F-MARC

Football Medicine Manual

2nd Edition

FIFA®

For the Game. For the World.

F-MARC
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Contents

Preface

Football is the most popular sport in the world. There are currently about 265 million players, 26 million of whom are women. The ever-increasing number of active players, leading in turn to more injuries with resultant costs for treatment and loss of playing time, has long demonstrated the need for an injury prevention programme.

The Fédération Internationale de Football Association (FIFA), world football's governing body, which currently unifies 208 member associations, has realised that it has the responsibility to guarantee a smooth management of the various competitions for all age groups and levels of play and for both sexes in the spirit of fair play. **FIFA is also aware of its responsibility for the health of its football players and has therefore decided to play an active role in developing and supporting research on football.** In 1994, following the initiative of today's FIFA President, Joseph S. Blatter, the FIFA Medical Assessment and Research Centre (F-MARC) was established to provide the scientific evidence to facilitate the decision-making of the FIFA Executive Committee, the Football and Strategic Committees of FIFA as well as the International Football Association Board (IFAB). Apart from assessing the epidemiological data, the individual risk factors for injury and the environmental influences on players' health, **F-MARC's focus is on developing a football-specific injury prevention programme and on improving the standards of care.**

It is important that the FIFA administration, the confederations and the member associations are all informed about the medical advances for the benefit of football players. Reducing or preventing injuries during match play or training, being aware of the current status regarding state-of-the-art diagnosis and treatment of injuries and other football-related complaints are of primary importance. It is also important that the governing bodies are appropriately informed in a timely manner about any negative aspects or developments in the game so that they can act accordingly, particularly when adapting the Laws of the Game is recommended to protect players' health.

As part of a sustainable development of football medicine, the idea of establishing independent medical centres for football players has been further elaborated. The first **FIFA Medical Centre of Excellence** was inaugurated at the Schulthess Clinic in Zurich in May 2005, close to FIFA's headquarters. Since then, nine further FIFA Medical Centres of Excellence have been accredited in the different confederations.

In order to have an effective tool to disseminate the latest advances in knowledge among the 208 member associations, **F-MARC is permanently extending its network of FIFA medical officers**, who offer the best possible medical care to football players in all member countries. The first edition of the Football Medicine Manual was the first step in the process of developing a standardised educational curriculum for physicians and physiotherapists. To improve the quality of the educational process, basic and new information is continuously made available to the participants of instructor courses, FUTURO III football medicine courses and, henceforward, also on the FIFA football medicine extranet (www.FIFA.com/medical).

FIFA has a strong opinion and a clearly defined strategy in the fight against doping. To date, the annual FIFA anti-doping statistics have shown no indication of systematic doping in football. There is no scientific evidence to assume that doping considerably improves the overall performance of football players at any skill level. It is not only of limited use, but more importantly, it is potentially harmful to the individual player. FIFA has developed a worldwide network of FIFA doping control officers, who are required to be physicians. **Continuing anti-doping education by the medical profession is of great importance to players and all related personnel.** A better understanding among the football community of the risks and limits of doping in football is more important to FIFA than the development of further control mechanisms, even though these are necessary, as these serve more as a monitoring system.

This second edition of the F-MARC Football Medicine Manual is being launched to mark the FIFA Medical Conference in Zurich in October 2009. The FIFA President, together with the FIFA Executive Committee, has invited all 208 presidents of FIFA's member associations, together with the chairman of the medical committee or medical director of each member association, to this unique event in order to provide them with the opportunity to learn about the current body of knowledge in football medicine and to take it home and use it for the maximum benefit of each individual association. The authors and all collaborators involved in this second edition of the Football Medicine Manual present this book to the football community as a gift on the occasion of 15 years of F-MARC, and for the years to come, with the following objective:

To protect the players' health and to promote football as a health-enhancing leisure activity, thereby laying the foundation for playing for fun, playing fair and, above all, contributing to a better world.

Zurich, May 2009

FIFA Medical Assessment and Research Centre (F-MARC)


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18

1. Background information

1.1 The F-MARC concept of prevention

F-MARC's approach to reducing the incidence and minimising the short and long-term consequences of acute and overuse/gradual-onset injuries in football is based on the principle of risk management, which is summarised in Figure 1.1.1.

The three stages described in Figure 1.1.1 as "Identify risk factors", "Estimate the risk" and "Evaluate the risk" are referred to collectively as risk assessment.

Risk factors

There are formal directives contained in the Laws of the Game about the size of a football pitch, the number of players in a team, the equipment that can be used and the match officials required for controlling competitive games of football. However, the minimum requirements for most people to play football for fun and exercise are any playing area, two goals, a ball and sufficient people to create two teams. Irrespective of whether participation in football is for fun, exercise or as a professional player, there are a number of factors that can impact on a player's risk of injury. These risk factors are sub-divided into extrinsic (situation or event-based) and intrinsic (player-based) factors.

Intrinsic risk factors include issues that are specifically related to the player, such as gender, age, body mass, joint range of motion and flexibility. F-MARC has investigated a wide range of intrinsic risk factors, including age, gender, nutrition, tackling mechanisms and previous injury.

Extrinsic risk factors include all other issues that are not directly related to the player, such as the Laws of the

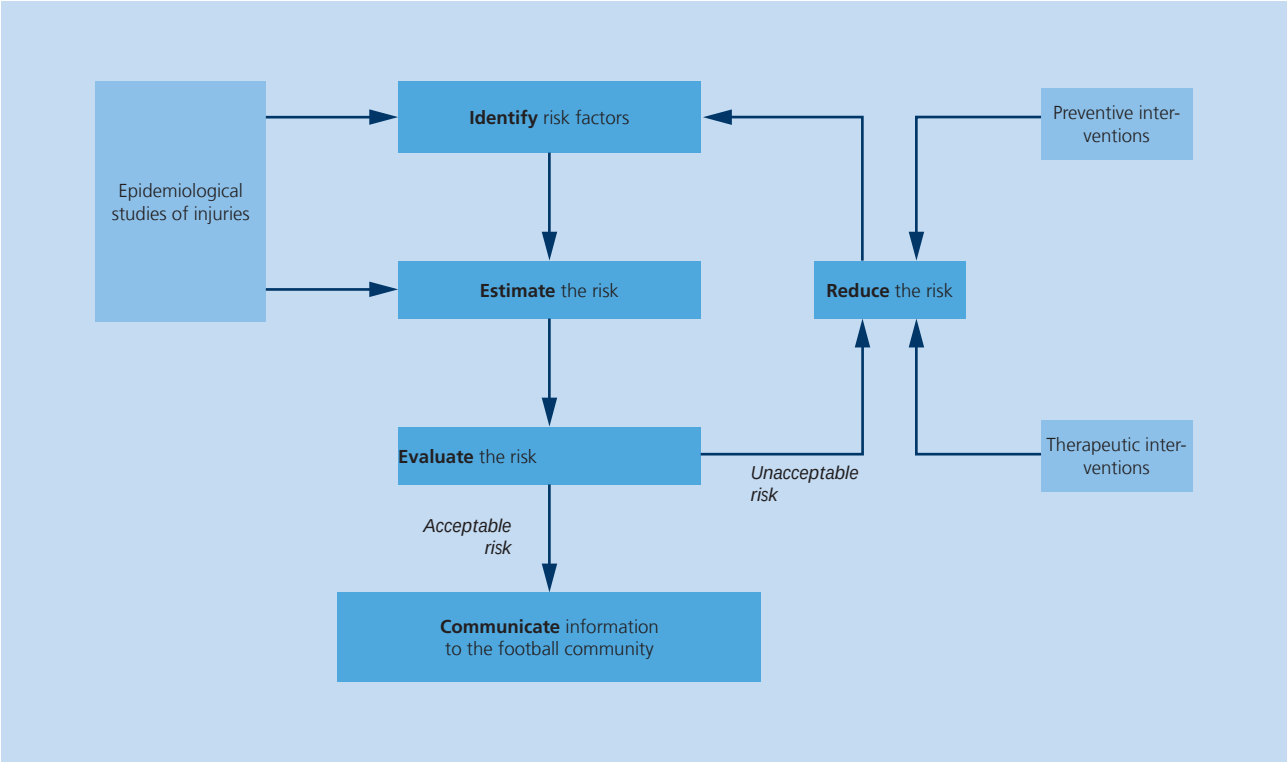


Figure 1.1.1 Framework for managing the risk of injury in football

Game, the effectiveness of protective equipment, the design of football boots, being tackled and the condition of playing surfaces. F-MARC has investigated the impact on injury of many extrinsic risk factors, including the use of artificial turf, foul play, playing at altitude, and referees' performance in penalising dangerous tackles according to the Laws of the Game.

Understanding which, how and why risk factors can have a significant impact on the incidence and nature of injury amongst different groups of players is the first stage in developing effective injury prevention programmes.

Risk estimation

Epidemiological studies of football injuries are employed to define baseline levels of risk amongst players, to identify risk factors and to investigate the effectiveness of injury prevention strategies on the incidence and nature of injury. Identifying significant risk factors and understanding how their effects can be minimised is an important priority for FIFA. Consequently, F-MARC has undertaken and published the results of many epidemiological studies, including studies carried out at all FIFA tournaments since 1998: these studies provide data that enable long-term injury trends in football to be identified. In order to improve the quality and consistency of epidemiological data in football, F-MARC gathered nine experienced researchers from seven countries together at FIFA headquarters in 2006 in order to reach an agreement on injury definitions and procedures for studies of injuries in football. By producing a consensus agreement on the criteria that should be used for measuring the risk of injury, it should now be possible to provide consistent and comparable data about the level, nature and significance of injuries in football, wherever the epidemiological studies are undertaken.

Risk evaluation

Just monitoring the level of injury risk in football is not sufficient – it is also necessary to evaluate whether the levels of risk measured in the sport are acceptable to players and to the sport's governing bodies. There will always be an element of injury risk associated with any sport and this must be balanced against the beneficial health effects provided by sport and exercise. Although some sports present a higher risk and some a lower risk of injury, football undoubtedly provides an invaluable source of entertainment and exercise for enormous numbers of people in countries all around the world. By implementing a consistent injury surveillance methodology, it is possible to measure risk levels in each of these countries so that all stakeholders can reach evidence-based decisions about the costs and benefits of playing football in any environment.

Risk reduction

If the level of injury risk in football is deemed to be too high, it is necessary to examine means of eliminating or at least reducing the risks. Because the risk of injury to players is a function of the incidence and the consequence of the injuries sustained, it is important to address both of these aspects by considering the introduction of preventive and therapeutic interventions. Interventions can be generalised in two ways: on a time basis, in terms of whether they occur pre-event (e.g. preparation of playing surface), in-event (e.g. boot design) or post-event (e.g. ice treatment of players' injuries); and by the type of intervention, in terms of whether they involve physical (e.g. use of a neck brace to manage neck injuries), management (e.g. team training programmes) or human (e.g. player behaviour) control measures.

Preventive interventions – these approaches are primarily aimed at reducing the incidence of injury, although they may also have a beneficial effect on the severity of injuries. Preventive interventions include changing the Laws of the Game (an in-event management control measure), changing players' behaviour to one of fair play (an in-event human control measure), and ensuring that playing surfaces have been inspected and cleared of dangerous matter prior to the start of play (a pre-event physical control measure). F-MARC researchers have investigated a number of preventive interventions, including the F-MARC 11 injury prevention programme and pre-participation assessment programmes for players at FIFA tournaments.

Therapeutic interventions – these approaches are aimed at reducing the consequences of players' injuries. Many sports physicians and surgeons working within football have developed new techniques to improve the

assessment, treatment and repair of injuries, whilst other specialists have developed innovative injury rehabilitation programmes that reduce the time away from play and/or the number of re-injuries sustained by players.

Risk communication

One important objective for F-MARC is communication of research results to the football community. In this respect, F-MARC has to date published over 120 research papers openly in peer-reviewed sports medicine journals. The results of F-MARC research studies are also regularly presented at national and international sports medicine conferences around the world. In addition, F-MARC research findings are regularly reported on the FIFA web pages. This free exchange of information enables all stakeholders in football to engage in informed debates about injury prevention, treatment and rehabilitation, which benefits the health of all players. FIFA's open approach to research and the discussion of injury risks in football has led FIFA to make representations to the International Football Association Board to amend some Laws of the Game in order to further safeguard players' health and safety and to obtain acceptance of artificial turf as a playing surface for all levels of the game. Furthermore, the F-MARC risk management approach to injury prevention has led, for example, to the development of training programmes for players and the preparation of health-related information and guidance, which are freely available to every player and coach via the FIFA website.

1.2 The team physician in football

1.2.1 The roles of the team physician in football

In football teams, either national teams or club teams, there is a uniform organisational structure within which the coaching staff, administrative staff, medical team and players function. The team physician (TP) is the head of the medical team. Medical teams in different leagues form a continuum, with the simplest form consisting of a resident physiotherapist working with a consulting doctor acting as a TP. In more advanced forms, a medical team consists of a resident TP with physiotherapists, trainers, masseurs, a nutritionist and a podiatrist. In the absence of an elaborate medical team, the TP would have to perform numerous tasks, e.g. advise players on nutrition including meal selection and assist in some cases with physiotherapy and ankle strapping, especially when on tour.

Regardless of the composition of the medical team, the TP is ultimately responsible for the short- and long-term health condition of the players and staff. Injuries and illnesses that occur beyond the expertise of the doctor should be referred to a suitable specialist and a referral network should be established both at home and when the team is travelling.

The TP advises the coach about the health status of the players and makes the final decision with respect to the players' eligibility for competitive football. The TP must respect players' confidentiality, especially when addressing the media.

The TP as described in this chapter is in keeping with a simple medical team comprising a doctor, physiotherapist and possibly a trainer. The roles of a TP mainly refer to eight broad activity fields:

- Injury management
- Illness management
- Prevention of injury and illness
- Emergency care
- Acclimatisation issues: jet lag, heat and humidity, altitude
- Anti-doping issues including prevention of doping infringements
- Optimising performance: nutrition, hydration, screening for overtraining of players
- Counselling players and staff

Injury management

One of the major roles of a TP is to determine that a player is injured, accurately diagnose the complaint and institute treatment. Most injuries in football are mild and involve the soft tissues. Diagnosis may rely on the use of clinical acumen in simple injuries but sometimes complex imagining is required for more complicated ones. The TP heads the rehabilitation team and this team plans the treatment for the injured player. The TP is responsible for notifying the coaching staff of the injury and the expected time that the player will be out of team training. He is also responsible for declaring that the injured player has recovered and is sufficiently well to resume full training after undergoing rehabilitation.

Illness management

Footballers and other athletes suffer illnesses that occur commonly in the general population such as asthma, upper respiratory tract infections, skin rashes and diarrhoea. The TP is responsible for diagnosing and treating such conditions. The TP should travel with medications that would be used to treat the common medical conditions seen in general practice. However, it is of particular importance that he knows about the applicable restrictions on the use of prohibited substances by players subject to doping control. He should be aware of the different types of common conditions that could be encountered when travelling to foreign countries, e.g. malaria and travellers' diarrhoea, and be prepared to treat these conditions. The TP again notifies the coaching staff about the illness of the player and

the time the player is expected to be out of full training or competition.

Staff members who become ill while on tour are also evaluated and treated. Staff members are usually older than the players and so may suffer from chronic diseases like hypertension and diabetes and this must be catered for, especially when travelling. It is recommended that both athletes and staff declare to the TP what chronic illnesses they suffer from in advance so that the TP can be well prepared to handle any conditions that may arise.

Prevention of injury and illness

Part of the role of the TP is to carry out or assist in carrying out pre-participation physical examinations for musculoskeletal conditions that could predispose a player to injury, as well as underlying cardiovascular conditions that could predispose a player to sudden cardiac death. Such risk factors, when identified, may need correction where possible. In permanent cardiac conditions, e.g. structural anomalies of the heart of players, a cardiologist may need to be included in the management of the case (see 2.1.3). It may mean disqualification from participating in sport if the defect cannot be corrected and if the player is at high risk of sudden cardiac death.

The implementation of evidence-based injury prevention programmes, e.g. "The 11+", during the warm-up would require the coach's and trainer's permission. Anterior Cruciate Ligament (ACL) prevention programmes and strapping of previously injured ankles should be promoted where practical.

When travelling to foreign countries, endemic diseases should be identified and recommended immunisation or prophylaxis against disease instituted prior to departing the home country. This could be a challenge for a national team whose players may play in different countries. Communication with the various team physicians, advising of the need for such immunisation or prophylaxis, could be very helpful in assisting selected players to receive the same. Precautions against travellers' diarrhoea and sexually transmitted diseases should be recommended and implemented for both players and staff.

Provision of emergency care

The TP should be able to provide emergency care for the team members. Although severe injuries and cardiac arrest are uncommon in football, when incidents do occur, they usually assume national and even international importance. The TP is responsible for the initial evaluation, stabilisation, treatment and evacuation of such injured or ill players. Basic emergency equipment and drugs should always be at the touch line (see below). It is recommended that the TP, together with other members of the medical team, have regular mock exercises simulating dealing with unconscious players or cardiac arrest and have certification in short courses involving advanced cardiac and trauma care.

Anti-doping issues

The TP is the person most likely to be contacted by players to determine whether a substance is banned or not. The TP therefore needs to be knowledgeable of the updated WADA list of banned substances and methods and be able to advise players on doping issues. The use of medications and supplements prescribed to and used by players should be monitored regularly to keep players from doping violations. The TP has an important role to play in preventing doping violations. There should be regular reminders to players about checking medications prior to use and the possibility of nutritional supplements being contaminated with pro-hormones of anabolic steroids. The TP should be confident in the procedure of applying for therapeutic use exemptions (TUE) for the use of medications on the Prohibited List that players may need for justifiable medical use to treat medical conditions. Having the TP present at doping control is reassuring for the players and this should be encouraged where practical.

Acclimatisation to foreign conditions

The TP has an important role to play in advising the team on acclimatisation to foreign conditions, e.g. heat and humidity and jet lag when travelling. The TP should research the temperature, humidity, altitude and time zone and common communicable diseases prior to travel. The TP should also advise the coaching staff of the possible time needed to adapt to the new conditions and possible strategies needed to acclimatise to the conditions based on current medical evidence or practice.

Performance optimisation: nutrition, hydration, screening for overtraining

Players should be advised about the importance of adequate hydration, proper nutrition including

carbohydrate ingestion, and rest. The timing and composition of meals should be established on a match-by-match basis. On long tours, players may overtrain and this could affect their performance. Screening for overtraining with profile of mood state (POMS) questionnaires in tired athletes could identify athletes at risk of overtraining. Athletes complaining of persistent tiredness should have an enforced period of rest or reduced training for a short period of time. Those with persistent tiredness should have a history, physical examination and blood investigations (metabolic screen, viral titres) to determine the cause of the fatigue. If no cause is found and this condition is reversed with a short period of rest, it is called functional overreaching (FO). If this state takes longer than expected to resolve, it is referred to as non-functional overreaching (NFO). Only if this state is severe and persists for prolonged periods is it referred to as overtraining syndrome (OTS).

Screening of tired athletes with decreased performance for overtraining can be done with serial POMS questionnaires. This could identify athletes at risk of overtraining and is easy to administer by the TP. This questionnaire looks at five scales: depression, anger, fatigue, tension and vigour. Normal athletes show higher vigour scores and lower fatigue, anger and depression ratings than athletes with overreaching or overtraining. Players with a high chance of NFO or OTS should then be subjected to the testing of hormonal responses to two maximal exercise tests. The Recovery-Stress Questionnaire for athletes (RESTQ-sport) can also be used to screen for overtraining and this has been used more commonly in recent studies looking at overtraining.

Counselling players and staff

Players and staff may have psycho-social issues such as a fear of being dropped from the squad or relationship problems that could affect their performance on the field. The TP has a role to play in counselling players and staff on such issues. The TP should be receptive to players with social issues. This requires a doctor-player relationship that encourages mutual trust and respect. Players can be very apprehensive towards medical staff, but with repeated consultations, such fear is reduced. This occurs quickly, especially if the player realises that there is confidentiality in such consultations and that the player's best interest is sought at all times.

1.2.2 Contents of medical bags in football

The contents of a medical bag can be divided into: a small emergency bag (e.g. the small FIFA Fair Play pouch, see Figure 1.2.2.1), which is usually taken onto the field of play when a physician is called on the pitch by the referee to evaluate an injured player, and a larger main bag (e.g. the FIFA Fair Play medical bag), which is usually kept at the sideline. Both of these bags should be waterproof. In addition, depending on the duration of the tour and the country being toured, additional items not readily available could be taken, e.g. splints and crutches. The bag should be replenished locally as depending on the location, supplies abroad could prove disappointing. The TP must be familiar with the contents of the bag and should have packed it to ensure knowledge of where items are located, especially if items are needed rapidly. The TP should also have TUE forms available with a fax number to send them to when completed. Some countries still do not have a reliable internet service so depending on downloading these in such situations can be insufficient. The TP must be familiar with the latest FIFA Anti-Doping Regulations and should also have a copy of same.

The TP depends on the organisers of a competition to provide equipment like oxygen, stretchers and spine boards, suctions, intubation equipment and a defibrillator to be used in an emergency, and these should be readily available at the touch line during a match.

The contents of the emergency bag include emergency drugs and equipment:

- Adrenaline 1:1000 solution injection
- Atropine injection 1mg
- Chlorpheniramine 10mg injection
- Hydrocortisone injection
- Local anaesthetic (1% lidocaine with and without adrenaline)
- Pocket face mask with one-way valve (for mouth-to-mouth resuscitation)
- Oral airway
- Rigid cervical collar (see Figure 1.2.2.1)
- Coolant spray
- Quick Clottm – granules to cease bleeding immediately
- Tape – cohesive and non-cohesive

– Gloves
– Triangular sling
– Cotton, gauze, eye pads
– Adhesive sutures
– Scissors
– Alcohol swabs

The contents of the **main bag** include:
– Eye pack: eye pads, fluorescein eye strips, anaesthetic eye drops, eye wash, penlight with blue filter (to identify corneal abrasions)
– Basic medical equipment (thermometer, stethoscope, otoscope/ opthalmoscope)
– Sutures and suture set, plasters
– Universal splints (see Figure 1.2.2.1)
– Adhesive felt/foam
– Ice bags
– Condoms
– Intravenous fluids, cannulas and giving sets
– Crutches

Drugs: (This may be more easily remembered by using categories of drugs according to the various body systems)

Analgesics/Anti-inflammatories/Antipyretics

1. Non-steroidal anti-inflammatory drugs, e.g. diclofenac (oral and injectable), ibuprofen
2. Tramadol injection
3. Paracetamol and paracetamol/codeine preparations
4. Muscle relaxants
5. Injectable depot glucocorticoids, e.g. triamcinolone
6. COX-2 Inhibitors

Figure 1.2.2.1 FIFA Fair play pouch, rigid cervical collar

Gastrointestinal

7. Antiemetics (oral, injectable)
8. Antidiarrhoeal preparations, e.g. loperamide
9. Stool softener
10. Oral rehydration salts
11. Antacids, omeprazole and ranitidine
12. Chewable vitamin C

Respiratory

13. Inhaled beta-2 agonist/inhaled corticosteroid, e.g. salbutamol inhaler, beclamethasone inhaler
14. Cough expectorants

Wound and skin-cleansing solutions

15. Hydrogen peroxide, SavlonTM or Dettol™ solution, isopropyl alcohol solution

Ears, nose and throat

16. Wax softener
17. Eye and ear antibiotics
18. Antihistamine eye drops
19. Antihistamines, e.g. fexofenadine, loratidine
20. Nasal sprays: decongestant, e.g. xylometazoline and steroidal, e.g. fluticasone
21. Throat lozenges

Antibiotics

22. Ciprofloxacin, penicillin (amoxillicin/clavulate), macrolide (claritromycin), cephalosporin

Topical preparations

23. Anti-fungal and steroid creams
24. Mosquito repellent
25. Heat rub

Sedatives

26. Midazolam tablets

Optional

Automatic External Defibrillator (AED)

2. Prevention

2.1 General medical assessment

Before a player starts participating in any sport, he should be medically examined to ensure that he is physically fit to cope with the demands of training and playing. Therefore, medical assessments aimed at risk factor detection are generally advocated by physicians and sports organisations. While in principle a standardised, validated medical assessment that meets quality criteria and provides sensitive screening of potential participants in sport is highly desirable, it might be more adequate to tailor the medical assessment to specific sports and populations.

Playing football is associated with a certain risk of injury for the participating players. Several studies have analysed the risk factors for football injuries. The most important risk factor for a future injury is a previous injury in the same body part. Other risk factors are inadequate rehabilitation and musculoskeletal complaints. Some studies indicate that mechanical instability, general joint laxity and functional instability are also risk factors for injury. Therefore, players should be asked about musculoskeletal injuries and current complaints, and an orthopaedic examination should be performed by an experienced physician.

In addition, the very small number of individuals with unknown or undiagnosed heart conditions such as congenital cardiomyopathy face a risk that exercise might trigger the manifestation of their disease and might, in the worst case, cause sudden cardiac death. This small part of the population should be identified in order not to expose a player to any disproportionate risk.

2.1.1 General history

Before the medical history, a player should be briefly asked for his position, his dominant leg and the number of matches he has played in the last 12 months. These initial questions help to indicate the physical and mental demands on a player as exerted by his individual responsibilities within the team, the level of play, and his training and match schedules. The general medical history covers the personal and family history of a player, and includes standard questions on general health (e.g. allergies, medication). The general medical assessment should include questions aimed at detecting risk factors manifested through any suspicious symptoms in the past and present, particularly with respect to exercise. It has been suggested that a proper medical history alone may identify up to 75% of the problems that affect athletes. While medical history is divided into three sections (general, orthopaedic, cardiovascular) in this manual, it might be helpful to combine the general history (see 2.1.1.1) with the more specific history, depending on the focus of the physical examination (see 2.1.2.1, 2.1.3.1, 2.1.3.2).

Apart from the specific orthopaedic and cardiovascular examination, a general medical examination (including height, weight, palpation of the thyroid gland, lymph nodes/spleen and abdomen, percussion of the lung) is always indicated. The analysis of basic blood parameters is also recommended (see form 2.1.1.2).

2.1.2 Orthopaedic examination

History

Knowledge of the medical history of the musculoskeletal system may help to identify risk factors for injury. The player should be asked about previous injuries, especially those leading to a time-loss of more than four weeks, and about any operations on the musculoskeletal system. It should be determined whether or not the player suffers from any current complaints, aches or pain; whether there are known diagnoses; and what treatment is applied for these. A short standardised orthopaedic history for football players is presented on ‘Documentation form for the orthopaedic history’ (Figure 2.1.2.1).

Competition history

Position on the field ☐ goalkeeper ☐ defender
☐ midfielder ☐ striker

Dominant leg ☐ left ☐ right ☐ both

Number of matches in the last 12 months _____

Medical history – present and past complaints

General	*no*	*yes, **within** the last 4 weeks*	*yes, **prior to** the last 4 weeks*
Flu-like symptoms	☐	☐	☐
Infections (esp. viral)	☐	☐	☐
Rheumatic fever	☐	☐	☐
Heat illness	☐	☐	☐
Concussion	☐	☐	☐
Allergies to food, insects	☐	☐	☐
Allergies to drugs	☐	☐	☐

Routine medication within last 12 months

	no	*yes*
Non-steroidal anti-inflammatory drugs	☐	☐
Asthma medication	☐	☐
Antihypertensive drugs	☐	☐
Lipid lowering drugs	☐	☐
Anti-diabetic drugs	☐	☐
Psychotropic drugs	☐	☐
Other _____	☐	☐

Form 2.1.1.1 Documentation form for the general history

General physical examination

Height _____ cm/_____ inches	Weight _____kg/_____ lbs	
Thyroid gland	□ normal	□ abnormal
Lymph nodes/spleen	□ normal	□ abnormal
Lungs		
Percussion	□ normal	□ abnormal
Breath sounds	□ normal	□ abnormal
Abdomen		
Palpation	□ normal	□ abnormal

Blood results (fasting)

Haemoglobin	_____ mg/dL
Haematocrit	_____ %
Erythrocytes	_____ mg/dL
Thrombocytes	_____ mg/dL
Leukocytes	_____ mg/dL
Sodium	_____ mmol/L
Potassium	_____ mmol/L
Creatinine	_____ µmol/l
Cholesterol (total)	_____ mmol/L
LDL cholesterol	_____ mmol/L
HDL cholesterol	_____ mmol/L
Triglycerides	_____ mmol/l
Glucose	_____ mmol/l
C-reactive protein	_____ mg/l

Form 2.1.1.2 Documentation form for the general physical examination and blood analysis

Severe injury leading to more than four weeks of limited participation or absence from play/training:

			Last occurrence
☐ no	☐ yes,	☐ groin strain	when?_____ (year)
		☐ strain of m. quadriceps femoris	when?_____ (year)
		☐ strain of hamstring muscles	when?_____ (year)
		☐ ligament injury of the knee	when?_____ (year)
		☐ strain of calf	when?_____ (year)
		☐ ligament injury of the ankle	when?_____ (year)
		☐ others, please specify_____	when?_____ (year)

For others, please provide diagnosis _____

Operations of the musculoskeletal system

☐ no	☐ yes,	☐ hip joint	when?_____ (year)
		☐ groin (due to pubalgia)	when?_____ (year)
		☐ knee ligaments	when?_____ (year)
		☐ knee meniscus or cartilage	when?_____ (year)
		☐ Achilles tendon	when?_____ (year)
		☐ ankle joint	when?_____ (year)
		☐ other operations	when?_____ (year)

For others, please provide diagnosis _____

Current complaints, aches or pain

☐ no ☐ yes, please specify body parts

		right	*left*	
☐ head / face	☐ shoulder	☐	☐	hip
☐ cervical spine	☐ upper arm	☐	☐	groin
☐ thoracic spine	☐ elbow	☐	☐	thigh
☐ lumbar spine	☐ forearm	☐	☐	knee
☐ sternum / ribs	☐ wrist	☐	☐	lower leg
☐ abdomen	☐ hand	☐	☐	Achilles tendon
☐ pelvis / sacrum	☐ fingers	☐	☐	ankle
		☐	☐	foot, toe

Current diagnosis and treatment

☐ no	☐ pubalgia	☐ rest	☐ physiotherapy	☐ surgery
	☐ hamstring strain	☐ rest	☐ physiotherapy	☐ surgery
	☐ quadriceps strain	☐ rest	☐ physiotherapy	☐ surgery
	☐ knee sprain	☐ rest	☐ physiotherapy	☐ surgery
	☐ meniscus lesion	☐ rest	☐ physiotherapy	☐ surgery
	☐ calf strain	☐ rest	☐ physiotherapy	☐ surgery
	☐ tendinosis of Achilles tendon	☐ rest	☐ physiotherapy	☐ surgery
	☐ ankle sprain	☐ rest	☐ physiotherapy	☐ surgery
	☐ concussion	☐ rest	☐ physiotherapy	☐ surgery
	☐ low back pain	☐ rest	☐ physiotherapy	☐ surgery
	☐ others, specify_____	☐ rest	☐ physiotherapy	☐ surgery

Form 2.1.2.1 Documentation form for the orthopaedic history

Examination of the spine

Spinal form

The player stands with the back towards the physician. The physician identifies the spinal process of the seventh cervical vertebra and palpates the spinal process of the thoracic and lumbar spine with the index finger. Visual and palpated deviations from normal are identified as related to the curvature of the spine (thoracic kyphosis, lumbar hyperlordosis, flat back in the region of thoracic and/or lumbar spine scoliosis).

C7 palpation

Thoracic palpation

Lumbar palpation

Example of flat thoracolumbar spine

Example of flat thoracolumbar spine

Example of lumbar hyperlordosis

Pelvic level

The player stands in an upright position with his feet parallel and as close together as possible. From behind, the physician places both hands on the pelvic crest. If there is any difference between the levels of the two sides of the pelvic crest, boards are placed under one foot so that the levels are equalised and the difference can be determined.

Examination of pelvic level

Example of leg length difference

Sacroiliac joint

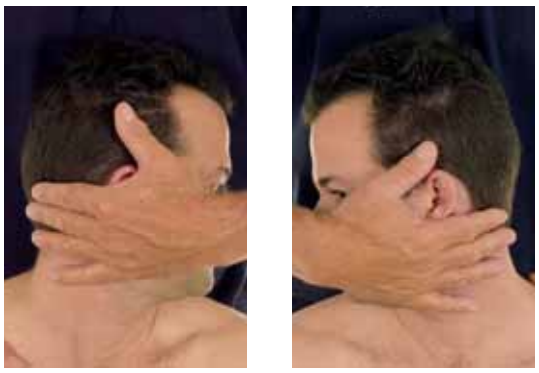
The player lies in a supine position on an examination table with both legs extended. The physician palpates the inferior borders of both medial malleoli, and assesses the symmetry of their position. The player is then asked to sit up, keeping his legs extended on an examination table. During the movement, the physician keeps his thumbs on the bony landmarks and assesses again the symmetry of the malleoli. A change in the position of the malleoli towards each other (from supine to long sit) is an indication of a sacroiliac joint dysfunction (hypo-mobility or blockage).

Sacroiliac joint testing

Cervical spine range of motion

The player sits in an upright position. The physician stands behind the player. The physician fixes the left shoulder with one hand and with the other hand he rotates the cervical spine to the right; exploring the full range of motion. The range of motion is normal when 90° is measured. If rotation is below 60° then rotation out of flexion is to be performed or further assessment of the cervical spine is needed. The examination is repeated for the left side accordingly.

