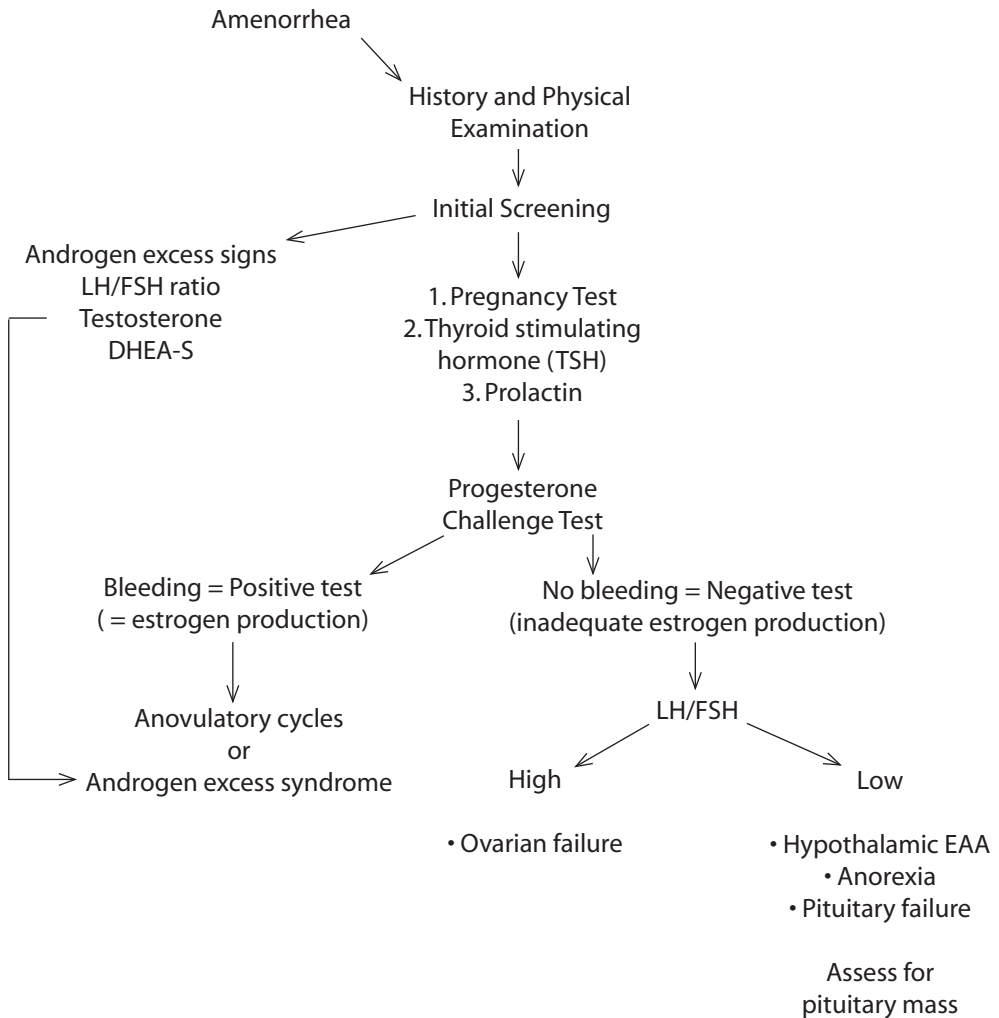


Figure 13-2. Flow chart for assessing amenorrhea.

Estrogen replacement is essential if other measures do not alleviate the condition within a few months. Estrogen therapy is best accomplished by the use of low-dose oral contraceptive pills. Although athletes may be concerned about adverse effects on performance, this is a minor problem with modern formulations and can be alleviated by trying different preparations. Recent studies show estrogen therapy has no effect on aerobic capacity, reaction time, speed, or strength. Unless there is a change in lifestyle, medications should be continued. Discontinuance may be tried, but hormonal therapy should be resumed if there is no menses in 3 to 6 months.

B. Dysmenorrhea

Pre-menstrual symptoms of bloating, weight gain, depression, and abdominal cramping affect the athlete's sense of well being and ability to perform. Exercise is helpful in reducing some of these effects, but it does not totally alleviate the

problems. Simple analgesics may be sufficient in mild cases, but non-steroidal anti-inflammatory drugs are more effective as prostaglandin inhibitors, especially if begun just prior to the anticipated onset of symptoms.

More complete control can be obtained by suppressing ovulation with hormones, such as oral contraceptives. These can be used to regulate the timing of the menstrual cycle to avoid major competitions as well as to control symptoms. Use of these agents has been shown to produce minimal if any weight gain and no changes in performance-related factors.

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GENDER VERIFICATION AND SEX REASSIGNMENT POLICY

A. History

Gender verification has been required of all female athletes since the early 1960s, when allegations surfaced that men and perhaps “hermaphrodites” were participating in women’s sports events. Although there was no strong evidence to support these claims, the IOC and other international sports governing bodies initiated gender verification procedures.

At first, athletes were physically examined by a panel of female physicians. Because this caused considerable embarrassment, athletes and officials quickly embraced the buccal smear (Barr body) method, which can separate XX (genetic female) from XY (genetic male) individuals. However, geneticists have shown that a number of genetic defects in hormone synthesis or recognition can render an XY individual anatomically and physiologically female. Labeling these women as “male” has caused irreparable harm.

In 1991, the IAAF abandoned the X-chromatin (buccal smear) test and adopted the recommendation that female and male athletes undergo a general physical examination by their team physician as part of a general “health check” prior to international competition. Thus, questions concerning sexual identity could be resolved in the athlete’s own country. This procedure, however, was nearly impossible to standardise or verify at the international level.

B. Current IAAF Policy

In 1992, the Medical Committee recommended and the Council adopted the current policy on gender verification, which states:

1. The general “health check” is strongly recommended, but no longer required.
2. Visual examination of the genitalia during the delivery of a urine specimen in the women’s doping control station is a sufficient method of determining whether the athlete is male or female. The risk of a male being discovered during the doping control procedure is sufficient deterrent to prevent males from attempting to compete as females.
3. The Medical Delegate at international competitions has the authority to initiate additional examinations if there is a question or ambiguity concerning an athlete’s gender.

There is currently concern among some athletes and officials that genitalia are not being examined adequately in doping control stations, due to lack of training of station personnel, and perhaps due to cultural constraints as well. Hence, further clarification or changes may be forthcoming. (See also Appendix 13, *Process for the Management of Gender-Related Issues*.)

C. Sex Reassignment—IOC Consensus Statement

The IAAF Medical /Anti-Doping Commission has adopted the IOC Medical Commission’s statement on sports participation for athletes who have undergone sex reassignment.

1. Before Puberty

“Individuals undergoing sex reassignment of male to female before puberty should be regarded as girls and women.” Similarly, this also applies to female to male reassignment, and they should be regarded as boys or men.

2. After Puberty

Individuals undergoing sex reassignment from male to female, or the reverse, after puberty are eligible to participate in their reassigned gender under the following conditions:

- a. Surgical anatomic changes have been completed, including external genitalia changes and gonadectomy.
- b. Legal recognition of their assigned sex has been conferred by the appropriate official authorities.
- c. Hormonal therapy appropriate for the assigned sex has been administered in a verifiable manner and for sufficient length of time to minimise gender-related advantages in sport competitions.

Further guidelines:

- a. Eligibility should begin no sooner than two years after gonadectomy.
- b. A confidential case-by-case evaluation will occur.
- c. In the event that the gender of a competing athlete is questioned, the medical delegate (or equivalent) of the relevant sporting body shall have the authority to take all appropriate measures for the determination of the gender of a competitor.

Reference

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CARDIOVASCULAR EVALUATION OF ATHLETES

Recommendations concerning participation in athletics by persons with possible cardiovascular disorders arise in three contexts: first, the eligibility of potential athletes with a known cardiovascular disorder to participate in athletics; second, the athlete with symptoms that suggest a possible cardiovascular abnormality; finally, non-symptomatic, “healthy” young people may be found to have a possible cardiac abnormality during routine pre-participation examination.

Although the risk of sudden death is small, a few athletes die from cardiovascular disease each year, and the impact of these deaths is great. Persons with severe aortic stenosis, hypertrophic cardiomyopathy, and some congenital lesions are at increased risk of sudden death during and after exercise. Thus it is the physician’s responsibility to make participation by these athletes as safe as possible. Also, the possible effects of exercise on the progression of existing heart disease must be recognised, assessed, and addressed.

A. Diagnostic Techniques

Most valvular and septal defects can be detected and diagnosed by physical examination. In order to make an informed opinion regarding sports participation, however, additional diagnostic tests are usually needed to determine the severity of the problem. The test to assess the severity of the defect is echocardiography, with electrocardiogram and chest X-ray providing additional measures of heart size. Other non-invasive tests must be used in specific situations. For example, two-dimensional and M-mode echocardiography can be used to assess chamber size, wall thickness, and valve motion. Doppler flow echocardiography is valuable in quantifying obstruction to flow or regurgitation. Radio-nuclide angiography can assist in determining coronary circulation and cardiac output.

Exercise electrocardiography may be necessary to determine an individual’s response to exercise, especially if an arrhythmia or ischemia is suspected. Exercise testing should be modified to simulate the athlete’s event or sport. If an intermittent rhythm or conduction disturbance is suspected, continuous ECG (Holter) monitoring will be needed.

Finally, cardiac catheterisation may be required to determine the degree of valvular stenosis, shunting, or pressure gradient.

B. The “Athlete’s Heart Syndrome”

Athletes who participate regularly in athletics often develop changes in their heart, circulation, and electrocardiogram that may be difficult to distinguish from those associated with true cardiac pathology. This complex of changes is often referred to as the “athlete’s heart syndrome.” It is seen most often in endurance athletes. The athlete’s heart syndrome is characterised by an increase in para-

sympathetic tone, especially vagotonia and consequent bradycardia, as well as a variety of conduction changes. Characteristics of the syndrome may include:

1. Increased left ventricular volume. This is seen as cardiomegaly during physical examination and X-ray. It is usually accompanied by a mild to moderate degree of ventricular wall thickness (hypertrophy).
2. Resting bradycardia
3. Electrocardiographic changes:
 - a. Left ventricular hypertrophy
 - b. Sinus bradycardia
 - c. Anterior wall S-T and T wave changes
 - d. Atrio-ventricular conduction abnormalities:
 - i. First degree A-V block
 - ii. Wandering pacemaker
 - iii. Second degree A-V block, of Mobitz Type I (Wenckebach) variety
 - e. Bundle branch block:

Partial or complete *right* bundle branch block (the occurrence of left bundle branch block is usually indicative of cardiac pathology).

Athletes whose training includes primarily resistive exercises, such as strength training (weight lifting) or wrestling show evidence of left ventricular wall hypertrophy, but little or no increase in ventricular volume. However, the ventricular wall thickness does not exceed 13 mm, but a “gray zone” of 13–15 mm may exist in some large male athletes, and requires evaluation by additional criteria to exclude the possibility of hypertrophic cardiomyopathy (Figure 14-1). Female athletes have not been found to have a ventricular wall thickness of more than 13 mm, but those who train extensively with high intensity resistance exercise have not been studied.

C. Athletes with Known Cardiovascular Disorders

Athletes may exhibit a wide variety of disorders, but most fall into six general categories:

1. Congenital Disorders
2. Acquired Valvular Disease
3. Cardiomyopathies and Mitral Valve Prolapse
4. Hypertension
5. Rhythm and Conduction Disturbances
6. Ischemic Heart Disease

A careful, detailed medical history can help the physician determine the nature of a disorder and advise the athlete about participation in strenuous activities.

A thorough discussion of all possible defects in each category is beyond the scope of this brief review, but the high-risk conditions in each category are as follows:

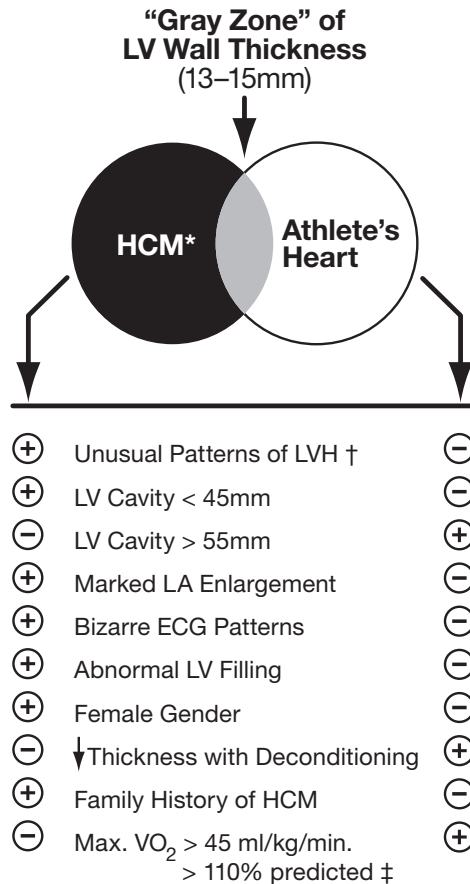


Figure 14-1. Chart showing criteria used to favour or distinguish hypertrophic cardiomyopathy (HCM) from athlete's heart when maximal left ventricular (LV) wall thickness is within shaded gray zone of overlap (i.e., 13 to 15 mm), consistent with both diagnoses. *Assumed to be the non-obstructive form of HCM (under resting conditions) in this discussion because the presence of substantial mitral valve systolic anterior motion would confirm the diagnosis of HCM in the athlete. † May involve a variety of abnormalities, including heterogeneous distribution of left ventricular hypertrophy (LVH) in which asymmetry is prominent and adjacent regions may show greatly different thicknesses with sharp transitions evident between segments, as well as unusual patterns in which the anterior ventricular septum is spared from the hypertrophic process and LV thickening may be in posterior portion of septum or anterolateral or posterior free wall or apex. ‡ Assessed with cardiopulmonary (metabolic) exercise testing. ↓ = decreased; ECG = electrocardiogram; LA = left atrial. Adapted from Maron, B. J., and D. P. Zipes (2005).

1. Congenital Heart Disease

Most congenital heart disorders are discovered in childhood, and repair will have been carried out before participation in sports. Mild defects that do not affect cardiac function or that have a good long-term prognosis should not affect the ability to participate in sports. In other cases, the ability to participate in sports will be determined by the extent of the defect, the success of the repair, and any residual

effects, such as pulmonary hypertension, chamber hypertrophy, or shunting and arterial desaturation.

2. Acquired Valvular Diseases

a. Aortic Valve Disease

i. Stenosis

Sudden death can occur in individuals with severe stenosis but is rare in those with mild degrees of obstruction. Those with mild stenosis may participate in competitive athletics, but the disorder should be re-assessed periodically with Doppler echocardiography and possible catheterisation, as the lesion may progress in severity.

ii. Regurgitation

Individuals with any degree of aortic regurgitation should not participate in vigorous exercise.

b. Mitral Valve Disorders

Athletes with mild degrees of stenosis or insufficiency, and with normal left ventricular size and normal sinus rhythm, may participate in all sports.

3. Cardiomyopathies and Mitral Valve Prolapse

a. Hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is the commonest cause of sudden death in young athletes, whereas coronary artery disease is the most frequent cause in older athletes. HCM is a relatively common genetic abnormality, occurring in approximately 0.2% (1:500) of the population. It is inherited as a mendelian autosomal dominant caused by a mutation in one of 12 genes (over 400 mutations have been found), each encoding proteins of the cardiac sarcomere that have contractile, structural, or regulatory functions. Due to the heterogeneity of the many identified mutations, there is considerable clinical variance in the presentation of this disorder. Genotyping of suspected individuals is available, but is time-consuming and expensive.

The clinical presentation of HCM usually does not occur before the late teens. The diagnosis may be suspected if there is a systolic murmur associated with outflow tract obstruction, but this is not common. Other indications are a family history of premature sudden death, new symptoms of chest pain or dyspnea, an arrhythmia, or an abnormal ECG. A major clinical problem is one of distinguishing between the cardiac changes seen in highly trained athletes, and those due to cardiac diseases such as HCM, dilated cardiomyopathy, and arrhythmogenic right ventricular cardiomyopathy (ARVC).

The ventricular wall thickness in trained athletes seldom exceeds 13 mm. However, some large male athletes who participate in resistance sports (weightlifting, wrestling) may have a ventricular wall thickness in the 13–15 mm “gray zone.” Similarly, some endurance athletes may have dilated left ventricular (LV) chambers that exceed the end-diastolic dimension of

60 mm or more, with low-normal LV function. Criteria for distinguishing between HCM and the “Athlete’s Heart” are listed in Figure 14-1.

Cautions:

Athletes with probable or diagnosed HCM should not participate in most competitive sports. Prior treatment with drugs, surgical interventions, alcohol septal ablation, an implantable pacemaker or implantable defibrillator, should not be criteria for allowing sports participation. The stresses of the competitive milieu present unique challenges for which these interventions may not be able to adapt, or the devices may malfunction. Further, the presence of an automated external defibrillator (AED) at sports activities should not be considered as a protection against sudden death, or as a rationale for allowing participation of athletes with HCM or other high-risk cardiac disorders.

b. Marfan’s Syndrome

Marfan’s Syndrome is an autosomal dominant disorder of connective tissue. Over 400 mutations have been found in the gene that encodes fibrillin-1 (FBN-1). It has an estimated prevalence of 1:5000–1:10000 in the general population.

Those with Marfan’s Syndrome are typically tall and thin, with hyper-extensible joints, mitral valve prolapse, aortic valve regurgitation, aortic dilatation, and a potential for aortic dissection. Dissection can result in sudden death. The degree of aortic dilation must be assessed by echocardiography. Individuals without aortic root dilation or mitral valve prolapse may participate in low-intensity activities; a few may engage in activities with a high dynamic and low static demands (i.e., endurance activities).

c. Mitral Valve Prolapse (MVP)

This common, usually benign lesion affects 2–3% of the general population and rarely causes sudden death. Contra-indications to exercise include:

- i. History of syncope
- ii. Family history of sudden death due to MVP
- iii. Chest pain, worse with exercise
- iv. Repetitive ventricular ectopy or supra-ventricular tachycardia, worse with exercise
- v. Moderate to marked mitral valve regurgitation
- vi. Associated dilated aorta, with Marfan’s Syndrome
- vii. A prior embolic event

d. Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

This is a rare disorder, but it has been associated with sudden death in young athletes. It has a broad phenotypic spectrum, and is characterised by a loss of myocytes in the right ventricular myocardium, with replacement by fatty or fibro-fatty cells. There is thinning of the ventricular wall, and it is often associated with myocarditis. Diagnosis is difficult—a family history,

ventricular tachyarrhythmias, and T wave inversion in leads V1–V3 are diagnostic clues. These individuals should not participate in competitive sports.

e. Congenital Long Q-T Syndrome

The diagnosis of this syndrome is complex, as many individuals with this genotype may have a normal corrected Q-T interval (QTc), while normal persons (approximately 25%) may have a Q-T interval of 440 ms, which previously has been considered as the upper limit of normal. A Q-T interval of 470 ms in males, and 480 ms in females requires further study. A patient with the Long Q-T Syndrome (LQTS) and a resting QTc of 500 ms or longer is considered at risk for severe arrhythmias.

There are at least 150 mutations in 7 cardiac ion-channel genes that are responsible for 75% of these cases, and a diagnostic test is now available. Other diagnostic criteria include: family history, T wave abnormalities, and symptoms such as syncope, in addition to the finding of a long QTc.

All competitive sports should be prohibited in those who have had an out-of-hospital cardiac arrest, or a suspected LQTS-precipitated syncopal episode. Athletes who are genotype-positive/phenotype-negative (i.e., mutations but no symptoms and a non-diagnostic QTc) may be allowed to compete. Non-symptomatic patients with a baseline QT (QTc 470 ms in males, 480 ms in females) should be restricted to mild activities.

f. Other Inherited Arrhythmias

Other inherited arrhythmias include the Brugada Syndrome (J wave in V1-3, ST segment elevation, and negative T wave), Short Q-T Syndrome (QTc less than 300 ms), and Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT), which are caused by mutations in various conduction and receptor systems. All of these may precipitate a sudden fatal arrhythmia during exercise or with a rise in body temperature, and must disqualify the subject from participation in vigorous exercise until further studies are carried out and the extent of the abnormality is determined.

g. Anomalous L. Coronary Artery from the R. Sinus of Valsalva

This rare condition is difficult to diagnose pre-mortem, but can cause sudden death. After surgical repair, participation is permissible if an exercise ECG is normal.

4. Hypertension

Secondary causes of hypertension, including coarctation of the aorta and polycystic renal disease, must be excluded.

Sports participation depends on the extent of organ involvement (left ventricular hypertrophy, renal, or eye disease) and the ability to control blood pressure with appropriate medications. Calcium channel blockers or angiotension-converting enzyme inhibitors are the most suitable for those who participate in athletics. Blood pressure control should be assessed during exercise or immediately post-exercise.

5. Arrhythmias/Conduction Disturbances

Arrhythmias are often transient and difficult to diagnose, and are not always reproduced by exercise. Autonomic “tone,” which is higher in trained athletes, affects arrhythmia occurrence. Changes in sinus node function and low-grade A-V conduction delays may result from chronic endurance exercise itself.

a. Ventricular Pre-excitation

This disorder is characterised by a P-R interval of less than 120 ms and a QRS that exceeds 120 ms and has a slurred, slowly rising onset and secondary ST-T changes. It is often associated with tachy-arrhythmias, i.e., the Wolff-Parkinson-White Syndrome (WPW). The tachycardia is usually 150–250 beats per minute.

An individual whose accessory pathway has a short refractory period (less than 200 ms) is at risk of very rapid heart rates and sudden death. Those with a history of palpitations or syncope should be examined to exclude structural heart diseases, and have 24-hour monitoring and an electro-physiologic study to determine the properties of the accessory pathway. Recently, surgery has been used to ablate the accessory pathway.

b. Ventricular Dysrhythmias (PVCs, Ventricular Tachycardia, Ventricular Flutter)

Individuals whose dysrhythmia is precipitated by exercise, who have underlying heart disease, or who have the “Prolonged QT Syndrome” should not participate in intense exercise.

c. Heart Block

First-degree heart block and Mobitz Type I block (Wenckebach) may occur as a result of endurance training itself. Athletes with these disorders who have no heart disease and whose block is not worsened by exercise may compete in sports. Persons with Mobitz Type II block or *acquired* complete (3rd degree) heart block should be treated with a pacemaker. Those with *congenital* complete block, a satisfactory response to exercise, and no dysrhythmias may participate in athletics.

d. Bundle Branch Block

Partial Right Bundle Branch Block (BBB) is common in endurance athletes. Left Bundle Branch Block is more often associated with structural heart disease, especially in older patients. Athletes with complete BBB, no structural defects, and no ventricular dysrhythmias may participate in all sports.

6. Atherosclerotic Coronary Artery Disease

Atherosclerotic coronary artery disease (CAD) is the commonest cause of cardiac events in older athletes, especially those over age 35–40. Although exercise has beneficial effects on the healthy as well as those with diagnosed CAD, vigorous exercise transiently increases the risk of a cardiac event (myocardial infarction or

sudden death). Plaque rupture or plaque erosion may be responsible for these events, even though the coronary arteries may not have been significantly narrowed.

Known risk factors such as hypertension, hyperlipidemia, obesity, and a positive family history, as well as other unknown factors, must be considered in evaluating the older adult athlete. As older athletes who have exercise-related CAD events may have less extensive vascular disease than the average, standard risk assessment is more difficult. Variations in the resting and exercise ECG may make interpretation difficult. Further, more precise imaging techniques such as computerised tomographic calcium scoring may identify early atherosclerotic lesions. Coronary calcium increases substantially with age, so that nearly 50% of males age 40 and older show measurable calcium in their coronary arteries. Presently, there are no clear guidelines as to how well coronary calcification per se can be used as an indicator to govern or restrict exercise in non-symptomatic individuals. However, progressively increasing calcification is associated with rising CAD risk.

The prognosis for patients with known CAD worsens depending on the extent of disease, left ventricular systolic dysfunction, inducible ischemia, and electrical instability. Therefore, older persons who contemplate or participate in a competitive athletics programme should have a maximal exercise test that simulates as closely as possible the metabolic demands of the training programme and competitive event, even though such testing cannot entirely replicate the actual stresses of training and competition. Any evidence of LV dysfunction, ischemia, or electrical instability should lead to a limitation of physical activity and further cardiovascular evaluation.