Cervical spine range of motion



Cervical spine range of motion

Example of cervical spine hypermotility
(rotation more than 90°)

Spinal flexion (stand and reach test)

The player stands with his feet as close together as possible and his knees extended. He bends forward as far as possible, keeping his legs straight. He has to remain in this bent position for approximately five seconds, before the physician measures the distance between fingertip and floor in centimetres.

Good flexibility of lumbar spine and hip muscles

Hypomobility (normally 0-10 cm distance)

Examination of the upper extremity

The following four global active tests represent a quick functional screening to detect potential asymmetries and limitations in the movements of the upper extremity, and to note any symptoms which may require further investigations.

Active elevation

The player sits in an upright position on the edge of the examination table. He is asked to fully elevate both arms above his head with extended elbows and the hands in a thumbs-up position to the maximum position. A normal range of motion is about 180°.

Active elevation of the shoulders and arms

Active external rotation

The player sits in an upright position on the edge of the examination table. He is asked to flex his elbows and to grasp both hands behind his neck. He should then move back both elbows ("opening position") as far as he can, followed by the opposite movement ("closing movement").

Active external rotation

Active internal rotation

The player sits in an upright position on the edge of the examination table and is asked to flex his elbows and to bring both hands behind his body. The back of the hands should point to the spine, and the player should attempt to position both thumbs as high as he can.

Active internal rotation

Active "compression"

The player sits in an upright position on the edge of the examination table and is asked to flex his right elbow and to bring his right hand to his left shoulder. He should then repeat the movement with his left arm. In this position, the shoulder joint and its capsular structures are compressed and stressed.

Active compression

Findings may be documented on the "Documentation form for the spine and upper extremity" (Figure 2.1.2.2)

Spine

Spine form	☐ normal	☐ flat ☐ hyperkyphosis ☐ hyperlordosis ☐ scoliosis
Pelvic level	☐ even	_____ cm lower ☐ right ☐ left
Spinal flexion		
Distance fingertips to floor		_____ cm
Sacroiliac joint	☐ normal	☐ abnormal
Cervical rotation		
right	☐ normal	☐ 80° - 60° ☐ < 60° painful ☐ no ☐ yes
left	☐ normal	☐ 80° - 60° ☐ < 60° painful ☐ no ☐ yes

Upper extremity

Active elevation		
right	☐ normal	☐ abnormal, please specify: _____
left	☐ normal	☐ abnormal, please specify: _____
Active external rotation		
right	☐ normal	☐ abnormal, please specify: _____
left	☐ normal	☐ abnormal, please specify: _____
Active internal rotation		
right	☐ normal	☐ abnormal, please specify: _____
left	☐ normal	☐ abnormal, please specify: _____
Active "compression"		
right	☐ normal	☐ abnormal, please specify: _____
left	☐ normal	☐ abnormal, please specify: _____

Form 2.1.2.2 Documentation form for the examination of the spine and upper extremity

Examination of the hip, groin and thigh

Hip flexion

The player lies in a supine position on the examination table. The player is then asked to flex and elevate his left knee towards his chin. First the active flexion and then the passive flexion are measured. Hip flexion measurements usually range between 90° (active) and 130-140° (passive). The examination is then repeated for the right leg.

Active hip flexion

Passive hip flexion

Hip extension

The player lies in a prone position on the examination table. He is then asked to elevate his right leg with a straight knee. The range of motion of the hip extension is recorded. The active range should be between 10 and 40°; passive motion should add approximately 10°. The examination is then repeated for the left leg.

Active hip extension

Passive hip extension

Inward rotation

The player lies in a supine position on the examination table with his right hip joint and knee joint bent at 90°, so that the thigh lies in a neutral position (i.e. no ab- or adduction components). The physician stabilises the right knee with his left hand. With his right hand, the physician then rotates the foot outwards in an arc (axis of rotation is the thigh), placing the knee in a valgus position to achieve internal rotation of the hip. The angle between the median line of the body and the axis of the lower leg is measured using a goniometer. The normal range is between 30 and 45°. The examination is then repeated for the left leg.

Internal rotation

Outward rotation

The player lies in a supine position on the examination table with his right hip joint and knee joint bent at 90° so that the thigh lies in a neutral position (i.e. no ab- or adduction components). The physician stabilises the knee with his left hand. With his right hand, the physician then rotates the foot inwards in an arc (axis of rotation is the thigh), placing the knee in a varus position to achieve external rotation of the hip. The angle between the median line of the body and the axis of the lower leg is measured using a goniometer. The normal range is approximately 40 to 50°. The examination is then repeated for the left leg.

External rotation

Abduction

The player lies in a supine position on the examination table with his knees and hips extended. The physician stabilises the iliac crest using his left hand. The player abducts his left leg and the angle between the median line of the body and the axis of the lower leg is measured using a goniometer. Measurement normally ranges between 30 and 45°. The examination is repeated for the right leg.

Abduction

Tenderness on groin palpation

The player lies in a supine position on the examination table with both legs extended (optional with a pillow under the knees). The physician palpates bilaterally (starting from the pubic symphysis) along the bony landmarks, the surrounding soft tissues in the inguinal area, and records any symptoms or pain elicited by this palpation. Any tenderness in this region indicates a groin problem that may require further examinations.

Hernia

Examination of the inguinal region is either performed with the player laying or standing and the physician seated on a stool facing him. Observation of the groin area with the patient relaxed and then actively coughing may reveal a bulge or an abnormal motion. The physician should then stand to the side of the player with his fingers lightly applied to the groin, his left hand on the patient's left side and his right hand on the patient's right side. With his fingers placed over the femoral region, the external inguinal ring, and the internal ring, the physician should have the player cough. A palpable bulge or impulse located in any one of these areas may indicate a hernia. In the male, with the physician having returned to the sitting position, the scrotum on each side is inverted with the examining index finger entering the inguinal canal. The size of the external ring can be ascertained by palpating just lateral to the pubic tubercle.

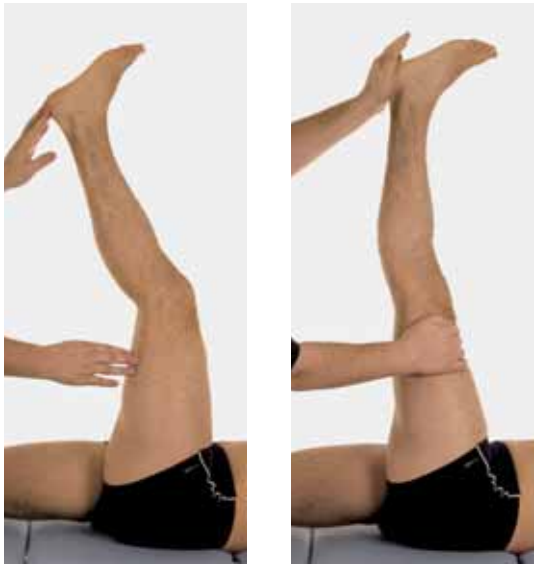
Adductor muscles

The player lies in a supine position on the examination table and is asked to place the soles of his feet against each another and bring his feet towards the buttocks. This position causes a maximal flexion and external rotation of the hip. If the player cannot achieve at least 45°, this indicates shortened or tight adductor muscles. In addition, pain elicited by this movement is recorded.

Adduction

Hamstring muscles

The player lies in a supine position on the examination table. The physician bends the left leg with flexed knee to 90° of hip flexion. Slowly, the knee is passively extended from 90° to 0°. If the player cannot achieve 90° hip flexion with an extended knee, the hamstrings on the left side are considered shortened or tight. In addition, pain elicited by this movement is recorded. The examination is then repeated for the right leg.



Hamstring muscles

Iliopsoas muscle

The player lies in a supine position on the examination table with his left leg bent over the end of the table in a resting position. The right hip is flexed to at least 90°. If the left hip flexes spontaneously, this indicates shortened or tight iliopsoas muscles on the left side. In addition, pain elicited by this movement is recorded. The examination is then repeated for the right leg.

Iliopsoas muscle

Rectus femoris muscle

The player lies in a supine position on the examination table with the right leg bent over the end of the table in a resting position. The right hip is flexed to at least 90°. The left knee is now flexed from 90 to 120°. If 120° cannot be achieved, this indicates a shortened or tight rectus femoris muscle on the left side. In addition, pain elicited by this movement is recorded. The examination is then repeated for the right leg.

Rectus femoris muscle

Tensor fascia latae muscle (iliotibial band)

The player is lying on his left side on the examination table. The lower leg is slightly flexed in order to maintain stability. The player is asked to first hold the right leg in a horizontal position (hip extension and neutral abduction), and then to drop it towards the edge of the table until the point the leg stops to move. If the right knee does not reach the edge of the table, this indicates a shortened or tight tensor fascia latae muscle (iliotibial band) on the right side. In addition, pain elicited by this movement is recorded. The examination is then repeated for the left leg.

Tensor fascia latae muscle test

Findings may be documented on the "Documentation form for the hip, groin and thigh" (Figure 2.1.2.3)

Flexibility of the hip

Flexion (passive)					
right	□ normal	□ limited _____°	painful	□ no	□ yes
left	□ normal	□ limited _____°	painful	□ no	□ yes
Extension (passive)					
right	□ normal	□ limited _____°	painful	□ no	□ yes
left	□ normal	□ limited _____°	painful	□ no	□ yes
Inward rotation (in 90° flexion)					
right		_____°	painful	□ no	□ yes
left		_____°	painful	□ no	□ yes
Outward rotation (in 90° flexion)					
right		_____°	painful	□ no	□ yes
left		_____°	painful	□ no	□ yes
Abduction					
right		_____°	painful	□ no	□ yes
left		_____°	painful	□ no	□ yes
Tenderness on groin palpation					
right	□ no	□ pubis □ inguinal canal			
left	□ no	□ pubis □ inguinal canal			
Hernia					
right	□ no	□ yes, please specify _____			
left	□ no	□ yes, please specify _____			

Muscles

Adductor muscles					
right	□ normal	□ shortened	painful	□ no	□ yes
left	□ normal	□ shortened	painful	□ no	□ yes
Hamstring muscles					
right	□ normal	□ shortened	painful	□ no	□ yes
left	□ normal	□ shortened	painful	□ no	□ yes
Iliopsoas muscles					
right	□ normal	□ shortened	painful	□ no	□ yes
left	□ normal	□ shortened	painful	□ no	□ yes
Rectus femoris muscle					
right	□ normal	□ shortened	painful	□ no	□ yes
left	□ normal	□ shortened	painful	□ no	□ yes
Tensor fascia latae muscle (iliotibial band)					
right	□ normal	□ shortened	painful	□ no	□ yes
left	□ normal	□ shortened	painful	□ no	□ yes

Form 2.1.2.3 Documentation form for the examination of the hip, groin and thigh

Examination of the knee

Knee joint axis

The player stands with his feet as close together as possible. The knees are fully extended. The axis of the knee joint and the lower leg is visually assessed for each leg. If there is no contact between the epicondyli of the femur, this indicates a genu varum. When contact between the epicondyli of the femur can only be accomplished with a distance between the malleoli, this indicates a genu valgum.

Genu varum

Genu valgum

Flexion

The player lies in a supine position on the examination table and is asked to move his right heel to his buttocks (active flexion). Then the physician slightly lifts the heel of the player from the examination table and further flexes the knee (passive flexion). The examination is then repeated for the left knee.

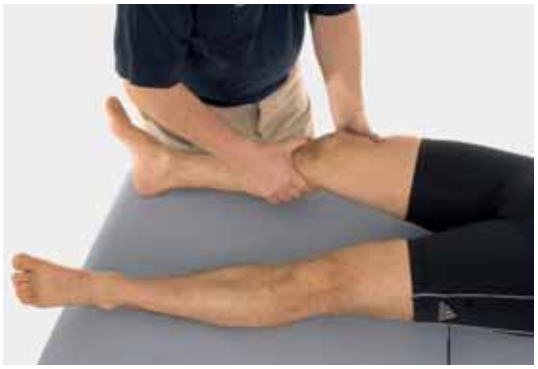
Extension

The player lies in supine position on the examination table with extended knees. The player is asked to extend his right knee further with the thigh on an examination table (active extension). Then the physician slightly lifts the heel of the player from the table and further extends the knee (passive extension). The examination is then repeated for the left knee.

Lachman test

The player lies in a supine position on the examination table with his right knee in 20-30° flexion. The physician fixes the distal femur of the right knee from the lateral side with one hand and proximal tibia from the medial side with the other hand. The player is asked to relax, especially his leg muscles. The physician makes a swift drawer movement with the upper tibia from its resting position in the ventral direction. The examination is then repeated for the left knee.

A difference in the drawer movement between the two legs of 5mm or more is pathological. The test should be considered normal when there is no difference between the right and left side.



Lachman test

Anterior drawer sign

The player lies in a supine position on the examination table with the right knee joint bent at 90° and the tibia in neutral rotation. The physician sits on the front of the player's foot with both hands around the upper tibia. The player is asked to relax, especially his leg muscles. The physician then carries out a ventral drawer movement. The examination is then repeated for the left knee.

More than 5mm movement or a difference in the anterior drawer movement as compared to the other leg is a pathological result. Anterior laxity with a stiff end point that is equal for the right and left knee is considered normal. An anterior drawer with the tibia in external rotation is a sign of instability of the medial collateral ligament and joint capsule. An anterior drawer with the tibia in internal rotation is a sign of an anterior cruciate ligament injury.

Anterior drawer test

Posterior drawer sign

The player lies in a supine position on the examination table with the right knee joint bent at 90° and the tibia in neutral rotation. The physician sits on the player's forefoot with both hands around the upper tibia. The player is asked to relax, especially his leg muscles. The knee should be checked for spontaneous posterior drawer. The physician then pushes with both hands on the upper tibia to perform the posterior drawer. The examination is then repeated for the left knee.

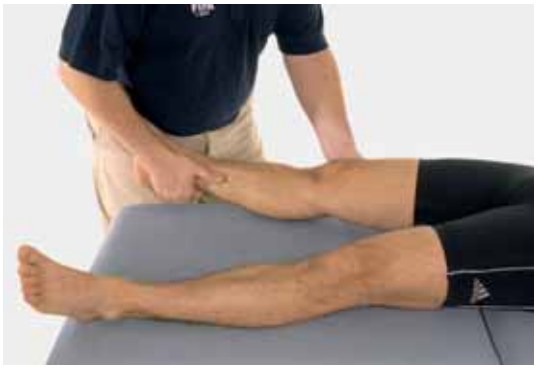
More than 5mm movement or a difference in the posterior drawer movement as compared to the other knee is a pathological result.

Posterior drawer test

Valgus stress in extension

The player lies in a supine position on the examination table with both knee joints fully extended. The physician puts one hand on the right lateral femoral condyle above the joint line and the other hand medial around the right ankle. He then applies a medially directed stress to the knee joint. The examination is then repeated for the left knee.

More than 5mm movement is a pathological result. An increased valgus in extension is a sign of a medial collateral ligament injury and concomitant injury to the postero-medial capsule which might also include an anterior cruciate ligament injury.



Valgus stress in extension

Varus stress in extension

The player lies in supine position on the examination table with both knee joints fully stretched and the thigh muscles completely relaxed. The physician places one hand on the right medial femoral condyle above the joint line and the other hand lateral around the right ankle. Then the physician applies a laterally directed stress to the knee. The examination is repeated for the left knee accordingly.

More than 5mm movement is a pathological result. An increased varus in extension is a sign of lateral collateral ligament injury and concomitant injury to the posterolateral capsule which might also include an anterior cruciate ligament injury.

Varus stress in extension

Valgus stress in flexion

The player lies in a supine position on the examination table with the right knee in 30° flexion and with the thigh muscles completely relaxed. The physician puts one hand on the lateral femoral condyle above the joint line and the other hand medial around the right ankle. He then applies a medially directed stress to the knee joint. The examination is then repeated for the left knee.

More than 5mm movement is a pathological result and a sign of medial collateral ligament injury.

Valgus stress in flexion

Varus stress in flexion

The player lies in a supine position on the examination table with the right knee in 30° flexion and with the thigh muscles completely relaxed. The physician places one hand on the right medial femoral condyle above the joint line and the other hand lateral around the right ankle. Then the physician applies a laterally directed stress to the knee. The examination is then repeated for the left knee.

More than 5mm movement as compared to the other side is a pathological result. Note, however, that there is always some laxity in this test. Side-to-side comparison is essential. An increased varus is a sign of a lateral collateral ligament injury.

Varus stress in flexion

Meniscus test

The player lies in a supine position on the examination table with the right knee in 90° flexion and with the thigh muscles completely relaxed. The physician monitors the joint line with a pincer grip (thumb and index finger palpating the medial and lateral joint space), and progressively flexes the knee, performing internal and external tibia rotation. These passive rotation motions are performed with as much flexion as possible (towards end-range flexion, the menisci – especially their posterior horns - are compressed and stressed with additional rotations). If pain or symptoms are elicited by these movements, this indicates a possible meniscus problem.

Meniscus test in knee flexion with tibia in internal rotation

Meniscus test in knee flexion with tibia in external rotation

Findings may be documented on the "Documentation form for the examination of the knee" (Figure 2.1.2.4)

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Knee joint axis

right	□ normal	□ genu varum	□ genu valgum
left	□ normal	□ genu varum	□ genu valgum

Flexion (passive)

right	□ normal	□ limited _____ °	painful	□ no	□ yes
left	□ normal	□ limited _____ °	painful	□ no	□ yes

Extension (passive)

right	□ 0°	□ limited _____ °	painful	□ no	□ yes
	□ hyper-extension	_____ °			
left	□ 0°	□ limited _____ °	painful	□ no	□ yes
	□ hyper-extension	_____ °			

Lachman test

right	□ normal	□ +	□ ++	□ +++
left	□ normal	□ +	□ ++	□ +++

Anterior drawer sign (knee joint in 90° flexion)

right	□ normal	□ +	□ ++	□ +++
left	□ normal	□ +	□ ++	□ +++

Posterior drawer sign (knee joint in 90° flexion)

right	□ normal	□ +	□ ++	□ +++
left	□ normal	□ +	□ ++	□ +++

Valgus stress, in extension

right	□ normal	□ +	□ ++	□ +++
left	□ normal	□ +	□ ++	□ +++

Valgus stress, in 30° flexion

right	□ normal	□ +	□ ++	□ +++
left	□ normal	□ +	□ ++	□ +++

Varus stress, in extension

right	□ normal	□ +	□ ++	□ +++
left	□ normal	□ +	□ ++	□ +++

Varus stress, in 30° flexion

right	□ normal	□ +	□ ++	□ +++
left	□ normal	□ +	□ ++	□ +++

Meniscus test

right	□ normal	□ abnormal	painful	□ no	□ yes
left	□ normal	□ abnormal	painful	□ no	□ yes

Figure 2.1.2.4 Documentation form for the examination of the knee

Examination of the lower leg, ankle and foot

Tenderness of Achilles tendon

The player lies in a prone position on the examination table with his feet overlapping the table. The physician carefully palpates the right Achilles tendon between the heel (calcaneus; tendinous insertion) and the lower border of the calf muscle (musculotendinous insertion). The palpation can elicit pain or symptoms of the Achilles tendon structure: tenderness, thickening and crepitus ("crackling" feeling) may be found. These are pathological findings which require further examination.

Triceps surae muscle

The player stands in a step position facing a wall. He is asked to press the heel of the posterior leg against the floor (the foot should be perpendicularly positioned to the wall). He places his hands on the wall in front of him. He then moves his trunk slowly forward – with his back straight – until he feels the calf muscle of the posterior leg stretched to a point that hinders movement (the heel must remain in contact with the floor, the knee extended). If a dorsal extension in the ankle joint of about 30° cannot be achieved, this indicates a shortened or tight triceps surae muscle on the right side. The examination is then repeated for the other leg.

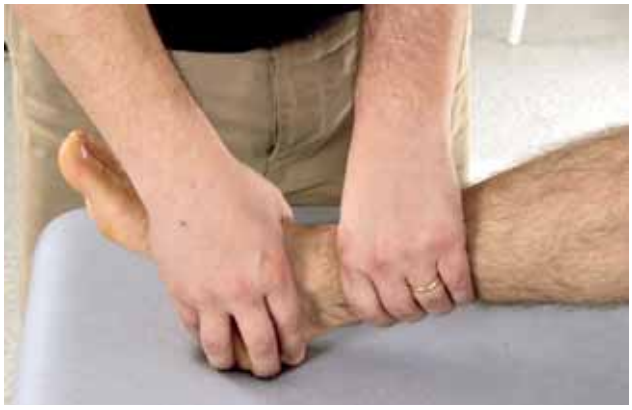
Triceps surae muscle test

Anterior drawer sign of the ankle

The player lies in a supine position on the examination table with the ankle in 10° plantar flexion (relaxed position). The physician lifts the right leg from the table, flexes the knee to 30°, holding the malleolus fork with one hand, and carries out an anterior drawer movement with the other hand around the talus and calcaneus. The examination is then repeated for the left ankle.

Any difference observed in the anterior drawer movement in comparison to the other ankle or greater than normal movement (3-5mm) on both sides is a pathological result.

Anterior drawer test normal finding



Anterior drawer test pathological finding

Dorsal flexion of the ankle

The player lies in a supine position on the examination table with both feet overlapping the table. The upper ankle of his right foot is in a neutral position and the knee joint is extended. The navicular tuberosity is palpated. The pivotal point of the goniometer lies on the medial side of the talus. The physician fixes the leg and moves the ankle joint dorsally and measures the angle between the axis of the tibia and the tuberosity of the navicular bone. The examination is then repeated for the left ankle.

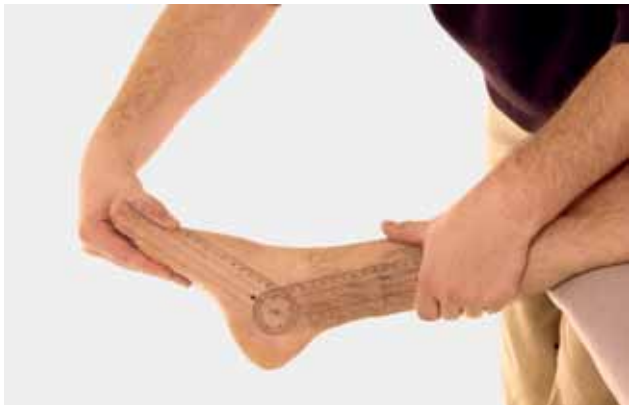
Less than 15° dorsal extension indicates either a shortened triceps surae muscle or pathological changes of the ankle joint.

Dorsal flexion

Plantar flexion of the ankle

The player lies in a supine position on the examination table with both feet overlapping the table. The upper ankle of his right foot is in a neutral position and the knee extended. The tuberosity of the navicular bone is palpated. The pivotal point of the goniometer lies on the medial side of the talus. The physician fixes the leg and moves the ankle joint passively plantar, measuring the angle between the axis of the tibia and the tuberosity of the navicular bone. The examination is then repeated for the left ankle.

Less than 20° plantar flexion indicates either a muscular or articular problem.



Plantar flexion

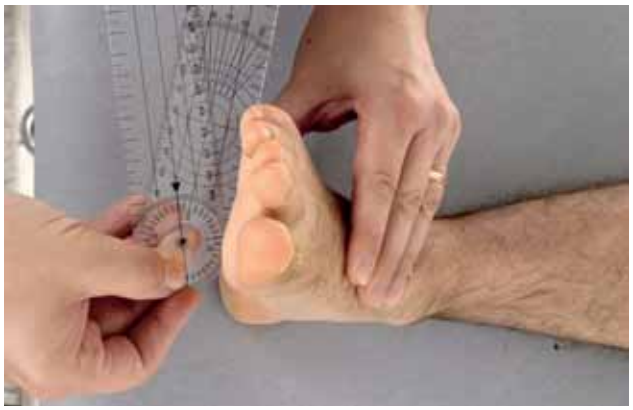
Total supination in the foot joints

The player lies in a supine position on the examination table with both knees extended and both feet overlapping the table. The upper ankle of his right foot is in a neutral position. The physician carries out a passive supination movement of the front of the right foot. The angle between the sole of the foot (metatarsal heads) and the perpendicular to the body axis is measured using a goniometer. The examination is then repeated for the left ankle. More significant restrictions of joint mobility require individual assessment.

Total supination in the foot joints

Total pronation in the foot joints

The player lies in a supine position on the examination table with extended knees and the upper ankle of his right foot in a neutral position. The physician carries out a passive pronation movement of the front of the right foot. The angle between the sole of the foot (metatarsal heads) and the perpendicular angle to the body axis is measured using a goniometer. The examination is then repeated for the left ankle. More significant restrictions of joint mobility require individual assessment.



Total pronation in the foot joints

Metatarsophalangeal joint I

The player lies in a supine position on the examination table with relaxed, extended legs. The physician fixes the right ankle/foot with one hand and then moves the metatarsophalangeal joint I ("big toe joint"). The angle between the axis of the metatarsal bone and the axis of phalangeal bone is measured using a goniometer. The extension ranges between 70-90°, and the flexion should be around 45°. The examination is repeated for the left metatarsophalangeal joint I accordingly.

Metatarsophalangeal joint test

Findings may be documented on the "Documentation form for the examination of the lower leg, ankle and foot" (Figure 2.1.2.5)

Tenderness of Achilles tendon

right	□ no	□ yes
left	□ no	□ yes

Triceps surae muscle

right	□ normal	□ abnormal	painful	□ no	□ yes
left	□ normal	□ abnormal	painful	□ no	□ yes

Anterior drawer sign

right	□ normal	□ +	□ ++	□ +++
left	□ normal	□ +	□ ++	□ +++

Dorsi flexion

right	_____°	painful	□ no	□ yes
left	_____°	painful	□ no	□ yes

Plantar flexion

right	_____°	painful	□ no	□ yes
left	_____°	painful	□ no	□ yes

Total supination

right	□ normal	□ decreased	□ increased
left	□ normal	□ decreased	□ increased

Total pronation

right	□ normal	□ decreased	□ increased
left	□ normal	□ decreased	□ increased

Metatarsophalangeal joint I

right	□ normal	□ limited flexion	□ limited extension
left	□ normal	□ limited flexion	□ limited extension

Form 2.1.2.5 Documentation form for the examination of the lower leg, ankle and foot

2.1.3. Cardiac examination

In the following sub-chapters, the principal diagnostic tools of cardiac assessment, together with the specific requirements and pitfalls of their performance, are discussed. Any cardiac assessment in football should be performed by a sufficiently trained, experienced and skilled physician from the same cultural background as the player to reliably identify the clinical symptoms and signs associated with critical cardiovascular disease in sport.

Personal and family history

History taking is a cornerstone in the assessment of young competitive players. The personal and family history has its strengths especially in diseases that escape from other primary diagnostics (e.g. the arrhythmogenic right ventricular cardiomyopathy (ARVC) or coronary anomalies).

According to the Lausanne Recommendations, issued under the umbrella of the Medical Commission of the International Olympic Committee (IOC), personal history has to address exertional chest pain or discomfort, syncope or near-syncope, irregular heartbeat or palpitations, and presence of shortness of breath or fatigue out of proportion to the degree of exertion.

As the most common causes of sudden cardiac death (SCD) in sports are inherited diseases following an autosomal dominant pattern, family history is of great importance in identifying players at risk. Family history is considered positive when a close relative has suffered from premature heart attack or sudden death (<55 years of age in males and <65 years in females), or when cardiomyopathy, Marfan syndrome, long QT syndrome, Brugada syndrome, coronary artery disease or any other disabling cardiovascular diseases run in the family.

Present and past complaints		within the last 4 weeks		prior to last 4 weeks	
Heart and lung	**no**	**at rest**	**during/after exercise**	**at rest**	**during/after exercise**
Chest pain or tightness	☐	☐	☐	☐	☐
Shortness of breath	☐	☐	☐	☐	☐
Asthma	☐	☐	☐	☐	☐
Cough	☐	☐	☐	☐	☐
Bronchitis	☐	☐	☐	☐	☐
Palpitations	☐	☐	☐	☐	☐
Arrhythmias	☐	☐	☐	☐	☐
Other heart problems	☐	☐	☐	☐	☐
Dizziness	☐	☐	☐	☐	☐
Syncope	☐	☐	☐	☐	☐
	no	yes, within the **last 4 weeks**		yes, **prior** to the last 4 weeks	
Hypertension	☐	☐	☐	☐	☐
Heart murmur	☐	☐	☐	☐	☐
Abnormal lipid profile	☐	☐	☐	☐	☐
Seizures, epilepsy	☐	☐	☐	☐	☐
Advised to give up sport	☐	☐	☐	☐	☐
More quickly tired than team-mates	☐	☐	☐	☐	☐
Diarrhoea illness	☐	☐	☐	☐	☐

Figure 2.1.3.1 Documentation form for personal cardiovascular history

Physical examination

A focused clinical examination is another universally accepted cornerstone of medical screening in sport. Physical examination should start with a detailed inspection of the player. Obvious clinical findings like cyanosis, clubbing of the fingers, facies mitralis or peripheral signs of an endocarditis are rare in young players whereas affections of the skin (rash), joints (rheumatologic diseases) or general habitus can hint at systemic disease.

Focus should particularly be on stigmata of the *Marfan syndrome* as one of the most common inherited connective tissue disorders (incidence of 1 in 3,000 to 5,000 individuals). Affected individuals are prone to aortic dissection. Although phenotypic expression is variable, some features involving the skeletal, cardiovascular, and ocular systems are considered major and minor manifestations of the disease *("Ghent criteria")*. Some of these criteria, which are more easily detectable on physical examination, are listed in Table 2.1.3.1.

Diagnosis of Marfan syndrome is established by genetic analysis or a positive family history. In the absence thereof, one major criterion of two systems and the involvement of a third system with either one major or minor manifestation are needed to confirm the diagnosis of the syndrome.

Evaluation of general vital signs

Body temperature: Anamnesis or first clinical impression may raise suspicion of fever, which is easily confirmed on examination. Players should withdraw from competitive football and even intensive training when febrile as myocarditis may be deleterious.

Heart rate and pulse status: Heart rate should be assessed in a supine position after at least five minutes of rest. Peripheral pulses should be checked bilaterally with two fingers for rate and rhythm. Assessing the heart rate over one whole minute is more accurate than palpating the pulse only for a few seconds, and detection of arrhythmias is more likely. Detection of diminished and/or delayed pulses may hint at artery stenoses, valvular heart disease or aortic isthmus stenosis.

A clinical assessment of jugular venous pressure and pulse provides information on the heart's preload and the volume status. Examination of the internal jugular veins should be performed with the head of the player in the midline position at 30° to 45°. Venous pressure is obtained

Family history (male relatives <55 years, female relatives <65 years)

	no	father	mother	sibling	other
Sudden cardiac death	□	□	□	□	□
Sudden infant death	□	□	□	□	□
Coronary heart disease	□	□	□	□	□
Cardiomyopathy	□	□	□	□	□
Hypertension	□	□	□	□	□
Recurrent syncope	□	□	□	□	□
Arrhythmias	□	□	□	□	□
Heart transplantation	□	□	□	□	□
Heart surgery	□	□	□	□	□
Pacemaker/Defibrillator	□	□	□	□	□
Marfan syndrome	□	□	□	□	□
Unexplained drowning	□	□	□	□	□
Unexplained car accident	□	□	□	□	□
Stroke	□	□	□	□	□
Diabetes	□	□	□	□	□
Cancer	□	□	□	□	□
Others (arthritis etc.)	□	□	□	□	□

Figure 2.1.3.2 Documentation form for family history with regard to cardiovascular disease

by noting the height of pulsation. Normally, the filling level of the veins should be a maximum of about three to four centimetres above the sternal angle. In the horizontal position, the venous pulsation is usually visible in the neck. If the neck veins collapse in the horizontal position, subnormal right atrial pressure is to be suspected. Additional information can be gained by assessing hepato-jugular reflux: press firmly over the abdominal right upper quadrant (liver) or the centre of the abdomen for 10-60 seconds. A positive result is defined as a sustained rise in the JVP of at least four centimetres or more (or a fall of four centimetres or more after the examiner releases pressure).

Peripheral oedemas, palpated over the ventral tibia, ankle and the arch of the foot, should be evaluated for consistency and distribution.

The *heart examination* should start with a classification of heart rhythm as regular, irregular (due to sinus arrhythmia or extrasystoles) or absolutely irregular (due to atrial fibrillation).

To assess *blood pressure* (BP), the player should ideally be examined in a sitting position. Systolic pressure tends to be 2-3 mmHg higher and the diastolic pressure a similar degree lower in the supine position. The player should rest for at least five minutes before BP is measured with the arm supported at the level of the heart. When the arm hangs down, systolic pressures elevated by 10 to 12mmHg will result due to the added hydrostatic pressure. To establish the diagnosis of hypertension, multiple readings should be taken at various times. Mercury sphygmomanometers provide more accurate measurements than aneroid sphygmomanometers. Oscillometric blood pressure measuring devices give readings that are typically lower than BP values obtained with the auscultatory method.

Use of a proper-sized cuff is essential: if the cuff is too small, the pressure generated by inflating the cuff manifests higher than intra-arterial pressure. As a rule of thumb, the length of the cuff bladder should be 80%, and the width at least 40% of the circumference of the upper arm. The cuff should be inflated to a pressure approximately 30mmHg greater than estimated systolic pressure (by pulse palpitation) to avoid the "auscultatory gap". The cuff should be deflated slowly at a rate of 2 to 3mmHg per heartbeat. The systolic pressure is equal to the pressure at which the pulse is first heard by auscultation ("Korotkoff phase I") and the brachial pulse can first be palpated again. As the cuff is further deflated, the pulse is first abruptly muffled (phase IV) before it disappears (phase V). The diastolic pressure is generally equal to phase V, except in high cardiac output (children, athletes, during pregnancy or in hyperthyreotic state).

BP should be measured on both arms. Measurements with the player standing should always complete the assessment to detect postural hypotension.

	Major criteria	Minor criteria
Skeletal	Reduced upper to lower segment ratio Arm span exceeding height (ratio >1.05) Arachnodactyly of fingers and toes with positive wrist sign (thumb and fifth digit overlap circling the wrist) and thumb sign (distal phalanx protrudes beyond border of clenched fist) Pectus carinatum or excavatum Scoliosis >20° or spondylolisthesis	Pectus excavatum of moderate severity Joint hypermobility High arched palate Facial features: dolichocephaly, malar hypoplasia, enophthalmos, retrognathia, down-slanting palpebral fissures
Ocular	Ectopia lentis	Myopia, retinal detachment
Other		Striae distensae

Table 2.1.3.1 Marfan criteria

Auscultation

Although strongly dependent on the expertise of the examiner, auscultation has a reported sensitivity of 70% and a specificity of 98% for detection of valvular heart disease. Ideally, cardiac auscultation should firstly evaluate heart sounds and secondly evaluate heart murmurs.

Heart sounds

First heart sound (S1): The first heart sound (S1), normally best heard over the cardiac apex, consists of two high-frequency components: the first component of S1 is attributed to the dominant mitral valve closure (M1) and the second to closure of the tricuspid valve (T1). The intensity of valve closure sound is increased with an increased transvalvular gradient (mitral valve obstruction as in mitral stenosis or atrial myxoma), increased transvalvular flow (left-to-right shunt in patent ductus arteriosus, ventricular septal defect, and high output state), shortened diastole (tachycardia) or short PR intervals (preexcitation syndrome). Restricted valve mobility decreases the intensity of S1, as it happens in case of fibrosis or calcification of the mitral valve. S1 may also be reduced when the valve leaflets are already half-closed at the onset of systole, as it occurs with left ventricular dysfunction.

Abnormal splitting of S1 can result from delayed closure of the tricuspid valve (e.g. in patients with atrial septal defect). A widely split S1 can also occur in complete right bundle branch block (or during left ventricle (LV) pacing or ectopic beats of LV origin).

Second sound (S2): The second heart sound is made up of two components: aortic (A2) and pulmonary (P2) valve closure. P2 is best heard over the upper left sternal border, whereas A2 is widely transmitted to the right second interspace, along the sternal border and to the cardiac apex.

While S2 is usually single during expiration, separation of A2 and P2 occurs during inspiration, which allows comparison of the relative intensities of the two components and is best heard over the left second interspace. Increased intensity of S2 occurs in systemic hypertension, coarctation of the aorta or ascending aortic aneurysm (due to louder A2) or in pulmonary hypertension (due to louder P2). Decreased intensity of S2 occurs in very low arterial diastolic pressure (e.g. in significant aortic regurgitation), a relatively immobile aortic valve due to calcification (e.g. in patients with calcified aortic stenosis), and relatively low arterial pressure in significant aortic stenosis.

Increased separation of A2 and P2 during inspiration may result from complete right bundle branch block (or premature beats or idioventricular rhythm of LV origin), the Wolff-Parkinson-White syndrome with LV preexcitation and haemodynamic causes like pulmonary arterial hypertension of any aetiology. Fixed splitting of S2 is most commonly due to interatrial communication (large atrial septal defect, common atrium) and left-to-right or bidirectional shunt, or to any condition with severe right ventricle (RV) failure. Paradoxic splitting occurs when A2 follows P2 during expiration, and is mostly seen with left bundle branch block (premature beats of RV origin or preexcitation of the RV in Wolff-Parkinson-White syndrome, respectively), in aortic valve stenosis and in some patients with hypertension.

Third (S3) and fourth (S4) heart sounds: S3 occurs as passive filling begins after relaxation of the ventricle and is a normal finding in healthy young adults. Over the age of 40 it suggests an enlarged LV with increased volume load. In contrast, S4, which coincides with atrial systole and suggests increased LV pressure, is a normal finding in healthy elderly patients with prolonged PR interval and an unusual finding in children and adults. S3 and S4 are low-frequency diastolic sounds and are therefore best heard over the cardiac apex or over the lower left sternal border. Aortic ejection sounds, which can occur in association with a deformed but mobile aortic valve and with aortic root dilatation are found in patients with bicuspid aortic valve, aortic valve stenosis, aortic regurgitation, aneurysm of the ascending aorta and in some patients with systemic hypertension.

Prolapse of the mitral valve is the most common cause of a midsystolic click. In mitral-valve prolapse, it is important to exclude possible concomitant cardiovascular anomalies such as Marfan syndrome, atrial septal defect, hypertrophic cardiomyopathy, musculoskeletal abnormalities and systemic lupus erythematosus. A pericardial rub is generated by the friction of the two inflamed layers of the pericardium. The rub can be heard during atrial systole, ventricular systole, and the rapid-filling phase of the ventricle (three-component rub).

Heart murmurs

The character of a murmur should be described by its different features: intensity, frequency, shape, timing, location (and radiation).

Intensity of a murmur should be classified by six grades:

– Grade I (1/6) is the faintest murmur that can be heard (with difficulty)

- Grade II (2/6) murmur is also a faint murmur but can be identified immediately
- Grade III (3/6) murmur is moderately loud
- Grade IV (4/6) murmur is loud (possibly associated with a palpable thrill)
- Grade V (5/6) murmur is very loud (cannot be heard without the stethoscope)
- Grade VI (6/6) murmur is the loudest (can be heard without a stethoscope)
- The time course of murmur intensity corresponds to the "shape" of a diagram of murmur intensity over time, as in a phonocardiogram. A number of configurations or shapes of murmurs are recognised:
- Crescendo (increasing)
- Decrescendo (diminishing)
- Crescendo-decrescendo (increasing-decreasing or diamond or spindle shaped)
- Plateau (unchanged in intensity)

A *systolic murmur* generally starts with or after S1 and terminates before or at S2 and is therefore recognised by identifying S1 and S2. A *diastolic murmur* starts with or after S2 and ends at or before S1. A *continuous murmur* begins in systole and continues to diastole without interruption, encompassing the S2.

Mid-systolic murmurs are most commonly benign flow murmurs due to physiologic flow, increased flow rate across a normal semilunar valve or due to aortic valve sclerosis. A systolic murmur is present in up to 60% of patients but in 90% of these it is associated with a normal echocardiogram. Benign "flow" murmurs also occur when flow across the semilunar valve is significantly increased, as occurs in anaemia, pregnancy, and thyreotoxicosis. The "benignity" of a mid-systolic murmur should be established based on the absence of other abnormal findings. Examples of mid-systolic murmurs are Still's murmur (mainly in children), valvular aortic stenosis (AS), a bicuspid aortic valve (before leaflet thickening and calcification), subvalvular LV outflow obstruction (usually due to hypertrophic cardiomyopathy HCM). To distinguish between fixed valvular aortic stenosis (AS) and dynamic LV outflow obstruction (obstructive HCM) some clinical tests are established. With the patient starting in a squatting position then assuming a standing position, the intensity of the murmur in HCM increases but it decreases the murmur of valvular aortic stenosis. In the straining phase of a Valsalva manoeuvre the murmur of HCM increases in intensity. Both the intensity of the murmur and the carotid pulse volume decline with Valsalva in AS.

It can be difficult to distinguish between a long mid-systolic murmur and a holosystolic regurgitant murmur in certain situations. Handgrip manoeuvre may help to distinguish between a mitral regurgitation murmur, where the intensity of the murmur increases (increased afterload effect), and an AS murmur, where intensity usually decreases.

Early systolic murmurs most often result from mitral regurgitation (MR) and generally have a decrescendo configuration.

Late systolic murmurs are most commonly caused by mitral valve prolapse. They are best heard with the diaphragm of the stethoscope and are usually preceded by single or multiple clicks (For diagnostic manoeuvres see also "heart sounds" section).

There are three classical causes of **holosystolic murmurs** – MR, tricuspid regurgitation and ventricular septal defect (VSD) – and they usually occur when blood flows from a chamber whose pressure throughout systole is higher than pressure in the chamber receiving the flow.

The holosystolic murmur of MR is high pitched and is therefore best heard with the diaphragm of the stethoscope and the patient in the left lateral decubitus position.

Early diastolic murmurs are most often due to aortic or pulmonary regurgitation.

Mid-diastolic murmurs result from turbulent flow across the atrioventricular valves during the rapid filling phase because of mitral ("opening snap") or tricuspid valve stenosis and an abnormal pattern of flow across these valves. In mitral valve stenosis (MS) it can be stated that the longer the duration of the murmur, the more severe the MS might be.

Late diastolic or presystolic murmurs usually have a crescendo configuration and result from increased flow across the mitral or tricuspid valve (atrial fibrillation, mitral stenosis, tricuspid stenosis, left-to-right shunts), complete heart block and left or right atrial myxomas.

Continuous murmurs do not necessarily need to occupy the total duration of systole and diastole. Continuous murmurs result from blood flow from a higher pressure chamber or vessel to a lower system associated with a persistent pressure gradient between these areas during systole and diastole (patent ductus arteriosus, as a relatively common cause of a continuous murmur in

adults, an aortopulmonary window, congenital or acquired arteriovenous fistulas).

12-lead resting ECG

Performing 12-lead resting ECG

After at least five minutes of rest, the ECG should be performed supine with the 12 leads in the standard positions with the peripheral electrodes positioned on the upper and lower limbs (Einthoven I, II, III and Goldberger aVR, aVL, aVF).

Analysing 12-lead resting ECG

The following systematic approach to assessing pathological findings is recommended:

1. Rhythm:

– is there a rhythm other than sinus rhythm?
– if sinus rhythm, pathologic P wave configuration indicates:
  - *left atrial enlargement when* negative portion of the P wave in lead V1 ≥ 0.1mV in depth and ≥ 0.04s in duration
  - *right atrial enlargement* with peaked P wave in leads II and III or V1 ≥ 0.25mV in amplitude.

V1: Fourth intercostal space at the right sternal border.
V2: Fourth intercostal space at the left sternal border.
V3: Mid-way between V2 and V4.
V4: Fifth intercostal space in the mid-clavicular line.
V5: Left anterior axillary line at the level of V4.
V6: Left mid-axillary line at the level of V4.

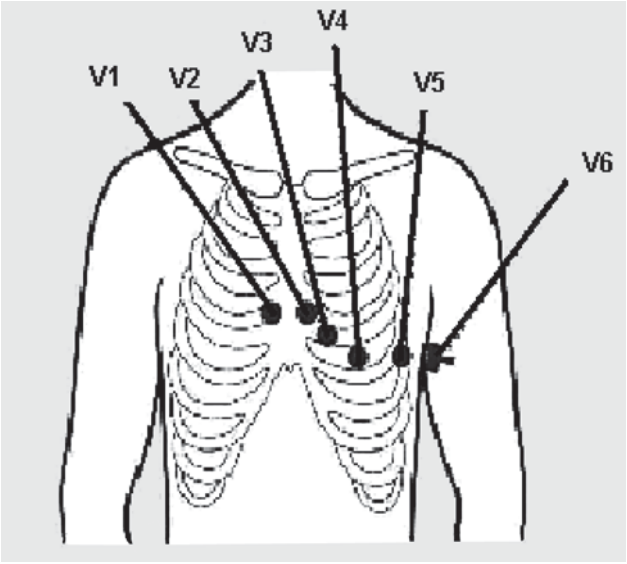


Figure 2.1.3.3 Precordial lead positions in 12-lead resting ECG

2. QRS complex

– axis: frontal plane axis deviation: right ≥ +120° or left –30° to –90°
– increased voltage: amplitude of R or S wave in a standard lead ≥ 2mV, S wave in lead V1 or V2 ≥ 3mV, or R wave in lead V5 or V6 ≥ 3mV
– abnormal Q waves ≥ 0.04s in duration or ≥ 25% of the height of the ensuing R wave or QS pattern in two or more leads
– right or left bundle branch block with QRS duration ≥ 0.12s
– R or R' wave in lead V1 ≥ 0.5mV in amplitude and R/S ratio ≥ 1

3. Repolarisation, ST-segment, T-wave, QT interval

– ST-segment depression or T-wave flattening or inversion in two or more leads
– prolongation of heart rate corrected QT interval >0.44s in males and >0.46s in females (or shortening of the corrected QT interval <330ms)

4. Rhythm and conduction abnormalities

– premature ventricular beats or more severe ventricular arrhythmias
– supraventricular tachycardias, atrial flutter, or atrial fibrillation
– short PR interval (<0.12s) with or without "delta" wave
– sinus bradycardia with resting heart rate ≤40 beats/min (only if increasing less than 100/min in stress test)
– first degree (PR≥0.21s) AV block (only if not shortening through hyperventilation or exercise test)
– second or third degree AV block

Disease	P wave	PR interval	QRS complex	QTc interval	ST interval	T-wave	Arrhythmias
HCM	(left atrial enlargement)	normal	increased voltage in mid-left precordial leads; abnormal Q waves in inferior and/or lateral leads; (LAD, LBBB); (delta wave)	normal	down-sloping (up-sloping)	inverted in mid-left precordial leads; (giant and negative in the apical variant)	(atrial fibrillation); (PVB); (VT)
ARVC	normal	normal	Prolonged (>110ms) in right precordial leads; epsilon wave in right precordial leads; reduced voltages (≤0.5mV) in frontal leads; (RBBB)	normal	(up-sloping in right precordial leads)	inverted in right precordial leads	PVB with LBBB pattern; (VT with LBBB pattern)
Dilated cardiomyopathy	(left atrial enlargement)	(prolonged ≥0.21s)	LBBB	normal	down-sloping (up-sloping)	inverted in inferior and/or lateral leads	PVB; (VT)
Long QT syndrome	normal	normal	normal	prolonged (♂ >440ms, ♀ >460ms)	normal	bifid or biphasic in all leads	(PVB); (torsade de pointes)
Brugada syndrome	normal	prolonged ≥ 0.21s	S1S2S3 pattern; (RBBB/LAD)	normal	Type I: Up-sloping and coved-type in right precordial leads, Type II/III: Saddle-back-type	Type I: Inverted in right precordial leads, Type II: biphasic or positive, Type III: positive	(polymorphic VT); (atrial fibrillation); (sinus bradycardia)
Lenègre disease	normal	prolonged ≥ 0.21s	RBBB; RBBB/LAD; LBBB	normal	normal	secondary changes	(2nd or 3rd degree AV block)
Short QT syndrome	normal	normal	normal	shortened <300ms	normal	normal	atrial fibrillation; (polymorphic VT)
Pre excitation syndrome (WPW)	normal	shortened <0.12s	Delta wave	normal	secondary changes	secondary changes	supraventricular tachycardia; (atrial fibrillation)
Coronary artery diseases (CAD or CCA)	normal	normal	(abnormal Q waves)	(prolonged)	(down- or up-sloping)	inverted in ≥2 leads	PVB; (VT)

Table 2.1.3.2 Common ECG findings in heart disease

Less common ECG findings at presentation are reported in brackets. QTc: QT interval corrected for heart rate by Bazett's formula. LBBB: left bundle branch block; RBBB: right bundle branch block. LAD: left axis deviation of -30° or more. PVB: either single or coupled premature ventricular beats. VT: either non-sustained or sustained ventricular tachycardia. CAD: coronary artery disease. CCA: congenital coronary anomalies. (Adapted from: Corrado et al., Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol (Consensus Statement), European Heart Journal 2005; 26: 516–524)

Echocardiography

Two-dimensional trans-thoracic echocardiography is the principal diagnostic tool for clinical recognition of many cardiac pathologies. While being a cornerstone in the diagnosis and follow-up of HCM, it can also detect other relevant abnormalities responsible for SCD in young athletes, such as valvular heart disease, aortic root dilatation, and left ventricular dysfunction (with myocarditis and dilated cardiomyopathy). Arrhythmogenic right ventricular cardiomyopathy (ARVC) is often difficult to diagnose by echocardiography alone, but electrocardiography may add some helpful information. Although echocardiography may raise strong suspicion of congenital coronary anomalies (mainly the left main coronary artery from the right sinus of Valsalva) in selected young athletes, CT-angiography, magnetic resonance imaging (MRI) or even coronary arteriography are usually necessary to diagnose this entity.

Echocardiography in trained athletes carries the potential for false-positive results, which may arise from borderline values for left ventricular wall thicknesses and cavity size mainly in the differentiation of physiological adaptations from HCM. In the vast majority of competitive athletes, absolute left ventricular wall thickness is normal or only mildly increased (≤12mm). However, in some athletes, the increase in left ventricular wall thickness may be more substantial (up to 16mm), which makes HCM more likely.

The echocardiographic hallmark of HCM is a hypertrophic and non-dilated left ventricle in the absence of other cardiac or systemic disease that could cause the hypertrophy. Although, in general, patients with HCM show a marked increase in left ventricular wall thickness (average wall thickness reported in most echocardiographic studies is ≈20mm, ranging to >50mm) an important minority of patients with HCM, especially young, adolescent patients < 18 years of age, show relatively mild left ventricular hypertrophy with a wall thickness in the range of ≈12 to 15mm. In this diagnostic "grey area" only careful analysis of several echocardiographic characteristics in addition to clinical and electrocardiographic features permits the diagnosis.

Left ventricular hypertrophy appears to be rather homogenous in athletes (even if there can be a predominantly thickened anterior septum) with differences in wall thickness of only 1-2mm between segments. By contrast, in patients with HCM the pattern of wall-thickening is heterogenous and asymmetric with the anterior portion of the septum (or other particular segments) being the region of maximal hypertrophy. Moreover, in HCM contiguous segments of the left ventricle show strikingly different wall thicknesses with abrupt transition between such areas.

Considering left ventricular cavity size improves specificity of the echocardiographic diagnosis of HCM: more than one third of highly trained elite male athletes show an enlarged left ventricular end-diastolic cavity dimension (>55mm). Conversely, the end-diastolic cavity dimension is small in most patients with HCM (usually <45mm) and it is enlarged (>55mm) only in those patients with end-stage disease.

Left ventricular diastolic filling pattern further helps to distinguish HCM from physiologic changes in athletes: most patients with HCM, including those with relatively mild hypertrophy, show abnormal Doppler diastolic filling pattern of the left ventricle, whereas trained athletes have invariably demonstrated normal left ventricular filling patterns.

Highly trained female athletes rarely show left ventricular wall thicknesses that lie within the "grey area", which means that a female player with "borderline" left ventricular wall thickness (in the presence of normal cavity size) is most likely to have HCM. An enlarged left atrium (LA) is another echocardiographic finding that is not compatible with physiological hypertrophy and provides further evidence of HCM. A regression of left ventricular hypertrophy after athletic deconditioning (of at least three months) demonstrates the physiological origin of wall thickening due to athletic training. A decrease in LV wall thickness is not observed in HCM.

Usually, distinguishing an "athlete's heart" from cardiopathies other than HCM is not that difficult: even when there are increases in left ventricular end-diastolic cavity dimension extending the "normal range" of 53-58mm (<3.2cm/m² in women and <3.1cm/m² in males), the absence of left ventricular systolic dysfunction is usually sufficient to distinguish this physiological ventricular enlargement induced by training from that due to dilated cardiomyopathy.

Because highly trained athletes may sometimes demonstrate right ventricular enlargement, the need for differential diagnosis between an athlete's heart and right ventricular cardiomyopathy may arise. Differentiation by echocardiography may be difficult because of technical limitations in imaging right ventricular structures and

assessing function in these athletes. Magnetic resonance imaging (MRI) enhances the non-invasive diagnosis of this condition, demonstrating the pathophysiological hallmark of fibro-fatty replacement of myocardium.

Diameters of the aorta (particularly in suspected Marfan syndrome) have to be referenced to individual conditions (age, body surface area, height, etc.).

Exercise test

The primary indication for exercise testing is the detection of exercise-induced myocardial ischaemia in individuals with a history indicative of cardiovascular disease. It is most useful in subjects with an intermediate "pre-test probability" (probability of disease as estimated without testing). In subjects with low pre-test probability, which will apply for the majority of young football players, the rate of false positive results is increased. That is the main reason why exercise testing is not recommended as part of routine screening in sport, e.g. as part of a pre-competition medical assessment.

Exercise testing performed as a self-limited stress test in the cardiac examination of young football players can, however, be used to assess the following:

*1. Rhythm disorders**

- class I: congenital complete AV-block
- class IIa: expected exercise-induced rhythm disorders
- class IIb: evaluation of ventricular premature beats in middle-aged patients without evidence of coronary artery disease and evaluation of young patients with first-degree AV block, type I (Wenckebach) second degree AV block, left bundle branch block, right bundle branch block, or isolated ectopic beats prior to participation in competitive sports
- class III: as part of the routine evaluation of isolated ectopic beats in young patients

*strength of recommendation based on the approach of the American Heart Association: Class I – Intervention is useful and effective; Class IIa – Weight of evidence/opinion is in favour of usefulness/efficacy; Class IIb – Usefulness/efficacy is less well established by evidence/opinion; Class III – Intervention is not useful/effective and may be harmful

2. Heart rate and blood-pressure response to exercise

Blood pressure (BP) should be measured at rest (supine and standing) and continuously during exercise. While systolic blood pressure normally rises during exercise, diastolic pressure falls or remains unchanged. Exercise-induced hypertension is defined as a peak systolic blood pressure of ≥210mmHg in men and ≥190mmHg in women, whereas exertional hypotension is defined as a fall in systolic blood pressure below the baseline values that were measured standing/sitting at rest (>10mmHg). Hypotension immediately after exercise is sometimes seen in healthy individuals due to venous pooling in the lower extremities. A "cool down" period of unloaded pedalling can prevent this phenomenon.

3. Exercise capacity

To assess these questions in football players, it is necessary to perform a self-limited stress test, in which the test person ensures an adequate workload. In fact,

Absolute contraindications	Relative contraindications
• Unstable angina pectoris • Acute myocardial infarction (within two days) • Uncontrolled symptomatic heart failure • Uncontrolled arrhythmias causing symptoms of haemo-dynamic compromise • Symptomatic severe aortic stenosis • Active endocarditis, acute mycarditis or pericarditis • Acute aortic dissection • Acute pulmonary or systemic embolism • Acute noncardiac disorders that may affect exercise performance or may be aggravated by exercise (e.g. infection, fever, thyreotoxicosis)	• Left main coronary stenosis or equivalent • Moderate stenotic valvular disease • Electrolyte abnormalities • Severe hypertension (systolic ≥200mmHg and/or diastolic ≥110mmHg) • Tachyarrhythmias or bradyarrhythmias, including atrial fibrillation with uncontrolled ventricular rate • Hypertrophic cardiomyopathy or other forms of outflow tract obstruction • Mental or physical impairment leading to inability to cooperate • High degree atrioventricular block (except congenital complete heart block)

Table 2.1.3.3 Absolute and relative contraindications for exercise testing

the test often fails to achieve significant results because of insufficient workload (see also "When to stop").

Contraindications

Depending on the individual situation, exercise testing may not be without risk. Careful risk and benefit calculation should be respected.

Technical requirements

- Bicycle or treadmill (calibrated, serviced)
- ECG (12-lead, at minimum V2, V4, V5), continuous monitoring
- BP measuring (continuously)
- Emergency drugs, defibrillator, skilled physician on site
- Room temperature: 18-22°, humidity: 30-60%

Treadmill or bicycle?

Whereas exercise testing on a treadmill is more popular in North America, bicycle testing is preferred in Europe. While in general, the way of exercise testing should be adapted to the individual sport, both methods have their advantages:

- The measured workloads are higher and also more accurately adjustable on the treadmill.
- Treadmill protocols are more flexible than bicycle protocols because both speed and inclination can be varied independently.
- The bicycle offers "cleaner" ECG data, especially at high workloads, because of fewer motion artefacts, which is also an advantage in blood pressure measurement.
- In case of injuries or disability, performance may be better on the bicycle than on the treadmill.

A 12-lead ECG at rest is usually obtained both supine and standing/sitting on the bike and should be assessed prior to the exercise test. An ECG obtained during exercise should be compared with the resting standing/sitting ECG. Diagnostic ECG testing may not be useful if the resting ECG shows left bundle branch block or other ECG abnormalities with marked ST-T wave changes.

Exercise test protocols

Players should be instructed not to eat, drink or smoke for two hours prior to the examination. In principle, workload should progress from low intensity to higher intensity until a predetermined end point is reached (workload, target heart rate or exhaustion in a self-limited test).

Treadmill testing

The Bruce protocol is widely used for office-based exercise testing (see table 2.1.3.4).

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Stage	Grade	Speed (mph)	Time (min)	METS	VO_2 (ml/min x kg)
(1/2	5	1.7	3	3	11)
1	10	1.7	3	4.5	17
2	12	2.5	3	7	25
3	14	3.4	3	10	35
4	16	4.2	3	13	47

Table 2.1.3.4 Bruce protocol

The Balke protocol increases intensity by adjusting the grade of the slope: while speed is fixed at 3.3mph (which corresponds to 5.3kmph) the grade of the slope is increased by 2.5% every minute. The "modified Bruce protocol" and the Cornell and Naughton protocols are more suitable for patients with known coronary heart disease.

Bicycle testing

Bicycle testing according to the "ramp protocol" starts after a resting period of one to three minutes with a "reference phase" of unloaded pedalling (two to three minutes). During the exercise period, workload is continuously increased to the point of exhaustion. Alternatively, increments of 15 to 50 watts every one to three minutes, depending upon the player's heart rate response, can be used ("step protocol").

After a brief cool-down phase, with the player still on the treadmill or sitting on the bicycle, a resting or recovery period of at least five to six minutes completes the examination while ECG recording and blood pressure measurement are continued. This is important as many significant ECG abnormalities develop after exercise.

When to stop

Widely accepted exercise-test endpoints for terminating an exercise test are:

Physician determined:

- Personal impression of the player (pallor, confusion, cyanosis, etc.; absolute indication)
- Exertional hypertension: BP systolic >250mmHg, diastolic > 115mmHg (relative indication)
- Exertional hypotension: BP systolic >10mmHg below baseline BP, despite an increasing workload, with (absolute indication) or without other evidence of ischaemia (relative indication)
- ECG endpoints:
  - ST segment elevation (>1.0mm) in leads without diagnostic Q waves (other than V1 or aVR; absolute indication), indicative of a severe high-grade lesion or coronary spasm
  - ST or QRS changes such as excessive ST depression (>2mm of horizontal or down-sloping of ST segment depression) or marked axis shift (relative indication)
  - Sustained (absolute indication) or non-sustained (relative indication) ventricular tachycardia, and other arrhythmias, such as multifocal ventricular premature beats, ventricular triplets, supraventricular tachycardia, heart block or bradyarrhythmias (relative indication)
  - Development of bundle branch block or intraventricular conduction delay that cannot be distinguished from ventricular tachycardia (relative indication)
- Equipment failure with technical difficulties to monitor ECG or systolic blood pressure (absolute indication)

Player determined:

- Player's request to stop (absolute indication)
- Moderate to severe angina (absolute indication) or increasing chest pain which is not typical or just mild and recognised as the usual angina-of-effort pain or discomfort (relative indication)
- Increasing nervous system symptoms, e.g. dizziness/near syncope, ataxia (absolute indication)
- Fatigue, shortness of breath, wheezing, leg cramps or leg claudication (relative indication)

Other diagnostic tests

Subjects who have positive findings at this basal cardiac evaluation should be referred for adequate additional testing, such as 24-hour ambulatory ECG monitoring, trans-oesophageal echocardiography, CT scan/CT angiography, magnetic resonance imaging, coronarography, etc.

2.1.4 Assessment of performance

Introduction

In order to understand how to evaluate performance, the physical requirements of football have to be known. The typical distance covered by a top-level outfield player during a match is 10-13km, with the midfield players covering longer distances as compared to players in other positions. However, the majority of the distance is covered by walking and low-intensity running, and in terms of energy production requirements, it is mainly the high-intensity exercise periods that are important. Thus, it seems clear that the amount of high-intensity exercise separates the elite players from players at a lower level. Computerised time-motion analysis demonstrated that top-class international players performed 28% more high-intensity running (2.43km v. 1.90km) and 58% more sprinting (650m vs. 410m) than professional players at a lower level.

Football is an intermittent sport in which the aerobic energy system is highly taxed with average and peak heart rates around 85% and 98% of maximal values, respectively. These values together with body temperature measurements suggest that the average oxygen uptake for elite football players is around 70% of maximum oxygen uptake (VO_2max). This may partly be explained by the 150-250 brief intense actions that an elite player performs during a match, which also indicates that the rate of anaerobic energy production is frequently high during a match. Muscle glycogen is probably the most important substrate for energy production, and fatigue towards the end of a match may be related to depletion of glycogen stores in some muscle fibres. Fatigue also appears to occur temporarily during a match.

A match naturally provides the best test for a player but it is difficult to isolate the various components of football and obtain objective measures of performance. Fitness testing can provide relevant information about specific parts of a sport. This chapter describes the different tests to be used in football with a focus on field tests as they are more applicable to match play than laboratory tests.

Objectives of testing

Before selecting a test, clear objectives of any such test should be defined. There may be a number of reasons for testing a player:

– To study the effect of a training programme;
– To motivate a player to train more;
– To give objective feedback to a player;
– To make a player more aware of the aims of the training;
– To evaluate whether a player is ready to compete;
– To determine the performance level of a player during a rehabilitation period;
– To plan short- and long-term training programmes;
– To identify the weaknesses of a player.

To obtain useful information from a test, it is important that the test is relevant and resembles the conditions of football. For example, a cycle test is of minor relevance for a football player but may be used if the player is not able to run.

Laboratory test

There are a number of validated laboratory tests to evaluate various aspects of performance. These include the determination of maximum oxygen uptake to evaluate the player's ability to take up and utilise oxygen (VO_2 max). However, the determination of VO_2 max is of rather limited use in football (see below). Another test often used is the Wingate test, which consists of 30 seconds of maximal cycling exercise, aimed at determining the maximum anaerobic power and ability to maintain a high power output. However, the test is of little relevance for football, since the exercise is very different from the activities during a football match. Strength measurements in the laboratory in which the strength of an isolated muscle group is measured either during isometric, concentric or eccentric contractions are also often used. Such tests provide general information about the strength capacity and peak power output of different muscle groups and may separate different performance levels of players. However, they reflect the performance of the player during competition only to a limited extent. For example, Figure 2.1.4.1 shows that for 20 top-class football players there was no relationship between knee-extensor strength and kick performance, suggesting that the strength of the knee extensors alone does not determine the final impact on the ball in a kick. Strength of other muscle groups, such as the hip muscles, may be important. Technical skills are also a predominant factor in the football kick, which incorporates a complex series of synergistic muscle movements, involving the antagonistic muscles as well. Therefore, such tests are more often adapted for the follow-up of specific training programmes such as preventive neuromuscular hamstring exercises (see 3.3), rather than for evaluating global physical demands in football.

Field tests

Tests which are more specific than laboratory tests will increase the validity of the test, i.e. the test result better reflects the performance of the player. A number of relevant football tests are provided below, focusing on the ability to sprint and perform repeated intense exercise. The tests are simple to organise and require little material.

Repeated sprint test

The ability to run fast and to perform repeated sprints can be easily tested by having the player sprint a given distance a number of times, separated by a period of recovery that leads to a decrease in performance. In relation to the latter aspect, it has been observed that performance in a 30-metre sprint could be maintained when subjects have a recovery period of 120 seconds between each sprint, but a marked decrease was found when the recovery time was 30 seconds or less. This means that in order to evaluate a player's ability to perform repeated sprints, the rest period between 30-metre sprints should be 30 seconds or shorter. In a test measuring the ability to sprint and at the same time change direction, athletes perform seven sprints, each lasting about seven seconds, separated by 25-second rest

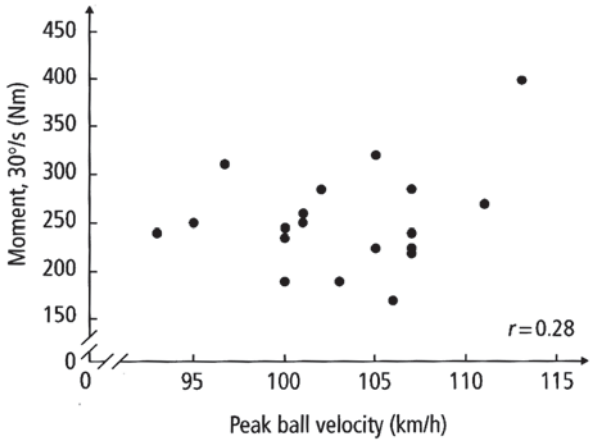


Figure 2.1.4.1
Individual relationship between kick performance (peak ball velocity) and maximum knee extensor torque (Nm) under isokinetic loading at a velocity of 30°/second for elite football players.

periods. Figure 2.1.4.2 shows how the performance of 25 professional football players changed during the preparation period. The significant decrease in the mean sprint time shows that the test can reveal changes in the player's ability to recover after a period of change in training, i.e. a period with speed training. Thus, it is suggested to perform 5 x 30 metres separated by a 30-second recovery period where players are walking back to the starting line. If the time is measured with a stopwatch, the test leader counts down "3-2-1-go", at which point the player starts the sprint. If an electronic device is used, the player should start 1.5 metres behind the starting line at a signal from the test leader.