D. Athletes with Symptoms Suggestive of Cardiovascular Disorder

Athletes with known heart disease may exhibit symptoms of syncope, faintness, or palpitations, which suggest a cardiac origin. A careful history and physical examination are needed to clarify the condition; for example, to differentiate among vagal fainting, hyperventilation, and true syncope. Careful auscultation may detect valvular or other congenital defects. Selected non-invasive tests may also be necessary to clarify the problem. A baseline 12-lead ECG and an echocardiogram can reveal conduction or structural abnormalities. An exercise ECG may be needed to elicit the underlying problem, and 24-hour monitoring is often used if an arrhythmia is suspected.

Continued participation in sports should depend on the findings. Restrictions on participation may be necessary until the nature of the problem is identified.

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ASTHMA AND EXERCISE-INDUCED BRONCHOSPASM (EIB)

A. Definitions

According to the American Thoracic Society, asthma is a clinical lung disease characterised by reversible airway obstruction as a consequence of a wide variety of stimuli. The term “current asthma” is used when at least one asthmatic episode occurred during the last year.

Asthma is characterised by a variety of symptoms, including dyspnoea, shortness of breath, wheezing, cough, excess mucus, breathlessness, and chest tightness. The symptoms might be mild or severe; intermittent or continuous; and more frequent in the morning and at night. Upper or lower airway obstructions are reversible with or without therapy.

The generic definition can also include bronchial or airway hyperresponsiveness (BHR or AHR), that is, an above-normal airway constriction upon exposure to physical stimuli or sensitising agents.

A transient airway narrowing occurring in susceptible individuals during or after exercise is defined as EIB (exercise induced bronchospasm) when observed in a non-asthmatic and non-atopic population; or EIA (exercise induced asthma) when including asthmatic individuals. Here we will use EIB to also include those with EIA.

Exercise induced asthma or bronchospasm is a transient, reversible, and intermittent narrowing of small and large airways, occurring about 5–15 minutes after intense exercise (aerobic activity more than anaerobic), performed for 8–10 minutes. A post-exercise fall in forced expiratory volume in 1 second of $>10\%$ is required for diagnosis. After an attack lasting 20–60 minutes, a complete recovery usually occurs.

In 50% of affected athletes, a refractory phase starts less than 1 hour after initial exercise; this may last up to 3 hours, with less than half the intensity of exercise bronchospasm. For this reason, the warm up period might be useful to ensure a refractory phase during competition. Sometimes, 6–8 up to 12 hours after the exercise, a late phase, less severe state with cough and wheezing is observed in 30% of subjects with EIB. The aetiology of the refractory period is probably due to the depletion of local mediators, or increased sympathetic activity.

EIB occurs in 12–15% up to 20–25% of the normal population, increasing to 35–40% in subjects with allergic rhinitis or hay fever and/or eczema, and up to 90% of those with asthma. The variability of the statistical data depends mainly on the method used (clinical or laboratory evaluation or epidemiologic questionnaires based on self reported symptoms or statistics performed in main competitions, based also on declared use of beta-2 agonists).

Sometimes, EIB is erroneously diagnosed in athletes who have respiratory stridor during inspiration, typical of vocal cord dysfunction (VCD, statistically up to 5%, by paradoxical narrowing of the vocal cords during inspiration or as described by patients, while “getting air in”), or with other upper respiratory disorders. Anxiety and hyperventilation syndrome may mimic asthma or EIB.

EIB is more prevalent in female athletes, and twice as prevalent in endurance athletes as in sprinters, jumpers and throwers in track and field. Furthermore, evidence exists that top athletes are at an increased risk of developing asthma or EIB during their career, particularly in endurance events.

High-level exercise performed on a regular basis by previously unaffected elite athletes, particularly in endurance activities, is liable to increase the incidence of asthma and airway hyperresponsiveness (AHR).

B. Aetiology

A basic genetic component associated with atopy is presupposed, but many other factors are involved and play a significant role both in asthma and AHR, particularly in athletes:

1. Recurrent airways infections or inflammations, in which many local cells and their related mediators are involved (mast cells, macrophages, eosinophils, neutrophils, etc.). Endurance athletes are particularly susceptible to upper respiratory tract infection and impaired function of the immune system after intense repeated training, with decreased activity of lymphocytes, neutrophils, macrophages, and natural killer (NK) cells and diminution of lymphokines and IgA levels.
2. Drug allergy (aspirin or NSAIDs are common), food allergy, or other allergic or anaphylactic medical conditions.
3. Environmental exposure to airborne allergens. Prolonged hyperventilation during intense training increases the possibility of exposure to different allergens, mainly in seasonal asthmatic patients. These include pollens or other allergens, or irritant pollutants such as cigarette smoke and sulphur dioxide (SO₂), carbon monoxide (CO), nitrous and nitric oxide (NO₂, NO₃) in smog, and—very important in track and field—also herbicides, pesticides, insecticides, and fertilisers. Training sessions in contaminated situations can cause chronic bronchial inflammation triggered by smoke, pollens, irritants, and allergens (IgE production).
4. Cold and dry air inhalation. Hyperventilation, mainly during endurance efforts, induces a loss of heat and water in the bronchial system: the bronchoconstriction response is due both to the direct effect of “cooling” in bronchial mucosa, and to the “hyperosmolarity” of mucosal fluid, inducing the release of pro-inflammatory mediators (histamine, leukotrienes, prostaglandins, neutrophil chemotactic factors). In addition, the “rewarming” after exercise generates a vasodilator effect on the pulmonary capillary system with vascular bronchial congestion, increased vascular permeability and oedema with bronchoconstriction.
5. Parasympathetic hyperactivity is a typical compensatory response to a prolonged sympathetic stimulation by intense and prolonged training sessions, which may increase the bronchomotor tone (normally and basically the parasympathetic system is dominant over the sympathetic system in the

bronchial apparatus), and may explain the higher incidence of AHR in endurance athletes.

C. Diagnosis

1. Self-Reported Symptoms

Self-reported symptoms are not valid to confirm the presence of asthma or EIB. The misinterpretation of post-exercise fatigue, prolonged recovery time, under-performances, or inadequate training might induce an erroneous diagnosis.

2. History

The history is very important, and permits, as a first step, the evaluation and discrimination of other possible diseases:

- a. Chronic bronchitis, pulmonary fibrosis, lymphadenopathy
- b. Seasonal asthma
- c. Infectious diseases
- d. Laryngeal dysfunction; throat or nasal problems; upper airways obstruction; allergic rhinitis; hypertrophic turbinates; nasal polyps; sinusitis; hay fever
- e. Cardiac problems with effort dyspnoea
- f. Allergic conditions (medicines, food) or anaphylactic reactions
- g. Blood disorders (anaemia)
- h. Thyroid dysfunction
- i. Gastro-oesophageal reflux
- j. Anxiety

3. Basal Screening

Many clinical instruments are useful in basal screening, including:

- a. Family history of asthma and allergic diseases
- b. Personal history of atopy or asthma; use of bronchodilator substances; other, also occasional, allergic disorders
- c. Physical and complete examination (lung auscultation is often quite normal)
- d. Evaluation of presence or influence of environmental factors (cigarette smoke, smog, pollen, animal dander, dust mites, cold or dry air)
- e. Blood cell count and erythrocyte sedimentation rate (infections)
- f. Chest radiography (chronic pulmonary diseases, fibrosis, lymphadenopathy, cardiomegaly)
- g. Skin allergy tests, IgE and RAST (allergic problems)
- h. Heart clinic and instrumental evaluation (ECG and/or echocardiogram)

4. Diagnostic Tests

Pulmonary function testing is the next diagnostic step. Note that the pulmonary function tests should be performed on days free from asthma symptoms and concurrent problems (rhinitis, allergies, sinusitis), without any prior short-acting

bronchodilator therapy in the last 8–12 hours, and any long-acting bronchodilator therapy in the last 12–24 hours. Antileukotrienes should be suspended in the last 48–96 hours prior to the test; cromolyn compounds in the last 12–24 hours; and anti-histamines in the last 48 hours. In addition, inhaled steroids should not be administered on the day of the test; no caffeine should be taken the morning of the test; and no vigorous exercise should be performed in the last 4–6 hours prior to the test, or preferably on the day of the test at all.

Laboratory basal spirometry is good for a simple and standard evaluation (Flow Volume Curve, FVC, FEV₁, FEV₁/FVC, PEF, FEF 25–75).

In athletes with solitary EIB, the basal FEV₁ will be normal, over 80% of predicted normal value, while in asthmatic athletes it will be lower than 80%.

For daily, practical, and on-field self-evaluation, athletes can use small and inexpensive peak flow meters.

Exercise challenge tests are commonly performed in the laboratory, using a treadmill, stationary cycle, or rowing equipment; sometimes an exercise test performed running free and outside, as in natural conditions, is practical but is less controlled in its intensity. However, for athletes, the chance to perform the specific field test of their sport is optimal for diagnosis. For exercise challenge tests:

- a. No warm up is allowed to avoid a direct bronchospasm.
- b. The intensity and duration of aerobic exercise should be 80–90% of the maximum heart rate for 6–10 minutes, preferably without crossing the anaerobic threshold, to avoid the exhaustion of the athlete and the release of catecholamines.
- c. The inhaled air should have a relative humidity below 50% and an ambient temperature of 20°–25°C; the use of inhaled cold air during the exercise test increases the sensitivity in diagnosing EIB, without decreasing specificity.
- d. After the exercise test, spirometric measurements are conducted every 3–5 minutes for 15–30 minutes, and possibly after 4–12 hours in late responders.
- e. A decrease of FEV₁ of 10% or more is considered positive for EIB. The severity of disease is classified as mild (10–20%), moderate (20–40%), and severe (more than 40%). Reversibility of bronchospasm after an inhaled bronchodilator will confirm the diagnosis.

Pharmacological challenge tests can be used to assess asthma, while being less specific and sensitive for EIB. The methacholine test, which is more sensitive and less specific, induces bronchoconstriction mainly in the distal bronchi, and increases airway inflation pressure and contraction of the trachealis muscle; histamine causes airway obstruction by activation of bronchial smooth muscle and mediator receptors.

Both for histamine and methacholine, cut-off levels are defined, in terms of concentration or cumulated dose, able to induce a 20% reduction of FEV₁ (PC-20 or PD-20).

Osmotic tests include the dry powdered mannitol inhalation challenge and the nebulised hypertonic saline challenge. Increased doses of the stimulating substance are followed by pulmonary function tests until cut-off levels. Both tests act by altering the osmolarity of airway surface liquid (ASL) with release of mediators from sensitised mast cells. The osmotic tests are sensitive and specific, easy to perform, and economical.

The eucapnic voluntary hyperventilation (EVH) challenge can, in susceptible individuals, induce bronchoconstriction with increased ventilation rate by drying the airway surface liquid and changing the osmolarity of the mucosal surface membrane components. In athletes, the respiration rate achieved should be almost 85% of MVV (maximal ventilation rate), about 35 times FEV₁. The test is performed with a dry air mixture containing 4.5% CO₂ to ensure eucapnia and protect from the hypocapnia induced by hyperventilation; this latter, in fact, may cause indistinct bronchoconstriction both in EIB positive and negative subjects. The inspired air can also be chilled, although chilling is not necessary.

The bronchodilator test is an indirect but limited method used to detect airway obstruction by airways reversibility to inhaled short-acting bronchodilators (terbutaline or salbutamol), when the resting FEV₁ is below 70% of normal. The response is variable, and the cut-off criteria for a positive test is 15% FEV₁ increase or, as recently stated by the European Respiratory Society, 12% increase of FEV₁ expressed as % predicted.

D. Non-Pharmacologic Treatment

Non-pharmacologic treatment should be the primary focus of the problem, and is based on the following criteria:

1. Education of athletes, coaches, and families is a primary component: the disorder is frequent and common in the population, and does not limit performance when adequately treated.
2. Prevention is the main method: avoiding cold or dry air, training indoors, or covering the mouth and nose with a scarf during winter, or using a mask to warm and humidify the air may be helpful.
3. Predominant nasal, more than mouth breathing, may reduce EIB by inhaling more warmed and humidified air.
4. Exercise conditioning to lower ventilation rate decreases airway responsiveness.
5. Warming up well before exercise, with repeated short and high intensity exercises, may induce a refractory period and partially reduce the need for pre-medication.
6. Avoid training sessions that risk possible exposure to environmental situations of airborne allergens or irritants.
7. Avoid consumptions of foods with possible allergens, at least in the last 4 hours before an exercise or competition session.

8. Reduce training when exacerbation periods of rhinitis, sinusitis, or allergy are present.
9. Stop training during viral respiratory infections or acute bronchial exacerbations.

E. Pharmacologic Treatment

The treatment of seasonal allergic rhinitis by non-sedating anti-histamines or local intranasal glucocorticosteroids is an important step in preventing bronchial hyperreactivity. Generally, the correct use of medications, when needed, will help the patients to train well and live better, without pharmaceutical addiction and with limited adverse effects, both of which are possible risks of inhaled beta-2 agonists.

Proper inhalation of the medication will result in a better deposition of the substances into the bronchial system: after slow expiration, controlled breathe while inhaling to total lung volume and holding the breath for 10 seconds will enhance deposition. A pause of 30 second between the two inhalations will increase the quantity of drug delivered into the lungs.

While describing beta-2 agonists, we will mention only those permitted by inhalation with a previous exemption request:

Short acting beta-2 agonists (salbutamol/albuterol, terbutaline) administered by inhalation 20–30 minutes before exercise have a peak bronchodilator effect within 60 minutes, and maximal duration of 3–4 hours. They are effective in 90% of EIB, but sometimes they induce tachyphylaxis, worsening asthma, and, with continuous use, become ineffective in 2–3 years.

Long acting beta-2 agonists (formoterol and salmeterol) need to be administered well before exercise, because they have their maximal effect in 4 hours and may last up to 12 hours, permitting the prevention of EIB and asthma attacks in prolonged exercise sessions and in the night (particularly when used with glucocorticosteroids).

The beta-2 agonists, both short and long acting, work by increasing intracellular concentration of cyclic adenosine monophosphate (cAMP), which modulates the relaxation of bronchial smooth muscle and inhibits the release of mediators from mast cells. While beta-2 selective, they interact (short acting more than long acting) with alpha- and beta-1 receptors, causing tachycardia, tremors, and palpitations.

Inhaled glucocorticosteroids (beclomethasone dipropionate, budesonide, fluticasone propionate, flunisolide, mometasone, triamcinolone acetonide, etc.) are practically long-term drugs. They are not effective on an as-needed basis, but are useful for chronic asthma, or in EIB if used for at least one month, when the beta-2 agonists are not effective when used individually. They suppress the production of cytokines, reducing the eosinophils, and prevent inflammatory mediators release. Adverse effects are dysphonia, oral irritation, and candidiasis.

Cromolyn compounds (Cromolyn sodium and Nedocromil sodium) are mast-cell stabilisers without bronchodilator effects, which are useful—when inhaled 20 minutes before exercise—in preventing EIB and EIA symptoms in 80% of patients. They may also prevent the late-phase response of EIB. They can be used many times a day in the absence of adverse effects, and may be additive when beta-2 agonists

are not completely effective. Adverse effects include bad taste, throat irritation, cough, nausea, vomiting, and abdominal pain.

Antileukotrienes are used for long-term asthma therapy. Montelukast and Zafirlukast are leukotriene antagonists, while Zileuton is an inhibitor of biosynthesis by 5-lipoxygenase; they are effective in preventing EIA in chronic asthma. Leukotrienes are products of arachidonic acid metabolism, and increase eosinophil migration, mucus production, and bronchial oedema, with bronchoconstriction response 1000 times greater than histamine. The antileukotrienes, normally used orally for long-term therapy, offer 24-hour protection with very low adverse effects (dyspepsia, nausea).

Inhaled anticholinergics (ipratropium bromide, oxitropium bromide, tiotropium bromide) are not useful in preventing EIB, and are only used in chronic obstructive pulmonary diseases and chronic bronchial infections complicated by asthmatic attacks.

Antihistamines (astemizole, cetirizine, chlorpheniramine, desloratidine, phexophenadine, terphenadine, etc.) exhibit little effect; they might be useful in allergic asthma only when the disease is combined with allergic rhinitis due to air pollens. The oral dryness and some mild sedative effects are not helpful for athletes. Further, they are sometimes combined with stimulants not permitted by antidoping rules. Ketotiphen is a more widely used antihistamine substance; like cromolyn compounds, it acts on mast cells as a stabiliser, with fewer adverse effects.

Antibiotics are used in the presence of infections—sinusitis, rhinitis, bronchitis—that increase bronchial sensitivity.

Methylxanthines (theophylline and aminophylline or theophylline ethylenediamine) work by decreasing the metabolism of cAMP by inhibition of phosphodiesterase; they also have adrenergic effects. They are used systemically (oral or injections), mainly in chronic asthma and in acute exacerbations, under strict medical control for possible adverse effects (tachyarrhythmias, hypertension, peptic ulcers, hyperthyroidism, seizure disorders).

Systemic glucocorticosteroids and *beta-2 agonists* are restricted to more serious conditions and are administered in emergency situations by medical prescription.

Epinephrine (adrenaline) is administered subcutaneously, under strict medical control, only in life-threatening emergency situations.

F. Doping Related Issues

Since 1993, the beta-2 agonists have been submitted to restrictions by the IOC and IAAF, based on their possible effects as anabolic agents (see Chapter 15, *Drugs in Sports/Doping Control*). However, a large increase in the number of athletes with declared use of beta-2 agonists was reported in high-level competitions and Olympic Games, perhaps as a consequence of an incomplete or erroneous diagnosis and, sometimes, due to an over-use of and/or acquired tolerance to beta-2 agonists, with an under-use of inhaled glucocorticosteroids.

Further, the beta-2 agonists can potentially have positive ergogenic effects on skeletal muscle anabolism, and on aerobic and anaerobic performance. For this reason the control of their use in accordance with sport rules is becoming more strict. Also, glucocorticosteroids by inhalation have been subjected since January 1, 2004 to more limited use, and only after application for a therapeutic use exemption (TUE).

In accord with IAAF and World Anti Doping Agency (WADA) rules and IAAF Procedural Guidelines for doping control:

1. Glucocorticosteroid by inhalation are banned “in” competition, and submitted in competition to the “Abbreviated Therapeutic Use Exemption” application process.
2. Beta-2 agonists by inhalation, are prohibited both “in” and “out” of competition, and are submitted to the “Abbreviated Therapeutic Use Exemption” application process.
3. Systemic glucocorticosteroids in competition and systemic beta-2 agonists both in and out of competition are banned, and a “Standard Therapeutic Use Exemption” application must be applied for before use.

Note that particularly in the case of an abbreviated TUE application to the IAAF for the use of beta-2 agonists by International Level athletes, and in accord with the IAAF beta-2 agonists protocol, the medical notification justifying the therapeutic necessity must be accompanied by the athlete’s detailed medical records and by a positive bronchial provocation test with graphic evidence, conforming to the IAAF specific protocol (see also Chapter 15, *Drugs in Sports/Doping Control*).

All of the doping rules are updated yearly by IAAF, in accordance with the yearly WADA prohibited list. The current list may be found on the websites of WADA (www.wada-ama.org), IAAF (www.iaaf.org), and IOC www.olympic.org. The list goes into effect three months after publication.

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HEADACHES AND EXERCISE-INDUCED ANAPHYLAXIS

A. Headaches

Headaches may occur with exercise, and are usually benign. However, exercise-related headache may occasionally signal the presence of an underlying organic disorder.

1. Benign Exertional Headache

These may occur with exercise, but also with coughing, sneezing, bending, and lifting. No distinct pattern is observed. Pain can occur at any site, and radiate in a variety of patterns.

2. Effort Migraine

Migraine can be precipitated by strenuous exercise, and has an atypical migraine pattern, with sudden onset, scotoma, photo-sensitivity, nausea, and vomiting, and unilateral headache that is often retro-orbital. It usually lasts 20–60 minutes. Treatment should initially consist of analgesics, but drugs such as ergotamine tartrate may be required.

3. Organic Disorders

a. Pheochromocytoma

Pheochromocytoma may appear as a pounding headache of sudden onset, accompanied by nausea and vomiting. The flushing, sweating, and tremor typical of this disease may be obscured by exercise. A rise in blood pressure is often associated with the symptoms, even at rest. Urinary VMA and catechols are elevated.

b. Vascular Malformations and Tumours

These rarely present with exertional headache. However, persistent headache, either diffuse or localised, warrants a thorough neurological examination, and possibly an EEG and or CT scan.

B. Exercise-Induced Anaphylaxis

This is an unusual type of physical allergy, but one that can become life-threatening.

1. Manifestations

- a. Flushing, sensation of warmth
- b. Giant urticaria (wheals 10–15 mm diameter)
- c. Angioedema, respiratory distress, vascular collapse

2. History

There is a prior personal or family history in over half of the subjects. The condition occurs unpredictably, and under variable circumstances associated with exercise. Prior food allergy may be a factor.

3. Differential Diagnosis

EIA must be distinguished from Cholinergic Urticaria (CU). This latter is uniformly brought on by exposure to heat or exercise, and has punctate urticaria and wheezing, but is not life-threatening.

4. Treatment

Stop exercise at once, and administer epinephrine subcutaneously. Subjects should be taught to carry and administer their own medication. Exercise with a companion who is aware of the problem and who can administer the drug.

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DRUGS IN SPORTS

A. Introduction

The use of drugs in sports has had a long and well documented history. The IAAF became the first International Sporting Federation to prohibit doping, doing so in 1928 by including the following wording in its Handbook:

“Doping is the use of any stimulant not normally employed to increase the pposer of action in athletic competition above the average. Any person knowingly acting or assisting as explained above shall be excluded from any place where these rules are in force or, if he is a competitor, be suspended for a time or otherwise from further participation in amateur athletics under the jurisdiction of this Federation.”

However, soon after World War II it became clear that many athletes in a wide range of sports were using drugs to enhance their performance. This practice was widespread, while measures to resist such use were limited. The death of athletes in cycling events in 1960 and 1967, which was traced to doping, aroused strong reactions and the demand was made that sports authorities should intervene.

The Council of Europe first defined doping in 1963 as the use of certain substances or the use of methods that could have the effect of unnaturally improving the physical and/or mental condition of a contestant before or during competition and thus enhance his or her sports performance. Although the danger associated with the use of drugs was the initial incentive for doping control, doping is now no less regarded as cheating and unethical.

In general, athletes have used drugs both to speed up development during training and to enhance their performance in the competition itself. The International Olympic Committee (IOC) established a Medical Commission in 1967 and approved a ban on doping in 1968. The Committee defined the list of Prohibited Substances and the first tests for stimulants were performed at the Winter Olympics in 1968. Steroids only became detectable in 1974.

International Sports Federations (IFs) also initiated doping controls at their own events and IAAF became the first IF to perform systematic out-of-competition tests, which are considered the most effective form of testing.

The authorities (such as National Anti-Doping Agencies) of various countries have taken over doping controls within their own borders and in some countries doping has been banned by law, and is therefore also punishable as such.

In 1998, several doping incidents occurred in various parts of the world, and several governments declared their dissatisfaction with the current doping control situation. As a result, the IOC called a conference in Lausanne in early 1999 with the participation of National Olympic Committees (NOCs), Government authorities, IFs, and athletes. More stringent measures were approved and “The Lausanne Declaration” was issued and the decision was made to establish The World Anti-Doping Agency (WADA), with the participation of the IOC, IFs, and governments.

The purpose of WADA is to harmonise and strengthen anti-doping actions and rules across all sports and countries.

At a conference in Copenhagen in March 2003 “The World Anti-Doping Code” was formally approved and replaced the IOC anti-doping rules. The “Code” sets stricter anti-doping aims, rules, and controls than were previously in effect.

WADA also took over the role of publishing the list of Prohibited Substances, which is continually under review and formally updated on 1 January each year. A substance or method is considered for inclusion on the List if WADA determines it meets any two of the following three criteria: a) it is performance enhancing, b) be dangerous to the athlete’s health, c) be contrary to the spirit of sport. A substance or method can also be added to the list if WADA determines it has the capacity to mask the use of other prohibited substances or methods.

The scope of the doping problem continues to shift and expand as new compounds, chemical and pharmacological classes, and methods of doping are embraced by succeeding generations of athletes, coaches, and unscrupulous chemists. As a result, anti-doping analytical laboratories have evolved continuously to face these new challenges.

In order to meet numerous legal challenges to the anti-doping rules and regulations, more-detailed legal definitions and clarifications were devised. Today’s anti-doping regulations, testing procedures, and adjudication processes are developed and refined constantly by teams of legal, medical, and pharmacological-analytical experts.