Cooper test

One of the most widely used field tests is the Cooper test, where the player runs as far as possible within 12 minutes. It is simple to perform, but the type of exercise is very different from the typical activities in football. The main disadvantages are that it requires players to know how to perform the test tactically (pacing strategy) in order to obtain the best test result, and that a track of at least 200m is needed. Its popularity is probably due to the fact that it is simple and a correlation between performance in general and VO_2 max has been observed. However, this relationship between the test and VO_2 max may not be too useful, since VO_2 max is a poor marker of physical performance during match play in competition (see below).

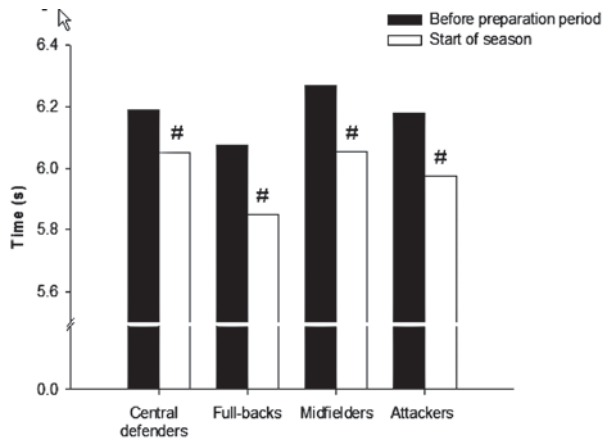


Figure 2.1.4.2
Repeated sprint performance before and after the pre-seasonal preparation period of elite football players at various playing positions. #: Denotes a significant difference in the test results before and after the pre-seasonal preparation period.

Yo-Yo intermittent tests

The Yo-Yo intermittent endurance test (Yo-Yo IE) and the Yo-Yo intermittent recovery test (Yo-Yo IR) are tests which evaluate various aspects of intermittent exercise performance quickly and easily. Two markers are positioned at a distance of 20 metres and a third marker is placed 2.5 (Yo-Yo IE) or 5 (Yo-Yo IR) metres behind one of the cones. A CD is placed in a CD player and the test is performed with the player running like a yo-yo back and forth between the 20-metre markers at a given speed controlled by the CD. The shuttles are interrupted by 5- and 10-second active recovery periods for Yo-Yo IE and Yo-Yo IR, respectively, in which the athletes run around the cone behind the starting line. The speed is regularly increased, and the test is over when the player is no longer able to maintain the speed. The test result is determined as the distance covered during the shuttle runs. Both tests can be used by anyone irrespective of their training status, since each of the tests has two levels. The Yo-Yo intermittent tests provide information on a high number of players within a relatively short time (30 players in 15 minutes), and both have higher validity regarding performance during competition than laboratory tests.

The Yo-Yo IE test lasts 10-20 minutes and consists of five- to 18-second intervals of running interspersed by regular five-second rest periods. The test evaluates an individual's ability to repeatedly perform exercise intervals over a prolonged period of time. The Yo-Yo IR test lasts 2-15 minutes and focuses on the ability to recover after intense exercise. Between each exercise period (5-15 seconds), there is a ten-second pause. The Yo-Yo IR1 test evaluates an individual's ability to repeatedly perform intermittent exercise with a high aerobic component towards the end of the test, whereas during the Yo-Yo IR2 test, both the aerobic and anaerobic energy systems are highly taxed.

The Yo-Yo tests have been shown to have a high reproducibility, sensitivity and validity for football. Some studies have examined whether there is a relationship between performance in the Yo-Yo IR tests and performance in a match. It should be emphasised that this type of comparison is complicated by the fact that in football, it is difficult to obtain a precise measure of physical performance. Nevertheless, for professional football players, a significant correlation was observed between Yo-Yo IR1 performance and the amount of high-intensity exercise during a match (Figure 2.1.4.3 A), which has been suggested to be the best measure of endurance performance during a football match.

Yo-Yo IR1 performance of elite female football players has also been observed to be significantly correlated (r=0.81, n=14) with the amount of high-intensity running performed at the end of each half of a match. Thus, the test appears to be useful in evaluating the match-related physical capacity of a football player. Similarly, a positive relationship between the Yo-Yo IR1 test result and match performance was observed for top-class football referees. In addition, in these referees, a 31% increase in performance of the Yo-Yo IR1 test after a 12-week training period was associated with a 23% higher amount of high-intensity work during a match, and a significantly lower reduction of high-intensity running towards the end of the match. It was also observed that the referees showing the greatest improvement in Yo-Yo IR1 performance also had the largest training-induced increase in high-intensity exercise during matches. The Yo-Yo IR1 test has also been able to detect performance differences between age groups for both girls and boys (Figure 2.1.4.4).

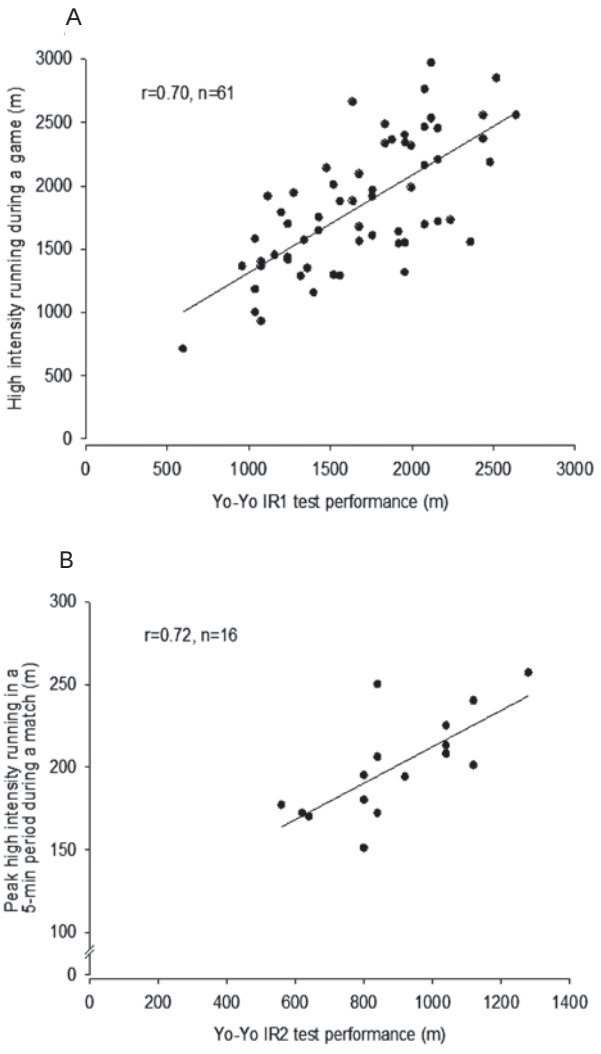


Figure 2.1.4.3 A and B
Relationship between the Yo-Yo IR1 performance and the amount of high intensity running (>15 km·h^{-1}) during a football match (A) (r=0.71; n=61; p<0.05) and the Yo-Yo IR2 performance and the peak high intensity distance covered in a 5-min period during a match for professional football players (B) (r=0.72; n=16; p<0.05).

For the Yo-Yo IR2 test, a significant relation between performance and the longest distance covered in a five-minute period during a match was observed (Figure 2.1.4.3 B). This might appear to be logical since the Yo-Yo IR2 test focuses on evaluating the ability to perform intense exercise and to recover, which are both essential components during the intense periods of a match. Nevertheless, the results underline the accuracy of the tests to evaluate these aspects.

Both the Yo-Yo IR1 and IR2 test are sensitive to alterations in the players' ability to perform repeated high-intensity exercise. During pre-season, this ability is commonly observed to improve by 25-40%, whilst during the season, the level is usually lower. However, it is not possible to generalise since major individual variations are found. Figure 2.1.4.5 shows Yo-Yo IR2 performance data for 10 football players. Whereas all showed an improvement in pre-season, there were major differences in the response during the season: four players improved their test performance, but nine players showed a decrease in performance ranging from 40 to 440 metres. These results illustrate that the Yo-Yo IR tests can also detect changes in the performance level of players during the season. The Yo-Yo IR tests provide far more sensitive measures of change in performance than VO_2 max. Thus, a considerable number of studies have demonstrated much greater changes in Yo-Yo IR test performance (25-40%) compared to change in VO_2 max (0-5%). It should also be mentioned that whilst VO_2 max determinations are time consuming and expensive, the Yo-Yo tests can be operated at low cost and quickly.

Non-exhaustive testing

It is also possible to perform the Yo-Yo intermittent tests without exhausting the players. In this case, the test is stopped after a given time and the heart rate is measured to evaluate the development of the cardiovascular system. The lower the heart rate, the higher the capacity of the individual. In players of the Danish national football team, there was a relationship between heart rate after six minutes of performance of the Yo-Yo IE2 and the amount of high-intensity running during critical parts of matches.

Consequently, the heart rate of the Danish national team's players preparing for EURO 2004 was significantly higher at the start of the preparation period compared to during the season and ten days prior to the start of the competition (Figure 2.1.4.6). Collectively, these observations suggest that heart-rate measurements during a submaximal version of the Yo-Yo IE2 test provide useful information about the fitness level of an individual. Such non-exhaustive tests can be used frequently and are especially useful for players in rehabilitation.

Summary

Laboratory tests provide only limited information about a football player's physical condition. Preferably, field tests which reflect the activities in football should be used. The repeated sprint test evaluates a player's maximal speed capacity as well as his ability to recover from a number of sprints. To examine intermittent endurance, the Yo-Yo intermittent endurance test is recommended, and to evaluate the recovery capacity of a player, the Yo-Yo intermittent recovery test should be used. A number of studies have shown the sensitivity of the Yo-Yo tests in identifying players' performance at various competitive levels, in different playing positions, and after periods of different training types. In addition, their reliability and validity are well known and strong correlations were found for instance between Yo-Yo test performance and the amount of high-intensity running during a football match. Submaximal testing using heart-rate measurements may be used for frequent testing.

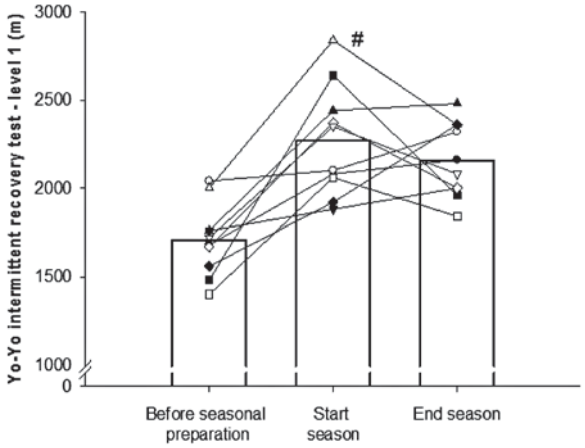


Figure 2.1.4.5
Yo-Yo IR1 performance for ten elite football players prior to pre-seasonal preparation, at the start of the season and at the end of the season. #: Denotes a significantly better performance at the start of the season as compared to before the pre-seasonal preparation.

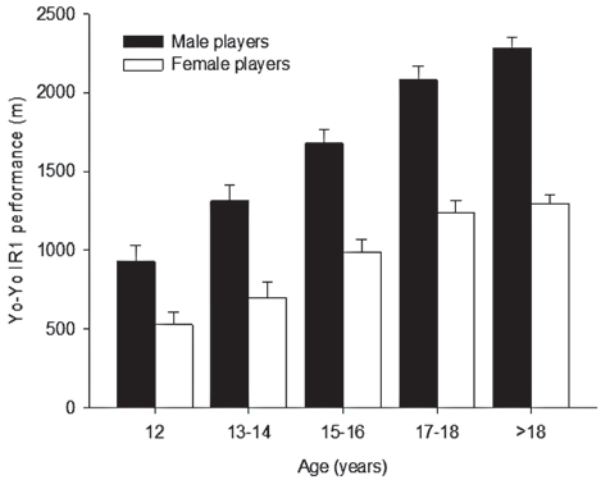


Figure 2.1.4.4
Yo-Yo IR1 performance in relation to age (12, 13-14, 15-16, 17-18, >18) for male (■; n= 58, n=60, n=94, n=58, n=72, respectively) and female players (□; n= 30, n=58, n=47, n=84, n=114, respectively). Values are mean ± SEM.

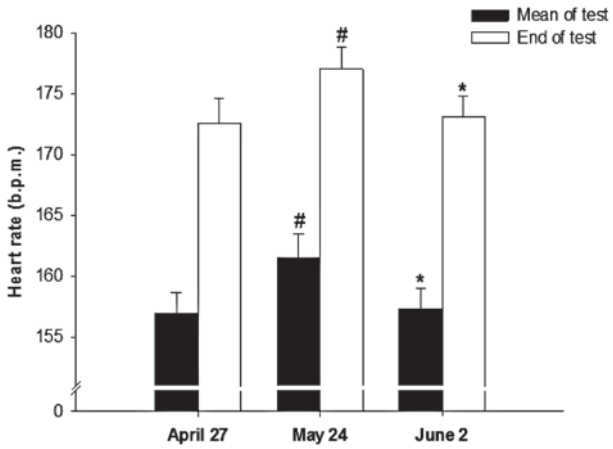


Figure 2.1.4.6
Mean and end heart rate values of 18 players of the Danish national football squad during a sub-maximal Yo-Yo IR2 performed 46, 19 and ten days prior to the start of the European Championship 2004. #: Denotes a significantly higher heart rate during the test performed at May 24 as compared to April 27. *: Denotes a significantly lower heart rate during the test performed at June 2 as compared to May 24.

2.2 Prevention of injury

When applying a scientific approach to implementing injury prevention programmes, the first step is to establish the extent of the problem, and then to analyse the causes of and risk factors for injury. Epidemiological data on the incidence, mechanism, location and severity of injuries offers useful information and assistance for developing specific preventive strategies.

In view of the fact that the greatest risk factor for injury is previous injury, especially if this has not been fully rehabilitated, it is crucial to conduct systematic medical assessments and carefully document previous injuries and current complaints.

Finally, issues relating specially to certain target groups (such as referees, females and youth players) need to be considered.

2.2.1 Registration of injury

Theoretical considerations

A standardised approach for the assessment of sports injuries not only provides important epidemiological information but also a basis for the development of injury prevention programmes and the opportunity to monitor long-term changes in the frequency, characteristics and causes of injury. Furthermore, injury reporting may be regarded as a part of prevention as it increases the awareness of the underlying problem. Studies should be of a prospective, cohort design to minimise the occurrence of errors associated with recall, which is a problem with retrospective study designs. Cohort studies that record players' exposures enable relationships between the incidence of injury and risk factors within the study population.

Definition of injury

When investigating the incidence of injuries, the first task is to define an "injury". Recently a consensus statement on injury definitions and data collection procedures in studies of football (soccer) injuries was published by an international expert group (see reference 2.2.2).

An **injury** is defined as any physical complaint sustained by a player that results from a football match or football training, irrespective of the need for medical attention or time loss from football activities. This injury definition includes three important aspects: (1) all injuries (not only time loss or reduced performance), (2) newly incurred (exclusion of pre-existing and not fully rehabilitated injuries), and (3) exclusion of illnesses and diseases.

A **recurrent injury** is defined as an injury of the same type and at the same site as an index injury and which occurs after a player's return to full participation from the index injury. A recurrent injury occurring within two months of a player's return to full participation is referred to as an "early recurrence"; one occurring two to 12 months after a player's return to full participation as a "late recurrence"; and one occurring more than 12 months after a player's return to full participation as a "delayed recurrence." An injury that results in a player receiving medical attention is referred to as a **"medical-attention" injury** and an injury that results in a player being unable to take a full part in future football training or match play as a **"time-loss" injury**.

The advantage of a broad injury definition is that it becomes possible to assess the effect of the full spectrum of injuries from mild contusions to fractures. This might be of importance in assessing the long-term consequences of injuries, as an analysis of injury sequences shows that minor injuries are often followed by moderate or major ones, and acute complaints are a predictor of subsequent injuries. In addition, athletes sometimes compete despite an injury. The documentation of additional information regarding time loss (estimated duration of subsequent absence from sport) allows expression of the incidence of time-loss injury and the possibility of comparing the results with studies that use that definition.

Besides the location, type and diagnosis of injury, the distinction between **traumatic and overuse injuries** is important for the description of sports injuries. A traumatic injury refers to an injury resulting from a specific, identifiable event and an overuse injury to one caused by repeated micro-trauma without a single, identifiable event responsible for the injury.

Injury reporting systems

The feasibility and quality of an injury-reporting system are not only dependent on the definition of injury but also on the source of information, the characteristics of the injury documentation form, the methods, frequency and duration of data collection and on the availability of exposure data. The precise information to be documented is mainly determined by the specific aim of the study. However, some basic characteristics of the injury should always be described, such as the diagnosis (e.g. body part and type of injury), circumstances/mechanism/causes (e.g. time in the match, contact, foul play) and consequences (e.g. referee's sanction of foul play, treatment of injury, absence from sport). An injury reporting system should allow a grading of the severity of the injury based on the duration of absence or the medical diagnosis. It should be acceptable to those expected to comply with it and be widely applicable. Finally, information about the exposure time is indispensable for the calculation of risk-related incidences.

Injuries should be diagnosed and reported by qualified medical personnel (team physician, physiotherapist) to ensure valid information on the characteristics of the injury and a comparable standard of data. The documentation forms presented in this section (Figure 2.2.1.1) provide a core set of information, but these might need to be extended depending on the specific research question. The selection of locations and types of injury is based on the consensus statement for studies on football injuries and allow comparison with other established coding systems such as the Orchard Sports Injury Classification System and the Sports Medicine Diagnostic Coding System.

To enable a comparison with other studies, different groups or types of sport, it is important to relate the number of injuries to the duration of exposure to the given activity. The incidence of injury is defined as the number of injuries divided by the time that all players spent in matches and/or training sessions. Usually, the incidence is calculated as the number of injuries per 1,000 hours of exposure. It is recommended that the incidences of injuries in matches and in training are reported and compared separately.

Summary

Injury reporting system:

- use **standardised documentation** forms
- **diagnoses** should be **established by a physician** or a specially trained physiotherapist
- registration of **exposure time**
- **separate** calculations for the incidence of **match and training injuries**

Methods of injury documentation

The methods of injury documentation and the related injury report forms differ for injuries during an entire season and during a football competition. If injuries during a season are analysed, the exposure time for both training and match must be documented and the evaluation of baseline information (see 2.1.2 and Figure 2.2.1.2) is recommended. For injuries during tournaments, the exposure time can be easily calculated by multiplying the number of matches, the average duration of a match and the number of players per match.

Injuries during the season

Since the incidence of injury varies during the football season, it is recommended to study football injuries during a whole season, including the period of pre-season training. The timing (pre-season or competition period) and circumstances (outdoor, indoor, match or training) of the injury should also be documented. All injuries should be documented by the team physician using a standardised injury report form (see Figure 2.2.1.1). Definitions of all parameters to be documented should be described in detail in a related manual for the physicians reporting the injuries. The team physician should ask all players about injuries and complaints at least once a week (preferably always on the same day of the week). All players reporting injuries or complaints should be examined by the physician. An injury report form should be completed for each newly incurred injury (and all re-injuries) immediately after the injury has occurred. If the diagnosis changes or can be more precisely described at a later date, the new information should be added to the documentation. A player is defined as injured as long as he/she does not participate as usual in training sessions and/or matches (i.e. any restriction in the exercises performed or duration of match play).

Injury report form

(Team) Player-ID: Date:

1. Injured body part

		right	left	
☐ head / face	☐ shoulder	☐	☐	hip
☐ neck / cervical spine	☐ upper arm	☐	☐	groin
☐ thoracic spine	☐ elbow	☐	☐	m. adductor
☐ lumbar spine	☐ lower arm	☐	☐	hamstrings
☐ sternum / ribs	☐ wrist	☐	☐	m. quadriceps
☐ abdomen	☐ hand	☐	☐	m. abductor
☐ pelvis / sacrum	☐ finger / thumb	☐	☐	thigh
		☐	☐	knee
		☐	☐	lower leg
		☐	☐	achilles tendon
		☐	☐	ankle
		☐	☐	foot
		☐	☐	toe

2. Type of injury

☐ concussion with loss of consciousness	☐ rupture of tendon	☐ strain
☐ concussion without loss of consicousness	☐ ligamentous rupture with instability	☐ contusion
☐ fracture	☐ ligamentous rupture without instability	☐ tendonitis / bursitis
☐ dislocation	☐ lesion of meniscus	☐ dental injury
☐ rupture of mescle	☐ sprain	☐ deep wound
		☐ laceration / abrasion
		☐ others

3. Diagnosis: _____

4. Has the player had a **previous injury** of the same location and type?
☐ no ☐ yes, _____ months ago

5. Was the injury caused by **overuse** or **trauma?**
☐ overuse
☐ trauma

6. **When** did the injury occur?
☐ training Date _____
☐ match

7. Was the injury caused by **contact with another player?**
☐ no ☐ yes

8. Approximately **how long** will the player not be able to participate as usual in the training sessions or matches?
Approx.. _____ days

Figure 2.2.1.1 Injury report form during the season

Figure 2.2.1.2 Attendance record form

Attendance record form

Team: Week:

		MONDAY	TUESDAY	WEDNESDAY	THURSDAY	FRIDAY	SATURDAY	SUNDAY
		training/match	training/match	training/match	training/match	training/match	training/match	training/match
	Date							
Player No.	Name							
T1/01								
T1/02								
T1/03								
T1/04								
T1/05								
T1/06								
T1/07								
T1/08								
T1/09								
T1/10								
T1/11								
T1/12								
T1/13								
T1/14								
T1/15								

Please mark whether it was a training session or a match and note how many minutes each player participated.

If a player did not particiapte in training or match as usual, please state the reason:

FI – no participation in training or match because of an injury caused by football

R – restriction in training or match because of an injury caused by football

0 – other reasons: diseases, personal reasons or injuries not caused by football

If symptoms re-appear after the player has been considered as rehabilitated, a new injury form should be completed for a re-injury (question 4).

Some details about the causes and circumstances of each injury should be noted on the form (questions 5-7). Training is defined as all coach-directed practical training sessions including training of match situations, but not matches against other teams. Matches are defined as all matches against other teams including friendly matches and all types of tournaments. The same definition of training and match applies to the attendance reports (see Figure 2.2.1.2) kept by the coaches. On this form, the coach should document the individual time that each player spends in training sessions and matches. If a player does not participate in training or matches as usual (i.e. any restriction in the exercises performed or duration of match play), the reason should be noted on the form.

Injuries during tournaments

The comprehensive F-MARC report form for injuries during tournaments (see Figures 2.2.1.3 and 2.2.1.4) has been applied in all international FIFA competitions as from the FIFA World Cup 1998™ in France. It has also been used for all team sports tournaments during the Olympic Games 2004 and was the basis for the IOC approach for injury surveillance in multi-sport events. In all FIFA tournaments, the physicians from all participating teams are requested to fill out a form after each match and return this directly to the FIFA medical officer. Confidentiality of all personal information is ensured. Compliance with this procedure has been excellent, and its implementation has quickly become routine for all participating teams as demonstrated by the response rate of almost 100% shortly after its introduction.

The injury report form comprises a single page on which all injuries, or where applicable the non-occurrence of injury during a given match, are described in tabular form. In contrast to other injury-reporting systems that require a form be filled out only if an injury has occurred, the F-MARC injury report form should be filled out after each match regardless of whether an injury has occurred or not. This method of documentation offers a number of advantages: the absence of injury can be distinguished from missing data, the response check is easier and the implementation of injury documentation is promoted as a matter of routine. The injury report form requires documentation on the following information: shirt number of the injured player, minute in the match, injured body part and type of injury, circumstances (non-contact, contact, foul play) and consequences of injury (e.g. referee's sanction, medical treatment), as well as an estimate of the duration of the player's likely absence from training and/or matches as a result of the injury. Definitions of all parameters to be documented are described on the back page of the injury report form (Figure 2.2.1.4).

2.2.2 Frequency and characteristics of injury

Incidence, nature and causes of injury

The growing importance of describing and understanding the incidence, nature and causes of injuries in football is demonstrated by the three-fold increase in the number of published research papers on football injuries since 1990. F-MARC has conducted an ongoing programme of injury surveillance at FIFA tournaments since the 1998 World Cup and has investigated a wide range of intrinsic and extrinsic risk factors, such as age, gender, skill level, foul play, tackle mechanisms, playing surfaces and refereeing, over this period. The benefit of using consistent methodologies in injury surveillance studies has long been recognised; for this reason, F-MARC facilitated the above mentioned international discussion meeting in Zurich in 2006 that resulted in the publication of the consensus agreement on definitions and procedures that should be used for studies of football injuries.

The information presented in the following sections is based mainly on injury surveillance studies conducted by F-MARC; namely, the two-season (2005 and 2006) study of match and training injuries sustained by male and female non-professional players aged 18 to 25 years and the study of match injuries sustained by male and female players at FIFA tournaments from 2000 to 2008. All the results presented were derived from data collected and analysed according to the international consensus statement on injury studies in football. An injury in the two-season study was, however, defined as "any physical complaint sustained by a player during a football match or training session that prevented the player from taking a full part in training or match play activities for one or more days following the day of injury". On average, most players can expect to sustain at least one match or training injury of this type every season.

Injury report form for matches

Team____________________ Match ____________ / ____________ Date ____/____/____

Team physician ____________________ Tel/Fax____________ e-mail____________

Please report: **All injuries** (traumatic and overuse) caused by football **regardless** of the consequences with respect to participation during training or match.
The information provided is for medical and research purposes and will be treated confidentially.

Player	**Time**	**Location**		**Diagnosis**		**Severity**	**Circumstances**		**Consequences**	
No.	minute of match	injured body part	code	type of injury	code	absence in days	contact	foul	referee´s sanction	treatment
							yes ☐ no ☐	yes ☐ no ☐	yes ☐ no ☐	yes ☐ no ☐
							yes ☐ no ☐	yes ☐ no ☐	yes ☐ no ☐	yes ☐ no ☐
							yes ☐ no ☐	yes ☐ no ☐	yes ☐ no ☐	yes ☐ no ☐
							yes ☐ no ☐	yes ☐ no ☐	yes ☐ no ☐	yes ☐ no ☐
							yes ☐ no ☐	yes ☐ no ☐	yes ☐ no ☐	yes ☐ no ☐
							yes ☐ no ☐	yes ☐ no ☐	yes ☐ no ☐	yes ☐ no ☐
							yes ☐ no ☐	yes ☐ no ☐	yes ☐ no ☐	yes ☐ no ☐
							yes ☐ no ☐	yes ☐ no ☐	yes ☐ no ☐	yes ☐ no ☐
							yes ☐ no ☐	yes ☐ no ☐	yes ☐ no ☐	yes ☐ no ☐

☐ **no injuries in this team**

Definitions and codes of location, diagnosis and severity see reverse

Figure 2.2.1.3 Injury report form for matches

Definitions and codes

Location of injury					
	TRUNK		**UPPER EXTREMITY**		**LOWER EXTREMITY**
1	Head / Face	11	Shoulder	21	Hip
2	Neck / Cervical spine	12	Upper Arm	22	Groin
3	Thoracic spine	13	Elbow	23	Thigh
4	Lumbar spine	14	Forearm	24	Knee
5	Sternum / Ribs	15	Wrist	25	Lower leg
6	Abdominal	16	Hand	26	Achilles tendon
7	Pelvis / Sacrum	17	Finger	27	Ankle
		18	Thumb	28	Foot
				29	Toe

Diagnosis					
1	Concussion with loss of consciousness	7	Ligamentous rupture with instability	13	Bursitis
2	Concussion without loss of consciousness	8	Ligamentous rupture without instability	14	Tendinitis
3	Fracture	9	Lesion of meniscus	15	Laceration / Abrasion
4	Dislocation	10	Sprain	15	Others
5	Muscle fibre rupture	11	Strain		
6	Tendon rupture	12	Contusion		

Severity of injury in days			
Estimated duration of absence from training or play for example:			
0	= 0 days	7	= 1 week
1	= 1 day	14	= 2 weeks
2	= 2 days	> 30	= more than 4 weeks

Circumstances and consequences	
Contact	with another player or an object (except ground)
Foul	judgement of the player, overt and hidden fouls
Referee's sanction	of the foul that caused the injury
Treatment	by a physician, physiotherapist or dentist (immediately or later)

Figure 2.2.1.4 Definitions and codes

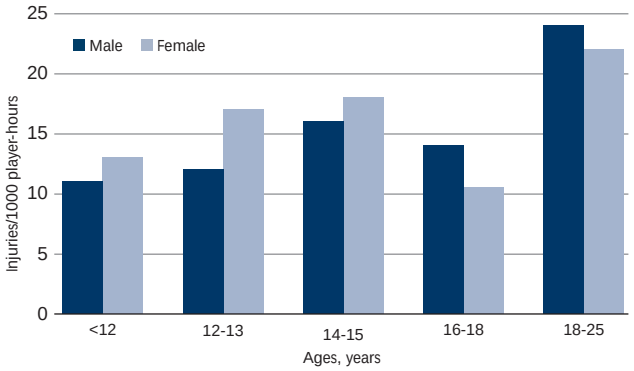


Figure 2.2.2.1 Incidence of match injuries as a function of age

Tournament	Incidence of injury (injuries/match)	
	Men	Women
World Cup	1.6	1.0
Olympic Football Tournament	1.3	1.0
U-19/20 World Cup	1.2	1.1
U-17 World Cup	1.0	0.5

Table 2.2.2.1 Incidence of match injuries in FIFA tournaments (2000 to 2008)

Severity category (number of days' absence)	Incidence of injury (injuries/1,000 player hours)	
	Men	Women
Minimal (1 to 3 days)	7.9	6.3
Mild (4 to 7 days)	7.0	5.9
Moderate (8 to 28 days)	5.9	4.0
Severe (>28 days)	3.1	4.6
ALL injuries	**24.2**	**21.4**

Table 2.2.2.2 Incidence of match injuries as a function of severity category

Match injuries

The incidence of injury during matches is around eight times higher than that observed during training, although the average severity of injuries is similar. It is normal to report the incidence of injury in football as the number of injuries sustained per 1,000 player hours of exposure. Therefore, because each team match represents 16.5 player hours of exposure (11 players on the pitch for 90 minutes), the incidence of match injuries equates approximately to the number of injuries that a team might expect to sustain on average in 60 matches. As many professional teams play around 60 matches per season, the incidence of injury also approximates to the number of match injuries a team might expect to sustain each season.

Incidence and severity of match injuries

The incidence of time-loss match injuries is ~24 injuries/1,000 player hours for men and ~21 injuries/1,000 player hours for women. The medical team at a club playing 60 matches in a season should therefore anticipate having to treat and rehabilitate between 20 and 25 match injuries each season. There is a general trend for the incidence of injury to decrease with decreasing age for both male and female players (Figure 2.2.2.1).

At international level, the incidence of time-loss injuries per match is higher amongst male than female players, and there is a trend for the incidence of injury to decrease with decreasing age for male players but this trend is not apparent for female players (Table 2.2.2.1). The average severity of match injuries is 15 days: the incidences of match injuries for men and women as a function of severity category are summarised in Table 2.2.2.2.

Over half of all match injuries sustained by players result in less than seven days' absence, so the majority of injured players do not miss their next scheduled match. However, teams should expect to treat and rehabilitate three to five players per season whose injuries will result in them being absent from training and play for over one month.

Nature of match injuries

Match injuries in football are predominantly located in the lower limb, with the knee and ankle the most commonly injured structures (Figure 2.2.2.2). The thigh and the head/neck are, however, also each responsible for over 10% of all injuries sustained by men and women. There are no significant differences in the locations of match injuries

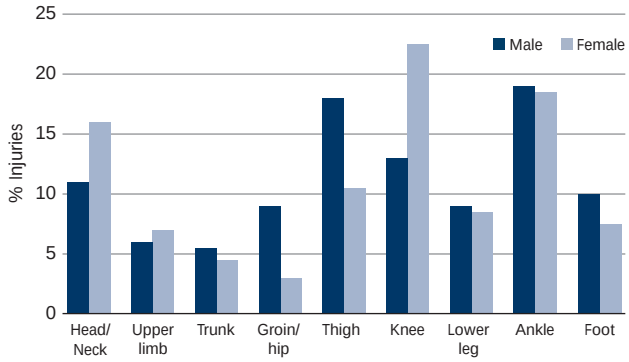


Figure 2.2.2.2 Distribution of match injuries by body location

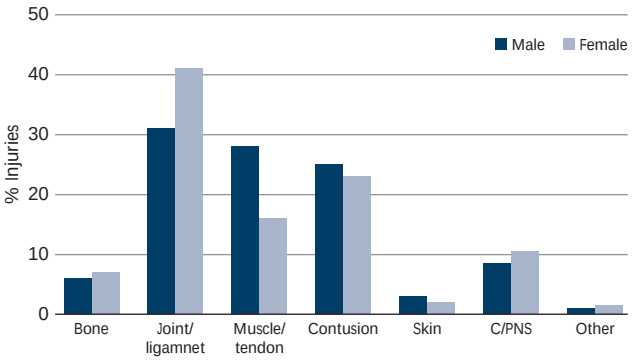


Figure 2.2.2.3 Distribution of match injuries by type (C/PNS: central/peripheral nervous system)

sustained by youth and international players compared to those shown in Figure 2.2.2.2.