Substances may be prohibited either in-competition, or both in- and out-of-competition, depending upon their short- or long-term potential to enhance performance or endanger the athlete’s health. The determination as to whether a substance or method is banned, or whether it is to be sought either in-competition or both in- and out-of-competition, or neither, may be updated from year to year, depending on current scientific knowledge and an evaluation of the extent to which a substance is being abused. Hence, it is essential that sports physicians, athletes, coaches, and sports administrators regularly apprise themselves of the contents of the WADA Code and List of Prohibited Substances and Methods, which is revised and published at least annually. Each new version comes into effect on 1 January.

The List is now published and revised by WADA and is made available to each member and published on the website (www.wada-ama.org) and also on the IAAF website (www.iaaf.org). WADA determination of the prohibited list and methods is not subject to legal challenge.

B. Prohibited Substances and Methods: A Brief History

Following is a brief history and selection of how the Prohibited List has evolved over the years. The classes of substance are listed by their current WADA Prohibited List titles, however these too have changed often.

The Prohibited List (the List) originally consisted only of:

- Psychomotor stimulant drugs
- Miscellaneous central nervous system stimulants

- Narcotic Analgesics
- Anabolic Steroids

At first, few individual drugs were specifically named on the List, which remained divided by substance class. The List has subsequently been under constant revision and changed considerably, with more individual substances mentioned within each group and some even deleted. In addition to Classes of Substances, the categories Prohibited Methods, Substances Prohibited Out-of-Competition, Specified Substances, and Substances Prohibited in Particular Sports have been added.

1. Anabolic Steroids

- a. *Anabolic steroids* initially included on the prohibited list were: methandienone, stanozolol, esters of nortestosterone, and related compounds.
- b. Testosterone and its esters were added in 1979 and in 1986 the list was expanded to include any substance that increased the testosterone:epi-testosterone (T:E) ratio, which was initially set at 6:1 by the IOC, but decreased to 4:1 in 2005. Further investigation may be needed to determine whether the ratio is due to a physiological or pathological condition.
- c. In 1993 Anabolic Agents were divided into two categories:
 - i. Androgenic Anabolic Steroids; and
 - ii. Other Anabolic Agents (e.g. beta-2 agonists)
- d. Dihydrotestosterone was added to the list in 1995 and deemed to be positive where the concentration in urine exceeds the range of normal values. A sample is not regarded positive for dihydrotestosterone or testosterone where an athlete proves that the ratio or concentration is attributable to pathological or physiological condition. This principle was also applied in 2002 to any Prohibited Substance that is capable of being produced by the body naturally. In 2000 it was stated that evidence obtained from metabolic profiles and/or isotopic ratio measurement may be used to draw definite conclusions.
- e. Epitestosterone was listed in 1995 under Prohibited Techniques.

2. Hormones and Related Substances

- a. 1990 the hCG, ACTH and hGH were included in the list
- b. In 1992 Erythropoietin (EPO) was added
- c. In 2000 the following were added as well: Pituitary and Synthetic Gonadotrophins (LH), Insulin-like Growth Factor (IGF-1) and Insulin (but permitted to treat insulin-dependent diabetes), all other erythropoiesis-stimulating proteins.
- d. In 2005 Mechano-Growth Factors (MGFs) were added to the prohibited list.

3. Beta-2 Agonists

- a. Beta-2 Agonists first appeared on the list in 1993 as Other Anabolic Agents with clenbuterol as an example.

- b. Salbutamol and Terbutaline by inhalation were permitted in 1995 as exceptions, when prescribed for therapeutic purposes by properly qualified medical personnel.
 - c. Salmeterol was added to the “permissible list” in 1996 and Formoterol in 2000.
 - d. In 2004 it was emphasised that all beta-2 agonists are prohibited except that formoterol, salbutamol, salmeterol, and terbutaline are permitted by inhalation only to prevent and/or treat asthma and exercise-induced asthma/bronchoconstriction. However, a concentration of salbutamol greater than 1000 ng/mL is considered an adverse finding, despite the granting of a Therapeutic Use Exemption (TUE). In 2004 Beta-2 Agonists were classified as a Specified Substance.
4. Agents with Anti-Oestrogenic Activity
- a. Agents with Anti-Oestrogenic Activity appear on the list in 2002 as Clomifene, cyclofenil, tamoxifen, and aromatase inhibitors (prohibited in males only).
 - b. In 2004 Selective Oestrogen Receptor Modulators were added.
5. Diuretics and Other Masking Agents
- a. Masking Agents were placed on the list as their own category in 2004, including but not limited to: diuretics, epitestosterone, probenecid, and plasma expanders.
 - b. In 2005 the name of the group was changed to Diuretics and other Masking Agents, and alpha-Reductase Inhibitors were added to the list.
 - c. Diuretics were classified as Specified Substance in 2004, but deleted from that list in 2005.
6. Stimulants
- a. Stimulant substances were initially classified in two groups as indicated previously;
 - i. in 1985 they were all grouped together and simply known as Stimulants;
 - ii. in 1990 they were divided into Amphetamines and Stimulants;
 - iii. and in 2004 they were again merged into one group as Stimulants.
 - b. Caffeine was placed on the list in 1983 but removed in 2004.
 - c. Ephedrine was on the original list and in 2002 a positive finding was stipulated as concentration of more than 10 mcg/ml in urine. In 2004 it became classified as a Specified Substance.
 - d. Cocaine was included in the list in 1990.
7. Narcotic Analgesics
- a. Only a few narcotic analgesics were listed initially.
 - b. In 1979 Codeine was “permitted for therapeutic uses.”
 - c. In 2006 analgesics are prohibited only in competition.

8. Cannabinoids

Cannabinoids were placed on the list in 2004 and classified as a specified Substance. They are prohibited in competition only.

9. Glucocorticosteroids

- a. Corticosteroids were put on the list in 1992 and prohibited by oral, intramuscular or intravenous application.
- b. Rectal administration was added in 2000.
- c. In 2006 Glucocorticosteroids are only prohibited in competition, and are classified as a Specified Substance.

10. Enhancement of Oxygen Transfer

- a. Prohibited techniques were first introduced as such in 1990 and defined as “blood doping and techniques.”
- b. Erythropoietin (EPO) was added in 1992; and blood plasma expanding products (e.g. HES) and artificial oxygen carriers were included in 2000.
- c. In 2002 any sort of blood transfusion was prohibited as well as all erythropoiesis-stimulating proteins.
- d. The term Blood Doping was changed to Enhancement of Oxygen Transfer in 2004 and defined as the use of autologous, homologous or heterologous or red blood products of any origin, other than for medical treatment.
- e. Use of products that enhance the uptake, transport, or delivery of oxygen is prohibited.

11. Chemical and Physical Manipulation

- a. Originally entitled “Techniques,” these were defined as the use of substances that alter the integrity of the urine samples such as catheterisation, urine substitution and/or tampering, or inhibition of renal excretion, e.g. by probenecid and related compounds.
- b. Epitestosterone was added in 1995, diuretics in 1996, and bromantan in 1998.
- c. In 2004 the term Prohibited Techniques was changed to Pharmacological, Chemical and Physical Manipulation.
- d. In 2005 intravenous infusions were prohibited except as legitimate acute medical treatment.

12. Gene Doping

Gene Doping appears on the list for the first time in 2004.

13. Substances Prohibited in Particular Sports

Substances Prohibited in Particular Sports were added to the List in 2005 and consist of Alcohol and Beta-blockers. Each International Sporting Federation is able to choose whether or not they prohibit these substances. Neither of these two substances are currently prohibited by the IAAF.

14. Specified Substances

Specified Substances were introduced in 2004 as substances that are susceptible to unintentional anti-doping rule violations because of availability in medicinal products. Athletes who test positive to these substances may receive a reduced sanction if they can prove they were not taken to enhance performance.

Examples of substances included on this list are: Ephedrine, L-methylamphetamine, Cannabinoids, Inhaled Beta-2 Agonists (except clenbuterol), Diuretics (deleted 2005), Probenecid, and Glucocorticosteroids.

DOPING CONTROL

A. Extracts of IAAF Anti-Doping Rules

At the 44th IAAF Congress in Paris, August 2003, it was decided to accept the World Anti-Doping Code as a basis for the fight against doping and adapt the existing IAAF anti-doping rules to the Code. Following is a brief outline of the IAAF Anti-Doping Rules.

Note: The latest IAAF Rules are available on the IAAF website and these should be referred to when required, as changes may occur.

1. Anti-Doping Roles

The Anti-Doping Rules must be incorporated into each Member Federation's rules, and specify that all athletes and support personnel are bound by them. Members must guarantee that national-level testing complies with IAAF rules.

IAAF Anti-Doping Organisation is overseen by the IAAF Council, which delegates authority to the Medical and Anti-Doping Commission, Doping Review Board, and the IAAF Anti-Doping Administrator.

The Commission meets once to twice a year to review its anti-doping activities. It publishes the Procedural Guidelines, which are fully compliant with the standards set by WADA. The Commission implements and monitors anti-doping programmes and education, publishes updated information on prohibited substances and methods, health consequences of doping, doping control procedures, and athletes' rights and responsibilities. It also grants TUEs, and establishes guidelines for the selection of athletes to be tested.

The Doping Review Board determines whether or not exceptional circumstances exist in the case of adverse analytical findings, decides on referral to the Court of Arbitration for Sports (CAS), and on acceptance of sanctions made by other sporting bodies.

The IAAF Anti-Doping Administrator is responsible for day-to-day management, implements the anti-doping programme, and conducts the results management process.

2. Testing and Sample Analysis.

Testing is done both in-competition and out-of-competition, and any athlete may at any time be subject to testing. It is a condition of membership of the IAAF that each Member includes in its constitution the authority for them as well as for IAAF to conduct out-of-competition testing on its athletes and that the IAAF has the authority to test at all National Championships if required. In-competition testing is the responsibility of IAAF at certain International Competitions and selection is based on final position/random basis and may include target testing and athletes breaking an Area and/or World Records.

The IAAF concentrates its out-of-competition testing efforts on international-level athletes who are required to provide their whereabouts information directly to

the IAAF in order for this testing to occur. There are sanctions in place if athletes do not comply with this requirement.

Analysis of samples shall be done at WADA-accredited laboratories to detect prohibited substances and methods and samples remain the property of the IAAF. When analysis indicates the presence of a prohibited substance or substances, the WADA laboratory informs the IAAF of this fact.

Results management in a case of an adverse analytical finding, is first done by reviewing the case to determine if there is a Therapeutic Use Exemption (TUE) on file, or a departure from the required collection process has occurred. If this is not the case, the athlete must be informed of the adverse finding and can ask for analysis of the B sample at which they are entitled to have a representative present. If the B sample confirms the A sample the athlete must bear the costs of this analysis.

An Anti-doping rule violation, i.e. doping is defined as:

- a. the presence of a banned substance or metabolite in an athlete's body;
- b. the use or attempted use of prohibited substances or methods;
- c. the refusal or failure to submit to doping control or undergo an anti-doping test;
- d. 3 missed out-of-competition tests in a period of 5 years;
- e. tampering or attempting to tamper with any part of the doping process;
- f. possession of a prohibited substance or methods, without TUE;
- g. trafficking in prohibited substance or method;
- h. the administration of a prohibited substance or method or assisting in an anti-doping violation;
- i. competing, or attempting to compete, whilst suspended or ineligible.

Standards of proof of doping are the burden of the IAAF or other prosecuting authority, which must establish that an anti-doping rule violation has occurred. The proof is a positive sample analysis by a WADA-accredited laboratory.

3. Disciplinary Procedures, Hearings, and Appeals

Disciplinary procedures if a doping violation has been committed involve:

- a. provisional suspension,
- b. hearing,
- c. sanction or exoneration.

Provisional suspension shall be imposed by the IAAF or Member if there is no adequate explanation for the cause of the adverse analytical finding, or this provisional suspension can be accepted voluntarily.

In all doping cases the athlete has the right to a hearing of their case before the relevant disciplinary body or tribunal, however he or she must confirm in writing within 14 days of notification that they would like a hearing, otherwise it is assumed that the athlete accepts that a violation has occurred. The hearing before a tribunal must be held within 2 months from the date of notification to the athlete. The athlete has the right to legal counsel, to call witnesses, and to have an interpreter (at the

athlete's expense). The decision of the tribunal will be sent to the IAAF. If the IAAF does not agree with the sanction the case will be reviewed by the IAAF Doping Review Board, which decides whether it appeals to CAS; if so, it may re-impose suspension. The athlete also has a right of appeal to CAS

Exceptional circumstances may occur but do not include:

- a. allegation that the substance was given to the athlete by another person without his or her knowledge;
- b. that the substance was taken by mistake;
- c. that it was contained in contaminated food supplements; or
- d. that it was prescribed by support personnel in ignorance.

If an athlete provides substantial evidence or assistance to IAAF or National Federation in other doping cases, this may be accepted as an exceptional circumstance and result in a reduced sanction.

If a National Tribunal decides in the case of an international athlete that exceptional circumstances exist, it shall be referred to the IAAF Doping Review Board. If the Doping Review Board determines that there are no exceptional circumstances, this determination is binding on the relevant tribunal, which shall impose sanctions. The athlete has the right to appeal to CAS.

4. Disqualification, Sanctions, and Return to Competition Requirements

Disqualification of the athlete shall be automatic from the event when a violation occurs in connection with an in-competition test, with forfeiture of titles, awards, medals, points, and prize and appearance money. When the athlete is part of a relay team the team shall be automatically disqualified. All competitive results from the date the sample was provided shall be annulled with resulting consequences for the individual and the team (unless fairness requires).

Sanctions against individuals:

- a. If prohibited substance is found or prohibited methods established the sanctions:
 - i. First violation, minimum 2 years;
 - ii. Second violation: ineligibility for life.
- b. For refusal to submit to doping or tampering with doping control:
 - i. First violation: minimum 2 years ineligibility;
 - ii. Second violation: ineligibility for life.
- c. For 3 missed out-of-competition tests or other whereabouts violations:
 - i. First violation: one year ineligibility;
 - ii. Second and subsequent violations: two years ineligibility.
- d. For trafficking or administration of prohibited substance or methods:
 - i. Ineligibility for life.

Elimination, reduction, or replacement of ineligibility period can be reduced to half of the minimum period and if life sanction to 8 years, where there are exceptional circumstances, such as no fault or provided substantial evidence or assistance.

Specified substances include a few medications, acknowledged to be susceptible to unintentional violation because of their general availability in medicinal products and not intended to enhance performance. For specified substances the following sanctions apply:

- a. First violation: public warning and disqualification from the event to maximum 1 year;
- b. Second violation: 2 years ineligibility;
- c. Third violation: ineligibility for life.

Commencement of ineligibility period shall start on the date of the hearing decision, with the period of any provisional suspension credited against the total period.

Status during ineligibility is such that no athlete or support personnel may participate in competition or activity other than education programmes whilst ineligible. While ineligible, the athlete is not entitled to any payment by virtue of appearance and/or performance. If he or she receives any payment contrary to this rule the athlete shall not be entitled to return to competition until it has been repaid.

Requirements for return to competition are that after any period of 2 years ineligibility the athlete shall undergo 3 out-of-competition tests at his or her cost with at least 4 months between each test, and immediately prior to the end of the period must undergo testing for the full range of prohibited substances and methods. If any of these tests reveal an adverse finding, it constitutes a separate violation leading to sanctions as appropriate. If the athlete has complied with these rules he or she shall automatically be re-eligible after the period has ended.

5. Member Federation Reporting Obligations and Sanctions against Members

- a. Members should report to the IAAF within 14 days any adverse finding and the name of the athlete associated with that finding.
- b. Members should report to the IAAF any TUE granted to their athletes.
- c. Members should report to the IAAF within the first 3 months of each year on the doping control conducted during the previous year.

Sanctions against members may be taken by the council against any Member in breach of the Anti-Doping Rules, such as:

- a. failure to guarantee athletes' eligibility;
- b. failure to hold a hearing within 2 months;
- c. failure to assist IAAF in whereabouts information;
- d. failure to report an adverse analytical finding.

If a Member is deemed to be in breach of its obligations the Council may, for instance:

- a. suspend or caution the member;
- b. issue fines;
- c. withhold grants; or
- d. exclude the Member's athletes from competitions.

B. Extracts of IAAF Procedural Guidelines for In- and Out-of-Competition Testing

Following is a brief outline of the IAAF Procedural Guidelines for Doping Control. All athletes and support personnel should acquaint themselves with the IAAF Procedural Guidelines for Doping Control, which should be followed as far as is reasonably practicable.

Note: The latest IAAF Procedural Guidelines can always be found on the IAAF website and these should be referred to when required as changes can occur.

1. In-Competition Testing

The Doping Control Station shall be clean and clearly identified and consist of a waiting room, working room, and WCs (men and women) equipped with all necessary material. Only authorised persons are allowed in the Doping Control Station.

Selection of athletes to be tested shall be done on a final position and/or random basis. Sample collection shall be conducted on any athlete who has broken or equalled an Area and/or World record. Tests for rh-EPO shall be conducted on any athlete who has broken or equalled Area or World record in races of 60 m upwards including multi-events and walks. Notification of the athlete shall be done appropriately by the Doping Control Officer (DCO) and the athlete's identity must be confirmed. The DCO shall inform the athlete:

- a. which type of sample collection he or she is required to undergo;
- b. his or her right to an assistant/representative;
- c. that he or she must remain within the sight of the DCO;
- d. that he or she must report to the doping station in no later than 60 minutes.

The athlete shall sign an appropriate form to accept the notification.

The DCO shall consider any request by the athlete to delay reporting to the Doping Control Station or to leave after reporting for testing for the following reasons only if the athlete can be continuously chaperoned:

- a. medals ceremony;
- b. media commitments;
- c. further competition;
- d. warm down;
- c. medical treatment;
- d. locating an appropriate witness.

The urine sample collection process shall be explained to the athlete after his or her arrival and the athlete is offered a choice of sample collection vessels when he or she is ready to provide a sample. The athlete shall check that all seals are intact and if not satisfied select another.

After selection, only the athlete and DCO of the same gender shall proceed to the WC. The DCO shall witness the sample leaving the athlete's body and record this fact. The athlete is required to disrobe as necessary for the witnessing to take place.

Athletes shall provide no less than 75 ml of urine. For EPO testing no less than 100 ml is required. Where the volume is insufficient the athlete shall be asked to provide more urine but the originally obtained sample shall be kept in a sealed container. The athlete shall remain under observation and be able to drink. When the athlete is ready to provide the remaining amount needed, the sample collection procedure shall be repeated.

Once a sample has been provided, the athlete him or herself shall pour the provided urine into the bottles he/she has previously selected. A small amount is retained to measure specific gravity. The athlete seals the bottles. The DCO tests the specific gravity; 1.010 or higher is recommended. If the specific gravity is too low the athlete is required to provide a further sample, but not until after one hour has elapsed. The collection procedure shall be the same. The athlete shall have fulfilled his or her duty to submit to doping control only after having delivered the required volume of acceptable urine, irrespective of the time necessary for this.

Blood sample collection may be required and begins after the procedure has been explained to the athlete and he or she has signed the consent form. If the athlete refuses this procedure, it shall be regarded as refusal to submit to doping control. The athlete may nevertheless be required to provide a urine sample.

The athlete chooses a sampling kit from a selection. The Collection official shall provide proof of his or her qualification to withdraw blood. The sample should be taken from a superficial vein only from the arm or hand. No more than 25 ml should be withdrawn and no more than three attempts made. The athlete is only entitled to refuse if the above criteria are not fulfilled.

2. Out-of-Competition Testing (OOCT)

The testing pool is established by IAAF, which may appoint a third party to conduct the testing. Registered athletes may be subject to no advance notice OOCT at any time. Athletes are required to inform IAAF of their whereabouts and notify IAAF of changes. If the athlete fails to provide the information or is unable to be located 3 times in 5 years, he or she will be evaluated for anti-doping rule violation.

Selection of athletes for testing is done by IAAF by random and targeted methods. Notification of athletes shall usually be the no advance notice method for OOCT. Only exceptionally will there be advance notice. For no advance notice OOCT, the DCO shall make reasonable attempts to locate and notify the selected athlete and include attempts at alternative times and locations. If the athlete cannot be contacted it shall be reported to the IAAF. When in exceptional cases advance notice is given, the DCO shall arrange with the athlete a time and place for the testing. It is the athlete's responsibility to check that there is no confusion over the agreed time and location. Identification of each party and the same sample procedures shall be applied as in competition.

Following the sample collection, any behaviour by the athlete or associated persons that may compromise the sample collection shall be recorded. The athlete shall have the opportunity to document any concerns. Detailed information shall be recorded on the Doping Control Form, such as: time of notification and sampling,

athlete's name, date of birth, gender, address and discipline, the code number and name of responsible officials, and signature of the athletes and officials.

C. Extracts of IAAF Therapeutic Use Exemptions (TUEs)

Athletes with a medical condition that requires the use of medication on the prohibited list can apply to IAAF or their National Authority for permission for their use, i.e. a Therapeutic Use Exemption or TUE. A TUE will be granted only in cases of clear need in accordance with the following criteria:

- a. that the application was submitted no less than 21 days before competition (this applies for standard applications, not for abbreviated ones);
- b. that the athlete would experience a significant impairment to his or her health if the substance was withheld;
- c. that the use would produce no additional enhancement other than return to normal health. The use of a substance to increase "low-normal" levels of hormones is not acceptable;
- d. there is no reasonable alternative;
- e. that the necessity for use is not a consequence of prior non-therapeutic use of any prohibited substance.

A TUE will not be granted if it might give the athlete a competitive advantage.

A retroactive TUE will not be granted except in cases where:

- a. emergency treatment was necessary;
- b. due to exceptional circumstances, there was insufficient time to submit an application.

A TUE will only be considered following the receipt of a completed application form and relevant documents and a statement by a physician attesting to the necessity of the substance and why an alternative medication cannot be used. A specific IAAF Commission of a minimum of three members reviews each application. No member of the Commission shall decide on a TUE from his or her own country. The decision of the commission will be conveyed to the athlete and respective authorities in writing. Each TUE will be for a specified duration. A TUE may be cancelled by the IAAF at any time; the athlete can nevertheless reapply or appeal this decision.

An abbreviated TUE process is used for Beta-2 agonist by inhalation and most glucocorticosteroids. An application for use of Beta-2 agonists by International athletes shall be accompanied by detailed medical records and provocation test results (see Chapter 14, Part 2, *Asthma and Exercise-Induced Bronchospasm*).

References

The Prohibited List is now revised and published by WADA. The current list may be found on the web sites of WADA (www.wada-ama.org) and the IAAF (www.iaaf.org).

Appendix 1

Olympic Movement Medical Code

In force as from 1 January 2006

PREAMBLE

"Fundamental Principles of Olympism

1 Olympism is a philosophy of life, exalting and combining in a balanced whole the qualities of body, will and mind. Blending sport with culture and education, Olympism seeks to create a way of life based on the joy of effort, the educational value of good example and respect for universal fundamental ethical principles.

2 The goal of Olympism is to place sport at the service of the harmonious development of man, with a view to promoting a peaceful society concerned with the preservation of human dignity."

Olympic Charter, September 2004

1. The Olympic Movement, in accomplishing its mission, should take care that sport is practised without danger to the health of the athletes and with respect for fair play and sports ethics. To that end, it takes the measures necessary to protect the health of participants and to minimise the risks of physical injury and psychological harm. It also protects the athletes in their relationships with physicians and other health care providers.
2. This objective can be achieved only through an ongoing education based on the ethical values of sport and on each individual's responsibility in protecting his or her health and the health of others.
3. The present Code recalls the basic rules regarding best medical practices in the domain of sport and the safeguarding of the rights and health of the athletes. It supports and encourages the adoption of specific measures to achieve that objective. It complements and reinforces the World Anti-Doping Code and reflects the general principles recognised in the international codes of medical ethics.
4. The Olympic Movement Medical Code is intended to apply to the Olympic Games, the various championships of the International Federations and all competitions to which the International Olympic Committee (IOC) grants its patronage or support, and to all sport practised within the context of the Olympic Movement, either during training or during competition.

1. General Principles

1.1. Athletes are entitled to the same fundamental rights as all patients in their relationships with physicians and health care providers, in particular the right to respect for:

- a. their human dignity;
- b. their physical and mental integrity
- c. the protection of their health and safety;

- d. their self-determination; and
- e. their privacy and confidentiality.

1.2. The relationship between athletes, their personal physician, the team physician and other health care providers must be protected and subject to mutual respect. The health and the welfare of athletes must prevail over the sole interest of competition and other economic, legal or political considerations.

2. Information

Athletes have the right to be informed in a clear and appropriate way about their health status and their diagnosis; preventive measures; proposed medical interventions, together with the risks and benefits of each intervention; alternatives to proposed interventions, including the consequences of non-treatment for their health and for their return to sports practice; and the prognosis and progress of treatment and rehabilitation measures.

3. Consent

3.1. The voluntary and informed consent of the athletes is required for any medical intervention.

3.2. Particular care should be taken to avoid pressures from the entourage (e.g. coach, management, family, etc.) and other athletes, so that athletes can make fully informed decisions, taking into account the risks associated with practising a sport with a diagnosed injury or disease.

3.3. Athletes have the right to refuse or to interrupt a medical intervention. The consequences of such a decision must be carefully explained to them.

3.4. Athletes are encouraged to designate a person who can act on their behalf in the event of incapacity. They can also define in writing the way they wish to be treated and give any other instruction they deem necessary.

3.5. With the exception of emergency situations, when athletes are unable to consent personally to a medical intervention, the authorisation of their legal representative or of the person designated by the athletes for this purpose is required, after they have received the necessary information.

When the legal representative has to give authorisation, athletes, whether minors or adults, must nevertheless assent to the medical intervention to the fullest extent of their capacity.

3.6. The consent of the athletes is required for the collection, preservation, analysis and use of any biological sample.

4. Confidentiality and Privacy

4.1. All information about an athlete's health status, diagnosis, prognosis, treatment, rehabilitation measures and all other personal information must be kept confidential, even after the death of the athlete.

4.2. Confidential information may be disclosed only if the athlete gives explicit consent thereto, or if the law expressly provides for this. Consent may be presumed

when, to the extent necessary for the athlete's treatment, information is disclosed to other health care providers directly involved in his or her health care.

4.3. All identifiable medical data on athletes must be protected. The protection of the data must be appropriate to the manner of their storage. Likewise, biological samples from which identifiable data can be derived must be protected.

4.4. Athletes have the right of access to, and a copy of, their complete medical record. Such access excludes data concerning or provided by third parties.

4.5. Athletes have the right to demand the rectification of erroneous medical data.

4.6. An intrusion into the private life of an athlete is permissible only if it is necessary for diagnosis, treatment and care, and the athlete consents to it, or if it is legally required. Such intrusion is also permissible pursuant to the provisions of the World Anti-Doping Code.

4.7. Any medical intervention must respect privacy. This means that a given intervention may be carried out in the presence of only those persons who are necessary for the intervention, unless the athlete expressly consents or requests otherwise.

5. Care and Treatment

5.1. Athletes have the right to receive such health care as is appropriate to their needs, including preventive care, activities aimed at health promotion and rehabilitation measures. Services should be continuously available and accessible to all equitably, without discrimination and according to the financial, human and material resources available for such purpose.

5.2. Athletes have the right to a quality of care marked both by high technical standards and by the professional and respectful attitude of health care providers. They have the right to continuity of care, including cooperation between all health care providers and establishments which are involved in their diagnosis, treatment and care.

5.3. During training and competition abroad, athletes have the right to the necessary health care, which if possible should be provided by their personal physician or the team physician. They also have the right to receive emergency care prior to returning home.

5.4. Athletes have the right to choose and change their own physician, health care provider or health care establishment, provided that this is compatible with the functioning of the health care system. They have the right to request a second medical opinion.

5.5. Athletes have the right to be treated with dignity in relation to their diagnosis, treatment, care and rehabilitation, in accordance with their culture, tradition and values. They have the right to enjoy support from family, relatives and friends during the course of care and treatment, and to receive spiritual support and guidance.

5.6. Athletes have the right to relief of their suffering according to the latest recognised medical knowledge. Treatments with an analgesic effect, which allow an athlete

to practise a sport with an injury or illness, should be carried out only after careful consideration and consultation with the athlete and other health care providers. If there is a long-term risk to the athlete's health, such treatment should not be given.

Procedures that are solely for the purpose of masking pain or other protective symptoms in order to enable the athlete to practise a sport with an injury or illness should not be administered if, in the absence of such procedures, his or her participation would be medically inadvisable or impossible.

6. Rights and Duties of Health Care Providers

6.1. The same ethical principles that apply to the current practice of medicine apply to sports medicine. The principal duties of the physicians and other health care providers include:

- a. making the health of the athletes a priority;
- b. doing no harm.

6.2. Health care providers who care for athletes must have the necessary education, training and experience in sports medicine, and must keep their knowledge up to date. They have a duty to understand the physical and emotional demands placed upon athletes during training and competition, as well as the commitment and necessary capacity to support the extraordinary physical and emotional endurance that sport requires.

6.3. Athletes' health care providers must act in accordance with the latest recognised medical knowledge and, when available, evidence-based medicine. They must refrain from performing any intervention that is not medically indicated, even at the request of the athletes, their entourage or another health care provider. Health care providers must also refuse to provide a false medical certificate concerning the fitness of an athlete to participate in training or competition.

6.4. When the health of athletes is at risk, health care providers must strongly discourage them from continuing training or competition and inform them of the risks. In the case of serious danger to the athlete, or when there is a risk to third parties (players of the same team, opponents, family, the public, etc.), health care providers may also inform the competent persons or authorities, even against the will of the athletes, about their unfitness to participate in training or competition.

6.5. Health care providers must oppose any sports or physical activity that is not appropriate to the stage of growth, development, general condition of health, and level of training of children. They must act in the best interest of the health of the children or adolescents, without regard to any other interests or pressures from the entourage (e.g. coach, management, family, etc.) or other athletes.

6.6. Health care providers must disclose when they are acting on behalf of third parties (e.g. club, federation, organiser, NOC, etc.). They must personally explain to the athletes the reasons for the examination and its outcome, as well as the nature of the information provided to third parties. In principle, the athlete's physician should be informed.

6.7. When acting on behalf of third parties, health care providers must limit the transfer of information to what is essential. In principle, they may indicate only the athlete's fitness or unfitness to participate in training or competition. With the athlete's consent, the health care providers may provide other information concerning the athlete's participation in sport in a way compatible with his or her health status.

6.8. At sports venues, it is the responsibility of the team or competition physician to determine whether an injured athlete may continue in or return to the competition. This decision may not be delegated to other professionals or personnel. In the absence of the competent physician, these individuals must adhere strictly to the instructions that he or she has provided. At all times, the priority must be to safeguard the health and safety of athletes. The outcome of the competition must never influence such decisions.

6.9. When necessary, the team or competition physician must ensure that injured athletes have access to specialised care, by organising medical follow-up by recognised specialists.

Chapter II: Protection and Promotion of the Athlete's Health during Training and Competition

7. General Principles

7.1. No practice constituting any form of physical injury or psychological harm to athletes is permissible. The members of the Olympic Movement ensure that the athletes' conditions of safety, well-being and medical care are favourable to their physical and mental equilibrium. They must adopt the necessary measures to achieve this end and to minimise the risk of injuries and illness. The participation of sports physicians is desirable in the drafting of such measures.

7.2. In each sports discipline, minimal safety requirements must be defined and applied with a view to protecting the health of the participants and the public during training and competition. Depending on the sport and the level of competition, specific rules are adopted regarding the sports venues, the safe environmental conditions, the sports equipment authorised or prohibited, and the training and competition programmes. The specific needs of each athlete category must be respected.

7.3. For the benefit of all concerned, measures to safeguard the health of the athletes and to minimise the risks of physical injury and psychological harm must be publicised in order to benefit all those concerned.

7.4. The measures for the protection and the promotion of the athletes' health must be based on the latest recognised medical knowledge.

7.5. Research in sports medicine and sports sciences is encouraged. It must be conducted in accordance with the recognised principles of research ethics, in particular the Helsinki Declaration adopted by the World Medical Association (Edinburgh, 2000), and the applicable law. It must never be conducted in a manner

which could harm an athlete's health or jeopardise his or her performance. The voluntary and informed consent of the athletes to participate in such research is required.

7.6. Advances in sports medicine and sports science must not be withheld, and must be published and widely disseminated.

8. Fitness to Practise a Sport

8.1. Except when there are symptoms or a significant family medical history, the practice of sport for all does not require undergoing a fitness test. The choice to undergo such a test is the responsibility of the personal physician.

8.2. For competitive sport, athletes may be required to present a medical certificate confirming that there are no apparent contraindications. The fitness test should be based on the latest recognised medical knowledge and performed by a specially trained physician.

8.3. A pre-participation medical test is recommended for high level athletes. It should be performed under the responsibility of a specially trained physician.

8.4. Any genetic test that attempts to gauge a particular capacity to practise a sport constitutes a medical evaluation to be performed solely under the responsibility of a specially trained physician.

9. Medical Support

9.1. In each sports discipline, guidelines must be established regarding the necessary medical support depending on the nature of the sports activities and the level of competition.

These guidelines must define, but not be limited to, the following points:

- the medical coverage of training and competition venues and how this is organised;
- the necessary resources (supplies, premises, vehicles, etc.);
- the procedures in case of emergencies;
- the system of communication between the medical support services, the organisers and the competent health authorities.

9.2. In the case of a serious incident occurring during training or competition, there must be procedures to provide the necessary support to those injured, by evacuating them to the competent medical services when needed. The athletes, coaches and persons associated with the sports activity must be informed of those procedures and receive the necessary training for their implementation.

9.3. To reinforce safety in the practice of sports, a mechanism must exist to allow for data collection with regard to injuries sustained during training or competition. When identifiable, such data must be collected with the consent of those concerned, and be treated confidentially and in accordance with the recognised ethical principles of research.

Chapter III: Adoption, Compliance and Monitoring

10. Adoption

10.1. The Code is intended to apply to all the members of the Olympic Movement, in particular the IOC, the International Sports Federations and the National Olympic Committees (hereafter the Signatories). Each Signatory adopts the Code according to its own procedural rules.

10.2. The Code is first adopted by the IOC. It is not mandatory but desirable that the other members of the Olympic Movement adopt it.

10.3. A list of all Signatories will be made public by the IOC.

11. Compliance

11.1. The Signatories implement the applicable Code provisions through policies, statutes, rules or regulations according to their authority and within their respective spheres of responsibility. They undertake to make the principles and provisions of the Code widely known, by active and appropriate means. For that purpose, they collaborate closely with the relevant physicians' and health care providers' associations and the competent authorities.

11.2. The Signatories ensure that the physicians and other health care providers caring for athletes within their spheres of responsibility act in accordance with this Code.

11.3. Physicians and other health care providers remain bound to respect their own ethical and professional rules in addition to the applicable Code provisions. In the case of any discrepancy, the most favourable rule that protects the health, the rights and the interests of the athletes shall prevail.

12. Complaints Procedure

12.1. Each Signatory designates a competent body to deal with complaints concerning alleged violations of the applicable Code provisions and with all other situations brought to its attention concerning the implementation of the Code. This body must have the power to take sanctions against the person or organisation at fault or to propose sanctions or the necessary measures to other authorised bodies.

12.2. The IOC Medical Commission designates a committee (hereafter: Complaints Committee), composed of three of its members, to deal with all cases of alleged violations of the applicable Code provisions occurring during the Games. This Committee also acts as a body to review decisions taken by the competent bodies of the Signatories pursuant to the Code. A request for a review may be submitted to this Committee by the person or organisation sanctioned, as well as by the claimant.

12.3. Decisions taken by the Complaints Committee in the first instance may be submitted to the IOC Executive Board for review. Decisions taken by the Complaints Committee as a review body and those taken by the IOC Executive Board are final.

12.4. The Signatories establish the necessary procedural rules, including the applicable sanctions in the event of a violation of the applicable Code provisions.

The competent bodies of the Signatories and the Complaints Committee have the power to act upon the filing of a complaint or under their own authority.

13. Monitoring

13.1. The IOC Medical Commission oversees the implementation of the Code and receives feedback relating to it. It is also responsible for monitoring changes in the field of ethics and best medical practice and for proposing adaptations to the Code.

13.2. The IOC Medical Commission may issue recommendations and models of best practice with a view to facilitating the implementation of the Code.

Chapter IV: Scope, Entry into Force and Amendments

14. Scope

14.1. The Code applies to all participants in the sports activities governed by each Signatory, in competition as well as out of competition.

14.2. The Signatories are free to grant wider protection to their athletes.

14.3. The Code applies without prejudice to the national and international ethical, legal and regulatory requirements that are more favourable to the protection of the health, rights and interests of the athletes.

15. Entry into Force

15.1. The Code enters into force for the IOC on 1 January 2006. It applies to all Olympic Games, starting with the 2006 Games in Turin.

15.2. The Code may be adopted by the other members of the Olympic Movement after this date. Each Signatory determines when such adoption will take effect.

15.3. The Signatories may withdraw acceptance of the Code after providing the IOC with written notice of their intent to withdraw.

16. Amendments

16.1. Athletes, Signatories and other members of the Olympic Movement are invited to participate in improving and modifying the Code. They may propose amendments.

16.2. Upon the recommendation of its Medical Commission, the IOC initiates proposed amendments to the Code and ensures a consultative process, both to receive and respond to recommendations, and to facilitate review and feedback from athletes, Signatories and members of the Olympic Movement on proposed amendments.

16.3. After appropriate consultation, amendments to the Code are approved by the IOC Executive Board. Unless provided otherwise, they become effective three months after such approval.

16.4. Each Signatory must adopt the amendments approved by the IOC Executive Board within one year after notification of such amendments. Failing this, a Signatory may no longer claim that it complies with the Olympic Movement Medical Code.

Adopted by the IOC Executive Board in Lausanne on 27 October 2005

Appendix 2

Planning International Travel

A. Before Departure

1. *Travel schedule.* Work with the Federation administration to arrange a travel schedule that minimises the effects of jet lag.
2. *Facilities.* Work with the administration to ensure that adequate medical care spaces (including sports psychology) are included in the team housing allocation.
3. *Licensure.* Determine whether temporary medical licensure is required by the host country, and comply if necessary.
4. *Drug importation.* Determine whether the host country requires permission for the importation of medications, and complete applications if necessary.
5. *Environmental health.* Review international health information resources to determine whether any unusual health situations or infectious disease outbreaks exist in the host country, and take appropriate preventive measures (immunisations, medications, etc.).
6. *Supplies and Equipment.* Work with the medical staff to ensure that adequate medical supplies and equipment are on hand to support the number of athletes and other team members.
7. Athlete Health
 - a. *Fitness.* Review athlete medical records, and, if possible, conduct examinations to ensure that athletes are fit and able to compete at the highest levels.
 - b. *Immunisations.* Ensure that all athletes have up-to-date immunisations, i.e., tetanus, hepatitis, influenza, etc., and any others that may be required for health in the host country.
 - c. *Medications.* Review all athlete medications, including over-the-counter, supplements, etc., which the athlete may be taking to be sure that none contain banned substances.

If restricted substances are being used, be sure that an appropriate Therapeutic Use Exemption has been filed and approved. If not, complete the required application and submit to IAAF.
 - d. *Hygiene and Jet Lag.* Educate athletes and staff concerning measures for personal hygiene, including food selection, hand washing, potable water and hydration, sexually transmitted diseases (STDs), and measures to ameliorate jet lag during and after travel. (See Appendix 10, *Recommendations for Minimising Jet Lag*, and Appendix 11, *General Health and Hygiene Recommendations for Athletes*).

B. During Travel

1. *Medical Supplies.* Carry a small kit with medications for emergency use, such as analgesics, anti-histamines, anti-nauseas, and anti-diarrheals.
2. *Jet Lag.* Remind athletes and staff to follow guidelines to minimise jet lag: frequent hydration (but avoid caffeine and alcohol), exercise and stretch often, re-set watches to arrival time zone and try to adapt to the new time.

C. Upon Arrival

1. Establish the Team Medical Care System
 - a. Allocation of work spaces.
 - b. Hours of operation.
 - c. Staffing assignments, including various training and competition venues. Present this information to the team at the initial team meeting. Reinforce the need to maintain personal hygiene and to report illnesses promptly.
2. Establish contact with the Local Organising Committee (LOC) Medical Committee to determine the details of the medical care system. Determine how to access ancillary medical facilities: emergency room, ambulance, hospital, pharmacy, X-ray, supplies, etc.

Arrange for an orientation tour of the LOC medical care system and facilities, including spaces allocated for the team.
3. Environmental Health and Sanitation
 - a. Review the food preparation system.
 - b. Ensure the availability of potable water at all venues.
 - c. Determine the contaminated waste disposal system.
 - d. Determine the procedures for isolation of ill patients in case of infectious diseases.
4. Record-Keeping.
 - a. Maintain treatment logs at all venues.
 - b. Maintain medical record forms for all treatments of illnesses and injuries.

D. During Training and Competition

1. Establish the medical care operation at team headquarters, and at all training and warm-up venues.
2. Establish a medical staffing rotation system for each venue; be sure to allow time off for recovery for the staff.

E. After-Action Responsibilities

1. *Trip report.* File a trip report to the Director of Athlete Services concerning the medical staff experiences and any unusual occurrences, such as major health problems, hospital admissions, etc. Make recommendations for improving the medical care system for future teams.
2. *Staff evaluations.* File a report to the Chief Medical Officer that includes the above, plus evaluations of medical staff members as to suitability for future teams, work ethic, professionalism, etc.
3. *Records.* Send all medical records to the Chief Medical Officer or designee. This may be needed for insurance purposes, and for future travel planning and other statistics of injury/illness incidence .

Appendix 3

Preparticipation Physical Evaluation

HISTORY FORM

DATE OF EXAM _____

Name _____ Sex _____ Age _____ Date of birth _____

Grade _____ School _____ Sport(s) _____

Address _____ Phone _____

Personal physician _____

In case of emergency, contact

Name _____ Relationship _____ Phone (H) _____ (W) _____

Explain "Yes" answers below.
Circle questions you don't know the answers to.

- | | |
|--|---|
| <p>1. Has a doctor ever denied or restricted your participation in sports for any reason? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>2. Do you have an ongoing medical condition (like diabetes or asthma)? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>3. Are you currently taking any prescription or nonprescription (over-the-counter) medicines or pills? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>4. Do you have allergies to medicines, pollens, foods, or stinging insects? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>5. Have you ever passed out or nearly passed out DURING exercise? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>6. Have you ever passed out or nearly passed out AFTER exercise? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>7. Have you ever had discomfort, pain, or pressure in your chest during exercise? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>8. Does your heart race or skip beats during exercise? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>9. Has a doctor ever told you that you have (check all that apply):</p> <p style="padding-left: 20px;"><input type="checkbox"/> High blood pressure <input type="checkbox"/> A heart murmur</p> <p style="padding-left: 20px;"><input type="checkbox"/> High cholesterol <input type="checkbox"/> A heart infection</p> <p>10. Has a doctor ever ordered a test for your heart? (for example, ECG, echocardiogram) <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>11. Has anyone in your family died for no apparent reason? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>12. Does anyone in your family have a heart problem? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>13. Has any family member or relative died of heart problems or of sudden death before age 50? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>14. Does anyone in your family have Marfan syndrome? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>15. Have you ever spent the night in a hospital? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>16. Have you ever had surgery? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>17. Have you ever had an injury, like a sprain, muscle or ligament tear, or tendinitis, that caused you to miss a practice or game? If yes, circle affected area below: <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>18. Have you had any broken or fractured bones or dislocated joints? If yes, circle below: <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>19. Have you had a bone or joint injury that required x-rays, MRI, CT, surgery, injections, rehabilitation, physical therapy, a brace, a cast, or crutches? If yes, circle below: <input type="checkbox"/> Yes <input type="checkbox"/> No</p> | <p>24. Do you cough, wheeze, or have difficulty breathing during or after exercise? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>25. Is there anyone in your family who has asthma? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>26. Have you ever used an inhaler or taken asthma medicine? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>27. Were you born without or are you missing a kidney, an eye, a testicle, or any other organ? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>28. Have you had infectious mononucleosis (mono) within the last month? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>29. Do you have any rashes, pressure sores, or other skin problems? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>30. Have you had a herpes skin infection? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>31. Have you ever had a head injury or concussion? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>32. Have you been hit in the head and been confused or lost your memory? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>33. Have you ever had a seizure? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>34. Do you have headaches with exercise? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>35. Have you ever had numbness, tingling, or weakness in your arms or legs after being hit or falling? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>36. Have you ever been unable to move your arms or legs after being hit or falling? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>37. When exercising in the heat, do you have severe muscle cramps or become ill? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>38. Has a doctor told you that you or someone in your family has sickle cell trait or sickle cell disease? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>39. Have you had any problems with your eyes or vision? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>40. Do you wear glasses or contact lenses? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>41. Do you wear protective eyewear, such as goggles or a face shield? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>42. Are you happy with your weight? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>43. Are you trying to gain or lose weight? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>44. Has anyone recommended you change your weight or eating habits? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>45. Do you limit or carefully control what you eat? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>46. Do you have any concerns that you would like to discuss with a doctor? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>FEMALES ONLY</p> <p>47. Have you ever had a menstrual period? <input type="checkbox"/> Yes <input type="checkbox"/> No</p> <p>48. How old were you when you had your first menstrual period? _____</p> <p>49. How many periods have you had in the last 12 months? _____</p> |
|--|---|
-
- | | | | | | | | |
|------------|------------|----------|-----------|-------|-----------|--------------|-----------|
| Head | Neck | Shoulder | Upper arm | Elbow | Forearm | Hand/fingers | Chest |
| Upper back | Lower back | Hip | Thigh | Knee | Calf/shin | Ankle | Foot/toes |
20. Have you ever had a stress fracture? Yes No
21. Have you been told that you have or have you had an x-ray for atlantoaxial (neck) instability? Yes No
22. Do you regularly use a brace or assistive device? Yes No
23. Has a doctor ever told you that you have asthma or allergies? Yes No

Explain "Yes" answers here: _____

I hereby state that, to the best of my knowledge, my answers to the above questions are complete and correct.

Signature of athlete _____ Signature of parent/guardian _____ Date _____

Preparticipation Physical Evaluation

PHYSICAL EXAMINATION FORM

Name _____ Date of birth _____

Height _____ Weight _____ % Body fat (optional) _____ Pulse _____ BP / / (/ , /)

Vision R 20/ _____ L 20/ _____ Corrected: Y N Pupils: Equal _____ Unequal _____

Follow-Up Questions on More Sensitive Issues

- | | Yes | No |
|--|--------------------------|--------------------------|
| 1. Do you feel stressed out or under a lot of pressure? | <input type="checkbox"/> | <input type="checkbox"/> |
| 2. Do you ever feel so sad or hopeless that you stop doing some of your usual activities for more than a few days? | <input type="checkbox"/> | <input type="checkbox"/> |
| 3. Do you feel safe? | <input type="checkbox"/> | <input type="checkbox"/> |
| 4. Have you ever tried cigarette smoking, even 1 or 2 puffs? Do you currently smoke? | <input type="checkbox"/> | <input type="checkbox"/> |
| 5. During the past 30 days, did you use chewing tobacco, snuff, or dip? | <input type="checkbox"/> | <input type="checkbox"/> |
| 6. During the past 30 days, have you had at least 1 drink of alcohol? | <input type="checkbox"/> | <input type="checkbox"/> |
| 7. Have you ever taken steroid pills or shots without a doctor's prescription? | <input type="checkbox"/> | <input type="checkbox"/> |
| 8. Have you ever taken any supplements to help you gain or lose weight or improve your performance? | <input type="checkbox"/> | <input type="checkbox"/> |
| 9. Questions from the Youth Risk Behavior Survey (http://www.cdc.gov/HealthyYouth/yrbs/index.htm) on guns, seatbelts, unprotected sex, domestic violence, drugs, etc. | <input type="checkbox"/> | <input type="checkbox"/> |

Notes:

	NORMAL	ABNORMAL FINDINGS	INITIALS*
MEDICAL			
Appearance			
Eyes/ears/nose/throat			
Hearing			
Lymph nodes			
Heart			
Murmurs			
Pulses			
Lungs			
Abdomen			
Genitourinary (males only)†			
Skin			
MUSCULOSKELETAL			
Neck			
Back			
Shoulder/arm			
Elbow/forearm			
Wrist/hand/fingers			
Hip/thigh			
Knee			
Leg/ankle			
Foot/toes			

*Multiple-examiner set-up only.

†Having a third party present is recommended for the genitourinary examination.