The majority of injuries sustained by men and women are joint/ligament injuries but high proportions of contusion (20-25%) and muscle/tendon (15-25%) injuries are also observed (Figure 2.2.2.3). There are no significant differences in the types of match injuries sustained by youth and international players compared to those shown in Figure 2.2.2.3.

For both men and women, the most common match injury is a tear of the ankle lateral ligament complex (~11%). The majority of thigh injuries sustained by men and women are hamstring and quadriceps muscle tears and quadriceps contusions. For the knee, men sustain a high proportion of ligament and contusion injuries but for women the great majority are ligament injuries. Women are three times more likely to sustain an anterior cruciate ligament injury than men. In the ankle, ligament injuries at the lateral ligament complex, tibiofibular syndesmosis and medial ligament are the most common injuries for both male and female players. A significantly higher incidence of adductor tears is observed amongst men than women. Although there is a higher incidence of concussion reported by women, this may reflect a greater readiness on the part of female players to report this type of injury compared to men, rather than indicating a real difference.

The most common match injuries observed as a function of severity category are shown in Table 2.2.2.3.

Severity category (number of days' absence)	Men	Women
Minimal (1 to 3 days)	Ankle lateral ligament complex tear and quadriceps contusion	Ankle lateral ligament complex tear and lower leg and quadriceps contusions
Mild (4 to 7 days)	Ankle lateral ligament complex tear	Ankle lateral ligament complex tear and concussion
Moderate (8 to 28 days)	Hamstring muscle and ankle lateral ligament complex tears	Concussion and ankle lateral ligament complex tear
Severe (>28 days)	Hamstring muscle and anterior cruciate ligament tears	Anterior cruciate ligament tear

Table 2.2.2.3 Most common match injuries as a function of severity category

Causes of match injuries

Over 95% of match injuries are reported to be acute with less than 5% of match injuries resulting from overuse/gradual onset. The causation of match injuries is summarised in Figure 2.2.2.4.

The major cause of match injuries is contact with other players (e.g. tackles, collisions); however, acute non-contact activities (e.g. running, jumping) and contact with the playing surface are also responsible for significant numbers of injuries. Male players sustain a significantly higher proportion of acute non-contact injuries than female players, which is largely related to the higher number of non-contact adductor and hamstring muscle tears sustained by men. Female players, on the other hand, suffer twice as many injuries through contact with the playing surface and a major reason for this is the substantially higher numbers of ankle and knee ligament injuries sustained by women from this cause. Three quarters of contact injuries are sustained by the tackled player and a quarter by the tackling player; almost half of these contact injuries result from foul tackles: two-footed tackles and tackles resulting in a clash of heads are particularly prone to cause injury.

The relationship between players' on-pitch and post-match medical attention is an important issue that medical personnel should be aware of. The proportion of players receiving on-pitch attention who subsequently require post-match medical attention is small (~25%). However, the proportion of players that require post-match medical attention who previously received on-pitch medical attention is also relatively low (~40%); this indicates that a significant number of injured players continue to play without medical evaluation or treatment during a game even though this may result in the player exacerbating a relatively minor injury.

Training injuries

Although the incidence of injuries is higher during match play than during training, players' exposure to training is very much higher than that for match play. As players might expect to train for ~5 hours per week for 40 weeks each season (200 training hours per player per season), the incidence of training injuries approximates to the number of training injuries that a group of five players might expect to sustain, on average, each season. For this reason, training injuries still represent a significant injury burden for teams so it is important that the medical support group are also aware of the incidence, nature and causes of training injuries.

Incidence and severity of training injuries

The incidence of time-loss training injuries is ~3 injuries/1,000 player-hours for both men and women. A medical team supporting a squad of 25 players should therefore anticipate having to treat and rehabilitate around 15 training injuries each season. The average severity of training injuries is 13 days: the incidences of training injuries for men and women as a function of severity category are summarised in Table 2.2.2.4.

The proportions of injuries in each severity category are similar to those presented for match injuries for both men and women.

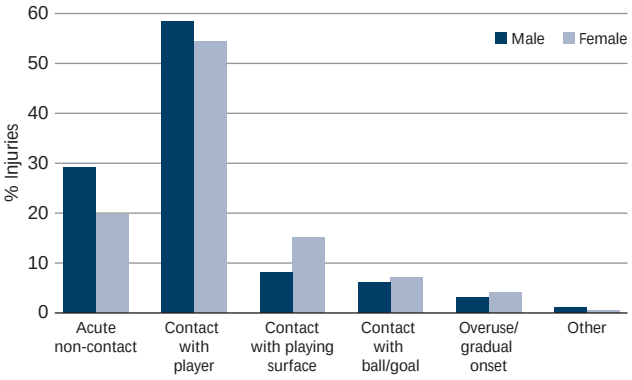


Figure 2.2.2.4 Distribution of match injuries by causation

Severity category (number of days' absence)	**Incidence of injury (injuries/1,000 player-hours)**	
	Men	Women
Minimal (1 to 3 days)	1.2	0.9
Mild (4 to 7 days)	0.7	0.6
Moderate (8 to 28 days)	0.6	0.7
Severe (>28 days)	0.5	0.5
ALL injuries	**3.1**	**2.8**

Table 2.2.2.4 Incidence of training injuries as a function of severity category

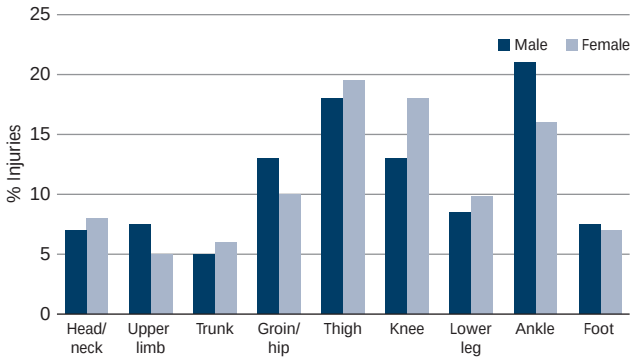


Figure 2.2.2.5 Distribution of training injuries by body location

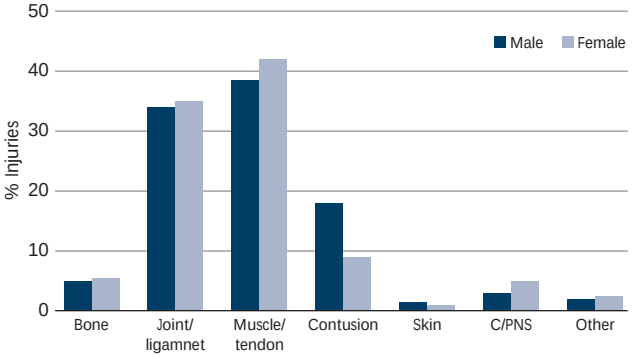


Figure 2.2.2.6 Distribution of training injuries by type
(C/PNS: central/peripheral nervous system)

Nature of training injuries

Training injuries are also predominantly located in the lower limb; however, there are some significant differences in the specific body locations of training injuries (Figure 2.2.2.5) compared to match injuries. In particular, there are higher proportions of injuries in the thigh and groin/hip regions and a smaller proportion of head injuries than that observed during matches for both male and female players.

There are substantially more muscle/tendon and substantially fewer contusions observed amongst training injuries than are found with match injuries for both male and female players (Figure 2.2.2.6); the proportion of joint/ligament training injuries is, however, similar to that found for match injuries.

There are differences in the specific injuries sustained in training compared to match injuries, which reflects the training activities undertaken by players compared to match activities. Nevertheless, an ankle lateral ligament complex tear (~12%) is also the most common training injury for both men and women. For women, other common training injuries are quadriceps (~11%), hamstring (~7%) and adductor (~6%) muscle strains, and for men, hamstring (~8%), adductor (~8%) and quadriceps (~7%) muscle strains. Women (~3%) are again three times more likely to sustain an anterior cruciate ligament training injury than men. The most common training injuries observed as a function of severity category are shown in Table 2.2.2.5.

Severity category (number of days' absence)	Men	Women
Minimal (1 to 3 days)	Ankle lateral ligament complex and adductor muscle tears	Ankle lateral ligament complex tear
Mild (4 to 7 days)	Ankle lateral ligament complex tear	Quadriceps muscle and ankle lateral ligament complex tears
Moderate (8 to 28 days)	Ankle lateral ligament complex tear	Ankle lateral ligament complex and quadriceps muscle tears
Severe (>28 days)	Ankle lateral ligament complex and hamstring muscle tears	Anterior cruciate ligament tear

Table 2.2.2.5 Most common match injuries as a function of severity category

Causes of training injuries

Although the great majority (>80%) of training injuries are still reported to be acute injuries, around 12% of injuries sustained by men and almost 20% of injuries sustained by women are reported as overuse/gradual onset injuries. In contrast to match injuries, the major cause of training injuries (Figure 2.2.2.7) for both men and women is acute non-contact injuries; however, contact with other training partners still accounts for a major proportion of training injuries. Female players are significantly less likely to sustain player-to-player contact injuries but more likely to sustain overuse/gradual onset injuries in training than male players.

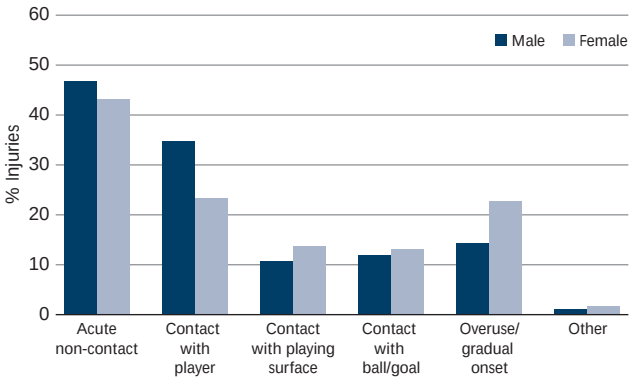


Figure 2.2.2.7 Distribution of training injuries by causation

General risk factors for injury

Intrinsic risk factors predispose players to injury, whilst extrinsic risk factors create situations that make players more susceptible to injury. A previous injury is perhaps the most important risk factor for sustaining an injury in football, especially for lower limb muscle/tendon and ligament injuries. Other intrinsic risk factors include inadequate rehabilitation, mechanical and functional instability of the knee and ankle, physical and mental fatigue and poor pre-match and training preparation. Extrinsic risk factors exist in most aspects of football, including training load, number of matches played, climate, altitude, playing equipment and playing conditions. Two large-scale epidemiological studies, one conducted amongst male elite professional footballers in Europe and the other amongst male and female non-professional players in the USA, investigated whether new generation artificial turf had an adverse effect on the incidence and nature of football injuries. The two studies produced very similar results with neither study identifying significant differences in the incidence or nature of injuries sustained on artificial turf compared to grass surfaces for either male or female players during match play or training activities.

Long-term risks of injury

Only a few studies have investigated the long-term health risks associated with playing football and these studies relate to two issues: osteoarthrosis of lower limb joints and neuropsychological impairment from heading a football. There is evidence to suggest that football, especially at the highest levels, is associated with an increased risk of osteoarthrosis of the ankle, knee and hip joints; however, significantly more detailed research is still required about this issue. Early studies, which claimed an increased prevalence of neurological and neuropsychological deficits amongst retired footballers, were based on play with older, water absorbent, leather footballs; unfortunately many of these earlier studies were also subject to methodological weaknesses. More recent studies of this issue, using modern footballs, have not identified any neurological damage resulting from heading a football. Epidemiological data in fact indicate that most concussions in football (~80%), for both men and women, are caused by head-to-head or arm-to-head contacts rather than player-to-ball contact.

Fuller CW, Ekstrand J, Junge A, Andersen TE, Bahr R, Dvorak J, Hägglund M, McCrory P, Meeuwisse WH (2006) Consensus statement on injury definitions and data collection procedures in studies of football (soccer) injuries. Simultaneous publication in British Journal of Sports Medicine 40(3):193-201, Clinical Journal of Sports Medicine 16(2):97-106 and Scandinavian Journal of Medicine & Science in Sports 16(2):83-92

2.2.3 Injury prevention programmes

Introduction

Playing football requires various skills and abilities, including endurance, agility, speed, technical and tactical understanding of the game. All these aspects will be addressed and improved during the training session, but playing football also entails a substantial risk of injury. Thus, training should also include exercises to reduce the risk of injury. Prevention programmes such as "The 11", "The 11+" und "PEP" have proven to be effective in reducing the injuries in scientific studies. Before these

programmes are described in detail, the key elements core strength, neuromuscular control and balance, eccentric training of the hamstrings, plyometrics and agility will be outlined. The described programmes can be easily incorporated into the warm-up part of the training session in a very time-efficient manner. This will allow the coaching staff to maintain their autonomy and continue to address the facets of play that they feel should be worked on during the training session.

Key components in exercise-based prevention

Core training

The "core" represents a functional unit, which includes muscles of the trunk (abdominals, back extensors) and the pelvic-hip region. The achievement and preservation of core stability is crucial for optimum functioning of the upper and lower extremities. Football players must possess sufficient strength and neuromuscular control in the hip and trunk musculature to provide sufficient core stability in all planes of motion (see Figure 2.2.3.1). Research has demonstrated the importance of the way different muscles groups work synergistically to achieve optimum core stability. Abdominal muscles and lumbar extensor muscles control the stability of the spine and the pelvis. The importance of hip abductors and hip rotators (especially external ones) in maintaining lower extremity alignment has been emphasised by several authors. Hip muscle activation directly affects the ability of the thigh muscles to generate and dissipate forces during jumping and landing. Core instability can have a dramatic negative influence on the alignment and neuromuscular control of the lower extremity (see Figure 2.2.3.2). A destabilised lumbo-pelvic unit typically demonstrates hip adduction and femoral internal rotation, knee valgus, associated external tibial rotation, lateral deviation of the patella and foot pronation (see Figures 2.2.3.3 and 2.2.3.4). In other words, core instability can directly contribute to positional pathokimematics that is so often associated with knee injury mechanisms (especially in a non-contact situation). There is growing scientific evidence that core stability has an important role in injury prevention.

Neuromuscular training

Neuromuscular control refers to the neural activation of muscles to control joint motion, which requires complex interactions in the sensorimotor system. Neuromuscular control does not represent a single entity, but complex interacting systems integrating different aspects of muscle actions (static, dynamic, reactive), muscle activations (eccentric more than concentric), coordination (multi-joint muscles), stabilisation, body posture, balance and anticipation ability. The sensorimotor system (see Figure 2.2.3.5) represents the composite of the physiological systems of the complex neurosensory and neuromuscular process, which has been frequently simplified and inappropriately described as proprioception. Peripheral afferents originate from sensory receptors located in the

Figure 2.2.3.1 Optimum core control for Dr. Socrates, Brazil (1982 FIFA World Cup Spain™)

Figure 2.2.3.2 Destabilised core and left lower extremity for F. Baresi, Italy (1994 FIFA World Cup USA™)

Figure 2.2.3.3 and 2.2.3.4 Poor core and lower extremity neuromuscular control during landing

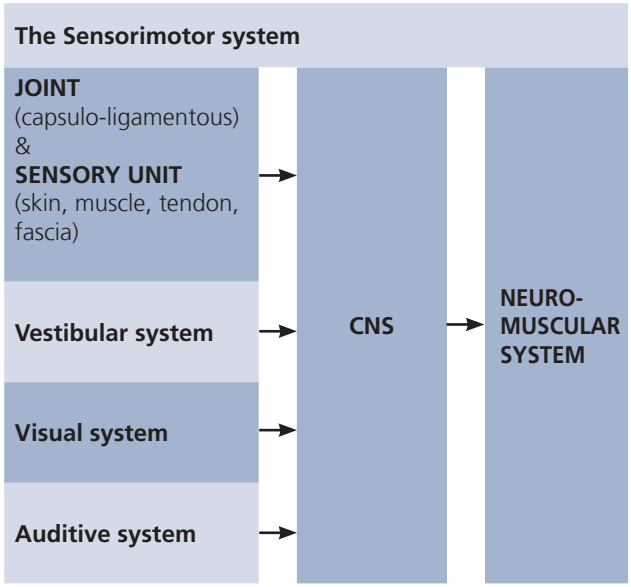


Figure 2.2.3.5 Neuromuscular control

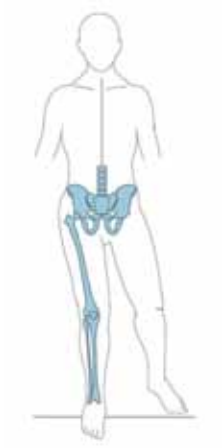


Figure 2.2.3.6 Model for optimum stabilisation of the core (Bizzini 2000)

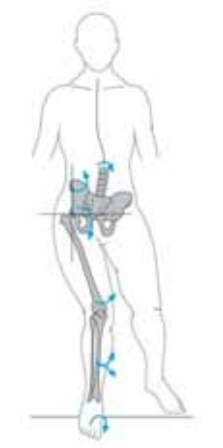


Figure 2.2.3.7 The medial collapse: cascade of de-stabilisation of the core (Bizzini 2000)

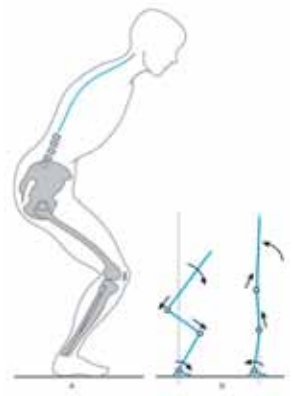


Figure 2.2.3.8 Model for optimal "ready position" of the core and the lower extremity for plyometrics and agility drills (Bizzini 2000)

joint (capsulo-ligamentous structures), muscle (muscle spindle, golgi tendon organ), myofascial and cutaneous tissue. Afferent input is also derived from the visual and vestibular systems. Balance is also a complex process involving coordination of multiple sensory (visual, vestibular, somatosensory), motor (coordinated stabilising neuromuscular strategies) and biomechanical components (body segments alignment against gravity) (see Figures 2.2.3.6-7).

Eccentric muscle activity is defined as a type of muscle loading that involves an external force application with resultant tension increase during physical lengthening of the musculo-tendinous unit. In sport, approximately 80% of all muscle activity is eccentric in nature; typical examples are deceleration and landing situations. Optimum eccentric strength of coordinated muscle activities greatly contributes to the neuromuscular control and stabilisation of the involved joints. Two-joint muscles, such as the hamstrings (acting on the hip and knee joint), are highly eccentrically loaded during football. There is strong empirical and growing scientific evidence that sport-specific neuromuscular training programmes can effectively prevent knee and ankle injuries.

Plyometric and agility training

Plyometrics is defined as exercises that enable a muscle to reach maximum strength in as short a time as possible. Eccentric muscle contractions are rapidly followed by concentric contractions in many sports skills. This type of muscle actions is also described as the "stretch shortening" cycle. All movement patterns in sport involve repeated stretch shortening cycles. The aim of plyometric training is to decrease the amount of time (also called "amortisation time phase") required between the yielding eccentric muscle contraction and the initiation of the overcoming concentric contraction. Plyometrics provide the ability to train specific movement patterns in a biomechanically correct manner, thereby strengthening the muscle, tendon and ligament more functionally. Agility is the ability to decelerate, accelerate, or change direction quickly with proper biomechanical control without loss in speed or strength (see Figure 2.2.3.8). Plyometrics and agility drills were the important components of programmes which proved effective in the prevention of anterior cruciate ligament (ACL) injuries in particular, but also of other knee and ankle injuries.

"The F-MARC 11" – the basic programme

Background

"The 11" is a simple, catchy and time-efficient preventive programme that comprises ten evidence-based or best-practice exercises (created by a group of international experts, under the leadership of FIFA/F-MARC) and the promotion of fair play. The programme is performed on the field, with the players wearing their usual equipment and football shoes. It requires no equipment except a ball and can be completed in 10-15 minutes (after a short period of familiarisation).

The main targets of the exercise programme are: core strength, neuromuscular control and plyometrics/agility. It is emphasised that "The 11" are basic exercises, which may have many variations. Specific programmes to address a specific pathology or dysfunction should be best designed and implemented by specialised sports physiotherapists and athletic trainers. The first two exercises, "the bench" and the "sideways bench" focus on core strength. These are really basic exercises meant to train strength and stabilisation of the dorsal, ventral and lateral core muscle groups. Other exercises of "The 11" also help to improve core stability, even if their primary aim is other muscle groups. Two exercises focus on the eccentric component of neuromuscular control: "hamstrings" and "cross-country skiing". In these exercises, the key is the maintenance of a stabilised body position during controlled motion(s). In "hamstrings", the focus is set on the eccentric work and stabilising activity of the two-joint muscles of the posterior thigh (=hamstring) on the knee, hip and lumbo-pelvic region. In "cross-country skiing" the focus is on the eccentric work and stabilising activity of the quadriceps muscle (with its two-joint rectus femoris muscle also acting on the pelvis) on the knee joint. For both exercises, optimum core stability is a must within the controlled body position: abdominal, back and hip muscles are also indirectly trained. Three exercises were chosen for the static, dynamic and reactive neuromuscular control of the lower extremity: "chest-passing in single-leg stance", "forward bend in single-leg stance" and "figures of eight in single-leg stance". In these three exercises the key component is the maintenance of an optimum stabilised body position: proper lower extremity alignment, core stability, head-neck control (aligned with the spine), balance and anticipation. For plyometric and agility training three exercises were chosen: "jumps over the line" and "bounding" focus more on plyometric skills, whereas "zigzag shuffle" puts more emphasis on agility skills. "Jumps over the line" improves lower extremity power, speed and reactive body control, whereas "zigzag shuffle" focuses on lower extremity reactivity, speed (foot movements), quickness and reactive body control and "bounding" improves lower extremity power, speed and stride length. The key for effective training in all these exercises is the maintenance of an optimum coordinated body position during dynamic performance. The symmetry of the lower limb actions, along with their proper alignment within the body position, is crucial in this type of exercise.

Football injuries can be prevented only partly by improved physical condition of the players. Knowing that a substantial amount of football injuries are caused by foul play, the observance of the Laws of the Game and especially fair play is an essential aspect in the prevention of injury. Fair play involves more than just complying with the existing rules; its essence comprises respect for the opponent and the spirit of the game. It should be promoted not only with regard to the players, but also in relation to the attitudes and behaviour of the coaches, referees and spectators. A positive attitude towards fair play can change intentional behaviour. An increased awareness of the importance of fair play may reduce the incidence of injury and make football a healthier game.

Description of "The F-MARC 11" – Instruction for players

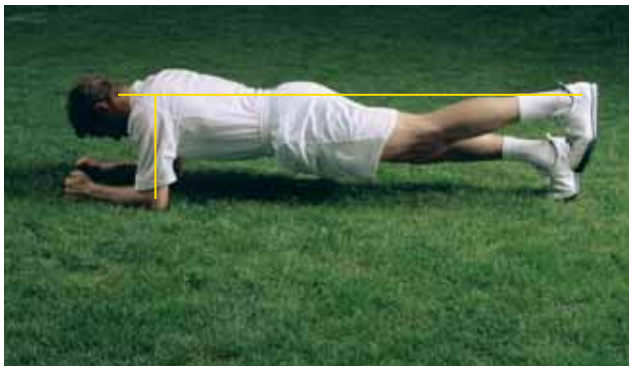
Before starting the training programme, always perform an adequate warm-up and dynamic stretching routine.

1 - The bench

- **Starting position:** Lie on your stomach. Support your upper body with your arms. Place your feet vertical to the ground.
- **Action:** Lift your stomach, hips and knees so that your body forms a straight line from your shoulders to your heels, parallel to the ground. Your elbows should be directly underneath your shoulders. Tighten the abdominal muscles and buttocks. Pull your shoulder blades towards the centre of your back so that your shoulder blades are level with your back. Lift the right leg a few centimetres from the ground and hold this position for 15 seconds. Return to the starting position, relax and repeat the exercise with the left leg. Perform 1-2 times on each leg.
- **Important:**
  - Your head, shoulders, back and pelvis should be in a straight line
  - Your elbows should be directly underneath your shoulders
  - Do not tilt your head backwards
  - Do not let your stomach drop
  - Do not move your hips upwards
  - When lifting your leg, do not let your pelvis tilt to the side

2 - Sideways bench

- **Starting position:** Lie on one side. Support your upper body with one arm so that your elbow is underneath your shoulder and your forearm is on the ground. Bend your bottom knee 90°. When viewed from above, the shoulders, elbow, hips and both knees should form a straight line.
- **Action:** Lift your top leg and hips until the shoulder, hip and top leg are in a straight line parallel to the ground and hold this position for 15 seconds. Return to the starting position, relax and repeat the exercise on the other side. Perform twice on each side.
- **Important:**
  - When viewed from the front, your upper shoulder, hips and upper leg should be in a straight line
  - Your elbow should be directly underneath your shoulder
  - When viewed from above, your shoulders, elbow, hips and both knees should be in a straight line
  - Do not rest your head on your shoulder
  - Do not let your hips drop
  - Do not tilt your upper shoulder, hips, pelvis or legs forwards or backwards



The bench

Sideways bench

3 - Hamstrings

– **Starting position:** Kneel down with a straight upright upper body. Knees and lower legs should be hip-width apart. Cross your arms in front of your body. Have a partner pin your ankles firmly to the ground with both hands.

– **Action:** Slowly lean forward keeping your upper body and hips straight. Thighs, hips and upper body stay in a straight line. Try to hold this straight body alignment as long as possible. When the body position can no longer be maintained by the hamstrings then use both hands to control the fall. Perform 5 times.

– **Important:**
  - Your partner must keep your ankles firmly on the ground
  - Your upper body, hips and thigh should be in a straight line
  - Do not bend at your hips
  - Do not tilt your head backwards
  - Perform the exercise slowly at first, but once you feel more comfortable, speed it up

Hamstrings

4 - Cross-country skiing

- **Starting position:** Stand on your right leg and let the other leg hang relaxed. Bend the knee and hips slightly so that the upper body leans forward. When viewed from the front, your hip, knee and foot of the supporting leg should be in a straight line.
- **Action:** Flex and extend the knee of your supporting leg and swing your arms in opposite directions in the same rhythm. Flex your knee as much as possible, but keep your weight balanced on the entire foot. On extension, never lock the knee. Keep your pelvis and upper body stable and facing forwards. Perform 15 times on the right leg, then 15 times on the left leg.
- **Important:**
  - When viewed from the front, the hip, knee and foot of your supporting leg should be in a straight line
  - Keep your upper body and pelvis stable and facing forward
  - Keep your pelvis horizontal and do not let it tilt to the side
  - Balance your weight across the whole foot
  - Do not let the knee of your supporting leg buckle inwards
  - Never let your knees meet

Cross-country skiing

5 - Chest-passing in single-leg stance

- **Starting position:** Two players face each other at a distance of 3m, both standing on their right leg. Knee and hips should be slightly bent. Keep your weight on the ball of your foot or lift your heel from the ground. When viewed from the front, the hip, knee and foot of your supporting leg should be in a straight line.
- **Action:** Throw a ball back and forth, standing on your right leg means throwing with your left arm and vice versa. Catch the ball with both hands and throw it back with one hand. The quicker the exchange of the ball, the more effective the exercise. Perform 10 times on the right leg, then 10 times on the left leg.
- **Important:**
  - When viewed from the front, the hip, knee and foot of your supporting leg should be in a straight line
  - Keep your upper body and pelvis stable and facing forward
  - Keep your pelvis horizontal and do not let it tilt to the side
  - Keep your hips and the knee of your supporting leg always slightly bent
  - Do not let your knee buckle inwards
  - Keep your weight on the ball of your foot or lift your heel fully off the ground

Chest-passing in single-leg stance

6 - Forward bend in single-leg stance

– **Starting position:** As in exercise 5, face your partner at a distance of 3m, each of you standing on your right legs.
– **Action:** As in exercise 5. Before throwing back, touch the ground with the ball without putting weight on it. Perform 10 times on the right leg, then 10 times on the left leg.
– **Important:**
  - When viewed from the front, the hip, knee and foot of your supporting leg should be in a straight line
  - Keep your pelvis horizontal and do not let it tilt to the side
  - Keep your hips and the knee of your supporting leg slightly bent throughout
  - Do not let your knee buckle inwards
  - Keep weight only on the ball of your foot, or lift your heel fully off the ground
  - When touching the ground with the ball, do not place your weight on the ball

7 - Figures of eight in single-leg stance

– **Starting position:** As in exercise 5, face your partner at a distance of 3m, each of you standing on your right legs.
– **Action:** As in exercise 5. Before throwing back, swing the ball in a figure of eight through and around both legs, first around your supporting leg with your upper body leaning forward and then around the other leg while standing as upright as possible. Perform 10 times on the right leg, then 10 times on the left leg.
– **Important:**
  - When viewed from the front, the hip, knee and foot of your supporting leg should be in a straight line
  - Keep your pelvis horizontal and do not let it tilt to the side
  - Keep your hips and the knee of your supporting leg slightly bent throughout
  - Do not let your knee buckle inwards
  - Keep weight only on the ball of your foot, or lift your heel fully off the ground
  - Your upper body should move up and down substantially during this exercise

Forward bend in single-leg stance

Figures of eight in single-leg stance

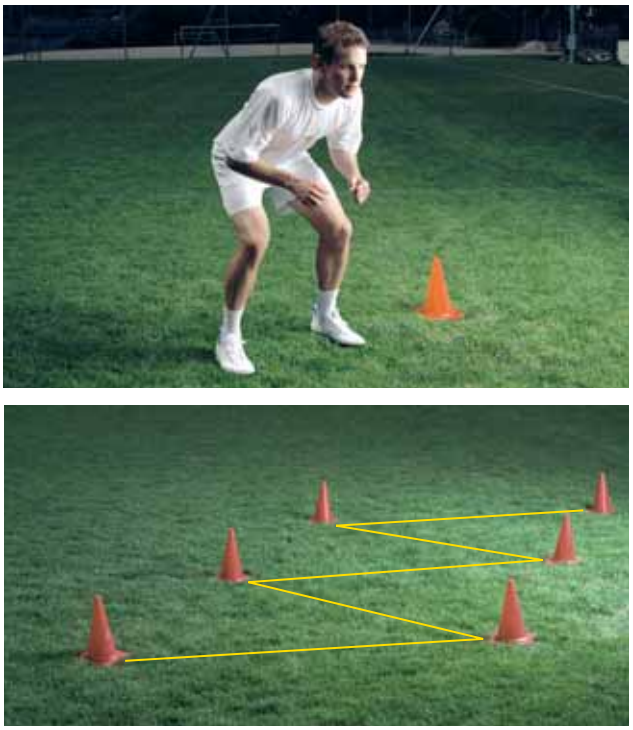
8 - Jumps over a line

- **Starting position:** Stand on both feet hip-width apart, about 20cm to the side of a line. Bend your knees and hips slightly so the upper body leans a little forward. When viewed from the front, your hip, knee and foot should be in a straight line. Arms are slightly bent and close to the body.
- **Action:** Jump with both feet, sideways over the line and back as quickly as possible. Land softly on the balls of both feet with slightly bent knees. Jump 10 times side to side, then 10 times forwards and backwards over the line.
- **Important:**
  - When viewed from the front, your hips, knees and feet should form two parallel lines
  - Keep your hips and knees slightly bent throughout
  - Never let your knees meet and do not let them buckle inwards
  - Push off both feet and land on the balls of both feet
  - Land softly with your knees bent to cushion impact
  - Never land with extended knees or on your heels
  - A soft landing and quick take-off are more important than the height of the jump

Jumps over a line

9 - Zigzag shuffle

– **Starting position:** Stand at the start of the zigzag course (6 marks set 10 x 20 m), legs shoulder-width apart. Bend your knees and hips so your upper body leans substantially forward. One of your shoulders should point in the direction of movement.
– **Action:** Shuffle sideways to the first mark, turn so that the other shoulder points to the next mark and complete the zigzag course as fast as possible. Always take-off and land on the balls of your feet. Complete the course twice.
– **Important:**
  - Always keep your upper body leaning forward with your back straight
  - Keep your hips and knees substantially bent
  - Push off and land on the balls of both feet
  - Land softly with your knees bent to cushion impact
  - Keep knees 'soft' throughout and do not let them buckle inwards
  - Never land with extended knees or on your heels



Zigzag shuffle

10 - Bounding

– **Starting position:** Stand on your take-off leg with your upper body upright. The arm of the same side is in front of the body. When viewed from the front, the hip, knee and foot of the take-off leg should be in a straight line.
– **Action:** Spring as high and far as possible off your supporting leg. Bring the knee of your trailing leg up as high as possible and the opposite arm bent in front of the body when bounding. Land softly on the ball of the foot with a slightly bent knee. Cover 30m twice.

Important:

– When viewed from the front, the hip, knee and foot of your take-off leg should be in a straight line
– Bring your trailing leg and the opposite arm up in the front of your body when bounding
– Land on the ball of your foot and with your knee bent to cushion impact
– Do not let your knee buckle inwards during take-off or landing
– Never land with extended knees or on your heels

11 - Fair play

Knowing that a substantial amount of football injuries are caused by foul play, the observance of the Laws of the Game and especially fair play are essential for the prevention of football injuries.