Notes:

Name of physician (print/type) _____ Date _____

Address _____ Phone _____

Signature of physician _____, MD or DO

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Appendix 4

American College of Sports Medicine Position Stand

Heat and Cold Illnesses During Distance Running

This pronouncement was written for the American College of Sports Medicine by: Lawrence E. Armstrong, Ph.D., FACSM, (Chair), Yoram Epstein, Ph.D., John E. Greenleaf, Ph.D., FACSM, Emily M. Haymes, Ph.D., FACSM Roger W. Hubbard, Ph.D., William O. Roberts, M.D., FACSM, and Paul D. Thompson, M.D., FACSM.

Summary

Many recreational and elite runners participate in distance races each year. When these events are conducted in hot or cold conditions, the risk of environmental illness increases. However, exertional hyperthermia, hypothermia, dehydration, and other related problems may be minimized with pre-event education and preparation. This position stand provides recommendations for the medical director and other race officials in the following areas: scheduling; organizing personnel, facilities, supplies, equipment, and communication; providing competitor education; measuring environmental stress; providing fluids; and avoiding potential legal liabilities. This document also describes the predisposing conditions, recognition, and treatment of the four most common environmental illnesses: heat exhaustion, heatstroke, hypothermia, and frostbite. The objectives of this position stand are: 1) to educate distance running event officials and participants about the most common forms of environmental illness including predisposing conditions, warning signs, susceptibility, and incidence reduction. 2) To advise race officials of their legal responsibilities and potential liability with regards to event safety and injury prevention. 3) To recommend that race officials consult local weather archives and plan events at times likely to be of low environmental stress to minimize detrimental effects on participants. 4) To encourage race officials to warn participants about environmental stress on race day and its implications for heat and cold illness. 5) To inform race officials of preventive actions that may reduce debilitation and environmental illness. 6) To describe the personnel, equipment, and supplies necessary to reduce and treat cases of collapse and environmental illness.

Introduction

This document replaces the position stand titled *The Prevention of Thermal Injuries During Distance Running* (4). It considers problems that may affect the extensive community of recreational joggers and elite athletes who participate in distance running events. It has been expanded to include heat exhaustion, heatstroke, hypothermia, and frostbite—the most common environmental illnesses during races.

Because physiological responses to exercise in stressful environments may vary among participants, and because the health status of participants varies from day to day, compliance with these recommendations will not guarantee protection from environmentally induced illnesses. Nevertheless, these recommendations should minimize the risk of exertional hyperthermia, hypothermia, dehydration,

and resulting problems in distance running and other forms of continuous athletic activity such as bicycle, soccer, and triathlon competition.

Managing a large road race is a complex task that requires financial resources, a communication network, trained volunteers, and teamwork. Environmental extremes impose additional burdens on the organizational and medical systems. Therefore, it is the position of the American College of Sports Medicine that the following RECOMMENDATIONS be employed by race managers and medical directors of community events that involve prolonged or intense exercise in mild and stressful environments.

1. Race Organization

a. Distance races should be scheduled to avoid extremely hot and humid and very cold months. The local weather history should be consulted when scheduling an event. Organizers should be cautious of unseasonably hot or cold days in early spring and late fall because entrants may not be sufficiently acclimatized. The wind chill index should be used to reschedule races on cold, windy days because flesh may freeze rapidly and cold injuries may result.

b. Summer events should be scheduled in the early morning or the evening to minimize solar radiation and air temperature. Winter events should be scheduled at midday to minimize the risk of cold injury.

c. The heat stress index should be measured at the site of the race because meteorological data from a distant weather station may vary considerably from local conditions (66). The wet bulb globe temperature (WBGT) index is widely used in athletic and industrial settings [see Appendix I;(87)]. If the WBGT index is above 28°C (82°F), or if the ambient dry bulb temperature is below -20°C (-4°F), consideration should be given to canceling the race or rescheduling it until less stressful conditions prevail. If the WBGT index is below 28°C, participants should be alerted to the risk of heat illness by using signs posted at the start of the race and at key positions along the race course [see Appendix I;(61)]. Also, race organizers should monitor changes in weather conditions. WBGT monitors can be purchased commercially, or Figure 1 may be used to approximate the risk of racing in hot environments based on air temperature and relative humidity. These two measures are available from local meteorological stations and media weather reports, or can be measured with a sling psychrometer.

d. An adequate supply of fluid must be available before the start of the race, along the race course, and at the end of the event. Runners should be encouraged to replace their sweat losses or consume 150—300 ml (5.3—10.5 ounces) every 15 min (3). Sweat loss can be derived calculating the difference between pre- and postexercise body weight.

e. Cool or cold (ice) water immersion is the most effective means of cooling a collapsed hyperthermic runner (25,48,49,59,88). Wetting runners externally by spraying or sponging during exercise in a hot environment is pleasurable but does not fully attenuate the rise in body core temperature (14,88). Wetting the skin can result in effective cooling once exercise ceases.

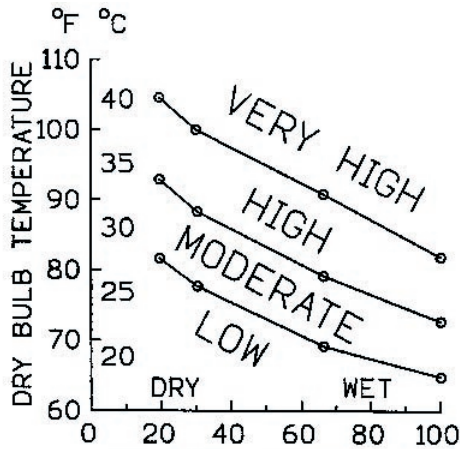


Figure 1. Risk of heat exhaustion or heat stroke while racing in hot environments. Figure drawn from data presented in American College of Sports Medicine Position stand: the prevention of thermal injuries during distance running. *Med. Sci. Sports Exercise* 19: 529-533, 1987.

f. Race officials should be aware of the warning signs of an impending collapse in both hot and cold environments and should warn runners to slow down or stop if they appear to be in difficulty.

g. Adequate traffic and crowd control must be maintained along the course at all times.

h. Radio communications or cellular telephones should connect various points on the course with an information processing center to coordinate emergency responses.

2. Medical Director

A sports medicine physician should work closely with the race director to enhance the safety and provide adequate medical care for all participants. The medical director should understand exercise physiology, interpretation of meteorological data, heat and cold illness-prevention strategies, potential liability, and the treatment of medical problems associated with endurance events conducted in stressful environments.

3. Medical Support

a. Medical organization and responsibility: The medical director should alert local hospitals and ambulance services and make prior arrangements to care for casualties, including those with heat or cold injury. Medical personnel should have the authority to evaluate, examine, and stop runners who display signs of impending illness or collapse. Runners should be advised of this procedure prior to the event.

b. Medical facilities: Medical support staff and facilities must be available at the race site. The facilities should be staffed with personnel capable of instituting immediate and appropriate resuscitation measures. The equipment necessary to institute both cooling therapy (ice packs, child's wading pools filled with tap water or ice water, fans) and warming therapy (heaters, blankets, hot beverages) may be necessary at the same event. For example, medical personnel treated 12 cases of hyperthermia and 13 cases of hypothermia at an endurance triathlon involving 2300 competitors; air temperature was 85°F, water temperature was 58°F.

4. Competitor Education

The physical training and knowledge of competitive runners and joggers has increased greatly, but race organizers must not assume that all participants are well-prepared or informed about safety. Distributing this position stand before registration, publicizing the event in the media, and conducting clinics or seminars before events are valuable educational procedures.

a. All participants should be advised that the following conditions may exacerbate heat illness: obesity (13,39,89), low degree of physical fitness (30,63,79,83), dehydration (23,34,69,83,84,95), lack of heat acclimatization (31,51,89), a previous history of heat stroke (82,89), sleep deprivation (5), certain medications, including diuretics and antidepressants (31), and sweat gland dysfunction or sunburn (31). Illness 1 wk prior to an event should preclude participation (32,96), especially those involving fever, respiratory tract infections, or diarrhea (41,46).

b. Prepubescent children sweat less than adults and have lower heat tolerance (11,12).

c. Adequate training and fitness are important for full enjoyment of the event and will reduce the risk of heat illness and hypothermia (22,64,67,85).

d. Prior training in the heat will promote heat acclimatization (6) and thereby reduce the risk of heat illness, especially if the training environment is warmer than that expected during a race (5,51). Artificial heat acclimatization can be induced in cold conditions (6).

e. Adequate fluid consumption before and during the race can reduce the risk of heat illness, including disorientation and irrational behavior, particularly in longer events such as a marathon (23,34,95).

f. Excessive consumption of pure water or dilute fluid (i.e., up to 10 liters per 4 hours) during prolonged endurance events may lead to a harmful dilutional hyponatremia (60), which may involve disorientation, confusion, and seizure or coma. The possibility of hyponatremia may be the best rationale for inclusion of sodium chloride in fluid replacement beverages (3).

g. Participants should be advised of the early symptoms of heat illness, which may include clumsiness, stumbling, headache, nausea, dizziness, apathy, confusion, and impairment of consciousness (41,86).

h. Participants should be advised of the early symptoms of hypothermia (slurred speech, ataxia, stumbling gait) and frostbite (numbness, burning, pain, paresthesia) on exposed skin (36). Wet clothing, especially cotton, increases heat loss and the risk of hypothermia (68).

i. Participants should be advised to choose a comfortable running speed and not to run faster than environmental conditions or their cardiorespiratory fitness warrant (43,71,91).

j. It is helpful if novice runners exercise with a partner, each being responsible for the other's well-being (71).

5. Responsibilities and Potential Liability

The sponsors and directors of an endurance event are reasonably safe from liability due to injury if they avoid gross negligence and willful misconduct, carefully inform the participants of the hazards, and have them sign waivers before the race (78). However, a waiver signed by a participant does not totally absolve race organizers of moral and/or legal responsibility. It is recommended that race sponsors and directors: 1) minimize hazards and make safety the first concern; 2) describe inherent hazards (i.e., potential course hazards, traffic control, weather conditions) in the race application; 3) require all entrants to sign a waiver; 4) retain waivers and records for 3 yrs; 5) warn runners of the predisposing factors and symptoms of environmental illness; 6) provide all advertised support services; 7) legally incorporate the race or organizations involved; 8) purchase liability insurance (18,78,80).

Race directors should investigate local laws regarding Good Samaritan action. In some states physicians who do not accept remuneration may be classified as Good Samaritans. Race liability insurance may not cover physicians (78), therefore the malpractice insurance policy of each participating physician should be evaluated to determine if it covers services rendered at the race.

Medical and race directors should postpone, reschedule, or cancel a race if environmental conditions warrant, even though runners and trained volunteers arrive at the site and financial sponsorship has been provided. Runners may not have adequate experience to make the decision not to compete; their safety must be considered. Downgrading the race to a “fun run” does not absolve race supervisors from their responsibility or decrease the risk to participants (15,66).

Background for this Position Stand

Dehydration is common during prolonged endurance events in both hot and cold environmental conditions because the average participant loses 0.5–1.5 quarts (0.47–1.42 liters) of sweat/hr, and fluid replacement is usually insufficient (2,42,69). Runners may experience hyperthermia [body core temperature above 39°C (102.2°F)] or hypothermia [body core temperature below 35°C (95°F)], depending on the environmental conditions, caloric intake, fluid consumption, and clothing worn. Hyperthermia is a potential problem in warm and hot weather race when the body's rate of heat production is greater than its heat dissipation (2). Indeed, on extremely hot days, it is possible that up to 50% of the participants may require treatment for heat-related illnesses such as heat exhaustion and heatstroke (1,66). Hypothermia is more likely to occur in cold or cool-windy conditions. Scanty clothing may provide inadequate protection from such environments, particularly near the end of a long race when running speed and heat production are reduced. Frostbite can occur in low air temperature and especially when combined with high wind speed. The race and medical directors should anticipate the above medical problems and be capable of responding to a large number of patients with adequate facilities, supplies, and support staff. The four most common heat and cold illnesses during distance running are heat exhaustion, heatstroke, hypothermia, and frostbite.

1. Heat Exhaustion

Body sweat loss can be significant in summer endurance races and may result in a body water deficit of 6–10% of body weight (41,95). Such dehydration will reduce the ability to exercise in the heat because decreases in circulating blood volume, blood pressure, sweat production, and skin blood flow all inhibit heat loss (41,81) and predispose the runner to heat exhaustion or the more dangerous hyperthermia and exertional heatstroke (41,66).

Heat exhaustion, typically the most common heat illness among athletes, is defined as the inability to continue exercise in the heat (7). It represents a failure of the cardiovascular responses to workload, high external temperature, and dehydration (16,41,42). Heat exhaustion has no known chronic, harmful effects. Symptoms may include headache, extreme weakness, dizziness, vertigo, “heat sensations” on the head or neck, heat cramps, chills, “goose flesh” (“goose bumps”), vomiting, nausea, and irritability (41,42). Hyperventilation, muscular incoordination, agitation, impaired judgment, and confusion also may be seen. Heat syncope (fainting) may or may not accompany heat exhaustion (41). The onset of heat exhaustion symptoms is usually sudden and the duration of collapse brief. During the acute stage of heat exhaustion, the patient looks ashen-gray, the blood pressure is low, and the pulse rate is elevated. Hyperthermia may add to the symptoms of heat exhaustion, even on relatively cool days (20,22,30,37,38,43,62,90).

Although it is improbable that all heat exhaustion cases can be avoided, the most susceptible individuals are those who either exert themselves at or near their maximal capacities, are dehydrated, not physically fit, and not acclimatized to exercise in the heat. It is imperative that runners be adequately rested, fed, hydrated, and acclimatized (7); they should drink ample fluids before, during, and after exercise (3). Also, repeated bouts of exercise in the heat (heat acclimatization) reduce the incidence of both heat exhaustion and heat syncope. Heat acclimatization can best be accomplished by gradually increasing the duration and intensity of exercise training during the initial 10–14 days of heat exposure (6).

Oral rehydration is preferred for heat exhaustion patients who are conscious, coherent, and without vomiting or diarrhea. Intravenous (IV) fluid administration facilitates rapid recovery (42,57). Although a variety of IV solutions have been used at races (42), a 5% dextrose sugar in either 0.45% saline (NaCl) or 0.9% NaCl are the most common (1). Runners may require up to 4 l of IV fluid if severely dehydrated (57).

2. Exertional Heatstroke

Heat production, mainly from muscles, during intense exercise is 15–20 times greater than at rest, and is sufficient to raise body core temperature by 1°C (1.8°F) each 5 min without thermoregulatory (heat loss) adjustments (56). When the rate of heat production exceeds that of heat loss for a sufficient period of time, severe hyperthermia occurs.

Heatstroke is the most serious of the syndromes associated with excess body heat. It is defined as a condition in which body temperature is elevated to a level

that causes damage to the body's tissues, giving rise to a characteristic clinical and pathological syndrome affecting multiple organs (32,83). After races, adult core (rectal) temperatures above 40.6° (105.1°F) have been reported in conscious runners (92,4,52,69,74,77), and 42–43°C (107.6–109.4°F) in collapsed runners (72–74,86,90). Sweating is usually present in runners who experience exertional heatstroke.

Strenuous physical exercise in a hot environment has been notorious as the cause of heatstroke, but heatstroke also has been observed in cool-to-moderate [13–28°C (55–82°F)] environments (5,32,74), suggesting variations in individual susceptibility (95,31,32). Skin disease, sunburn, dehydration, alcohol or drug use/abuse, obesity, sleep loss, poor physical fitness, lack of heat acclimatization, advanced age, and a previous heat injury all have been theoretically linked to increased risk of heatstroke (5,31,51,84). The risk of heatstroke is reduced if runners are well-hydrated, well-fed, rested, and acclimatized. Runners should not exercise if they have a concurrent illness, respiratory infection, diarrhea, vomiting, or fever (5,7,46). For example, a study of 179 heat casualties at a 14-km race showed that 23% reported a recent gastrointestinal or respiratory illness (70), whereas a study of 10 military heatstroke patients reported that three had a fever or disease and six recalled at least one warning sign of impending illness at the time of their heatstroke (5).

Appropriate fluid ingestion before and during prolonged running can minimize dehydration and reduce the rate of increase in body core temperature (23,34). However, excessive hyperthermia may occur in the absence of significant dehydration, especially in races of less than 10 km, because the fast pace generates greater metabolic heat (90).

The mortality rate and organ damage due to heatstroke are proportional to the length of time between core temperature elevation and initiation of cooling therapy (5,26). Therefore, prompt recognition and cooling are essential (1,5,22,42,48,51,62,74,83). A measurement of deep body temperature is vital to the diagnosis, and a rectal temperature should be measured in any casualty suspected of having heat illness or hypothermia. Ear (tympanic), oral, or axillary measurements are spuriously affected by peripheral (skin) and environmental temperatures and should not be used after exercise (8,75,76). When cooling is initiated rapidly, most heatstroke patients recover fully with normal psychological status (79), muscle energy metabolism (65), heat acclimatization, temperature regulation, electrolyte balance, sweat gland function, and blood constituents (5).

Many whole-body cooling techniques have been used to treat exertional heatstroke, including water immersion, application of wet towels or sheets, warm air spray, helicopter downdraft, and ice packs to the neck, underarm, and groin areas. There is disagreement as to which modality provides the most efficient cooling (7,47,97), because several methods have been used successfully. However, the fastest whole-body cooling rates (92,5,48,49,59,88) and the lowest mortality rates (25) have been observed during cool and cold water immersion. Whichever modality is utilized it should be simple and safe, provide great cooling power, and should not restrict other forms of therapy (i.e., cardiopulmonary resuscitation, defibrillation, IV

cannulation). The advantages and disadvantages of various cooling techniques have been discussed (47,75,97).

Heatstroke is regarded as a medical emergency that might be fatal if not immediately diagnosed and properly treated. Early diagnosis is of utmost importance and time-consuming investigation should be postponed until body temperature is corrected and the patient is evacuated to a nearby medical facility that is aware of such conditions.

3. Hypothermia

Hypothermia [body core temperature below 35°C (95°F)] occurs when heat loss is greater than metabolic heat production (94). Early signs and symptoms of hypothermia include shivering, euphoria, confusion, and behavior similar to intoxication. Lethargy, muscular weakness, disorientation, hallucinations, depression, or combative behavior may occur as core temperature continues to fall. If body core temperature falls below 31.1°C (88°F), shivering may stop and the patient will become progressively delirious, uncoordinated, and eventually comatose if treatment is not provided (10).

During cool or cold weather marathons, the most common illnesses are hypothermia, exhaustion, and dehydration. The most common medical complaints are weakness, shivering, lethargy, slurred speech, dizziness, diarrhea, and thirst (1,45). Runner complaints of feeling hot or cold do not always agree with changes in rectal temperature (74). Dehydration is common in cool weather (1,45). Runners should attempt to replace fluids at a rate that matches their sweat and urine losses. Cases of hypothermia also occur in spring and fall because weather conditions change rapidly and runners wear inappropriate clothing that becomes sweat-soaked during training or competition (19).

Hypothermia may occur during races, for example when distance runner complete the second half of the event more slowly than the first half (54). Evaporative and radiative cooling increase because wet skin (from sweat, rain, or snow) and clothing are exposed to higher wind speed at a time when metabolic heat production decreases. Hypothermia also occurs after a race, when the temperature gradient between the body surface and the environment is high. Subfreezing ambient temperatures need not be present and hypothermia may develop even when the air temperature is 10–18°C (50–65°F) (19,36,74). A WBGT meter can be used to evaluate the risk of hypothermia (see Appendix I). Cold wind increases heat loss in proportion to wind speed; i.e., wind chill factor. The relative degree of danger can be assessed [note: see Table 11-2 in Chapter 11, Part 1, *Heat and Cold* of this volume] (55). Wind speed can be estimated; if you feel the wind in your face the speed is at least 16 km/h (kph) [10 miles/h (mph)]; if small tree branches move or if snow and dust are raised, approximately 32 kph (20 mph); if large tree branches move, 48 kph (30 mph); if an entire tree bends, about 64 kph (40 mph) (9).

To reduce heat loss, runners should protect themselves from moisture, wind, and cold air by wearing several layers of light, loose clothing that insulate the skin with trapped air (17). An outer garment that is windproof, allows moisture to escape, and

provides rain protection is useful. Lightweight nylon parkas may not offer thermal insulation but offer significant protection against severe wind chill, especially if a hood is provided. Wool and polyester fabrics retains some protective value when wet; cotton and goose down do not (10). Areas of the body that lose large amount of heat (head, neck, legs, hands) should be covered (17).

Mild [$34\text{--}36^\circ$ ($93\text{--}97^\circ\text{F}$)] or moderate [$30\text{--}34^\circ\text{C}$ ($86\text{--}93^\circ\text{F}$)] hypothermia should be treated before it progresses. Wet clothing should be replaced with dry material (sweatsuit, blanket) that is insulated from the ground and wind. Warm fluids should be consumed if patients are conscious, able to talk, and thinking clearly. Patients with moderate and severe [$<30^\circ\text{C}$ (86°F)] hypothermia should be insulated in a blanket and evacuated to a hospital immediately (19,58). Although severe hypothermia should be treated in the field (27), it is widely recognized that life-threatening ventricular fibrillation is common in this state and may be initiated by physical manipulation, chest compression, or intubation (10,27,58,93). However, with conclusive evidence of cardiac standstill and breathlessness, emergency procedures (i.e., Basic Life Support, Advanced Cardiac Life Support) should be initiated. Life-support procedures (27) and commonly observed laboratory (i.e., electrolyte, acid-base) values (10,58) have been described by others.

4. Frostbite

Frostbite involves crystallization of fluids in the skin or subcutaneous tissue after exposure to subfreezing temperatures [-0.6°C (31°F)]. With low skin temperature and dehydration, cutaneous blood vessels constrict and circulation is attenuated because the viscosity of blood increases (55). Frostbite may occur within seconds or hours of exposure, depending upon air temperature, wind speed, and body insulation. Frostbitten skin can appear white, yellow-white, or purple, and is hard, cold, and insensitive to touch (55). Rewarming results in intense pain, skin reddening, and swelling. Blister formation is common and loss of extremities (fingers, toes, ears, hands, feet) is possible (36,55). The degree of tissue damage depends on duration and severity of the freezing and effectiveness of treatment.

No data have been published regarding the incidence of frostbite among athletes during training or competition. Since winter running races are rarely postponed when environmental conditions are harsh, and frostbite is the most common cold injury in military settings (35), it is imperative that runners be aware of the dangers. Cross-country ski races are postponed if the temperature at the coldest point of the course is less than -20°C (-4°F), due to the severe wind chill generated at race pace.

Runners risk frozen flesh within minutes if the air temperature and wind speed combine to present a severe wind chill. Because runners prefer to have unrestricted movement during races, and because they know that exercise results in body heating, they may not wear sufficient clothing. Runners can avoid frostbite and hypothermia in cold and windy conditions by protecting themselves by dressing adequately: wet skin or clothing will increase the risk of frostbite (21,29).

When tissue freezes [skin temperature $-2^\circ\text{--}0^\circ\text{C}$, ($28\text{--}32^\circ\text{F}$)], water is drawn out of the cells and ice crystals cause mechanical destruction of skin and subcutaneous

tissue (36). However, initial ice crystal formation is not as damaging to tissues as partial rethawing and refreezing (40). Therefore, the decision to treat severe frostbite in the field (versus transport to a hospital) should consider the possibility of refreezing. If there is no likelihood of refreezing, the tissue should be rapidly rewarmed (36,40) in circulating warm water (40–43.3°C, 104–110°F), insulated, and the patient transported to a medical facility. Research on animals suggests that topical aloe vera and systemic ibuprofen may reduce tissue damage and speed rehabilitation in humans (9). Other aspects of hospital treatment protocols are detailed elsewhere (9,36,40).

Race Organization

The following suggestions constitute the ideal race medical team. They are offered for consideration, but are not intended as absolute requirements. Staff and equipment needs are unique to each race and may be revised after 1–2 yr, in light of the distinctive features of each race. Depending on the weather conditions, 2–12% of all entrants will typically enter a medical aid station (1,45,50,74).

1. Medical Personnel

- a. Provide medical assistance if the race is 10 km (6.2 miles) or longer.
- b. Provide the following medical personnel per 1,000 runners: 1–2 physicians, 46 podiatrists, 1–4 emergency medical technicians, 2–4 nurses, 3–6 physical therapists, 3–6 athletic trainers, and 1–3 assistants. Approximately 75% of these personnel should be stationed at the finish area. Recruit one nurse (per 1,000 runners) trained in IV therapy.
- c. Recruit emergency personnel from existing organizations (police, fire-rescue, emergency medical service).
- d. One physician and 10–15 medical assistants serve as the triage team in the finish chute. Runners unable to walk are transported to the medical tent via wheelchair, litter, or two-person carry.
- e. Consider one or two physicians and two to four nurses trained in the rehabilitative medical care of wheelchair athletes.
- f. Medical volunteers should attend a briefing prior to the event to meet their supervisor and receive identification tags, weather forecast, instructions, and schedules. Supervisors from the following groups should be introduced: medical director; podiatry, nursing, physical therapy, athletic training, medical records, triage, wheelchair, athlete care, and medical security (optional: chiropractic, massage therapy). Medical volunteers should be distinguished from other race volunteers; luminous/distinctive vests, coats, or hats work well.

2. Medical Aid Stations

- a. Provide a primary medical aid station (250–1,500 ft² [23–139m²] for each 1,000 runners; see Table 1) at the finish area, with no public access. Place security guards at all entrances with instructions regarding who can enter.

Table 1: Suggested equipment and supplies per 1,000 runners.

Item	Secondary Aid Station ^b	Primary Aid Station ^c
Stretchers (at 10 km and beyond)	2–5	4–10
Cots	10	30
Wheelchairs	0	1
Wool blankets (at 10 km and beyond)	6–10	12–20
Bath towels	5–10	10–20
High and low temperature rectal thermometers (37–43°C; 99–110°F) and (22–37°C; 72–99°F) ^d	5	10
Elastic bandages (2, 4, and 6 inch)	3 each	6 each
Gauze pads (4 x 4 inch)	1/2 case	1 case
Adhesive tape (1.5 inch)	1/2 case	1 case
Skin disinfectant	1 l	2 l
Surgical soap	1/2 case	1 case
Band-aids	110	220
Moleskin	1/2 case	1 case
Petroleum jelly, ointments	1/2 case	1 case
Disposable latex gloves	80 pairs	175 pairs
Stethoscopes	1	2
Blood pressure cuffs	1	2
Intravenous (IV) stations ^d	1	2
IV fluids (D5:1/2 NS; 0.5 or 1 l) ^d	15 ^e	30 ^e
Sharps and biohazard disposal containers ^d	1	2
Alcohol wipes	50	100
Small instrument kits	1	1
Athletic trainer's kit	1	1
Podiatrist's kit	1–2	2–4
Inflatable arm and leg splints	2 each	2 each
Tables for medical supplies	1	2
Hose with spray nozzle, running water ^e	1	2
Wading pool for water immersions ^e	1	2
Fans for cooling	1	2–4
Oxygen tanks with regulators and masks	0	2
Crushed ice in plastic bags	7 kg	14 kg
Rehydration fluids	50 l ^e	100 l ^e
Cups (≥0.3 l, 10 oz)	1250	2250
Eye drops	1	1
Urine dipsticks ^d	10	20
Glucose blood monitoring kit ^d	1	2
Inhalation therapy for asthmatics ^d	1	1
EMS ambulance or ACLS station	1	1
Injectable and oral drugs ^d		

^aRevised from Adner, M. M., J. J. Scarlet, J. Casey, W. Robison, and B. H. Jones. The Boston Marathon medical care team: ten years of experience. *Physician Sportsmed.* 16:99-106, 1988; and Noble, H. B. and D. Bachman. Medical aspects of distance race planning. *Physician Sportsmed.* 7:78-84, 1979.

^bIncrease supplies and equipment if race is out and back. ^cAt finish area. ^dSupervised by a physician.

^eDepends on environmental conditions.

b. Position secondary medical aid stations along the route at 2- to 3-km (1.2- to 1.9-mile) intervals for races over 10 km, and at the halfway point for shorter races (see Table 1). Some race directors have successfully secured equipment and medical volunteers from military reserve or national guard medical units, the American Red Cross, and the National Ski Patrol.

c. Station one ambulance per 3,000 runners at the finish area and one or more mobile emergency response vehicles on the course. Staff each vehicle with a nurse and radio person or cellular telephone. Stock each vehicle with a medical kit, automatic defibrillator, IV apparatus, blankets, towels, crushed ice, blood pressure cuffs, rehydration fluid, and cups.

d. Signs should be posted at the starting line and at each medical station to announce the risk of heat illness or cold injury (see Appendix I).

e. A medical record card should be completed for each runner who receives treatment (1,74). This card provides details that can be used to plan the medical coverage of future events.

f. Provide personal protective equipment (gloves, gowns, face shields, eye protection) and hand washing facilities.

g. Provide portable latrines and containers for patients with vomiting and diarrhea.

h. Initial medical assessment must include rectal (not oral, aural, or axillary temperature; see ref. 8,76), central nervous system function, and cardiovascular function. Rehydration and cooling or warming are the cornerstones of treatment (32,41,42,50,74,94).

3. Universal Precautions

All medical personnel may encounter blood-borne pathogens or other potentially infectious materials, and should observe the following precautions (53,63):

a. Receive immunization against hepatitis B prior to the event.

b. Recognize that blood and infectious body fluids may be encountered from needle sticks, cuts, abrasions, blisters, and clothing.

c. Reduce the likelihood of exposure by planning tasks carefully (i.e., prohibiting recapping of needles by a two-handed technique, minimizing splashing and spraying).

d. Wear personal protective equipment such as gloves, gowns, face shields and eye protection. Remove this equipment and dispose/decontaminate it prior to leaving the work area.

e. Wash hands after removing gloves or other personal protective equipment.

f. Dispose of protective coverings, needles, scalpels, and other sharp objects in approved, labeled biohazard containers.

g. Do not eat, drink, smoke, handle contact lenses, or apply cosmetics/lip balm in the medical treatment area.

h. Decontaminate work surfaces, bins, pails, and cans [1/10 solution of household bleach (sodium hypochlorite) in water] after completion of procedures.

4. Fluid Stations

a. At the start and finish areas provide 0.34–0.45 l (12–16 oz) of fluid per runner. At each fluid station on the race course (2–3 km apart), provide 0.28–0.34 l (10–12 oz) of fluid per runner. Provide both water and a carbohydrate-electrolyte beverage in equal volumes.

b. In cool or cold weather [$\leq 10^{\circ}\text{C}$ (50°F)], an equivalent amount of warm fluid should be available.

c. Number of cups (≥ 0.3 l, 10 oz) per fluid station on the course = number of entrants + 25% for spillage and double use. Double this total if the course is out and back.

d. Number of cups at start and finish area = (2 x number of entrants) + 25% additional.

e. Cups should be filled prior to the race and placed on tables to allow easy access. Runners drink larger volumes if volunteers hand them cups filled with fluid.

5. Communications/Surveillance

a. Provide two-way radio or telephone communication between the medical director, medical aid stations, mobile vans, and pick-up vehicles.

b. Arrange for radio-equipped vehicles to drive the race course (ahead and behind participants) and provide communication with the director and his/her staff. These vehicles should be stationed at regular intervals along the course to search the course for competitors who require emergency care and encourage compromised runners to stop.

c. Place radio-equipped observers along the course.

d. Notify local hospitals, police, and fire-rescue departments of the time of the event, number of participants, location of aid stations, extent of medical coverage, and the race course.

e. Use the emergency response system (telephone number 911 [in U.S.] or 112 [international]) in urban areas.

6. Instructions to Runners

a. Advise each race participant to print name, address, telephone number, and medical problems on the back of the race number (pinned to the body). This permits emergency personnel to quickly identify unconscious runners. Inform emergency personnel that this information exists.

b. Inform race participants of potential medical problems at pre-race conferences and at the starting line. Signed registration forms should clearly state the types of heat or cold injuries that may arise from participation in this event.

c. Provide pre-event recommendations regarding training, fluid consumption, clothing selection, self-care, heat acclimatization, and signs or symptoms of heat/cold illness (88).

d. The race director should announce the following information to all participants by loudspeaker immediately prior to the race:

- Current and predicted maximum (or minimum) temperature, humidity, wind speed, and cloud cover;

- The WBGT category and risks for hyperthermia or hypothermia (see Appendix I);
- Location of aid stations, types of assistance, and fluid availability;
- Signs and symptoms of heat or cold illness;
- Recommended clothing;
- The need for fluid replacement before, during, and after the race;
- The policy of race monitors to stop runners who are ill;
- A request that runners seek help for impaired competitors who appear ill, who are not coherent, who run in the wrong direction, or who exhibit upper-body swaying and poor competitive posture;
- A warning to novice runners entering their first race that they should run at a comfortable pace and run with a partner;
- Warnings to runners who are taking medications or who have chronic illnesses (asthma, hypertension, diabetes, cardiovascular problems).

Acknowledgment

This position stand replaces the 1987 ACSM position paper, “The Prevention of Thermal Injuries During Distance Running.”

The pronouncement was reviewed for the American College of Sports Medicine by members-at-large, the Pronouncement Committee, and by: Arthur E. Crago, M.D., Stafford W. Dobbin, M.D., Mary L. O’Toole, Ph.D., FACSM, LTC Katy L. Reynolds, M.D., and John W. Robertson, M.D., FACSM.

References

See <http://www.acsm-msse.org/pt/re/msse/positionstandards.htm> for list of references (scroll to December 1, 1996 for pdf).

Appendix I. Measurement of Environmental Stress

Ambient temperature is only one component of environmental heat or cold stress; others are humidity, wind speed, and radiant heat. The most widely used heat stress index is the wet bulb globe temperature (WBGT) index (96):

$$\text{WBGT} = (0.7 T_{wb}) + (0.2 T_g) + (0.1 T_{db})$$

Where T_{wb} is the wet bulb temperature, T_g is the black globe temperature, and T_{db} is the shaded dry bulb temperature (28). T_{db} refers to air temperature measured with a standard dry bulb thermometer not in direct sunlight. T_{wb} is measured with a water-saturated cloth wick over a dry bulb thermometer (not immersed in water). T_g is measured by inserting a dry bulb thermometer into a standard black metal globe. Both T_{wb} and T_g are measured in direct sunlight.

A portable monitor that gives the WBGT index in degrees Celsius or degrees Fahrenheit has proven useful during races and in military training (28,44,87,96). The measurement of air temperature alone is inadequate. The importance of humidity

in total heat stress can be readily appreciated because T_{wb} accounts for 70% of the index whereas T_{db} accounts for only 10%.

The risk of heat illness (while wearing shorts, socks, shoes, and a t-shirt) due to environmental stress should be communicated to runners in four categories (see Fig. 1):

Very high risk: WBGT above 28°C (82°F);

High risk: WBGT 23–28°C (73–82°F);

Moderate risk: WBGT 18–23°C (65–73°F);

Low risk: WBGT below 18°C (65°F).

Large signs should be displayed, at the start of the race and at key points along the race course, to describe the risk of heat exhaustion and heatstroke (Fig. 1). When the WBGT index is above 28°C (82°F), the risk of heat exhaustion or heatstroke is very high; it is recommended that the race be postponed or canceled. High risk [WBGT index = 23–28°C (73–82°F)] indicates that runners should be aware that heat exhaustion or heatstroke may be experienced by any participant; anyone who is particularly sensitive to heat or humidity probably should not run. Moderate risk [WBGT index = 18–23°C (65–73°F)] reminds runners that heat and humidity during the course of the race if conducted during the morning or early afternoon. Low risk [WBGT index = below 18°C (65°F)] does not guarantee that heat exhaustion (even heat stroke, see ref. 5,32) will not occur; it only indicates that the risk is low.

The risk of hypothermia (while wearing shorts, socks, shoes, and a t-shirt) also should be communicated to runners. A WBGT index below 10°C (50°F) indicates that hypothermia may occur in slow runners who run long distances, especially in wet and windy conditions. Core body temperatures as low as 92°F have been observed in 65°F conditions (74).

Appendix 5

IAAF Policy on Fluid Replacement

Heat Stress and Heat Illness

Heat stress and heat illness occur when the body's heat production goes beyond the many factors responsible for heat loss.

Heat production is determined by the athlete's metabolic rate (energy expenditure), i.e., race pace, body weight, and running economy. About three-quarters of the energy produced by exercise is stored as heat. Thus, higher-intensity races such as a 10 kilometre race are more likely to lead to heat injury than longer races, which are run at a slower pace.

Many factors determine the body's ability to dissipate heat: environmental factors such as ambient temperature, relative humidity, and air currents, as well as the athlete's level of fitness and his or her degree of heat adaptation. The environmental factors can be assessed by measuring the Wet Bulb Globe Temperature (WBGT). It is advised to run distance races below 18 degrees of WBGT value.

One of the major factors responsible for cooling and maintaining the body's temperature in warm weather is the body's ability to evaporate sweat. Hence, adequate hydration is one of the most important elements in the prevention of heat injury, and the ability to maintain a high level of performance.

Performance begins to become impaired when the body loses more than 2–3% of body weight, primarily as fluid losses from sweating. The athlete's heart rate and core temperature will be increased. Thus, maintaining adequate hydration is important, but athletes must realise that hydration alone is not sufficient to prevent heat injury.

Athletes must learn to recognise thirst as a late indicator of dehydration. They should consume fluid before they feel thirsty. However, drinking excess amounts of fluid in the absence of thirst may lead to over-hydration and exertional hyponatremia (low blood sodium), especially if the fluids consumed are sodium-free. Exertional hyponatremia can be a life-threatening condition, and is more likely to occur in slower runners who are exercising for four hours or more. Their metabolic rate and heat production are lower, and due to their slower pace they are more able to consume more fluids than they need.

Pre-Race Preparation

1. Heat adaptation. Train under similar conditions as those expected during the race. This ideally may require 7–10 days. If this is not possible, train with additional clothing in order to raise core temperature. However, NEVER wear rubber suits or other clothing that inhibits sweat evaporation.
2. Practice drinking during training runs, so that you can drink comfortably while running. Use the same drinks in the training run and the race run. Observe weight changes during training sessions to get a feel for typical sweat rates, then develop a drinking plan that allows you to replace most

of these losses during the session. (For example, if you find that you sweat at a rate of ~1 litre per hour, drinking at a rate of 400–800 ml per hour is likely to be a good plan).

3. Salt food heavily for several days prior to the competition. Restore salt in the body.
4. Begin the race well-hydrated. Consume 500–600 ml of water or a sports drink during the 2–3 hours before the race, and another 300 ml 10–15 minutes before the start (300 ml of water can be absorbed within 15 minutes).
5. Be aware of WBGT, which will let you know the possibility of heat illness.
6. Do not use any non-steroidal anti-inflammatory drugs (NSAIDs) except acetaminophen. NSAIDs are thought to increase the possibility of hyponatremia while running long distances.

During the Race

1. Consume an adequate amount of fluids to prevent dehydration (see point 2 under race preparation).
2. Consume cool liquids that contain 0.2–0.45% sodium and 5% glucose or glucose polymer. This combination replaces electrolytes lost in sweat, aids in preventing hyponatremia, and provides carbohydrate for energy.
3. As sweat losses and the ability to absorb fluids may vary considerably among individuals, it is useful to determine one's individual needs by using the guidelines found in References 1 and 2 (see below).
4. It may not be possible or necessary to drink at a rate that completely replaces sweat losses during an event. In most cases, drinking to keep losses less than 2% of body mass is suitable. You should not drink at rates greater than sweat losses so that you gain weight over the session.

After the Race

1. Begin to re-hydrate and restore muscle glycogen as soon as possible after the race. Fluids containing electrolytes (sodium and potassium) and carbohydrates are needed to replace losses.
2. Optimal replenishment of muscle glycogen is best carried out in the first 2–4 hours post-competition.
3. If possible, the athlete should weigh him or herself before and after the race to determine the amount of fluid loss, and replace this loss with 1 1/4–1 1/2 times this amount.

Dehydration and hyponatremia can be prevented in distance running, and performance should be improved by proper fluid and salt replacement before and during the races.

This policy has been written by Dr. Fumihiko Yamasawa, Dr. Harmon Brown, and Professor Louise Burke on behalf of the IAAF Medical and Anti-Doping Commission.

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Appendix 6

Supplies for Injury Prevention/First Aid

On-Site Medical Supplies

Following are lists of medical bag items and medical supplies for contact/collision and high-risk sports.

General

- Alcohol swabs and povidone iodine swabs
- Bandage scissors
- Bandages, sterile/nonsterile, Band-Aids
- Disinfectant
- Gloves, sterile/nonsterile
- Local anesthetic/syringes/needles
- Paper/ Pen
- Plastic bags
- Sharps box and biohazard bag
- Sling
- Suture set/Steri-Strips
- Wound irrigation materials (e.g. , sterile normal saline, 10- to 50-cc syringe)

Cardiopulmonary

- Oropharyngeal airway
- Blood pressure cuff
- Epinephrine 1:1000 in a prepackaged unit
- Mouth-to-mouth mask
- Short-acting beta₂ agonist inhaler
- Stethoscope

Head and Neck/Neurologic

- Dental kit (e.g., cyanoacrylate, Hank's solution)
- Eye kit (e.g., blue light, fluorescein stain strips, eye patch pads, cotton tip applicators, ocular anesthetic and antibiotics, contact remover, mirror)
- Semirigid cervical collar
- Spine board and attachments
- Flashlight
- Pin or other sharp object for sensory testing
- Reflex hammer

General

- Access to a telephone
- Crutches
- Extremity splints
- Ice
- List of emergency phone numbers
- Injury and illness care instruction sheets for the athlete
- Benzoin
- Blister care materials
- Contact lens case and solution
- Ferric subsulfate solution (e.g., Munsell's for cauterizing abrasions and cuts)
- Nail clippers
- Nasal packing material
- Oral fluid replacement
- Oto-ophthalmoscope
- Razor and shaving cream
- Scalpel
- Skin lubricant
- Skin staple applicator
- Small mirror
- Tape cutter
- Tongue depressors
- Topical antibiotics
- Massage lotion

Cardiopulmonary

- Advanced Cardiac Life Support (ACLS) drugs and equipment
- IV fluids and administration set
- Tourniquet
- Automated external defibrillator (AED)

In addition, sideline medical supplies should include the following:

- Blanket/Crutches
- Sling psychrometer and temperature/humidity activity risk chart

Appendix 7

Cardiopulmonary Resuscitation (CPR)/Basic Life Support (BLS) Guidelines

Adult Basic Life Support

Adapted from the 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, *Circulation*. 2005; 112:IV-19–IV-34.

Basic life support (BLS) includes recognition of signs of sudden cardiac arrest (SCA), heart attack, stroke, and foreign-body airway obstruction (FBAO); cardiopulmonary resuscitation (CPR); and defibrillation with an automated external defibrillator (AED). This section summarizes BLS guidelines for lay rescuers and healthcare providers.

Introduction

Sudden cardiac arrest is a leading cause of death in the United States and Canada.¹⁻³ At the first analysis of heart rhythm, about 40% of victims of out-of-hospital SCA demonstrate ventricular fibrillation (VF).³⁻⁵ VF is characterized by chaotic rapid depolarizations and repolarizations that cause the heart to quiver so that it is unable to pump blood effectively.⁶ It is likely that an even larger number of SCA victims have VF or rapid ventricular tachycardia (VT) at the time of collapse, but by the time of first rhythm analysis the rhythm has deteriorated to asystole.⁷

Many SCA victims can survive if bystanders act immediately while VF is still present, but successful resuscitation is unlikely once the rhythm deteriorates to asystole.⁸ Treatment for VF SCA is immediate bystander CPR plus delivery of a shock with a defibrillator. The mechanism of cardiac arrest in victims of trauma, drug overdose, drowning, and in many children is asphyxia. CPR with both compressions and rescue breaths is critical for resuscitation of these victims.

The American Heart Association uses 4 links in a chain (the “Chain of Survival”) to illustrate the important time-sensitive actions for victims of VF SCA. Three and possibly all 4 of these links are also relevant for victims of asphyxial arrest.⁹

- Early recognition of the emergency and activation of the emergency medical services (EMS) or local emergency response system: “phone 911.”^{10,11}
- Early bystander CPR: immediate CPR can double or triple the victim’s chance of survival from VF SCA.^{8, 12-14}
- Early delivery of a shock with a defibrillator: CPR plus defibrillation within 3 to 5 minutes of collapse can produce survival rates as high as 49% to 75%.^{15, 16}
- Early advanced life support followed by postresuscitation care delivered by healthcare providers.

Bystanders can perform 3 of the 4 links in the Chain of Survival. When bystanders recognize the emergency and activate the EMS system, they ensure that basic and advanced life support providers are dispatched to the site of the emergency. In many communities the time interval from EMS call to EMS arrival is 7 to 8 minutes or longer.¹⁷ This means that in the first minutes after collapse the victim’s chance of survival is in the hands of bystanders.

Shortening the EMS response interval increases survival from SCA, but the effect is minimal once the EMS response interval (from the time of EMS call until arrival) exceeds 5 to 6 minutes.^{18,19} EMS systems should evaluate their protocols for cardiac arrest patients and try to shorten response intervals when improvements are feasible and re- sources are available. Each EMS system should measure the rate of survival to hospital discharge for victims of VF SCA and use these measurements to document the impact of changes in procedures.²¹

Victims of cardiac arrest need immediate CPR, which provides a small but critical amount of blood flow to the heart and brain. CPR prolongs the time VF is present and increases the likelihood that a shock will terminate VF (defibrillate the heart) and allow the heart to resume an effective rhythm and effective systemic perfusion. CPR is especially important if a shock is not delivered for 4 or more minutes after collapse.²² Defibrillation does not “restart” the heart; defibrillation “stuns” the heart, briefly stopping VF and other cardiac electrical activity. If the heart is still viable, its normal pacemakers may then resume firing and produce an effective ECG rhythm that may ultimately produce adequate blood flow.

Adult BLS Sequence

The steps of BLS consist of a series of sequential assessments and actions, which are illustrated in the BLS algorithm (Figure 1). The intent of the algorithm is to present the steps in a logical and concise manner that will be easy to learn, remember, and perform.

Before approaching the victim, the rescuer must ensure that the scene is safe. Lay rescuers should move trauma victims only if absolutely necessary (e.g., the victim is in a dangerous location, such as a burning building).

Check for Response

Once the rescuer has ensured that the scene is safe, the rescuer should check for response. To check for response, tap the victim on the shoulder and ask, “Are you all right?” If the victim responds but is injured or needs medical assistance, leave the victim to phone 911. Then return as quickly as possible and recheck the victim’s condition frequently.

Activate the EMS System

If a lone rescuer finds an unresponsive adult (i.e., no movement or response to stimulation), the rescuer should activate the EMS system (phone 911), get an AED (if available), and return to the victim to provide CPR and defibrillation if needed. When 2 or more rescuers are present, one rescuer should begin the steps of CPR while a second rescuer activates the EMS system and gets the AED. If the emergency occurs in a facility with an established medical response system, notify that system instead of the EMS system.