– **Important:** Play fair!

Bounding

"The 11+" – a complete warm-up programme

Background

"The 11+" is the advanced version of "The 11", integrating the basic characteristics of the "PEP" with the aim of providing a complete warm-up package for football players and teams. The programme is the result of cooperation between F-MARC, the Oslo Sports Trauma Research Center and the Santa Monica Orthopaedic and Sports Medicine Research Foundation. In a scientific study it was shown that female youth football teams using "The 11+" as a standard warm-up had a significantly lower risk of injuries than teams that warmed up as usual.

Description of "The 11+"

"The 11+" has three parts:

Part I: running exercises at a slow speed combined with active stretching and controlled partner contacts;

Part II: six sets of exercises, focusing on core and leg strength, balance, and plyometrics/agility, each with three levels of increasing difficulty; and

Part III: running exercises at moderate/high speed combined with planting/cutting movements. A key point in the programme is to use the proper technique during all of the exercises. Pay full attention to correct posture and good body control, including straight leg alignment, knee-over-toe position and soft landings. "The 11+" should be completed, as a standard warm-up, at least two to three times a week and should take approximately 20 minutes to complete.

PART 1: Running exercises (8 minutes)

The course is made up of six to ten pairs of parallel cones, approximately 5-6m apart. Two players start at the same time from the first pair of cones, jog along the inside of the cones and do the various exercises on the way. After the last cone they run back along the outside. On the way back, speed can be increased progressively as players warm up.

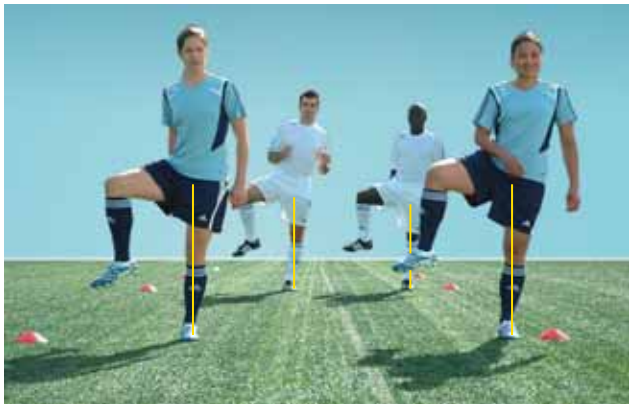
1 - Straight ahead (2 sets)

Jog straight to the last cone. Make sure you keep your upper body straight. Your hip, knee and foot are aligned. Do not let your knee buckle inwards. Run slightly more quickly on the way back.

Straight ahead

2 - Hip out (2 sets)

Jog to the first cone, stop and lift your knee forwards. Rotate your knee to the side and put your foot down. At the next cone, repeat the exercise on the other leg. Repeat until you reach the other side of the pitch.



Hip out

3 - Hip in (2 sets)

Jog to the first cone, stop and lift your knee to the side. Rotate your knee forwards and put your foot down. At the next cone, repeat the exercise on the other leg. Repeat until you reach the other side of the pitch.

Hip in

4 - Circling partner (2 sets)

Jog to the first cone. Shuffle sideways towards your partner, shuffle an entire circle around one other (without changing the direction you are looking in) and then shuffle back to the first cone. Repeat until you reach the other side of the pitch.

Circling partner

5 - Jumping with shoulder contact (2 sets)

Jog to the first cone. Shuffle sideways towards your partner. In the middle, jump sideways towards each other to make shoulder-to-shoulder contact. Land on both feet with your hips and knees bent. Shuffle back to the first cone. Repeat until you reach the other side of the pitch.

Jumping with shoulder contact

6 - Quick forwards and backwards (2 sets)

Run quickly to the second cone then run backwards quickly to the first cone, keeping your hips and knees slightly bent. Repeat, running two cones forwards and one cone backwards until you reach the other side of the pitch.

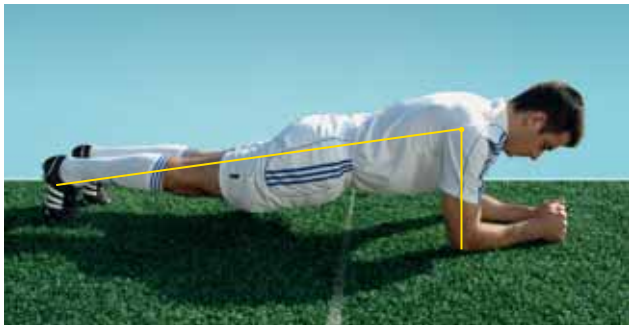
Quick forwards and backwards

PART 2: Strength - Plyometrics - Balance (10 minutes)

7 - The bench

Level 1: Static (3 sets)

- **Starting position:** Lie on your front, support your upper body with your forearms. Elbows directly under your shoulders.
- **Exercise:** Lift upper body, pelvis and legs up until your body is in a straight line from head to foot. Pull in stomach and gluteal muscles and hold the position for 20-30 sec.
- **Important:** Do not sway or arch your back. Do not move your hips upwards.



Static

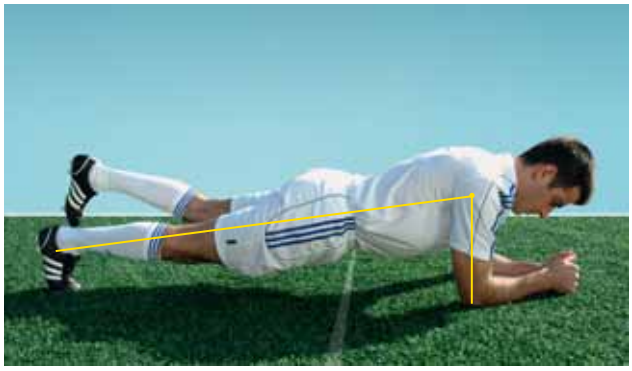
Level 2: Alternate legs (3 sets)

- **Starting position:** Lie on your front, support your upper body with your forearms. Elbows directly under your shoulders.
- **Exercise:** Lift your upper body, pelvis and legs up until your body is in a straight line from head to foot. Pull in your stomach and gluteal muscles. Lift each leg in turn, holding for a count of 2 sec. Continue for 40-60 sec.
- **Important:** Do not sway or arch your back. Do not move your hips upwards. Keep your pelvis stable and do not let it tilt to the side.

Alternate legs

Level 3: One leg lift and hold (3 sets)

- **Starting position:** Lie on your front, support your upper body with your forearms. Elbows directly under your shoulders.
- **Exercise:** Lift your upper body, pelvis and legs up until your body is in a straight line. Pull in your stomach and gluteal muscles. Lift one leg about 10-15cm off the ground and hold the position for 20-30 sec.
- **Important:** Do not sway or arch your back. Do not move your hips upwards. Keep pelvis stable and do not let it tilt to the side.



One leg lift and hold

8 - Sideways bench

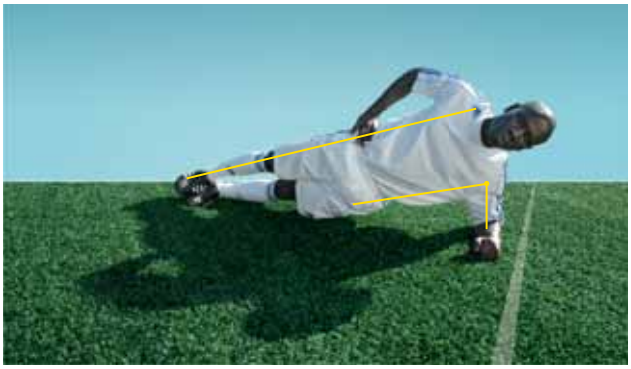
Level 1: Static (3 sets on each side)

- **Starting position:** Lie on your side with the knee of the lowermost leg bent to 90 degrees, support yourself on your forearm and lowermost leg. Elbow of your supporting arm directly under your shoulder.
- **Exercise:** Lift your pelvis and uppermost leg until they form a straight line with your shoulder and hold the position for 20-30 sec. Repeat on the other side.
- **Important:** Keep your pelvis stable and do not let it tilt downwards. Do not tilt your shoulders, pelvis or leg forwards or backwards.

Static

Level 2: Raise and lower hip (3 sets on each side)

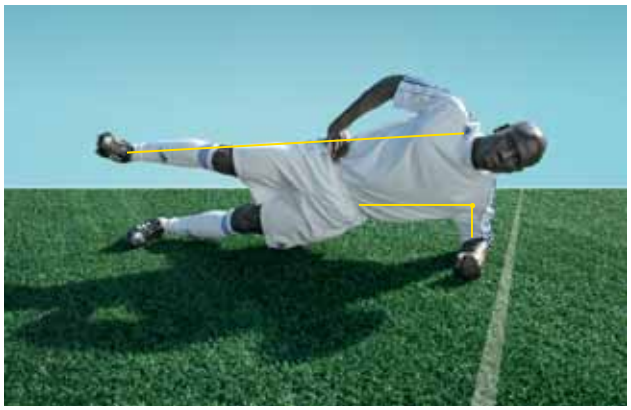
- **Starting position:** Lie on your side with both legs straight, support yourself on forearm. Elbow of your supporting arm directly under shoulder.
- **Exercise:** Raise yur pelvis and legs until your body forms a straight line from the uppermost shoulder to the uppermost foot. Lower your hips to the ground and raise them back up again. Continue for 20-30 sec. Repeat on the other side.
- **Important:** Do not tilt your shoulders or pelvis forwards or backwards. Do not rest head on your shoulder.



Raise and lower hip

Level 3: With leg lift (3 sets on each side)

- **Starting position:** Lie on your side with both legs straight, support yourself on your forearm and lower leg. Elbow of your supporting arm directly under shoulder.
- **Exercise:** Raise your pelvis and legs until your body forms a straight line from the uppermost shoulder to the uppermost foot. Lift your uppermost leg up and slowly lower it down again. Continue for 20-30 sec. Repeat on the other side.
- **Important:** Keep your pelvis stable and do not let it tilt downwards. Do not tilt your shoulders or pelvis forwards or backwards.



With leg lift

9 - Hamstrings

Level 1: Beginner (1 set)

- **Starting position:** Kneel with your knees hip-width apart; partner pins your ankles firmly to the ground with both hands.
- **Exercise:** Slowly lean forward, while keeping your body straight from the head to the knees. When you can no longer hold the position, gently take your weight on your hands, falling into a press-up position. 3-5 repetitions.
- **Important:** Do the exercise slowly at first, but once you feel more comfortable, speed it up.

Hamstrings

Level 2: Intermediate (1 set)

- **Starting position and exercise:** as described for level 1. Minimum 7-10 repetitions

Level 3: Advanced (1 set)

- **Starting position and exercise:** as described for level 1. Minimum of 12-15 repetitions.

10 - Single-leg stance

Level 1: Hold the ball (2 sets on each leg)

- **Starting position:** Stand on one leg, knee and hip slightly bent and hold the ball in both hands.
- **Exercise:** Hold your balance and keep your body weight on the ball of your foot. Hold for 30 sec., and repeat on the other leg. Exercise can be made more difficult by lifting the heel from the ground slightly or passing the ball around your waist and/or under your other knee.
- **Important:** Do not let your knee buckle inwards. Keep your pelvis horizontal and do not let it tilt to the side.

Hold the ball

Level 2: Throwing ball with partner (2 sets on each leg)

- **Starting position:** Stand on one leg, face a partner at a distance of 2-3m.
- **Exercise:** Keep your balance while you throw the ball to one another. Hold in your stomach and keep weight on the ball of your foot. Continue for 30 sec. and repeat on the other leg. Exercise can be made more difficult by lifting the heel from the ground slightly.
- **Important:** Do not let your knee buckle inwards. Keep your pelvis horizontal and do not let it tilt to the side.

Throwing ball with partner

Level 3: Test your partner (2 sets on each leg)

- **Starting position:** Stand on one leg, at arm's length from your partner.
- **Exercise:** Keep your balance while you and your partner in turn try to push the other off balance in different directions. Continue for 30 sec. and repeat on the other leg.
- **Important:** Do not let your knee buckle inwards. Keep your pelvis horizontal and do not let it tilt to the side.

Test your partner

11 - Squats

Level 1: With toe raise (2 sets)

- **Starting position:** Stand with your feet hip-width apart, hands on your hips.
- **Exercise:** Slowly bend your hips, knees and ankles until your knees are flexed to 90 degrees. Lean your upper body forwards. Then straighten your upper body, hips and knees and stand up on your toes. Then slowly lower down again, and straighten up slightly more quickly. Repeat for 30 sec.
- **Important:** Do not let your knee buckle inwards. Lean your upper body forwards with a straight back.

With toe raise

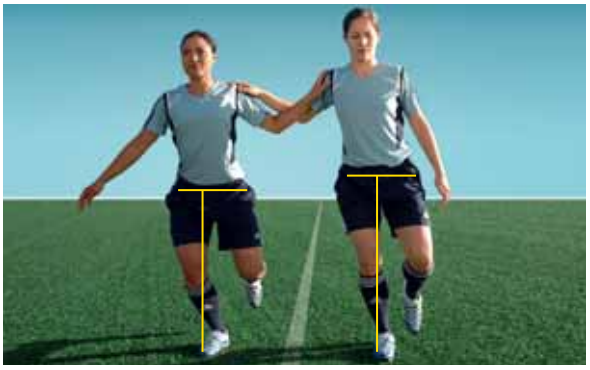
Level 2: Walking lunges (2 sets)

- **Starting position:** Stand with your feet hip-wide apart, hands on your hips.
- **Exercise:** Lunge forward slowly at an even pace. Bend your hips and knees slowly until your leading knee is flexed to 90 degrees. The bent knee should not extend beyond the toes. 10 lunges on each leg.
- **Important:** Do no let your knee buckle inwards. Keep your upper body straight and pelvis horizontal.

Walking lunges

Level 3: One-leg squats (2 sets on each leg)

- **Starting position:** Stand on one leg, loosely hold on to your partner.
- **Exercise:** Slowly bend your knee, if possible until it is flexed to 90 degrees, and straighten up again. Bend slowly then straighten slightly more quickly. Repeat on the other leg. 10 squats on each leg.
- **Important:** Do not let your knee buckle inwards. Keep your upper body facing forward and pelvis horizontal.



One-leg squats

12 - Jumping

Level 1: Vertical jumps (2 sets)

- **Starting position:** Stand with your feet hip-width apart, hands on your hips.
- **Exercise:** Slowly bend your hips, knees and ankles until your knees are flexed to 90 degrees. Lean your upper body forwards. Hold this position for 1 sec. then jump as high as you can, and straighten your whole body. Land softly on the balls of your feet. Repeat for 30 sec.
- **Important:** Jump off both feet. Land gently on the balls of both feet with your knees bent.

Vertical jumps

Level 2: Lateral jumps (2 sets)

- **Starting position:** Stand on one leg. Bend your hips, knee and ankle slightly and lean your upper body forwards.
- **Exercise:** Jump from your supporting leg approx. 1m to the side onto the other leg. Land gently on the ball of your foot and bend your hips, knee and ankle. Hold this position for about a second and then jump on the other leg. Repeat for 30 sec.
- **Important:** Do not let your knee buckle inwards. Keep your upper body stable and facing forward and your pelvis horizontal.

Lateral jumps

Level 3: Box jumps (2 sets)

- **Starting position:** Stand with your feet hip-width apart, imagine a cross you are standing in the middle of.
- **Exercise:** Jump with both legs forwards and backwards, from side to side, and diagonally across the cross. Keep your upper body slightly leaned forwards. Jump as quickly and explosively as possible. Repeat for 30 sec.
- **Important:** Land softly on the balls of both feet. Bend your hips, knees and ankles on landing. Do not let your knee buckle inwards.

Box jumps

PART 3: Running exercises (2 minutes)

Across the pitch

13 - Across the pitch (2 sets)

Run approx 40m across the pitch at 75-80% of maximum pace and then jog the rest of the way. Keep your upper body straight. Your hip, knee and foot are aligned. Do not let your knees buckle inwards. Jog easily back.

Bounding

14 - Bounding (2 sets)

Take a few warm-up steps then take 6-8 high bounding steps with a high knee lift and then jog the rest of the way. Lift the knee of the leading leg as high as possible and swing the opposite arm across the body. Keep your upper body straight. Land on the ball of the foot with the knee bent and spring. Do not let your knee buckle inwards. Jog back easily to recover.

Plant & cut

15 - Plant & cut (2 sets)

Jog 4-5 steps straight ahead. Then plant on the right leg and cut to change direction to the left and accelerate again. Sprint 5-7 steps (80-90% of maximum) before you decelerate and plant on the left foot and cut to change direction to the right. Do not let your knee buckle inwards. Repeat the exercise until you reach the other side of the pitch, then jog back.

"PEP" – prevention of ACL injuries

Background

The PEP (= Prevent injury, Enhance Performance) programme was developed by Holly Silvers and Bert Mandelbaum from the Santa Monica Orthopaedic and Sports Medicine Research Foundation (http://www.aclprevent.com/pepprogram.htm) for the prevention of anterior cruciate ligament (ACL) injuries in women's football. It has proven to reduce severe ACL injuries by 60 to 89% after a training period of approximately six to eight weeks.

This prevention programme consists of a warm-up, stretching, strengthening, plyometrics (training for power or explosiveness: jumping, bounding and hopping exercises) and football-specific activities to optimise the strength and coordination of the stabilising muscles around your knee joint. It is important to use the proper technique during all of the exercises. Pay full attention to correct posture, avoid excessive side-to-side movement when jumping and ensure soft, quiet landings. This programme should be completed at least two to three times a week and should take approximately 15 minutes to complete. It can easily be included in the training programme by substituting it for the usual warm-up (Figure 2.2.3.9).

Description of "PEP" – Instructions for players

Part 1. Warm-up

Warming up and cooling down are a crucial part of a training programme. The purpose of the warm-up section is to prepare your body for activity. By warming up your muscles first, you greatly reduce the risk of injury.

1A. Jog line to line (cone to cone)
Elapsed time 0-30 seconds
Purpose To ensure a good running technique. Keep your hip/knee/ankle in straight alignment without your knee caving in or your feet whipping out to the side.
Instructions Complete a slow jog from the near to the far sideline.

1B. Shuttle run (side to side)
Elapsed time 30-60 seconds
Purpose To engage your hip muscles at the inner and outer thigh. This exercise will increase speed. Carefully avoid inward caving of the knee joint.
Instructions Start in an athletic stance with a slight bend at the knee. Leading with the right foot, sidestep, pushing off with the left foot (back leg). When you drive off with the back leg, be sure your hip/knee/ankle are in a straight line. Switch sides halfway across.

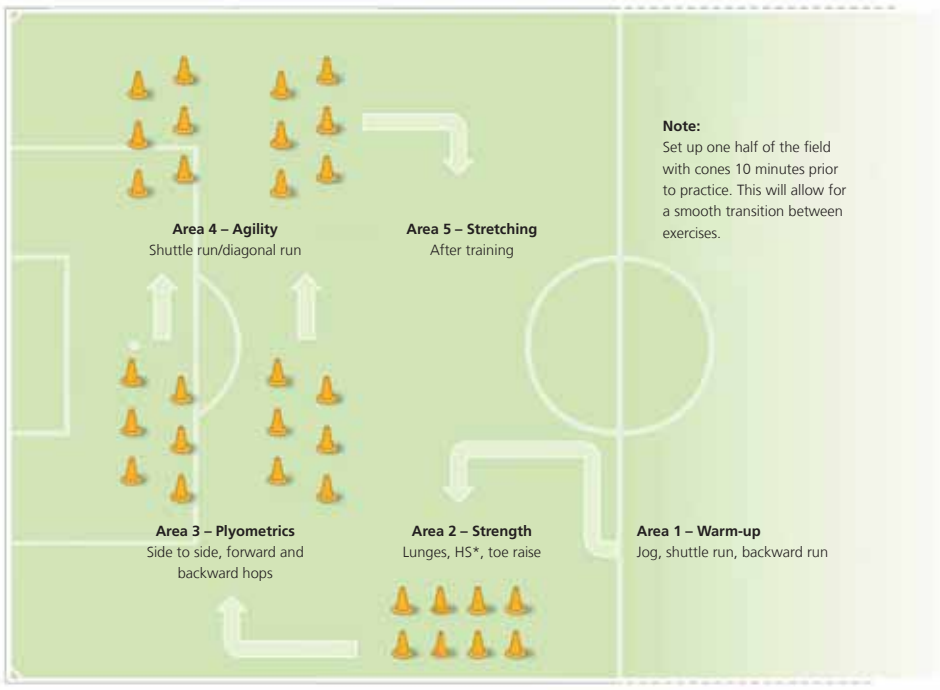


Figure 2.2.3.9 Field set-up for PEP

*HS = hamstrings

1C. Backward running
Elapsed time 1-1.5 minutes
Purpose To continue your warm-up and engage your hip extensors/hamstrings. Make sure you land on your toes. Watch for locking of your knee joint. As you bring your foot back, make sure you maintain a slight bend to your knee.
Instructions Run backwards from sideline to sideline. Land on your toes without snapping the knee back. Stay on your toes and keep the knees slightly bent at all times.

Part 2. Strength

This portion of the programme focuses on increasing leg strength. This will lead to improved leg strength and a more stable knee joint. Please note that here technique is everything! Make sure you perform the exercises accurately to avoid injury.

2A. Walking lunges (3 sets x 10 repetitions)
Elapsed time 6.5-7.5 minutes
Purpose To strengthen the thigh (quadriceps) muscle
Instructions Lunge forward, leading with your right leg. Push off with your right leg and lunge forward with your left leg. Drop the back knee straight down. Make sure that you keep your front knee over your ankle. Control the motion and try to avoid your front knee from caving inward. If you cannot see your toes on your leading leg, you are not doing the exercise correctly.

2B. Russian hamstring (3 sets x 10 repetitions)
Elapsed time 7.5-8.5 minutes
Purpose To strengthen your hamstrings
Instructions Kneel on the ground with your hands at your side. Have a partner hold firmly at your ankles. With a straight back, lean forward, leading with your hips. Your knee, hip and shoulder should be in a straight line as you lean towards the ground. Do not bend at the waist. You should feel the hamstrings in the back of your thigh working. Repeat the exercise in three sets of 10, or a total of 30 repetitions.

2C. Single toe raises (30 repetitions x 2)
Elapsed time 8.5-9.5 minutes
Purpose To strengthen your calf muscle and improve balance
Instructions Stand up with your arms at your side. Bend the left knee up and maintain your balance. Slowly rise up on your right toes with good balance. You may hold your arms out ahead of you in order to help. Slowly repeat 30 times and switch to the other side. As you become stronger, you may need to add repetitions to this exercise to build up the strengthening effect of the exercise.

Part 3. Plyometrics

These exercises are explosive and help to build power, strength and speed. The most important element when considering performance technique is the landing. It must be soft! When you land from a jump, you need to drop your weight softly on the balls of your feet, slowly rolling back to the heel with a bent knee and a straight hip. Although these exercises are basic, it is critical that you perform them correctly. Please take time to ensure that these exercises are carried out safely and correctly. You will notice that only time increments are given. Some individuals on your team will be able to complete more repetitions with proper technique than others. Please only do as many repetitions as you can within the given time period with perfect biomechanical form. If you do otherwise, you are reinforcing bad habits.

3A. Lateral hops over cone (30 seconds)
Elapsed time 9.5-10 minutes
Purpose To increase power/strength, emphasising neuromuscular control
Instructions Stand with a 15cm cone to your left. Hop to the left over the cone, landing softly on the balls of your feet and bending at the knee. Repeat this exercise, hopping to the right.

3B. Forward/backward hops over cone (30 seconds)
Elapsed time 10-10.5 minutes
Purpose To increase power/strength, emphasising neuromuscular control
Instructions Hop over the cone/ball, landing softly on the balls of your feet and bending at the knee. Now hop backwards over the ball, using the same landing technique. Be careful not to snap your knee back to straighten it. You need to maintain a slight bend in the knee.

3C. Single leg hops over cone (30 seconds)
Elapsed time 10.5-11 minutes
Purpose To increase power/strength, emphasising neuromuscular control
Instructions Hop over the cone/ball, landing on the ball of your foot and bending at the knee. Now hop backwards over the ball, using the same landing technique. Be careful not to snap your knee back to straighten it. You need to maintain a slight bend to the knee. Now stand on the left leg and repeat the exercise.

3D. Vertical jumps with headers (30 seconds)
Elapsed time 11-11.5 minutes
Purpose To increase height of vertical jump
Instructions Stand forward with your hands at your side. Bend the knees slightly and push off, jumping straight up. Remember the proper landing technique: drop the weight on the ball of your foot with a slight bend to the knee. Repeat for 30 seconds.

3E. Scissors jump (30 seconds)
Elapsed time 11.5-12 minutes
Purpose To increase power and strength of vertical jump
Instructions Lunge forward, leading with your right leg. Keep your knee over your ankle. Now push off with your right foot and propel your left leg forward into a lunge position. Be sure your knee does not cave in or out. It should be stable and directly over the ankle. Remember the proper landing technique: drop the weight on the ball of your foot with a slight bend to the knee.

Part 4. Agility

The agility portion of the programme incorporates sport-specific activities that reinforce safe movement patterns and encourage the use of musculature that reinforces the ACL. Remember to stay low to the ground (athletic stance), keep your toes visible to you and your buttocks back as if you were seated in a chair. Engage your buttock, outer hip and hamstring muscles.

4A. Shuttle run with forward/backward running
Elapsed time 12-13 minutes
Purpose To increase dynamic stability of the ankle/knee/hip complex
Instructions Starting at the first cone, sprint forward to the second cone, run backward to the third cone, sprint forward to the fourth cone (etc.).

4B. Diagonal runs (3 passes)
Elapsed time 13-14 minutes
Purpose To encourage proper stabilisation of the outside foot
Instructions Face forward and run to the first cone on the left. Pivot off the left foot and run to the second cone. Now pivot off the right leg and continue onto the third cone. Make sure that the outside leg does not cave in. Keep a slight bend to the knee and make sure the knee stays over the ankle joint.

4C. Bounding run (40m)
Elapsed time 14-15 minutes
Purpose To increase hip flexion strength/increase power/speed
Instructions Starting on the near sideline, run to the far side with your knees up towards the chest. Bring your knees up high. Land on the ball of your foot with a slight bend at the knee and a straight hip. Increase the distance as this exercise gets easier.
Begin your normal training session.

Part 5. Stretching

It is important to warm up prior to stretching – never stretch a cold muscle. By doing the stretches outlined here, you can improve and maintain your range of motion, reduce stiffness in your joints, reduce post-exercise soreness, reduce the risk of injury and improve your overall mobility and performance. Cooling down your body is a must. It allows the muscles that have been working hard throughout the training session to elongate and deters the onset of muscle soreness. The cool-down should take approximately ten minutes. It should begin with a slow jog to allow your heart rate to come down before stretching and be followed by some gentle strength training exercises.

In general:
- Do a large muscle warm-up such as brisk walking for five to ten minutes before stretching.
- Do not bounce or jerk when you stretch. Gently stretch to a point of tension and hold.
- Hold the stretch for 30 seconds.
- Concentrate on lengthening the muscles when you are stretching.
- Breathe normally. Do not hold your breath.

5A. Calf stretch (30 seconds x 2 repetitions)
Elapsed time 1.5-2.5 minutes
Purpose To stretch the calf muscle of your lower leg
Instructions Stand leading with your right leg. Bend forward at the waist and place your hands on the ground (V formation). Keep your right knee slightly bent and your left leg straight. Make sure your left foot is flat on the ground. Do not bounce during the stretch. Hold for 30 seconds. Switch sides and repeat.

5B. Quadriceps stretch (30 seconds x 2 repetitions)
Elapsed time 2.5-3.5 minutes
Purpose To stretch the quadriceps muscle of the front of your thigh
Instructions Place your left hand on your partner's left shoulder. Reach back with your right hand and take hold of the front of your right ankle. Bring your heel to the buttock. Make sure your knee is pointing down towards the ground. Keep your right leg close to your left. Do not allow your knee to wing out to the side and do not bend at the waist. Hold for 30 seconds and switch sides.

5C. Hamstring stretch (30 seconds x 2 repetitions)
Elapsed time 3.5-4.5 minutes
Purpose To stretch the hamstring muscles of the back of your thigh
Instructions Sit on the ground with your right leg extended out in front of you. Bend your left knee and rest the bottom of your foot on your right inner thigh. With a straight back, try to bring your chest towards your right knee. Do not curve your back. If you can, reach down towards your toes and pull them up towards your head. Do not bounce. Hold for 30 seconds and repeat with the other leg.

5D. Inner thigh stretch (20 seconds x 3 repetitions)
Elapsed time 4.5-5.5 minutes
Purpose To elongate the muscles of your inner thigh (adductors)
Instructions Remain seated on the ground. Spread your legs evenly apart. Slowly lower yourself to the centre with a straight back. You need to feel a stretch in the inner thigh. Now reach towards the right with the right arm. Bring your left arm over your head and stretch over to the right. Hold the stretch and repeat on the opposite side.

5E. Hip flexor stretch (30 seconds x 2 repetitions)
Elapsed time 5.5-6.5 minutes
Purpose To elongate the hip flexors in the front of your thigh
Instructions Lunge forward, leading with your right leg. Drop your left knee down to the ground. Placing your hands on top of your right thigh, lean forward with your hips. The hips should be square with your shoulders. If possible, maintain your balance, lift your left ankle and pull your heel to your buttocks. Hold for 30 seconds and repeat on the other side.

Part 6. Alternative exercises – warm-down and cool-down

Cooling down your body is a must. It allows the muscles that have been working hard throughout the training session to elongate and deters the onset of muscle soreness. The cool-down should take approximately ten minutes. It should begin with a slow jog to allow your heart rate to come down before stretching and be followed by some gentle strength training exercises. We recommend two strengthening exercises (A and B). Finally, stretch your hamstrings, calves, inner thigh, quadriceps and lower back, as described above. In addition to these basic stretches, try the additional stretches to target three muscle groups that are often forgotten, as described under C, D and E. Make sure you have a bottle of water by your side during the cool-down and drink enough fluid.

6A. Bridging with alternating hip flexion
(30 repetitions x 2)
Purpose To strengthen the outer hip muscles (hip abductors, flexors) and buttocks
Instructions Lie on the ground with your knees bent and feet on the ground. Raise your buttocks up off the ground and squeeze. Now lift your right foot off the ground and make sure that your right hip does not dip down. Lower your right foot and now lift your left foot, making sure your left hip does not dip down. Repeat 30 times on each side. As you become stronger, place your feet on top of a ball and repeat the exercise.

6B. Abdominal crunches (30 repetitions x 2)
Purpose To strengthen your abdominals
Instructions Lie on the ground with your knees bent. Place your hands behind your head with your elbows out wide. Support your neck lightly with your fingers. Take a deep breath and slowly contract your abdominal muscles as you exhale. Repeat 30 times. Drop your legs off to the right side. Slowly crunch up with your elbows out wide. You should feel your oblique muscles working on the side of your waist. Repeat 30 times and switch to the other side.

6C. Single and double knee to chest
(30 seconds x 2 repetitions)
Purpose To elongate your lower back muscles
Instructions Lie on your back. Bring your right knee towards your chest and hug firmly. Keep your left leg straight in front of you. You should feel a stretch along your lower back and into your buttocks. Hold the stretch for 30 seconds and switch sides. Now bring both knees to the chest. If you feel any pain in the lower back, discontinue the stretch and inform your coach/trainer.

6D. Figure four piriformis stretch – supine
(30 seconds x 2 repetitions)
Purpose To elongate the rotators of the hip
Instructions Lie on your back and bend both of your knees. Fold your left ankle over your right knee. Place your hands behind your right thigh and pull your right knee to your chest. You should feel a good stretch in the left buttock and the side of the thigh. Hold for 30 seconds and repeat on the other side. If you experience lower back pain with this stretch, slowly lower your legs down and let your coach/trainer know.

6E. Seated butterfly stretch – seated
(30 seconds x 2 repetitions)
Purpose To elongate your inner thigh muscles (adductors)
Instructions Sit up, bringing your feet in so that the soles of your feet are touching. Gently place your elbows on your knees and slowly push down. You should feel a good stretch of the inner thigh. Hold this for 30 seconds and repeat two to three times.

Prevention of ankle sprains

Background

Prevention research shows that ankle sprains can be prevented by using semi-rigid or air-supported braces, especially in players with a previous of ankle sprain. It appears that taping is not so effective at preventing ankle sprains. A previously sprained ankle should be protected for months after the injury. An incompletely rehabilitated injury is predictive of another injury that can be more severe than the first one.

Proprioceptive and coordination training, using ankle discs, has also proved to be effective in reducing ankle sprains. Balance training and proprioceptive training can be done on the ground or using a wobble board or ankle discs. These are boards with an unstable platform that the player stands on and tries to maintain balance.