Healthcare providers may tailor the sequence of rescue actions to the most likely cause of arrest.^{23,24} If a lone healthcare provider sees an adult or child suddenly collapse, the collapse is likely to be cardiac in origin, and the provider should phone

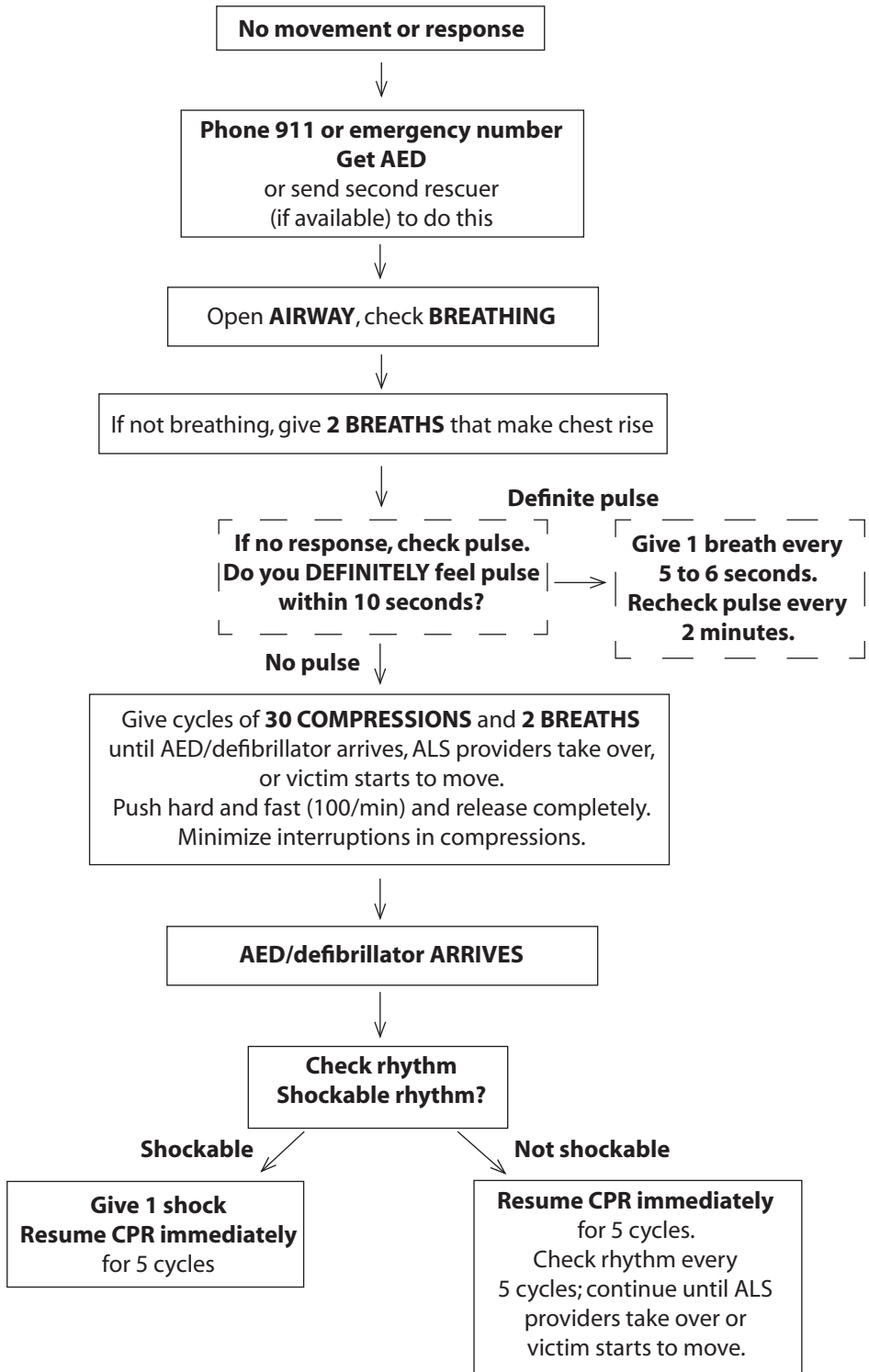


Figure 1. Adult BLS Healthcare Provider Algorithm. Boxes bordered with dotted lines indicate actions or steps performed by the healthcare provider but not the lay rescuer.

911, get an AED, and return to the victim to provide CPR and use the AED. If a lone healthcare provider aids a drowning victim or other victim of likely asphyxial (primary respiratory) arrest of any age, the healthcare provider should give 5 cycles (about 2 minutes) of CPR before leaving the victim to activate the EMS system.

When phoning 911 for help, the rescuer should be prepared to answer the dispatcher's questions about location, what happened, number and condition of victims, and type of aid provided. The caller should hang up only when instructed to do so by the dispatcher and should then return to the victim to provide CPR and defibrillation if needed.

Open the Airway and Check Breathing

To prepare for CPR, place the victim on a hard surface in a face up (supine) position. If an unresponsive victim is face down (prone), roll the victim to a supine (face up) position. If a hospitalized patient with an advanced airway (e.g., endotracheal tube, laryngeal mask airway [LMA], or esophageal-tracheal combitube [Combitube]) cannot be placed in the supine position (e.g., during spinal surgery), the healthcare provider may attempt CPR with the patient in a prone position.

Open the Airway: Lay Rescuer

The lay rescuer should open the airway using a head tilt– chin lift maneuver for both injured and noninjured victims. The jaw thrust is no longer recommended for lay rescuers because it is difficult for lay rescuers to learn and perform, is often not an effective way to open the airway, and may cause spinal movement.

Open the Airway: Healthcare Provider

A healthcare provider should use the head tilt– chin lift maneuver to open the airway of a victim without evidence of head or neck trauma. Although the head tilt– chin lift technique was developed using unconscious, paralyzed adult volunteers and has not been studied in victims with cardiac arrest, clinical²⁵ and radiographic evidence.^{26,27} have shown it to be effective. Approximately 2% of victims with blunt trauma have a spinal injury, and this risk is tripled if the victim has a craniofacial injury, a Glasgow Coma Scale score of 8 or both.²⁸ If a healthcare provider suspects a cervical spine injury, open the airway using a jaw thrust without head extension.²⁹ Because maintaining a patent airway and providing adequate ventilation is a priority in CPR, use a head tilt– chin lift maneuver if the jaw thrust does not open the airway.

Check Breathing

While maintaining an open airway, look, listen, and feel for breathing. If you are a lay rescuer and do not confidently detect normal breathing or if you are a healthcare provider and do not detect adequate breathing within 10 seconds, give 2 breaths (see below). If you are a lay rescuer and you are unwilling or unable to give rescue breaths, begin chest compressions.

Professional as well as lay rescuers may be unable to accurately determine the presence or absence of adequate or normal breathing in unresponsive victims³⁰ because the airway is not open³¹ or the victim has occasional gasps, which can

occur in the first minutes after SCA and may be confused with adequate breathing. Occasional gasps are not effective breaths. Treat the victim who has occasional gasps as if he or she is not breathing and give rescue breaths. CPR training should emphasize how to recognize occasional gasps and should instruct rescuers to give rescue breaths and proceed with the steps of CPR when the unresponsive victim demonstrates occasional gasps.

Give Rescue Breaths

Give 2 rescue breathes, each over 1 second, with enough volume to produce visible chest rise. This recommended 1-second duration to make the chest rise applies to all forms of ventilation during CPR, including mouth-to-mouth and bag-mask ventilation and ventilation through an advanced airway, with and without supplementary oxygen.

During CPR the purpose of ventilation is to maintain adequate oxygenation, but the optimal tidal volume, respiratory rate, and inspired oxygen concentration to achieve this are not known. The following general recommendations can be made:

1. During the first minutes of VF SCA, rescue breaths are probably not as important as chest compressions³² because the oxygen level in the blood remains high for the first several minutes after cardiac arrest. In early cardiac arrest, myocardial and cerebral oxygen delivery is limited more by the diminished blood flow (cardiac output) than a lack of oxygen in the blood. During CPR blood flow is provided by chest compressions. Rescuers must be sure to provide effective chest compressions (see below) and minimize any interruption of chest compressions.

2. Both ventilations and compressions are important for victims of prolonged VF SCA, when oxygen in the blood is utilized. Ventilations and compressions are also important for victims of asphyxial arrest, such as children and drowning victims who are hypoxemic at the time of cardiac arrest.

3. During CPR blood flow to the lungs is substantially reduced, so an adequate ventilation-perfusion ratio can be maintained with lower tidal volumes and respiratory rates than normal.³³ Rescuers should not provide hyperventilation (too many breaths or too large a volume). Excessive ventilation is unnecessary and is harmful because it increases intrathoracic pressure, decreases venous return to the heart, and diminishes cardiac output and survival.³⁴

4. Avoid delivering breaths that are too large or too forceful. Such breaths are not needed and may cause gastric inflation and its resultant complications.³⁵

The ECC Guidelines 2000³⁶ recommended a variety of tidal volumes, respiratory rates, and breath delivery intervals. But it is unrealistic to expect the rescuer to distinguish half-second differences in inspiratory times or to judge tidal volumes delivered by mouth-to-mouth or bag-mask ventilation. So these guidelines provide simple recommendations for delivery of rescue breaths during cardiac arrest:

- Deliver each rescue breath over 1 second.
- Give a sufficient tidal volume (by mouth-to-mouth/mask or bag mask with or without supplementary oxygen) to produce visible chest rise.
- Avoid rapid or forceful breaths.

- When an advanced airway (i.e., endotracheal tube, Combi-tube, or LMA) is in place during 2-person CPR, ventilate at a rate of 8 to 10 breaths per minute without attempting to synchronize breaths between compressions. There should be no pause in chest compressions for delivery of ventilations.

If you are delivering ventilation with a bag and mask, use an adult ventilating bag (volume of 1 to 2 L); a pediatric bag delivers inadequate tidal volume for an adult.^{37,38} When giving rescue breaths, give sufficient volume to cause visible chest rise. In 1 observational study trained BLS providers were able to detect “adequate” chest rise in anesthetized, intubated, and paralyzed adult patients when a tidal volume of approximately 400 mL was delivered.³³ It is likely, however, that a larger volume is required to produce chest rise in a victim with no advanced airway (e.g., endotracheal tube, Combitube, LMA) in place. We therefore recommend a tidal volume of 500 to 600 mL but emphasize that the volume delivered should produce visible chest rise. It is reasonable to use the same tidal volume in patients with asphyxial and arrhythmic cardiac arrest.

Mouth-to-Mouth Rescue Breathing

Mouth-to-mouth rescue breathing provides oxygen and ventilation to the victim.³⁹ To provide mouth-to-mouth rescue breaths, open the victim’s airway, pinch the victim’s nose, and create an airtight mouth-to-mouth seal. Give 1 breath over 1 second, take a “regular” (not a deep) breath, and give second rescues breathe over 1 second. Taking a regular rather than a deep breath prevents you from getting dizzy or lightheaded. The most common cause of ventilation difficulty is an improperly opened airway, so if the victim’s chest does not rise with the first rescue breath, perform the head tilt– chin lift and give the second rescue breath.

Mouth-to–Barrier Device Breathing

Despite its safety, some healthcare providers and lay rescuers may hesitate to give mouth-to-mouth rescue breathing and prefer to use a barrier device. Barrier devices may not reduce the risk of infection transmission,⁴⁰ and some may increase resistance to airflow.⁴¹ If you use a barrier device, do not delay rescue breathing. Barrier devices are available in 2 types: face shields and face masks. Face shields are clear plastic or silicone sheets that reduce direct contact between the victim and rescuer but do not prevent contamination of the rescuer’s side of the shield.⁴²

A rescuer with a duty to respond should use a face shield only as a substitute for mouth-to-mouth breathing. These responders should switch to face mask or bag-mask ventilation as soon as possible.⁴² Masks used for mouth-to-mask breathing should contain a 1-way valve that directs the rescuer’s breath into the patient while diverting the patient’s exhaled air away from the rescuer.⁴² Some masks include an oxygen inlet for administration of supplementary oxygen. When oxygen is available, healthcare providers should provide it at a minimum flow rate of 10 to 12 L/min.

Ventilation With Bag and Mask

Rescuers can provide bag-mask ventilation with room air or oxygen. A bag-mask device provides positive-pressure ventilation without an advanced airway and therefore may produce gastric inflation and its complications (see above). When using a bag-mask device, deliver each breath over a period of 1 second and provide sufficient tidal volume to cause visible chest rise.

The Bag-Mask Device

A bag-mask device should have the following^{43, 44}: a non jam inlet valve; either no pressure relief valve or a pressure relief valve that can be bypassed; standard 15-mm/22-mm fittings; an oxygen reservoir to allow delivery of high oxygen concentrations; a nonrebreathing outlet valve that cannot be obstructed by foreign material and will not jam with an oxygen flow of 30 L/min; and the capability to function satisfactorily under common environmental conditions and extremes of temperature.

Masks should be made of transparent material to allow detection of regurgitation. They should be capable of creating a tight seal on the face, covering both mouth and nose. Masks should be fitted with an oxygen (insufflation) inlet, have a standard 15-mm/22-mm connector,⁴⁵ and should be available in one adult and several pediatric sizes.

Bag-Mask Ventilation

Bag-mask ventilation is a challenging skill that requires considerable practice for competency.^{46, 47} The lone rescuer using a bag-mask device should be able to simultaneously open the airway with a jaw lift, hold the mask tightly against the patient's face, and squeeze the bag. The rescuer must also watch to be sure the chest rises with each breath. Bag-mask ventilation is most effective when provided by 2 trained and experienced rescuers. One rescuer opens the airway and seals the mask to the face while the other squeezes the bag. Both rescuers watch for visible chest rise.^{46, 47} The rescuer should use an adult (1 to 2 L) bag to deliver a tidal volume sufficient to achieve visible chest rise. If the airway is open and there are no leaks (i.e., there is a good seal between face and mask), this volume can be delivered by squeezing a 1-L adult bag about one half to two thirds of its volume or a 2-L adult bag about one-third its volume. As long as the patient does not have an advanced airway in place, the rescuer(s) should deliver cycles of 30 compressions and 2 breaths. The rescuer delivers the breaths during pauses in compressions and delivers each breathe over 1 second.

The healthcare provider should use supplementary oxygen (O₂ 40%, a minimum flow rate of 10 to 12 L/min) when available. Ideally the bag should be attached to an oxygen reservoir to enable delivery of 100% oxygen. Advanced airway devices such as the LMA145 and the esophageal-tracheal Combitube are currently within the scope of BLS practice in a number of regions (with specific authorization from medical control).⁴⁸ These devices may provide acceptable alternatives to bag-mask devices for healthcare providers who are well trained and have sufficient experience to use them. It is not clear that these devices are any more or less complicated to use

than a bag and mask; training is needed for safe and effective use of both the bag-mask device and each of the advanced airways.

Ventilation With an Advanced Airway

When the victim has an advanced airway in place during CPR, 2 rescuers no longer deliver cycles of CPR (i.e., compressions interrupted by pauses for ventilation). Instead, the compressing rescuer should give continuous chest compressions at a rate of 100 per minute without pauses for ventilation. The rescuer delivering ventilation provides 8 to 10 breaths per minute. The 2 rescuers should change compressor and ventilator roles approximately every 2 minutes to prevent compressor fatigue and deterioration in quality and rate of chest compressions. When multiple rescuers are present, they should rotate the compressor role about every 2 minutes.

Rescuers should avoid excessive ventilation by giving the recommended breaths per minute and limiting tidal volume to achieve chest rise.³⁴ A translational research study showed that delivery of 12 breaths per minute during CPR leads to increased intrathoracic pressure, impeding venous return to the heart during chest compressions.³⁴ Reduced venous return leads to diminished cardiac output during chest compressions and decreased coronary and cerebral perfusion.^{49, 50} It is critically important that rescuers maintain a ventilation rate of 8 to 10 breaths per minute during CPR and avoid excessive ventilation.^{34, 50}

Cricoid Pressure

Pressure applied to the victim's cricoid cartilage pushes the trachea posteriorly, compresses the esophagus against the cervical vertebrae, and can prevent gastric inflation and reduce the risk of regurgitation and aspiration.^{51, 52} Application of cricoid pressure usually requires a third rescuer, one who is not responsible for chest compressions or ventilations. Cricoid pressure should be used only if the victim is deeply unconscious (i.e., has no cough or gag reflex).

Pulse Check (for Healthcare Providers)

Lay rescuers fail to recognize the absence of a pulse in 10% of pulseless victims (poor sensitivity for cardiac arrest) and fail to detect a pulse in 40% of victims with a pulse (poor specificity). In the ECC Guidelines 2000³⁶ the pulse check was deleted from training for lay rescuers and de-emphasized in training for healthcare providers. There is no evidence, however, that checking for breathing, coughing, or movement is superior for detection of circulation.⁵³ For ease of training, the lay rescuer will be taught to assume that cardiac arrest is present if the unresponsive victim is not breathing. Healthcare providers also may take too long to check for a pulse, and have difficulty determining if a pulse is present or absent. The healthcare provider should take no more than 10 seconds to check for a pulse. If a pulse is not definitely felt within 10 seconds, proceed with chest compressions.

Rescue Breathing Without Chest Compressions

If an adult victim with spontaneous circulation (i.e., palpable pulses) requires support of ventilation, give rescue breaths at a rate of 10 to 12 breaths per minute, or about 1 breath every 5 to 6 seconds. Each breath should be given over 1 second regardless of whether an advanced airway is in place. Each breath should cause visible chest rise. During delivery of rescue breaths, reassess the pulse approximately every 2 minutes, but spend no more than 10 seconds doing so.

Chest Compressions

Chest compressions consist of rhythmic applications of pressure over the lower half of the sternum. These compressions create blood flow by increasing intrathoracic pressure and directly compressing the heart. Although properly performed chest compressions can produce systolic arterial pressure peaks of 60 to 80 mm Hg, diastolic pressure is low and mean arterial pressure in the carotid artery seldom exceeds 40 mm Hg.

Blood flow generated by chest compressions delivers a small but critical amount of oxygen and substrate to the brain and myocardium. In victims of VF SCA, chest compressions increase the likelihood that a shock (i.e., attempted defibrillation) will be successful. Chest compressions are especially important if the first shock is delivered 4 minutes after collapse.²²

Much of the information about the physiology of chest compressions and the effect of varying compression rates, compression-ventilation ratios, and duty cycles (percent of time the chest is compressed versus time allowed for chest recoil) is derived from animal models. Researchers at the 2005 Consensus Conference,⁵⁴ however, reached several conclusions about chest compressions:

1. “Effective” chest compressions are essential for providing blood flow during CPR.
2. To give “effective” chest compressions, “push hard and push fast.” Compress the adult chest at a rate of about 100 compressions per minute, with a compression depth of 1½ to 2 inches (approximately 4 to 5 cm). Allow the chest to recoil completely after each compression, and allow approximately equal compression and relaxation times.
3. Minimize interruptions in chest compressions.
4. Further studies are needed to define the best method for coordinating ventilations and chest compressions and to identify the best compression-ventilation ratio in terms of survival and neurologic outcome.

Technique

To maximize the effectiveness of compressions, the victim should lie supine on a hard surface (e.g., backboard or floor),⁵⁸ with the rescuer kneeling beside the victim’s thorax.⁵⁶ The safety and efficacy of over-the-head CPR (OTH- CPR) for lone rescuers and 2-person straddle CPR are unknown, but these techniques may be advantageous in confined spaces.⁵⁷ “CPR-friendly” deflatable mattresses have been

studied, and they do not provide an adequate surface on which to perform chest compressions.⁵⁷

The rescuer should compress the lower half of the victim's sternum in the center (middle) of the chest, between the nipples.⁵⁵ The rescuer should place the heel of the hand on the sternum in the center (middle) of the chest between the nipples and then place the heel of the second hand on top of the first so that the hands are overlapped and parallel.⁵⁸ Depress the sternum approximately 1½ to 2 inches (approximately 4 to 5 cm) and then allow the chest to return to its normal position. Complete chest recoil allows venous return to the heart, is necessary for effective CPR, and should be emphasized in training.⁵⁹ Compression and chest recoil/relaxation times should be approximately equal.⁶⁰

Lay rescuers should continue CPR until an AED arrives, the victim begins to move, or EMS personnel take over CPR. Lay rescuers should no longer interrupt chest compressions to check for signs of circulation or response. Healthcare providers should interrupt chest compressions as infrequently as possible and try to limit interruptions to no longer than 10 seconds except for specific interventions such as insertion of an advanced airway or use of a defibrillator. We strongly recommend that patients not be moved while CPR is in progress unless the patient is in a dangerous environment or is a trauma patient in need of surgical intervention. CPR is better and has fewer interruptions when the resuscitation is conducted where the patient is found. Allow the chest wall to recoil completely after each compression.

Rescuer fatigue may lead to inadequate compression rates or depth. Significant fatigue and shallow compressions are seen after 1 minute of CPR, although rescuers may deny that fatigue is present for 5 minutes. When 2 or more rescuers are available, it is reasonable to switch the compressor about every 2 minutes (or after 5 cycles of compressions and ventilations at a ratio of 30:2). Every effort should be made to accomplish this switch in 5 seconds. If the 2 rescuers are positioned on either side of the patient, one rescuer will be ready and waiting to relieve the "working compressor" every 2 minutes.

Compression-Ventilation Ratio

A compression-ventilation ratio of 30:2 is recommended and further validation of this guideline is needed.^{49, 50} In infants and children, 2 rescuers should use a ratio of 15:2. This 30:2 ratio is based on a consensus of experts rather than clear evidence. It is designed to increase the number of compressions, reduce the likelihood of hyperventilation, minimize interruptions in chest compressions for ventilation, and simplify instruction for teaching and skills retention. Once an advanced airway is in place, 2 rescuers no longer deliver cycles of CPR (i.e., compressions interrupted by pauses for ventilation). Instead, the compressing rescuer should give continuous chest compressions at a rate of 100 per minute without pauses for ventilation. The rescuer delivering ventilation provides 8 to 10 breaths per minute. The 2 rescuers should change compressor and ventilator roles approximately every 2 minutes to prevent compressor fatigue and deterioration in quality and rate of

chest compressions. When multiple rescuers are present, they should rotate the compressor role about every 2 minutes.

The compression rate refers to the speed of compressions, not the actual number of compressions delivered per minute. The actual number of chest compressions delivered per minute is determined by the rate of chest compressions and the number and duration of interruptions to open the airway, deliver rescue breaths, and allow AED analysis.⁶¹ Rescuers must make every effort to minimize these interruptions in chest compressions.

Defibrillation

All BLS providers should be trained to provide defibrillation because VF is the most common rhythm found in adults with witnessed, nontraumatic SCA.⁷ For these victims survival rates are highest when immediate bystander CPR is provided and defibrillation occurs within 3 to 5 minutes.^{8, 12–14}

Immediate defibrillation is the treatment of choice for VF of short duration, such as witnessed SCA.

The effect of CPR before defibrillation for prolonged VF SCA has largely been positive. When EMS arrived more than 436 to 537 minutes after dispatch, a brief period of CPR (1½ to 3 minutes) before defibrillation improved ROSC and survival rates for adults with out-of-hospital VF/VT in a before-after study.³⁶ Thus, for adult out-of-hospital cardiac arrest that is not witnessed by the EMS provider, rescuers may give a period of CPR (e.g., about 5 cycles or about 2 minutes) before checking the rhythm and attempting defibrillation. In settings with lay rescuer AED programs (AED on-site and available) and for in-hospital environments or if the EMS rescuer witnesses the collapse, the rescuer should use the defibrillator as soon as it is available.

Foreign-Body Airway Obstruction (Choking)

Death from FBAO is an uncommon but preventable cause of death.⁶³ Most reported cases of FBAO in adults are caused by impacted food and occur while the victim is eating. Most reported episodes of choking in infants and children occur during eating or play, when parents or childcare providers are present. The choking event is therefore commonly witnessed, and the rescuer usually intervenes while the victim is still responsive.

Recognition of Foreign-Body Airway Obstruction

Because recognition of airway obstruction is the key to successful outcome, it is important to distinguish this emergency from fainting, heart attack, seizure, or other conditions that may cause sudden respiratory distress, cyanosis, or loss of consciousness.

Foreign bodies may cause either mild or severe airway obstruction. The rescuer should intervene if the choking victim has signs of severe airway obstruction. These include signs of poor air exchange and increased breathing difficulty, such as a silent

cough, cyanosis, or inability to speak or breathe. The victim may clutch the neck, demonstrating the universal choking sign. Quickly ask, “Are you choking?” If the victim indicates “yes” by nodding his head without speaking, this will verify that the victim has severe airway obstruction.

Relief of Foreign-Body Airway Obstruction

When FBAO produces signs of severe airway obstruction, rescuers must act quickly to relieve the obstruction. If mild obstruction is present and the victim is coughing forcefully, do not interfere with the patient’s spontaneous coughing and breathing efforts. Attempt to relieve the obstruction only if signs of severe obstruction develop: the cough becomes silent, respiratory difficulty increases and is accompanied by stridor, or the victim becomes unresponsive. Activate the EMS system quickly if the patient is having difficulty breathing. If more than one rescuer is present, one rescuer should phone 911 while the other rescuer attends to the choking victim.

Although chest thrusts, back slaps, and abdominal thrusts are feasible and effective for relieving severe FBAO in conscious (responsive) adults and children 1 year of age, for simplicity in training we recommend that the abdominal thrust be applied in rapid sequence until the obstruction is relieved. If abdominal thrusts are not effective, the rescuer may consider chest thrusts. It is important to note that abdominal thrusts are not recommended for infants 1 year of age because thrusts may cause injuries.

Chest thrusts should be used for obese patients if the rescuer is unable to encircle the victim’s abdomen. If the choking victim is in the late stages of pregnancy, the rescuer should use chest thrusts instead of abdominal thrusts. Because abdominal thrusts can cause injury,⁶⁴ victims of FBAO who are treated with abdominal thrusts should be encouraged to undergo an examination by a physician for injury. Epidemiologic data⁶³ does not distinguish between FBAO fatalities in which the victims were responsive when first encountered and those in which the victims were unresponsive when initially encountered. However, the likelihood that a cardiac arrest or unresponsiveness will be caused by an unsuspected FBAO is thought to be low.⁶³

If the adult victim with FBAO becomes unresponsive, the rescuer should carefully support the patient to the ground, immediately activate EMS, and then begin CPR. A randomized trial of maneuvers to open the airway in cadavers and prospective studies in anesthetized volunteers show that higher sustained airway pressures can be generated using the chest thrust rather than the abdominal thrust.⁶⁵ ⁶⁶ Each time the airway is opened during CPR, the rescuer should look for an object in the victim’s mouth and remove it. Simply looking into the mouth should not increase the time it takes to attempt the ventilations and proceed to the 30 chest compressions.