Description of balance board exercises for players

Important:

- Do not begin balance board routines until your doctor has given you permission.
- Before starting any of the balance board routines, warm up for about ten minutes with light jogging, stretching, and range-of-motion activities for the trunk, lower back, hips, quadriceps, hamstrings, calves, Achilles tendons, shins and feet. When doing the balance board exercises, try to maintain an upright posture and use smooth, controlled movements. For the first few weeks, work on developing coordination and technique and try not to do many repetitions. As your skill improves, increase your movement speed, while keeping good balance and posture.
- Perform the exercises when you are rested and not tired. Fatigue can increase your risk of injury.
- The "ready position" for most athletic activities includes a little knee and hip flexion, ensuring that your toes are visible to you. So perform the balance board exercises with your knee(s) slightly flexed.
- When beginning a balance programme, stabilise yourself by placing the toes of the opposite (non-weight bearing) foot on the ground behind you during single-leg exercises. Using your hands for stability defeats the purpose of the balance board activities.
- Increase the difficulty of any balance board exercise by holding dumbbells in your hands, carrying a medicine ball, or by closing your eyes.

Beginners exercises

These first two exercises develop balance and coordination of the entire body. They also enhance the so-called "grip strength" of the feet and toes on the board, which will allow for progression into more difficult balance board exercises.

The two-leg stand and balance with instability from side to side: simply hold your position for 30 seconds without letting the edges of the board touch the ground.

The two-leg stand and balance with instability from front to back: complete the exercise by simply holding a balance position for 30 seconds, without letting the edges of the board touch the ground.

Side-to-side edge taps: slowly and deliberately allow the outside edges of the platform to touch or "tap" the ground (left edge, then right edge, left, right, etc.) for about one minute. This range-of-motion and strength exercise should be done under full control, without rapid swings of the board from side to side.

Front-to-back edge taps: slowly and deliberately allow the front and back edges of the platform to touch or "tap" the ground (front edge, then back edge, front, back, etc.) for approximately one minute. Once again, perform this exercise with smooth, rhythmic movements, without sudden jerks of the platform.

Beginners exercise

Beginners exercise

Beginners exercise

Beginners exercise

Intermediate exercises

Try the beginners exercises on one foot. These one-footed exercises may be too difficult to perform without losing your balance, so simply place the toe of your opposite (non-weight bearing) foot on the ground slightly behind the balance board. Once you are comfortable doing this with a little support, try them without touching the ground with the opposite toe.

Intermediate exercise

Advanced exercises

The following advanced balance board exercises develop coordination, balance, strength and mobility in the muscles of the feet, ankles, legs, hips and trunk. The advanced exercises require a high degree of body awareness and the skills needed to do them properly require repeated exposure so it is best to do them at least four to five times a week.

Side-to-side edge taps: place one foot directly in the middle of the platform using a board that is unstable in all directions. Slowly and deliberately allow the lateral edges of the platform to touch or "tap" the ground (left edge, right edge, left, right, etc.) for about one minute. Maintain full control at all times, avoiding rapid, uncontrolled motions of the balance board. If the exercise is too difficult at first, place the toes of your other foot on the ground behind the wobble board for better balance. Repeat the exercise on the opposite foot.

Advanced exercise

Front-to-back edge taps: these are the same, except that you are allowing the front edge of the balance board to touch the floor, then the back edge, etc. Do it for a minute then repeat with the other foot.

Advanced exercise

Edge circles: place your foot in the centre of the wobble board. Slowly and deliberately allow an edge of the platform to touch the floor then rotate the touching in a clockwise fashion, keeping the platform in contact with the floor at all times. The motion should be slow and controlled and for one minute without stopping. If too hard, place the toes of the opposite foot on the floor. Repeat with the other foot.

Advanced exercise

– Anti-clockwise edge circles: this is the same as the previous exercise, but going in an anti-clockwise direction.

Prevention of hamstring strains

Introduction

Hamstring strain injuries usually occur during maximum sprinting, when resisting knee extension, or at foot strike, when the muscle is close to its maximum length and eccentric power generation is at its maximum. An important risk factor is poor warm-up. Two other (less well-documented) risk factors are reduced range of motion and poor strength. In some players, a previous strain that caused scar tissue may result in reduced range of motion. If the hamstring muscles are weak in relation to the quadriceps muscles, the risk of hamstring injuries is also increased.

Description of exercises to prevent hamstring injuries – instructions for players

1) Warm-up exercises

Hamstring stretching: the goal is to prepare for maximum effort.

During warm-up before every single training session and match, especially before sprinting or shooting exercises, stretch your hamstring muscles.

Exercise: Use support, preferably from another partner or a firm object (stool, tree). Allow your ankle to relax. Press your heel against the ground for 5-10 seconds, to activate the hamstring muscles, then relax and use your hand to straighten out your knee. If necessary, bend forward slightly at your hip until you feel the stretch in the hamstrings, but be sure to keep your back straight. Stretch each leg three times.

2) Flexibility training

Hamstring stretching: the goal is to increase range of motion in the hip joint.

If your range of motion is limited, stretch your hamstring muscles regularly for 5-10 minutes at a time, at least three times a week during the preseason period and twice a week during the competitive season.

Exercise: Your partner lifts your leg with the knee slightly bent, until you feel stretching on the posterior side of your thigh. Hold this position for a while before actively pressing your leg against your partner's shoulder, so that your knee straightens. Hold for ten seconds. Then relax completely while your partner carefully stretches, by leaning forward. Hold that position for at least 45 seconds. It is important to relax your ankle, so you stretch the posterior side of the thigh, and not the lower leg. Stretch each leg three times.

3) Strength training

Eccentric strength training: the goal is to hold the descent as long as possible, to achieve maximum eccentric strength of the hamstrings. Perform eccentric strength training for the hamstring group regularly, at least three times a week during the preseason period and twice a week during the competitive season.

Exercise: The resistance exercises are partner exercises, in which your partner stabilises your legs. Lean forward in a smooth movement, keep your back and hips extended, and work at resisting the forward fall with your hamstring muscle as long as possible until you land on your hands. Go all the way down so that your chest touches the

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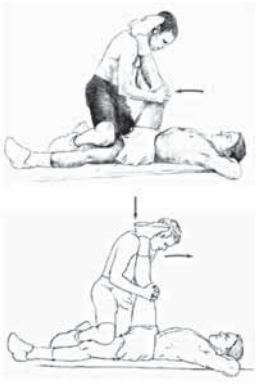
1) Warm-up



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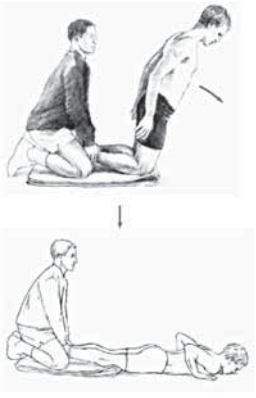
2) Flexibility



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3) Strength

ground and push off immediately with your arms until the hamstring muscles can take over and you can straighten up into a kneeling position again (this second part of the exercise is for individuals familiar with this type of training).

2.2.4 Protective equipment

Historically, various forms of protective equipment have been used to prevent injuries in sports. Examples of protective equipment from different sports include helmets, shoulder pads, mouth guards, etc. In football, the most commonly used protective equipment includes the following:

- Tape
- Braces (orthoses)
- Shin guards
- Mouth guards
- Padding
- Goalkeeper gloves

Taping and bracing

Taping and bracing are used ostensibly to protect joints from undesired, potentially harmful motion, yet at the same time allow desired motion. The two main indications for the use of tape and braces are:

Prevention – where taping or bracing is used as a measure to prevent, for example, ankle sprains.

Rehabilitation – where taping or bracing is used as a protective mechanism during the healing and rehabilitation phase of, for example, knee ligament injuries.

The obvious question is: does taping and bracing protect the joint from injury? To examine this, a randomised clinical study assessed the effect of a semi-rigid ankle stabiliser on the incidence of ankle sprains during one playing season. Senior male football players from South Africa were asked to participate in the study and they were randomly assigned to a control group (no treatment) or an intervention group who wore a semi-rigid orthosis (brace) during practice and games. The randomisation was stratified according to previous injury status and this resulted in four groups: two control groups (one with a history of previous ankle sprain and one with no history) and two intervention groups (one with a history of previous ankle sprain and one with no history). The results showed that wearing a brace significantly reduced the incidence of ankle sprains among players with a previous history of ankle sprains. The incidence was 0.46 per 1,000 playing hours among those who wore a brace and 1.16 per 1,000 playing hours among those who did not. However, among players with no previous history of ankle sprains there was no difference in the incidence of ankle injuries between those who wore orthoses and those who did not. Thus, a semi-rigid orthosis significantly reduced the incidence of recurrent ankle sprains in football players.

Similar results on ankle sprains were obtained in studies on male football players in Sweden and female college players in the United States. Thus, it seems reasonable to recommend taping or bracing in conjunction with a comprehensive rehabilitation programme to decrease the risk of recurrent ankle injuries. Similar results were obtained through a ten-week balance training programme. A reasonable strategy, therefore, is to use taping or a brace after an ankle sprain, at least until such a programme has been completed. As mentioned, note that there seems to be no protective effect of taping or bracing in healthy players, i.e. players with no history of a previous ankle sprain.

Whether knee braces (Figures 2.2.4.1 and 2.2.4.2) can prevent knee injuries is less clear. The use of a knee orthosis has been shown to prevent contact injuries to the knee in American football, particularly for medial and lateral collateral ligament injuries. However, they have not been effective in preventing anterior cruciate ligament injuries. Notably, their effect in primary prevention has not been examined in football, perhaps because knee bracing could be expected to interfere with performance through reduced skill and ball control. Nevertheless, knee braces

Figure 2.2.4.1 Aircast

Figure 2.2.4.2 Knee brace

play a very important role in protecting the joint during the rehabilitation phase after ligament injuries, while there is significant joint instability.

How tape and orthoses work is uncertain, but they may simply enhance sensorimotor control of the joint. This view is corroborated by the fact that their effect appears to be limited to players with previous injury, where postural control, position sense and postural reflexes seem to be reduced, and that orthoses do not seem to restrict motion enough to substantiate their prophylactic effect. If the protective effect were mechanical, one would expect an effect in healthy athletes as well. However, it may also be that the mechanism by which tape and orthoses work is by simply guiding the foot. In other words, the external ankle support may help to ensure that the player lands and plants with the knee and foot in the proper position.

Bracing is generally seen as being more comfortable than tape and more cost-effective with long-term use. However, in football – where knee and foot control is essential – some players may resist using braces and prefer tape. Most studies indicate that appropriately applied tape or orthoses do not adversely affect physical performance (measured as jumping ability or running speed).

There are many different tapes and bandages available for use by players. However, it is generally believed that only adhesive, non-stretch (rigid) tape is appropriate. Good tape should have excellent adhesive properties and be strong, non-irritant and easily torn by the therapist. Elastic tape is inappropriate for restricting motion, but can be used effectively in conjunction with rigid tape for protecting certain joints, e.g. the knee and thumb.

Custom-made braces can be formed by moulding thermoplastic material over the affected part. Such splints are commonly used for the hand and wrist and may make it possible for a player to train and even compete with minor undisplaced fractures, e.g. finger or metacarpal fractures. A similar approach with custom-fitted braces can be used for facial fractures, e.g. nose or maxillary fractures.

Shin guards

Historically, fractures of the lower leg (tibia fractures) have been a significant concern in football, especially among youth and adolescent players. Such fractures are usually caused by a direct kick to the anterior aspect of the shin.

According to FIFA regulations, shin guards (pads) are mandatory at all levels of competition to protect players from lower leg fractures and contusions. However, it is the responsibility of coaches and players to ensure that shin guards (Figure 2.2.4.3) are also worn during training and unofficial matches. This is particularly important among children and youngsters, who are more vulnerable to fractures because of an immature skeleton.

A multitude of different makes and models exist and FIFA has established a homologation system to ensure that shin pads that are marketed are properly designed. However, the key factor is that shin guards must be individually fitted for each player. They must be long enough to cover the entire tibia – not exposing an uncovered area below or above the padding. Also, they should be wide enough to afford as much protection as possible against kicks hitting the side of the shin.

The problem of shin pads that are too small is especially prevalent among youth and adolescents where they can outgrow their equipment during the course of one season. To save cost to players, clubs could establish equipment exchange programmes. This is especially important for safety equipment like shin pads.

Whenever tibia fractures occur, they usually result from a high impact kick to the leg. A limitation with most shin guards is that they do not protect against blows to the back of the leg. However, custom-fitted shin guards made from carbon fibre exist that claim to also protect the back of the leg. Unfortunately, these are very expensive.

Figure 2.2.4.3 Shin guards

Mouth guards

Dental injuries are not very frequent in football, but the treatment may be very costly. Dental injuries occur mainly through face-to-head or face-to-elbow collisions in heading duels, or from being kicked in the mouth when bending down to head the ball.

Mouth guards are commonly used in many sports, from individual sports like boxing to team sports such as rugby. Although they are not frequently used by football players, they do provide efficient protection against dental injuries. Players who have had extensive dental repairs may, depending on the type of work done, be at greater risk of re-injury and should consider wearing mouth guards during training and matches. Players with orthodontic braces on their teeth should wear mouth guards to prevent lacerations.

Mouth guards must be individually fitted and range from expensive models fitted by orthodontists to cheaper self-fit models that can be moulded by the player himself.

Padding

Goalkeepers are more exposed to contusions, abrasions and cuts than other players because of their role in the game – diving to save the ball. To protect their hips, elbows and shoulders, goalkeeper uniforms should be adequately padded (Figure 2.2.4.4). Kneepads are not regularly worn by goalkeepers at elite level during matches, probably because goalkeepers at this level feel that such padding would negatively affect their match performance. Nevertheless, the same goalkeepers normally wear long pants with extensive padding during training and it is recommended that goalkeepers at youth level wear long pants at all times during training and competition.

Goalkeepers should also wear gloves to protect their fingers from sprains, contusions and abrasions and there is a number of different makes and models available at a wide range of prices.

A final piece of equipment worn by male players is an athletic supporter, which is used to protect the groin area. Goalkeepers should, in addition, use a hard cup to protect them when hit in the groin by shots.

Figure 2.2.4.4 Padded goalkeeper shorts

2.3 Prevention of sudden cardiac death

2.3.1 Background and epidemiology

With its undoubted advantages in terms of mental, physical and social wellbeing and self-esteem, regular physical exercise is associated with well-documented health benefits. Epidemiological studies suggest that physical activity protects against cardiovascular, endocrinological and even psychiatric diseases. But, depending on the extent of physical activity, the type of sport and the individual underlying physical condition, athletes – including football players – are also exposed to a greater risk of injury, certain diseases and sudden death. The vast majority of sudden deaths related to physical activity are due to cardiovascular disease (>90%). In a landmark Italian survey, the overall incidence of sudden death from all causes was 2.3 (2.6 in males and 1.1 in females) in 100,000 athletes per year as compared to the inactive population. The overall incidence of sudden death from cardiovascular diseases was 2.1 in 100,000 athletes per year. It is worth noting that athletes had a 2.8 relative risk (RR) of sudden cardiac death (SCD) as compared to a 1.7 RR of non-cardiovascular sudden death.

However, the exact incidence of SCD in athletes is not known with certainty but varies from 0.5 to 2.3:100,000 per year in competitive athletes under the age of 35. The reasons for these differences in mortality rates include ethnic and genetic factors and different underlying pathological substrates, as well as differences in age, performance level and intensity. In fact, non-competitive, recreational athletes carry a risk of SCD related to physical activity which is about 2.5 times lower as compared to competitive athletes. There is no football-specific data.

SCD in athletes shows a clear predilection for the male gender. Although male athletes have a higher participation rate in competitive sports and generally exercise with greater intensity and ambition than their female counterparts, male gender itself was reported to be a risk factor for sports-related SCD. This was attributed to the greater prevalence and/or phenotypic expression of cardiac diseases placing the individual at risk of arrhythmic cardiac arrest, such as cardiomyopathies and premature coronary artery disease.

An ethnic preponderance of SCD is supported by the fact that in the United States, the single most common cause of SCD in athletes is hypertrophic cardiomyopathy (HCM), which occurs at a disproportionately high rate in African-Americans.

In Italy, where systematic pre-participation medical assessment of competitive athletes is required by law, more than 60% of disqualifications from competitive sports are due to cardiovascular findings, which corresponds to 1.8% of all assessed athletes. Other causes such as orthopaedic (12.7%), ophthalmological (12.3%), neurological (4.3%), respiratory and endocrinological conditions are much less common.

The prevention of SCD should therefore be the major objective of pre-competition medical assessment. As there are major differences in the aetiology and prognosis of SCD between *competitive* and *non-competitive* athletes, these terms need to be clearly defined. The Bethesda Conference has defined competitive athletes as follows:

"*Competitive athletes* are individuals of young and adult age, either amateur or professional, who are engaged in exercise training on a regular basis and participate in official sports competition. Official sports competition (local, regional, national, or international) is defined as an organized team or individual sports event that, placing a high premium on athletic excellence and achievement, is organized and scheduled in the agenda of a recognized Athletic Association. A characteristic of competitive sports, regardless of the level of achievement, is the strong proclivity for participants to exert themselves physically until their limits and improve performance."

Most of the studies concerning prevention of SCD have targeted young competitive athletes under the age of 35, which is commonly seen as a "cut-off" value.

This age group mainly corresponds to football players participating in national and international competitions and this chapter will therefore concentrate on this subgroup.

2.3.2 Causes of sudden cardiac arrest and sudden cardiac death

What makes physical activity potentially harmful? And which individuals may be prone to such harm? With the exception of commotio cordis, there is always a ***trigger*** (physical activity) that acts on a pre-existing ***substrate*** or underlying disorder. Thus, the major target of pre-competition cardiac assessment should be to seek to identify such underlying conditions in an individual.

In addition, the physician needs to consider secondary factors associated with sports activity such as electrolyte disturbances (particularly hypokalaemia and hypomagnesaemia), drug abuse, (over)activity of the autonomic nervous system and psychosocial factors which may trigger sudden cardiac arrest (SCA) or even SCD.

The primary causes of SCA and SCD are lethal arrhythmias such as ventricular tachyarrhythmias, in particular ventricular fibrillation, while bradyarrhythmias or asystoles are far less common. The latter are often due to the extension of a pathological process into the conduction system, causing complete heart block without a reliable escape focus. In addition, syncope may occur during exercise in certain congenital lesions with right-to-left shunts and cyanosis, but also due to dissections of the great vessels, particularly in patients with **Marfan syndrome**, leading to pericardial tamponade or deleterious major internal bleeding.

The underlying cardiac condition, either structural or not, that leads to the fatal event depends on the age of the athlete. In major surveys, **coronary heart disease** was by far (> 80%) the most common cause of SCD associated with sports in older athletes (> age of 35). Young and adolescent competitive athletes who died suddenly were mostly affected by cardiomyopathies (e.g. hypertrophic cardiomyopathy (HCM) and arrhythmogenic right ventricular cardiomyopathy (ARVC), among others), premature coronary artery disease, congenital coronary anomalies and hereditary conduction anomalies (such as long-QT-syndrome, Wolff-Parkinson-White (WPW) syndrome, Brugada syndrome, and catecholaminergic polymorphic ventricular tachycardia).

In studies in the United States, where 12-lead resting ECG is not part of the standard pre-competition medical assessment, HCM was the single most common cause of SCD in athletes, responsible for approximately one third of cases, followed by congenital coronary artery anomalies, ARVC and myocarditis (Figure 2.3.2.1). Conversely, more recent Italian studies have found ARVC to be the most common cause of SCD, whereas HCM was less common (Figure 2.3.2.2). This was attributed to the fact that the

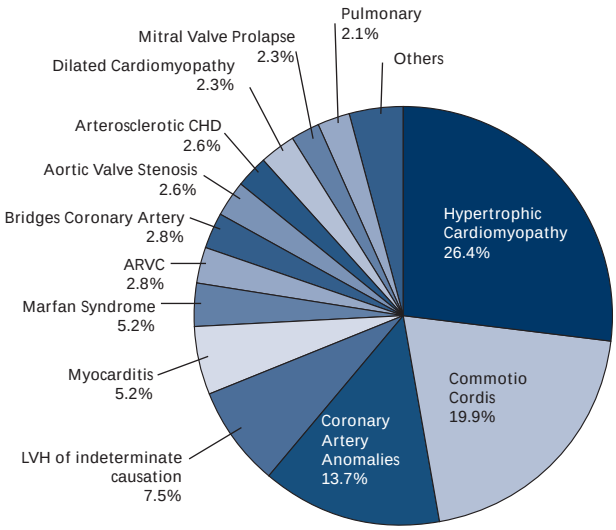


Figure 2.3.2.1: Causes of sudden death in 387 young athletes (Maron BJ. Sudden death in young athletes. N Engl J Med. 2003; 349811: 1064–75.)

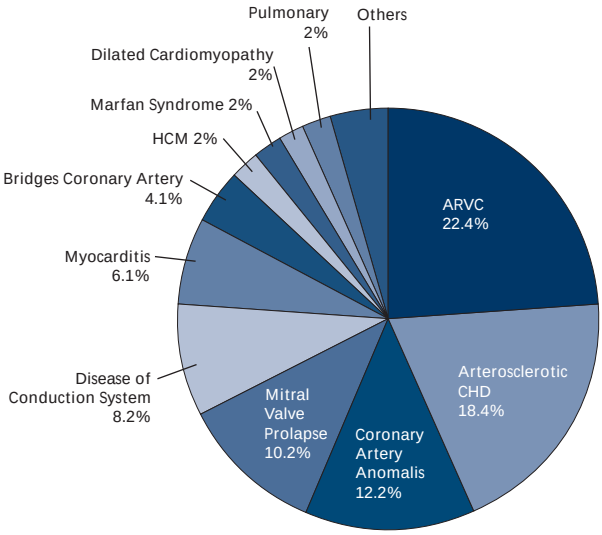


Figure 2.3.2.2 Causes of sudden death in athletes and non-athletes 35 years of age or less in the Veneto region of Italy 1979 to 1996 (Corrado D, Basso C, Schiavon M, et al. Screening for hypertrophic cardiomyopathy in young athletes. N Engl J Med. 1998; 339(6): 364-9.)

cohort of athletes examined in Italy consisted of much fewer black athletes, and that all subjects screened had undergone a 12-lead resting ECG. ECG changes due to HCM can be found in the majority of patients with this entity.

HCM is a relatively common genetic cardiac disease with a heterogeneous clinical, morphological and genetic expression and an incidence of 1:500 in the general population. Positive findings in the player's history (previous recurrent syncope/pre-syncope, or even cardiac arrest, sustained ventricular tachycardia or frequent non-sustained ventricular tachycardia and a positive family history of SCD) should raise suspicion of the disease and may indicate an increased risk of SCD. Clinical findings which are more or less specific can substantiate the suspicion, and include a bifid carotid pulse, a fourth heart sound (S4), an abnormal decrease in blood pressure during exercise and a harsh, crescendo-decrescendo systolic murmur varying in duration and intensity depending on left ventricular filling (see also 2.1.3). The vast majority of patients suffering from HCM also show electrocardiographic evidence of the disease, even though changes are not pathognomonic, such as: negative T-waves in precordial leads, Q waves in inferolateral leads, evidence of right or left ventricular enlargement, left axis deviation and a short PR interval with slurred upstroke (Figure 2.3.2.3). The hypertrophied and non-dilated left ventricle caused by a disorganised myocardial architecture can be detected by echocardiography but may be difficult to distinguish from physiological changes due to training (Figure 2.3.2.4, 2.1.3). HCM is potentially harmful even without an obstruction of the left ventricular outflow tract, though the risk of SCD might be higher in obstructive hypertrophic cardiomyopathy, based on a resting gradient greater than 30mmHg and provocable gradients greater than 50mmHg. In HCM, SCD is due to an electrically unstable and unpredictable myocardial substrate with re-entrant ventricular tachyarrhythmia. In obstructive hypertrophic cardiomyopathy, syncope during exertion may also occur due to a massive outflow tract gradient.

The hallmark of **ARVC** is a fibrofatty infiltration of the right ventricular myocardium predominantly involving the free wall. In rare cases, the left ventricle and in particular the apex may also be involved. It is an inherited progressive disease which may eventually lead to heart failure. However, lethal arrhythmias are quite often the first clinical manifestation of the disease. There may be predictors in a player's history: a positive family history as well as unspecific symptoms like exercise-induced palpitations, pre-syncope, and/or syncope (consistent with the catecholamine-sensitive nature of many of the associated tachyarrhythmias as well as the wall stretch observed in the right heart in response to the increased venous return occurring with exercise) might alert the physician to the possibility of underlying heart disease. Findings in the 12-lead resting ECG, primarily localised to the right precordial leads, include a QRS duration in V1 of more than 110 milliseconds, an epsilon wave due to delayed activation across the right ventricular myocardium in leads V1 or V2, and T-wave inversion in the right precordial leads (Figure 2.3.2.5 see also 2.1.3). Although echocardiography is the standard imaging technique for ARVC, in the early stages the detection of the typical right ventricular structural changes (dilatation, aneurysm, fibrofatty replacement of myocardium) may not

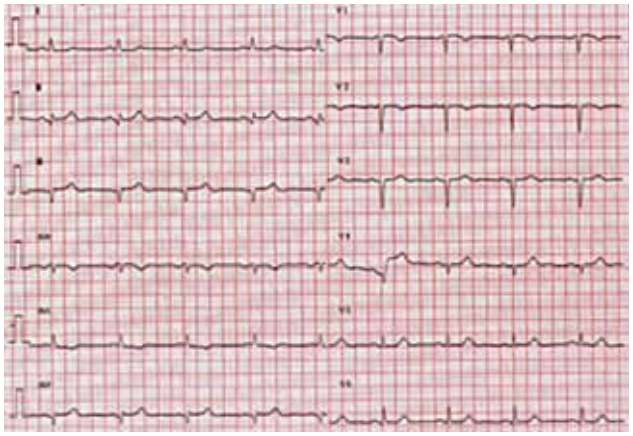


Figure 2.3.2.3 Some of the typical ECG changes in HCM: negative T-waves in precordial leads, Q waves in inferolateral leads, left axis deviation and short PR interval with slurred upstroke. (www.escardio.org)

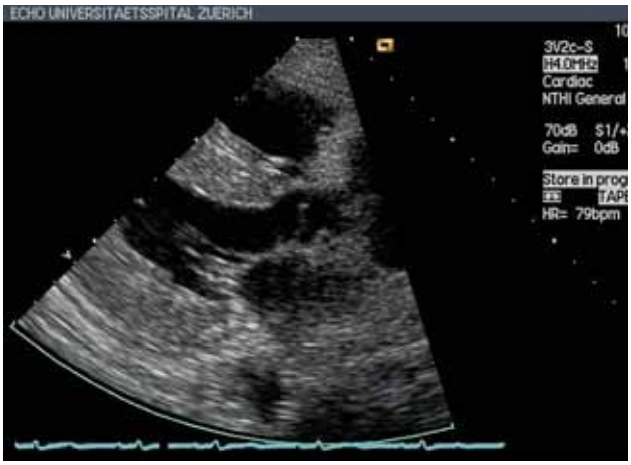


Figure 2.3.2.4 A massively hypertrophied left ventricle in a patient with hypertrophic cardiomyopathy. (Source: University Hospital Zurich/Switzerland)

yet be possible and an MRI may be required to confirm the diagnosis. Today's increasing numbers of disqualifications due to ARVC are partly due to under-diagnosis in the past.

Congenital coronary artery abnormalities are further conditions associated with SCD in athletes. The most common anomalies related to SCD are the origin of the left main coronary artery from the right sinus of Valsalva and the origin of the right coronary artery (RCA) from the left coronary sinus. Myocardial ischaemia secondary to an exaggeration of the sharp angle in the aberrant origin occurring with exercise, especially as the artery traverses an expanded aorta and pulmonary arterial trunk, may lead to anginal chest pain, syncope or pre-syncope. But unfortunately, SCD is often the first clinical manifestation. The possibility of detecting either premature coronary atherosclerosis or anomalous origin of the coronary artery (especially RCA that origins from the left sinus) is limited by the low sensitivity/specificity of baseline changes in resting and stress test ECGs. It is estimated that approximately one quarter of the young athletes who have died from coronary artery abnormalities had warning symptoms and/or ECG abnormalities at the pre-competition medical assessment that could have raised suspicion of cardiac disease.

Various non-invasive stress tests are available to detect exercise-related myocardial ischaemia. In order to detect anomalous coronary anatomy, coronary calcification or stenosis, non-invasive magnetic resonance coronary angiography (MRCA) and particularly coronary angiography by computed tomography (CTCA) are increasingly used. Coronary angiography is the gold standard for evaluation of the coronary arteries including their origin and course, but it is only used in athletes if a diagnosis has been made and therapeutic measures are considered.

Although the risks of SCD are higher in patients with structural heart disease, SCD also occurs in individuals with apparently normal hearts. So-called channelopathies, inherited arrhythmia syndromes due to mutations in genes coding for ion-channels within the conduction system, are of increasing importance. The **congenital long QT syndrome** (LQTS) can be diagnosed on the basis of a prolonged corrected QT (QTC) interval on the ECG. The QTC interval means a QT interval corrected for heart rate, because the QT interval is longer at slower rates and shorter at faster rates (Figure 2.3.2.6). This syndrome has been associated with a significant risk of SCD due to characteristic life-threatening cardiac arrhythmias, known as *torsade de pointes*. Exercise appears to increase the risk of SCD in some of the LQTS subtypes, particularly in LQTS 1. However, the QTc varies in response to a number of factors, such as autonomic state, electrolyte imbalance, drug intake, and diurnal changes. Thus, the sensitivity of measuring the QTc on a single ECG is limited. Furthermore, Bazett's formula, the most commonly used formula for heart-rate correction of the QT interval (QTc = QT interval (in milliseconds÷square root of the RR interval (in seconds)), is inaccurate at heart rate extremes and results in over-correcting at high rates and under-correcting at low ones. The normal range for the rate-corrected QT interval in children and adolescents is 0.37 to 0.44 seconds, <0.44 seconds in adult males and <0.46 seconds in women. Two clinical phenotypes have been described in congenital LQTS: the more common autosomal dominant form (Romano-Ward syndrome) has a purely cardiac

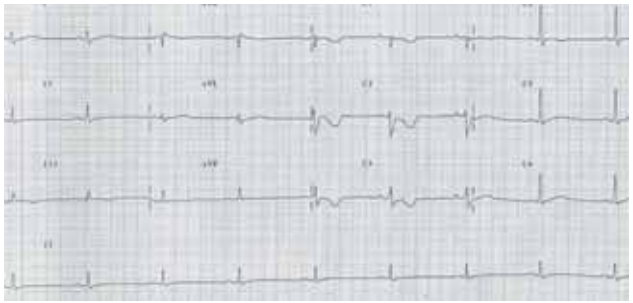


Figure 2.3.2.5 Electrocardiographic changes in arrhythmogenic right ventricular cardiomyopathy (ARVC) include inverted T-waves in the right precordial leads beyond V1 in the absence of a right bundle branch block, epsilon waves due to ventricular late potentials and QRS duration longer in the right (leads V1, V2 and V3) than in the left (V4, V5, V6) precordial leads. (Source: University Hospital Zurich/Switzerland)

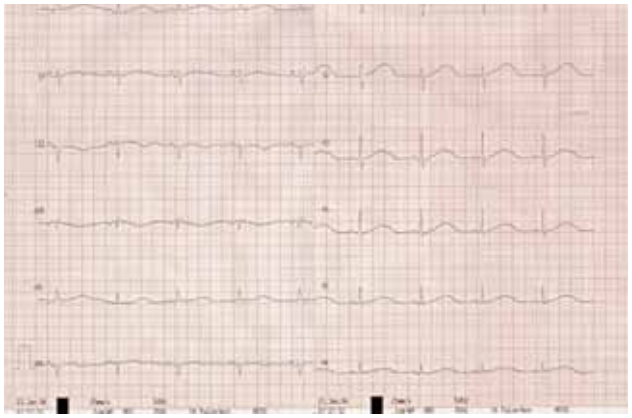


Figure 2.3.2.6 Patient with a prolonged QT interval (Source: University Hospital Zurich).

phenotype, whereas the autosomal recessive form (Jervell and Lange-Nielsen syndrome) is associated with LQTS and sensorineural deafness.

Brugada syndrome is a hereditary syndrome which shows a marked predominance in males and is characterised by alterations of the cardiac sodium channel which lead to electrocardiographic changes in leads V1 to V3 in the 12-lead resting ECG. There are three different types with varying ST-segment changes resembling right bundle branch block (RBBB; Table 2.3.2.1). Arrhythmic events (mostly polymorphic ventricular tachycardia) occur more commonly at night than during the day and more commonly during sleep than while awake. Thus, SCD in Brugada patients is not usually related to exercise. Characteristic ECG abnormalities may become manifest after application of a sodium channel blocker, thereby identifying those at risk. The Brugada ECG pattern may be an early manifestation of ARVC.

Patients with **catecholaminergic polymorphic ventricular tachycardia** typically present with polymorphic or bidirectional ventricular tachycardia (also seen with digitalis toxicity) or ventricular fibrillation when emotionally or physically stressed, with syncope or SCA/SCD often being the first manifestation of the disease. Arrhythmic events during swimming, previously considered to be specific to LQTS type 1, have also been described with this disorder.

Commotio cordis, in which SCA or SCD is precipitated by a direct trauma to the precordium, is a different issue and must be dealt with by making appropriate adjustments to the rules of the game and equipment. Commotio cordis occurs as a result of a blunt, non-penetrating blow to the chest (e.g. sports projectiles, such as baseballs and hockey pucks, or punches) unassociated with structural injury to the ribs, sternum, or heart that may elicit ventricular fibrillation. It is most common in children and adolescents (mean age 13), since these age groups characteristically have compliant chest walls that appear to facilitate the transmission of the energy from the chest blow to the myocardium. Animal experiments attempting to replicate commotio cordis have shown that a blow must hit the chest wall directly over the heart and occur within 15 to 30 milliseconds before the T-wave peak (about 1% of the duration of the cardiac cycle) which represents the vulnerable phase during repolarisation.