A healthcare provider should use a finger sweep only when the provider can see solid material obstructing the airway of an unresponsive patient. No studies have evaluated the routine use of the finger sweep to clear an airway in the absence of

visible airway obstruction.²² The recommendation to use the finger sweep in past guide- lines was based on anecdotal reports that suggested that it was helpful for relieving an airway obstruction.⁶⁴

Summary: The Quality of BLS

Methods should be developed to improve the quality of CPR delivered at the scene of cardiac arrest by healthcare providers and lay rescuers. These may include education, training, assistance or feedback from biomedical devices, mechanical CPR, and electronic monitoring. Components of CPR known to affect hemodynamics include ventilation rate and duration, compression depth, compression rate and number, complete chest recoil, and hands-off time. Systems that deliver professional CPR should implement processes of continuous quality improvement that include monitoring the quality of CPR delivered at the scene of cardiac arrest, other process-of-care measures (e.g., initial rhythm, bystander CPR, and response intervals), and patient outcome up to hospital discharge. This evidence should be used to maximize the quality of CPR delivered.

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Summary of BLS ABCD Maneuvers for Infants, Children, and Adults (Newborn Information Not Included)

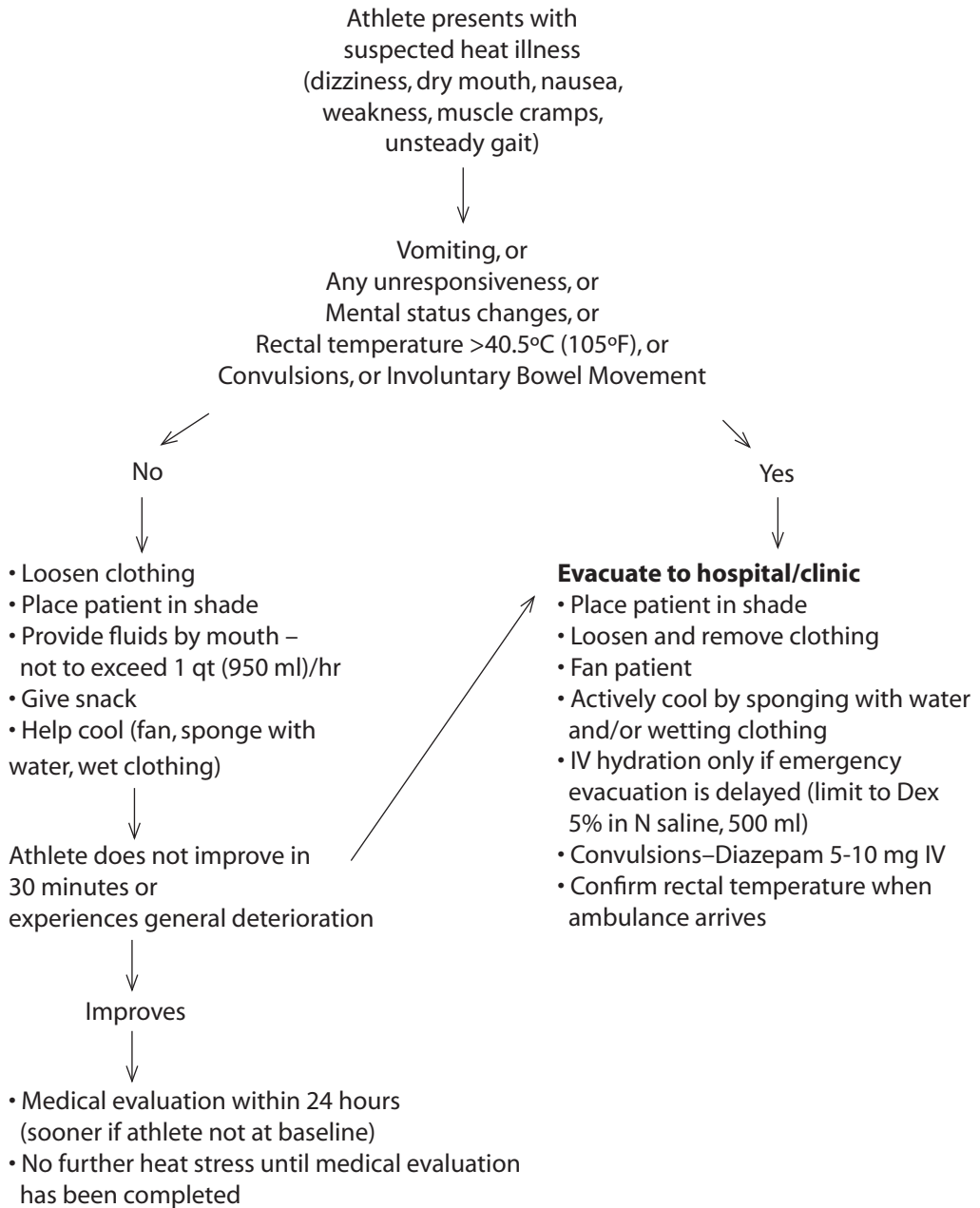
Maneuver	Adult Lay rescuer: ≥8 years HCP*: Adolescent and older	Child Lay rescuers: 1–8 years HCP: 1 year to adolescent	Infant Under 1 year of age
Airway	Head tilt–chin lift (HCP: suspected trauma, use jaw thrust)		
Breathing Initial	2 breaths at 1 second/breath	2 effective breaths at 1 second/breath	
HCP: Rescue breathing without chest compressions	10–12 breaths/minute (approx.)	12–20 breaths/minute (approximate)	
HCP: Rescue breaths for CPR with advanced airway	8–10 breaths/minute (approximately)		
Foreign-body airway obstruction	Abdominal thrusts		Back slaps and chest thrusts
Circulation HCP: Pulse check (≤10 sec)	Carotid		Brachial or femoral
Compression landmarks	Lower half of sternum, between nipples		Just below nipple (lower half of sternum)
Compression method Push hard and fast; allow complete recoil	Heel of one hand; other hand on top	Heel of one hand or as for adult	2 or 3 fingers HCP (2 rescuers): 2 thumb-encircling hands
Compression depth	1 1/2–2 inches	Approximately 1/3 to 1/2 the depth of the chest	
Compression rate	Approximately 100/minute		
Compression-ventilation ratio	30:2 (one or two rescuers)	30:2 (single rescuer) HCP: 15:2 (2 rescuers)	
Defibrillation AED	Use adult pads Do not use child pads	Use AED after 5 cycles of CPR (out of hospital)	No recommendations for infants <1 year of age
	Use pediatric system for child 1–8 years if available		

HCP: For sudden collapse (out of hospital) or in-hospital arrest, use AED as soon as available

***Note: Maneuvers used by only Healthcare Providers are indicated by HCP.**

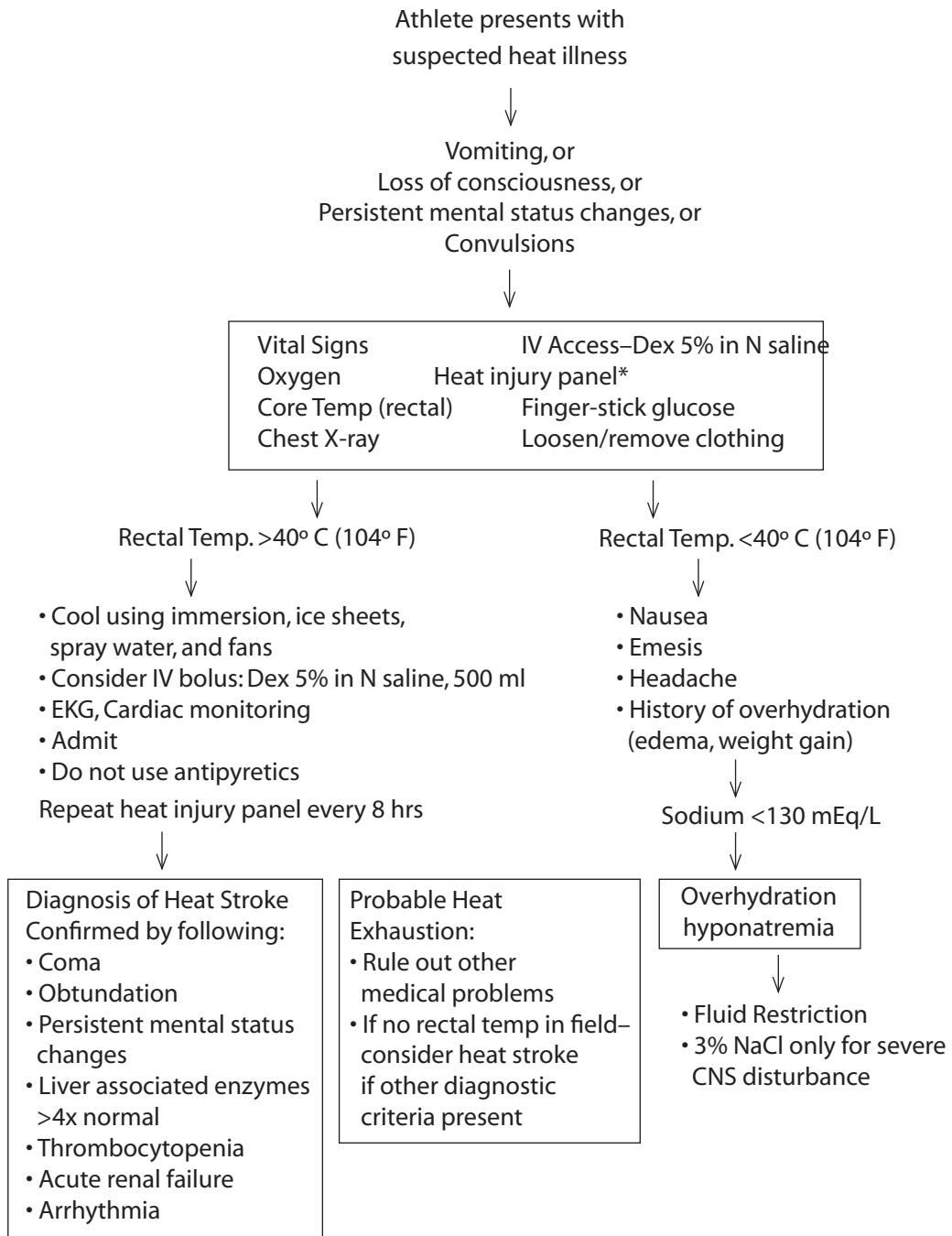
Appendix 8

Differential Diagnosis and Treatment of Exertional Heatstroke and Heat Exhaustion on Site



Appendix 9

Differential Diagnosis and Treatment of Exertional Heatstroke, Heat Exhaustion, and Hyponatremia in a Clinic Setting



*Heat injury panel: Liver, CPK, electrolytes, renal, CBC, urinalysis, PT/PTT

Appendix 10

Recommendations for Minimising Jet Lag

Three Days Before Travel

- On this day, go to bed at your usual time but avoid any sleep deprivation. Be sure to get a minimum of 7 1/2 hours of sleep.
- Begin to utilize a high protein/low carbohydrate breakfast and lunch, and a high carbohydrate dinner.

Two Days Before Travel

- Continue the pattern of high protein breakfast and lunch (no caffeine), and high carbohydrate dinner, but eat skimpier portions. At tea time, you can still have caffeine but reduce sweets. Snack during the day should consist of low calorie, high protein foods (for example, chicken soup with pieces of meat but no noodles). No evening snacks are permitted.
- If you are training, do not reduce your caloric intake too much. If you usually burn 2500 calories a day, do not fall below this level.
- Again, follow your usual sleep schedule, and avoid any sleep deprivation (get a minimum of 7 1/2 hours sleep).

One Day Before Travel

- You will likely be running around making last-minute preparations. This will stimulate you, so do this early in the day. If you do not have a travel kit, prepare one early in the day (see below for recommended contents). You may also want to prepare a high protein “picnic” for the flight. It will supplement food provided on the plane and may be your first meal on your new time. Remember, travel kit foods must either not need refrigeration or be eaten before they spoil. These foods are intended “for emergency use”; the airline will usually try to accommodate your schedule if you ask them.
- On this evening, say your goodbyes to friends and family, since you will have many other things to keep you busy on the travel day. You may want to remain awake a bit later than usual—feel free to do this.
- Delaying sleep activity helps adjust your biological clock. However, avoid any sleep deprivation, and get a minimum of 7 1/2 hours sleep.

Travel Day: West to East

- If your schedule allows it, sleep in. Try to get up to 8–9 hours of sleep.
- If you use caffeine, drink 2–3 cups of a strong caffeinated beverage immediately upon awakening (before 11 a.m.) but do not take in any more caffeine, if possible, for the rest of the day. The caffeine “jolt” in the morning will push your body in the right direction.
- If you can, take a nap after breakfast, and keep your activity to a minimum throughout the day. Avoid the morning sunlight as much as possible. When

you depart from your home city, feel free to sleep on the plane, and do not drink caffeine-containing beverages.

- **If you train** before departing, have a late, low calorie, high protein breakfast of just enough calories to get you through the workout. If possible, reduce your workout schedule this day and cut back your calories accordingly; be careful, however, not to cut back calories to the point that you feel weak. **If you do not train**, remember you are burning calories like a non-athlete, so consume a very skimpy, *high protein*, low calorie breakfast.
- **After breakfast** do not eat again until breakfast time in your destination city. Physical activity on this day should be kept to a minimum.
- It is suggested you put on two watches. Set one to destination time and one to home time. Use the watch for home time until you get on the plane to the destination city, but begin to think about what you would be doing if you were already on the destination time. Once you get on the plane, use the watch set to the destination time and take off the watch set to home time.
- Remember to carry your in-flight “travel kit” with you.
- Arrive at the airport early to avoid the rush. If sleeping is a normal activity on destination time, feel free to nap while waiting in the airport.
- Continue to fast until breakfast time in the destination city, and then, even if you are still in flight, break the fast with breakfast eaten on destination time. This large, high-protein breakfast begins a feast day, and represent the start of eating all your meals on destination time.
- If you arrive during the day, rest as much as possible on the plane. Grab a blanket and pillow, put on your blindfold, and insert your ear plugs to simulate night. Loosen restrictive clothing, or change into comfortable clothing, if possible.
- If it is night when you arrive, stay awake as much as possible on the plane. This helps you to be ready to sleep following your arrival.
- When your watch tells you it is time to be awake on destination time, get up and move around; take your travel kit to the toilet and wash your face, brush your teeth, etc.—that is, perform your usual morning rituals as much as possible. Interact with people to stimulate your body. If possible, walk up and down the aisle, and do simple isometric or stretching exercises.
- At breakfast and/or lunch time in the destination city, have high protein, high calorie meals. This helps push your body’s biochemistry into its new time frame. Eat the high protein picnic you brought with you if the airline doesn’t serve appropriate food at appropriate times. Do not worry if the foods look more like a dinner or snack; your body sees it as a source of protein. If eating the meal supplied by the airline, eat only the protein portions and ignore starches and sweets. Supplement this meal with your picnic. If you arrive in the destination only in time for lunch, you may eat

most of your picnic at this time, but keep some food in reserve in case you are detained by airport security or delayed between flights (if changing planes). Be sure that, according to the destination city time, you eat a high protein lunch on this day.

- During the flight, drink plenty of fluids, avoiding alcohol and caffeinated beverages. Humidity in jet cabins is very low, so liquid intake is important to avoid dehydration. Alcohol and caffeinated beverages are diuretics that cause the body to eliminate fluid. Water and fruit juices are the best fluids to drink.

Recommended Contents of In-Flight Travel Kit

- Essential toiletries: Toothbrush, toothpaste, razor, small hand towel, lotion, etc.
- High-protein snacks: Small package of cheese and crackers, hard boiled egg(s), low-sugar granola bars, peanut butter, nuts
- Eye mask or blindfold
- Ear plugs
- Light-weight slippers
- Books, cards, mp3 player, DVD player, or other items to entertain you during waking hours

Appendix 11

General Health and Hygiene: Recommendations for Athletes

The serious athlete must assume much of the responsibility for maintaining his/her health in order to train and compete well. These guidelines also apply to healthful living in general

A. Healthy Lifestyle Factors

- Rest and sleep adequately between periods of hard training or other intense physical activity. There is no hard and fast rule about the number of hours of sleep needed, although in general 6–8 hours of sleep should be sufficient. It's the soundness of the sleep, more than the number of hours, that's important. Each person has his/her own individual sleep requirements. Avoid unnecessary loitering (as per shopping trips, etc.) before competition time.
- Don't smoke. Using tobacco is detrimental to health in general and physical fitness in particular. Immediate acute effects include decrease in oxygen carrying capacity of the blood, elevation of pulse rate, increased coagulability of the blood, and spasm of the coronary blood vessels. Lung and heart disease as well as lung cancer are a few of the consequences of long-term tobacco addiction.
- Avoid other than moderate intake of alcohol.
- Say no to drugs. Drugs are no substitute for proper training. They are banned. You can be caught and the consequences to your athletic career are serious. Even if you are not caught, they have serious short and long-term effects on your health.
- Be sensible with respect to your sexual activities. Normal sex with legitimate partners is not detrimental to high level sports performance. However, stress associated with illicit or clandestine sexual encounters can affect performance. And remember, always practice safe sex (see C. 4).
- Eat a balanced diet commensurate with your type and level of activity. Follow the advice of your nutritionist, dietician, or doctor. Be sensible in your attitudes towards food fads, and avoid overindulgence. Adhere to strict timing of your meals prior to training or competition.

B. Basic Personal Hygiene

- Bathe regularly, use soap and dry your skin thoroughly afterwards. Launder and change your clothes regularly. This will minimise the risk of bacterial and fungal skin infection.
- Choose clothing to suit weather conditions. Clothes should fit properly to avoid chafing and abrasions.
- Abstain from using material/articles that contain substances that cause you allergic reactions or contact dermatitis.

- Choose appropriate and good quality footwear that fits properly to avoid blisters. Break in new shoes gradually, padding them at points of pressure or friction.
- Take care of your teeth. Brush and floss daily. See the dentist regularly to ensure early diagnosis of problems.

C. Infections (see also Appendix 12, *Respiratory Tract Infections*)

- A high level of fitness does not protect you against infections, especially viral infections. Avoid deliberate exposure to situations where airborne infections are facilitated. Minimise contacts with infected/sick people, animals, and contagious objects.
- When you have an infection, especially with a fever, refrain from strenuous physical activity.
- Food and waterborne bacteria, viruses, and protozoa can cause diarrhea. This is especially likely during travel to foreign countries. Eat only wholesome food that is freshly cooked under hygienic conditions. When the safety of the water supply is not assured drink only boiled or properly bottled water or beverages.
- Sexually transmitted diseases (STDs), including AIDS, are usually transmitted through sexual activity. Practice safe sex, that is, use latex condoms. AIDS and Hepatitis B can also be transmitted through contaminated needles and contaminated blood products. Should you become infected and have symptoms, seek proper medical attention immediately. Do not self-treat or share medicine with friends to avoid incomplete treatment or masking of other concomitant STDs.

D. Bronchial Asthma

It is not uncommon for good athletes to be asthmatic. Asthma by itself is not a contraindication to physical activity. Control your asthma through proper medical attention. Remember that some medications for asthma or other respiratory infections contain substances that are on the list of banned drugs. For this reason, it is best not to use over-the-counter medications.

E. Immunisations and Other Prophylactic Measures

Most countries have childhood immunisation programs against serious infectious diseases, such as TB, polio, diphtheria, whooping cough, tetanus, measles, and “German measles” (rubella) for females of childbearing age. In addition there are efficacious vaccines against hepatitis B, mumps, Japanese B encephalitis, and typhoid. Additional vaccines may be necessary when travelling to areas where these diseases may be endemic. Inquire through your local health department concerning outbreaks and recommended immunisations before you travel. Make sure that you are up to date with all vaccines needed at home and for travels.

Chemoprophylaxis, or prevention of disease through medications, should also be considered when travelling to areas where malarial infection is possible. This could also be considered for prevention of meningococcal meningitis and traveller's diarrhea.

Appendix 12

Respiratory Tract Infections (RTI)

A. Prevention

- Keep your distance from people who are coughing, sneezing or have a “runny nose.”
- Wash hands regularly, before meals, and after direct contact with potentially contagious people, animals, blood, secretions, public places, bathrooms, etc.
- Do not use other people’s drinking bottles, cups, cutlery, etc.
- Wear proper outdoor clothing and avoid getting cold and wet after exercise.
- Use disposable paper towels and limit hand to mouth/nose contact when suffering from respiratory tract infection (RTI) symptoms.
- Quickly isolate a team member with RTI symptoms and move his/her roommate to other accommodations.
- Check air conditioning/ventilation systems for potentially contagious material.
- Protect upper and lower airways from being directly exposed to cold and dry air during strenuous exercise, by using a face mask, etc., at temperatures below -15°C.
- Practice good recovery routines, including proper nutrition and rehydration.

B. Guidelines for Exercise During Episodes of RTI

First day of illness

- Cease strenuous exercise or competitions when experiencing RTI symptoms such as:
 - Sore throat or coughing
 - Runny or congested nose
- Cease all exercise when experiencing additional RTI symptoms such as:
 - Muscle/joint pain and headache
 - Fever and generalised feeling of malaise
- Drink plenty of fluids, keep from getting wet and cold, and minimise life-stress.
- Consider use of topical therapy with nasal drainage, decongestants, and analgesics if fever occurs.
- Report illness to a team physician or health care personnel and keep away from other athletes if you are part of a team training or travelling together.

Second day

- If you have a fever (temperature >37.5–38°C) or increased coughing:
No training!

- If no fever or malaise and no worsening of “above the collar” symptoms: Light exercise (pulse <120bpm) for 30–45 minutes by yourself (indoors during the winter).

Third day

- If fever and RTI symptoms persist: Consult your (team) physician by phone or at office.
- If no fever or malaise and no worsening of initial symptoms: Moderate exercise (pulse < 150bpm) for 45–60 minutes, preferably by yourself and indoors.

Fourth day

- If no symptom relief: Do not try to exercise but make an office visit to your doctor.
- If first day of improved condition: Follow the guidelines for “return to exercise after RTI,” below.

C. Guidelines for Returning to Exercise After RTI

- Make sure that you have one day without fever and with improvement of RTI symptoms before returning to exercise.
- Observe the body’s reaction to your first exercise session before starting on a new session.
- Stop physical exercise and consult your physician if:
 - A new episode with fever or worsening of initial symptoms occurs.
 - Persistent coughing and exercise-induced breathing problem occurs.
- Use the same number of days to step up to normal training as spent off of regular training because of illness.
- Observe closely your tolerance to increased exercise intensity and take an extra day off if you do not recover satisfactorily.
- Use proper outdoor clothing and specific cold air protection for airways when exercising in temperatures below -10°C the first week after RTI.

Adapted from: Ronsen, O. Prevention and management of of respiratory tract infections in athletes. New Studies in Athletics 20:3, 49-56, 2005.

Appendix 13

Process for the Management of Gender-Related Issues

1. Gender-related issues will be managed according to the IAAF Policy for Gender Verification, and the IOC Consensus Statement on Sex Reassignment.
2. Gender issues are likely to arise as a result of:
 - a. A “challenge” by another athlete or team as brought forward to authorities at an athletics event.
 - b. “Suspicion” raised as to an athlete’s gender as witnessed during the process of specimen collection during doping control.
 - c. An approach made to an IAAF/Regional AAA or National Federation by an athlete or his/her representative requesting advice or clarification.
3. The matter may be handled at various levels, including:
 - National Federation
 - The Medical Delegate of an athletics event
 - The IAAF Medical/Anti-Doping Commission
4. Procedures
 - a. The case is brought to the attention of the relevant medical authority at one of the above levels when it first arises.
 - b. The authority decides whether there is reason to investigate.
 - c. The authority determines who will investigate the matter, e.g., Special Panel at an international event.
 - d. The athlete is referred to the investigating authority in confidence for further investigation and advice.
 - e. The verdict is passed on to the National Federation with advice for further action, including appropriate advice to the athlete as to the need to “withdraw” from competition until the problem is definitely resolved through appropriate medical and surgical measures.
 - f. Evaluation of the effects of such measures to determine if and when the athlete can return to competition as per IOC Consensus guidelines.
5. The IAAF must establish a resource panel at the Medical/Anti-Doping Commission level that may be available for consultation if there is a need for resolution of complex cases.