Prognosis of SCA

Depending on the characteristics of the underlying cardiac condition and the circumstances of rescue, the survival rate of SCA is still very poor (11-16%). The success of resuscitation is highly dependent on the availability of an automatic external defibrillator (AED) to primary responders. Rapid conversion into sinus rhythm within minutes is crucial for the clinical outcome after SCA.

2.3.3 Primary prevention

The vast majority of players who die suddenly of cardiovascular disease do so during either competition or training. A general pre-participation medical assessment in football therefore seems an indispensable tool that should

	Type I	Type II	Type III
J wave amplitude	>= 2mm	>= 2mm	>= 2mm
T wave	negative	positive or biphasis	positive
ST-T configuration	coved type	saddleback	saddleback
ST segment (terminal portion)	gradually descending	elevated >= 1mm	elevated < 1mm

Table 2.3.2.1 ST segment abnormalities in the different types of Brugada syndrome

ideally identify any unknown risk in a player.

However, as mentioned previously, there is no consensus on the ideal pre-participation medical assessment for every athlete in all sports. For example, **medical history** (including personal and family history) and a focused **clinical examination** are widely accepted as the basis and cornerstone of every such assessment. It has been suggested that a proper medical history alone may identify up to 75% of problems that affect athletes. But whereas guidelines from the European Society of Cardiology (ESC) and the International Olympic Committee (IOC) recommend a **12-lead resting ECG** in addition to the player's history and clinical examination, American Heart Association (AHA) guidelines forego the ECG in "first-line" primary screening.

The "European modality" is mostly based on large Italian surveys in which it proved to be more sensitive for the detection of athletes at risk of SCA/SCD than the US protocol. In fact, the "European modality" showed a 77% greater capacity for detecting HCM as the most common cause of SCD and is expected to result in a corresponding additional number of lives saved (Figure 2.3.2.7).

Resting ECG

The 12-lead resting ECG shows pathological findings in more than 90% of patients with HCM as the major cause of SCD. In addition, the 12-lead ECG may detect or raise clinical suspicion of other "potentially" predisposing conditions, such as ARVC, dilated cardiomyopathy or channelopathies like the short QT- or long QT-syndrome, Brugada syndrome, WPW syndrome or Lenègre disease. Overall, these conditions, including HCM, account for up to 60% of SCDs in young competitive athletes. A major disadvantage of the ECG as a tool in screening is its relatively low specificity as a screening test in athletic populations mainly because of the high frequency of ECG

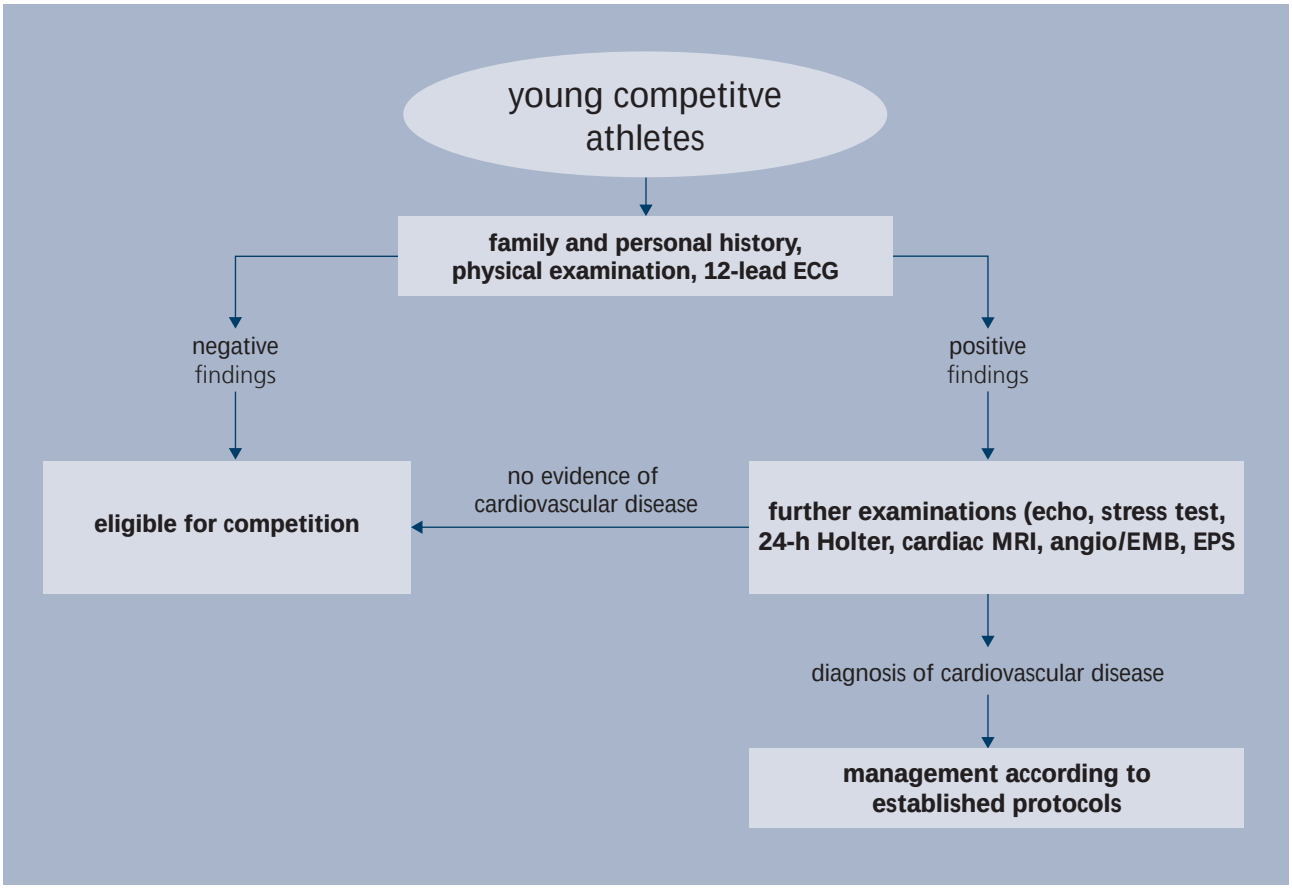


Figure 2.3.2.7: The "European modality" in cardiological screening of athletes (Corrado D, Pelliccia A, Bjornstad HH, et al. Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol. Consensus Statement of the Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. Eur Heart J. 2005; 26(5): 516–24.)

alterations associated with normal physiological adaptations in the trained athlete's heart. However, newer studies indicate that a number of formerly known "physiological" ECG alterations (e.g. ectopic ventricular beats or early repolarisation) may constitute a risk profile of their own. Most importantly, ECG abnormalities are crucial for the selection of athletes for further testing, in particular cardiac imaging that may lead to a final diagnosis.

Transthoracic echocardiography

The feasibility and cost-effectiveness of echocardiography in the pre-participation medical assessment of young athletes has been questioned, and its use in addition to the "first-line" screening tools has of late been considered not to significantly improve the efficacy in identifying HCM. However, echocardiography remains the gold standard in the diagnosis and follow-up of subjects with HCM and other relevant abnormalities responsible for SCD in young athletes, such as valvular heart disease (e.g. aortic stenosis and mitral valve prolapse), aortic root dilatation associated with Marfan syndrome, multifactorial dilated cardiomyopathy and other forms of left ventricular dysfunction, as well as, with certain limitations (see above), ARVC. Its selective use in those players with suspicious findings in history, clinical examination and/or ECG abnormalities is, however, generally indicated. Depending on resources, competition level and objectives, as well as in pilot studies to obtain specific data, the use of echocardiography might well be indicated. The debate on the value of this diagnostic tool in the primary prevention of SCD in sports is ongoing.

Exercise stress tests are not recommended as a primary diagnostic tool in pre-competition medical assessment, but may provide further information on specific clinical questions (see. 2.1.3).

Further diagnostic tools such as cardiac magnetic resonance imaging or computed tomography, ambulatory ECG, transesophageal echocardiography or coronary arteriography are currently only used to further investigate and clarify unclear clinical findings.

In an ideal world, screening by medical assessment should start at the beginning of competitive player activity. The screening should then be repeated on a regular basis every one to two years for the timely identification of diseases which are only detectable after progression over time. Once a significant pathology has been identified, adequate risk assessment and therapy have to be implemented and recommendations for physical activity following established guidelines (e.g. ESC, Bethesda) have to be given. However, the organisational, logistical, legal and not least financial issues and conditions in football worldwide might in fact not be in favour of pursuing this ideal strategy in all countries, and football has to investigate alternative feasible ways to protect players globally as much as possible.

At the same time, any declaration of ineligibility for football may represent a personal disaster for a talented or ambitious player. The higher the level of competition and the more parties and interests involved, the more complex decisions on eligibility become. If unexpected and based on findings in the ECG or echo during pre-competition medical assessment in an otherwise asymptomatic player, it can be postulated that the declaration of ineligibility will be met with a lack of understanding. Careful decision-making based on official and widely accepted recommendations developed by consensus groups is therefore indispensable (e.g. ESC, Bethesda Conferences (16, 26 and 36); Italian Committee for Sports Eligibility (COCIS)).

Besides the predisposing anomalies that have been discussed in detail in this chapter, other cardiovascular findings such as rhythm and conduction abnormalities, hypertension, congenital and rheumatic heart diseases, and peri-/myocarditis may also lead to non-eligibility for competitive sports. These conditions are also discussed in the consensus recommendations.

2.4 Special target groups

2.4.1 Female players – the female athlete triad

The female triad is a well-known phenomenon and frequently observed in sports like gymnastics or ballet, where a thin appearance is mandatory. However, recent findings show that football players, though less often than athletes in other sports, are also dieting and suffering from eating disorders, menstrual dysfunction and stress fractures. Therefore, team physicians should have a basic knowledge of how to prevent the triad and be aware of the warning signs.

The female athlete triad (or "the triad") refers to three interrelated conditions:

- disordered eating
- menstrual dysfunction
- osteopaenia/osteoporosis

A player may suffer from one, two or all three parts of the triad. Each of the components in itself causes considerable morbidity, but all three together have a synergistic negative effect on health. Elite players and those girls and women who train particularly hard are at the highest risk.

Prevalence in football

In a study directly comparing football players, handball players and endurance athletes, initial screening with a detailed questionnaire was followed by measurements of bone mineral density (BMD) and a clinical interview. Compared to the reported number of dieting athletes in other sports, dieting behaviour in football was low, and dieting players had body mass index (BMI) values within the recommended range (Table 2.4.1.1). A significantly lower percentage of football players compared to handball and endurance athletes reported eating disorders (Table 2.4.1.1). Also, considerably fewer football players (9.3%) than handball players (18.8%) and endurance athletes (27.9%) reported menstrual dysfunction. Both football and handball players had higher BMD values when compared to endurance athletes and controls.

Pathogenesis

Pressure to reduce weight is a common explanation for the frequent eating disorders among athletes. Further factors associated with eating disorders are: restrained eating together with extensive training, frequent

	Football (n=69)	Handball (n=60)	Endurance (n=115)	Controls (n=607)
Age (years)	19.58 ± 4.1	19.9 ± 3.1	22.31 ± 6.3	27.3 ± 7.9
BMI	21.5 ± 1.6	22.5 ± 2.0	20.5 ± 1.8	23.3 ± 4.2
Training hrs/week	12.3 ± 3.7	15.8 ± 4.2	13.1 ± 4.5	-------
Eating disorders*	5.9%**	22.4%	25.7%	21.1%
Menstrual dysfunction	9.3%	18.8%	27.9%	15.2%
Stress fractures	13.6%	23.2%	13.4%	12.4%

Table 2.4.1.1 Characteristics of football and handball players as compared to endurance sports and controls

* Self-reported

** Football significantly different from other groups ($p<0.05$)

weight-cycling, early start of sport-specific training, personality factors, injury, a sudden increase in training volume and certain coaching behaviours.

Coaches might recommend weight loss in order to improve performance. Some players would do almost anything to improve their performance. However, losing weight does not necessarily lead to improved performance but to unfavourable energy deficits. It has to be stressed that prolonged energy deficits cannot be sustained without harm to health and performance.

The body has no automatic mechanism for matching energy intake to activity-induced energy expenditure. Inadvertent energy deficits may sometimes occur without an eating disorder or even without any dietary restriction. Most athletes with energy deficits, however, consciously reduce dietary energy intake without reducing their energy expenditure on exercise, while others increase energy expenditure on exercise without increasing their energy intake.

The susceptibility of the reproductive axis to exercise- and diet-related stresses varies considerably among individuals. Irregular menstrual bleeding and amenorrhea due to suppressed levels of oestrogen can be a consequence of intense exercise and low energy intake and/or high psychological and physical stress. In the long run, irreversible damage to the reproductive system is to be expected.

Any factor that contributes to menstrual dysfunction can have a direct or indirect influence on bone density. While heredity is thought to explain between 60-80% of the variation in BMD among individuals, the peak bone mass at skeletal maturity is another influencing factor. Modifiable determinants influencing BMD include soft tissue composition (lean v. fat mass), lifestyle factors (smoking, alcohol consumption), medication, hormones, physical activity and nutrition.

Symptoms and signs

Disordered eating behaviour is characterised by disturbances in eating patterns, body image, emotions and relations. Some athletes practise abnormal eating behaviours including fasting, vomiting, diet pills, laxatives, diuretics and enemas. Anorexia nervosa is the extreme of restrictive eating where individuals continue to starve themselves when far below an ideal body weight. Bulimia refers to a cycle of food restriction or fasting followed by excessive eating, self-induced vomiting and use of laxatives. As opposed to anorectic patients, bulimic patients are more often of normal weight or might be even overweight.

Undernourishment and eating disorders may cause serious medical problems and can even be fatal. Low energy intake may delay recovery from exercise, impair adaptation to training stimulus and compromise the immune system and reproductive function. Complications may occur as a result of excessive eating with laxative abuse. The loss of fluids and electrolytes during purging can lead to dehydration, acid-base abnormalities and cardiac rhythm disturbances.

Menstruation disorders range from irregularities to missed periods and amenorrhea. In the long term, bone architecture may suffer irreversible damage. The loss of BMD, however, is a silent process and the player is usually unaware until a related injury, such as a stress fracture, occurs.

Influence on performance

In football, the relationship between carbohydrate intake and the ability to maintain high intensity work, especially in the second half of the match, is well known. Although there are individual differences depending on the player's position and playing style, a player typically performs intermittent work, with about 70% of a match consisting of low intensity with repetitive bouts of high intensity. Thus carbohydrates are highly important energy substrates during match play. Low carbohydrate availability reduces performance and can lead to reduced skill and judgement, leading to more errors during the match. Therefore, it is important to maximise glycogen storage before and during the match.

Prolonged energy deficits leading to menstrual dysfunction may cause muscle weakness, reduced performance and stress fractures. Dehydration and electrolyte abnormalities decrease coordination, balance and muscle function.

Prophylaxis

The team physician should continuously educate players on the importance of a diversified diet providing sufficient energy for match play, training and other activities. Healthy eating should be established as a social team event. Coaches and their or her players need to know that, contrary to popular belief, losing weight does not necessarily improve performance.

Physicians need to be aware that some players may consider menstruation as an unnecessary annoyance, and

welcome amenorrhea as a rather convenient condition. Therefore, they have to ensure that players understand the serious consequences of menstrual irregularities and how dangerous it is to shrug off months of missed periods.

It is well known that weight-bearing activity may slow or even reverse bone loss. Mechanical loading in football play is probably the main reason for the high BMD values observed in female players. It seems that the amount of bone loss is correlated with the severity and length of menstrual irregularities, nutritional status and the amount of mechanical loading during activity. That means that while playing football, particularly if combined with a high training load, in general poses the same risk of triad as in all other sports, it might at the same time be considered part of the prevention of at least one aspect of the triad.

Diagnosis

Screening for the triad can be done at the time of the pre-participation examination. It requires a basic evaluation of energy and nutrient intake, possible eating disorder behaviour, menstrual status and history, weight change and presence of cardiac arrhythmias including bradycardia. Team physicians should be alerted by any sign of energy deficiency or eating disorder behaviour as well as by irregular periods, fatigue, electrolyte imbalances, anaemia, depression and history of stress fractures. In cases of fractures occurring from minimal trauma, players inevitably need to be questioned about their menstruation and eating history. Players with one component of the triad should be screened for the other components.

The IOC proposes a specific questionnaire as a screening tool during the pre-competition assessment (Figure 2.4.1.1).

Only those identified as being at risk after these initial questions should undergo a targeted interview. This comprehensive history-taking and in particular the interview for identification of triad risk factors require knowledge and empathy on behalf of the interviewer. A more practical alternative for the team physician might be to identify some key questions and then refer the player in case of any suspicion.

	Yes	No
Are you satisfied with your eating pattern?	□	□
Do you worry about your weight/body composition?	□	□
Are you a vegetarian?	□	□
Do you lose weight to meet weight requirements for your sport?	□	□
Does your weight affect the way that you feel about yourself?	□	□
Do you worry that you have lost control over how much you eat?	□	□
Do you make yourself sick when you are uncomfortably full?	□	□
Do you currently suffer or have you ever suffered in the past with an eating disorder?	□	□
Do you ever eat in secret?	□	□
What was your age at your first menstrual period?	_____	
Do you have regular menstrual cycles?	□	□
How many menstrual cycles did you have in the last year?	_____	
When was your most recent menstrual period?	_____	
Have you had a stress fracture in the past?	□	□

Figure 2.4.1.1 IOC triad screening questionnaire

Nevertheless, the physical signs of the triad might be more readily detected during general physical examination, at least in underweight players (Table 2.4.1.3). If a player has experienced irregular menses for some time, bone density should be assessed using dual-energy X-ray absorptiomentry (DXA).

– Height, weight, BMI (in- or decreased)
– Blood pressure, pulse
– Physical signs of eating disorder: lanugo (anorexia), parotid gland enlargement (vomiting), jaundice (anorexia, food high in carotinoids, hypothyroidism)
– Body fat percentage (fat callipers in- or decreased)
– Skin: acne/male pattern hirsutism (oestrogen deficit)
– Musculoskeletal injury assessment

Table 2.4.1.3 Physical characteristics to be assessed in cases of suspected female triad

Treatment

The success of the treatment plan is based on a relationship of trust between the player and her care providers. This includes respecting the player's desire to be lean for optimum athletic performance and showing willingness to help the player be lean, but healthy at the same time.

If an energy deficit is the primary cause of menstrual dysfunction, ameliorating energy balance will improve overall nutritional status, reverse the menstrual dysfunction and achieve normal reproductive function. In female athletes with menstrual dysfunction, a calcium-rich diet seems the most appropriate dietary prescription to promote and support bone density. Players need to be informed about the detrimental effects of amenorrhoea on bone health with regard to both short- and long-term consequences. The diagnosis of osteopenia by DXA may help to initiate a change in a player's attitude and behaviour.

Suspension from training and competition is not recommended. If a player is suspended, she may train on her own, which might be even more dangerous because she will not be monitored. Furthermore, preventing a player from playing may further reduce her self-esteem. Finally, control is a key issue in eating disorders. A player may view suspension as an attempt by others to control her.

It is important to focus on normalising eating behaviour (and/or weight whenever necessary), body composition and the menstrual cycle, as well as to modify unhealthy mental procedures that maintain the disorder and to adequately deal with emotional issues. As comprehensive counselling and treatment of the female triad requires considerable expertise and often psychotherapy, the team physician should seek such qualified support whenever dealing with players suffering from this condition.

2.4.2 Youth players

Functional anatomy

The most significant differences between musculoskeletal injuries in children and adults are due to the stresses loaded on the epiphyses (separately ossifying ends of the long bones) and the epiphyseal cartilage (growth plate or cartilage, Figure 2.4.2.1). The growth plate consists of hyaline cartilage responsible for the longitudinal growth of the bone. Tendons and muscles insert near the apophyses, which

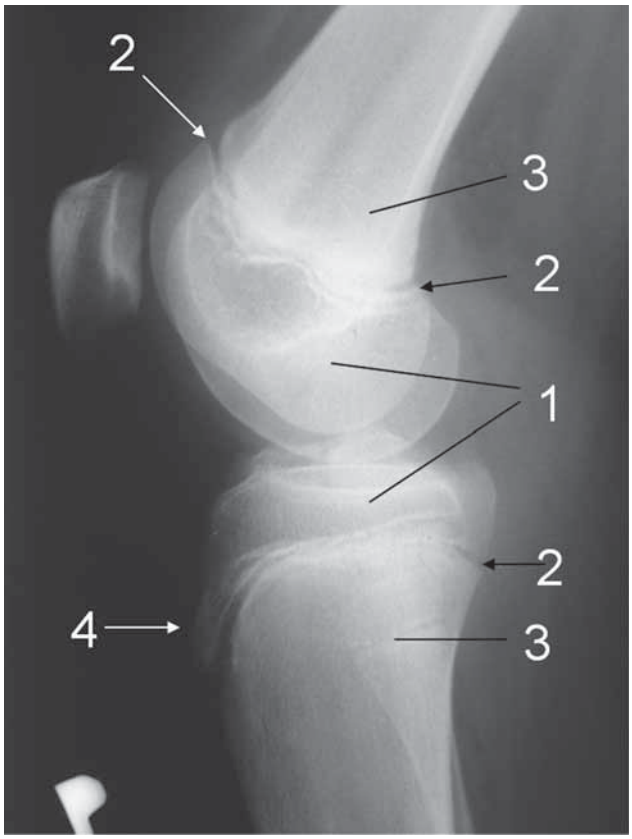


Figure 2.4.2.1 Functional anatomy of the immature skeleton of the knee. 1) epiphysis 2) epiphyseal cartilage (growth plate) 3) diaphysis 4) apophysis

are separate ossification nuclei. Apophyses are connected to the bone by layers of apophyseal cartilage. Epiphyseal and apophyseal cartilages are potentially weaker than the rest of the skeleton, the tendons, muscles and ligaments and therefore are more susceptible to injuries, leading to distinctively different injury patterns than in adults. Epiphyseal and apophyseal cartilages are weakest during puberty and towards the end of growth in adolescence. Growth plate injuries represent 15-30% of all fractures in children.

Principally, two groups of injuries in children need to be distinguished between: growth plate injuries and apophyseal injuries.

Growth plate injuries

Classification and grading

The classification of traumatic injuries to the growth plate mostly used in clinical practice is, according to Salter and Harris (1963) and Rang (1983), into six types (Figure 2.4.2.2). Prognosis of Salter I and II injuries are better because they do not cross the germinal layer of the growth plate and therefore the longitudinal growth of the bone remains mostly unaffected.

Salter III and IV fractures cross the germinal layer of the growth plate with consequent possible impairment of growth. Type V represents a particularly serious injury because of a crush of the growth plate, which almost inevitably leads to growth disturbance. A Type VI fracture was added by the classification of Rang, meaning a defect injury of the superficial peripheral part of the growth plate with subsequent growth disturbance.

A serious non-traumatic injury represents the slipped capital femoral epiphysis (SCFE) in adolescents aged 10-16 years, typically overweight boys, mostly unilateral, but sometimes bilateral at the time of presentation (20%). The proximal femoral epiphysis slips gradually in relation to the femoral neck, but a sudden slip may occur in some cases (Figure 2.4.2.3). It is important to differentiate between stable (weight-bearing possible) and unstable (no weight-bearing because of severe pain) SCFE.

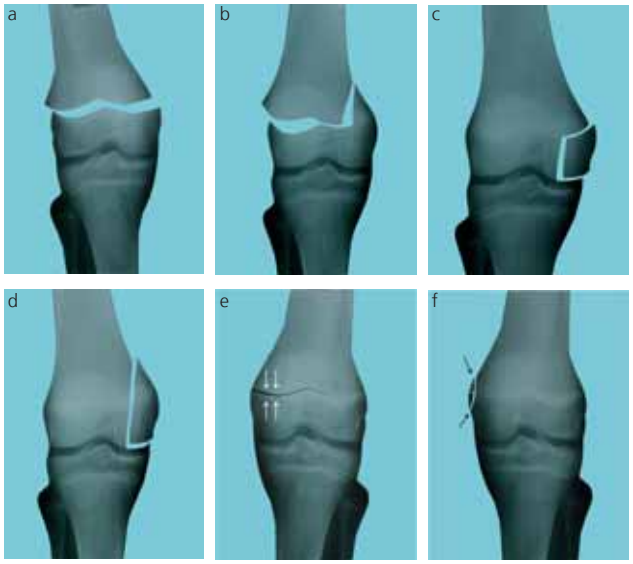


Figure 2.4.2.2
a) Salter Type I – separation of the epiphysis in the growth plate and slip of the epiphysis in relation to the bone (epiphyseolysis).
b) Salter Type II –- separation of the epiphysis in the growth plate and exiting to the metaphysis.
c) Salter Type III – fracture through the ephysis, exiting to the joint (epiphyseal fracture).
d) Salter Type IV – fracture through the epiphysis and metaphysis (epihyseal-metaphyseal fracture).
e) Salter Type V – crush injury of the growth plate (arrows).
f) Type VI (after Rang) – injury of the peripheral part (perichondral ring) of the growth plate (arrows).

Causes and mechanisms

The causes and mechanisms are direct or indirect impact in different anatomical locations, which in adults would lead to joint sprains, a rupture of the ligaments or fracture-dislocations. Although there are many theories, the true cause of SCFE is unknown. One explanation is a decrease in the mechanical resistance properties of the growth plate due to hormonal changes. In this situation, even a minor impact (running, jumping) may lead to a further gradual or sudden total slip of the femoral epiphysis.

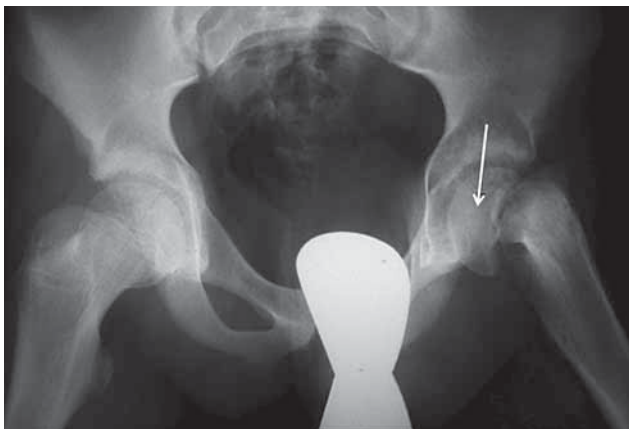


Figure 2.4.2.3 Severe slipped capital femoral epiphysis (SCFE) on the left side (arrow) in a boy aged 14 years.

Symptoms and signs

The symptoms vary according to anatomical location, type of injury and degree of displacement. In general, they are similar to the symptoms of joint sprains, ligament injuries and intra-articular dislocated fractures in adults. Localised intensive pain, immediate swelling and joint effusion, restricted and painful range of motion and in some cases visible dislocation are present. Disturbance of the blood supply (pallor) and innervation (anaesthesia and inability to move fingers/toes) indicate injuries to vessels and/or nerves, mainly by major dislocations. The symptoms of gradually developing SCFE are non-specific pain in the groin, which may be referred to as the anteromedial aspect of the thigh and knee, limited motion of the hip and shortening of the extremity. In an acute slip, symptoms similar to the traumatic injuries to the growth plate in other locations are present.

Examination and diagnosis

On the pitch, an immediate swelling of the injured area, joint effusion and possible angulation of the extremity are the important signs. After evacuation of the player, the points of maximum tenderness are identified and a more significant dislocation may be palpable. Any attempt to move the joint is extremely painful and should be omitted. Palpation of the peripheral arterial pulses and assessment of intact microcirculation (capillary reflux) of the extremity as well as sensory and motor innervation are absolutely necessary. X-ray and other diagnostic tools (CT, MRI) are usually required to confirm and define the correct diagnosis.

In SCFE, the ability to bear weight and the gait pattern should be assessed. Both hips should always be examined for range of motion, internal and external rotation, preferably in the prone position with knees flexed (90°). Internal rotation is almost invariably painful and decreased. Gentle passive hip flexion may lead to external rotation and abduction in SCFE (Drehmann sign).

On-field treatment

The player should immediately be removed from the pitch on a stretcher. The injured extremity is immobilised. The principles of Protection, Rest, Ice, Compression and Elevation (PRICE) are applied similarly as in adult fractures/dislocations, and the player is transported under supervision to the hospital as soon as possible. If signs of impairment of blood circulation or innervation of the extremity are present, or when there is suspicion of acute SCFE, transport to hospital care is urgent.

The principles of treatment are the same as those applied in the treatment of fractures in general, although there are a few important differences. The treatment modalities vary according to type of injury, degree of dislocation, bone age and remodelling potential of the bone. In all types of injuries, further damage of the growth plate needs to be avoided. Salter I and II injuries can be treated mostly conservatively with or without closed reduction to maintain or to achieve correct reduction of epiphysis. Some degree of dislocation is acceptable before adolescence because the remodelling potential of the bone will correct the deformity during further growth. Rotational dislocation, however, will usually not undergo spontaneous reduction and remodelling in most cases. Non-dislocated Salter III and IV and Salter V and VI are also treated conservatively. The extremity is immobilised in a cast for four to six weeks.

Operative treatment

Operative treatment in Salter I and II injuries is used only in major dislocation in child players or if no acceptable reduction is achieved in adolescent players (Figure 2.4.2.4). In Salter III and IV, even if minimally dislocated, it is absolutely vital to achieve anatomical reduction of fragments to restore the articular surface and to prevent osseous bridging across the growth plate (Figure 2.4.2.5). Minimally-invasive surgical techniques are used to avoid further damage of the growth plate. The slipped capital femoral epiphysis is always treated surgically, most often by percutaneous cannulated screw fixation of the slipped epiphysis. Prophylactic fixation of

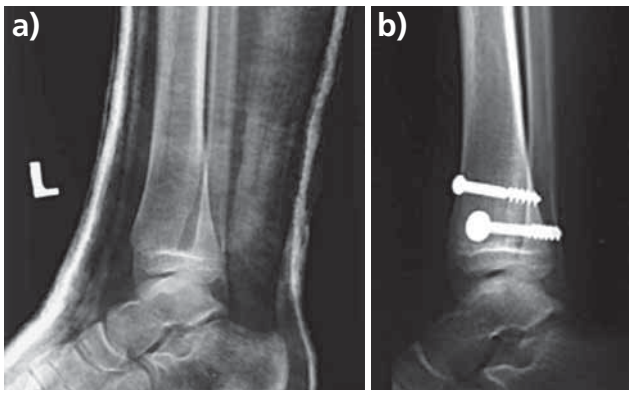


Figure 2.4.2.4 a) Dislocated epihyseolysis Salter. Type II of the distal tibia. b) Normal anatomical appearance after reduction and internal fixation of the fragments.

the contralateral side is a controversial subject and should be considered in younger patients (10-12 years) because a slip of the contralateral side may subsequently develop in approximately 40% of patients.

Protected partial weight-bearing with crutches is required in all cases for at least six weeks to three months.

Rehabilitation programme

After removing the cast, partial weight-bearing is allowed and the rehabilitation programme starts in order to increase the range of motion. Once the full range of motion is achieved, rehabilitation is targeted on strengthening the muscles and on the re-establishment of neuromuscular control. Full weight-bearing is usually allowed three months after injury. During this period, sports-specific exercises are applied to ensure gradual progression to training.

Prognosis and return to play

Return to training is possible after achievement of full range of motion and weight-bearing, adequate muscle strength and neuromuscular control. The player must follow the recommendation of the trauma or orthopaedic surgeon. The prognosis of the injury may be influenced by common complications like malunion, infection or osteonecrosis. A complication unique to growth plate injury is partial or total growth arrest due to premature ossification of the growth plate, resulting in leg length discrepancy or angular deformity (Figure 2.4.2.6). Malunion and infection are usually evident within two to six months after injury/surgery, but osteonecrosis and growth arrest may develop only later (up to one year). Both the player and parents should be warned about this potential problem. Treatment is mostly surgical. SCFE generally needs a long time before return to play is possible, and it can even be a career-ending injury. Misjudged and/or not properly treated SCFE may lead to the permanent restriction of hip function.

Apophyseal injuries

Injuries of the apophysis are typical injuries of the pre-adolescent and adolescent age from ten to 18 years. Two entities need to be distinguished: firstly, apophysitis as a chronic overuse injury due to repetitive overload of the musculo-tendinous junction with the bone; and, secondly, apophyseal avulsions (apophyseal fractures, avulsion fractures) occurring due to a sudden forceful overload/impact. In some cases, repetitive traction overloading may lead to avulsion fractures, mainly in the pelvis region.

Apophysitis

Classification

These conditions commonly occur in adolescent football players during the puberty growth spurt when height increases rapidly and muscle strength develops quickly. They are caused by overloading of the tendon insertion sites, predominantly where the major tendons insert in the lower extremity. The three major sites for these injuries are the lower patellar pole (the origin of the patellar tendon; Sinding-Larsen-Johansson disease), the tibial tuberosity (the insertion of the patellar tendon; Osgood-Schlatter disease) and the calcaneus (the insertion of the Achilles tendon; Sever's disease, also referred to as calcaneal apophysitis).

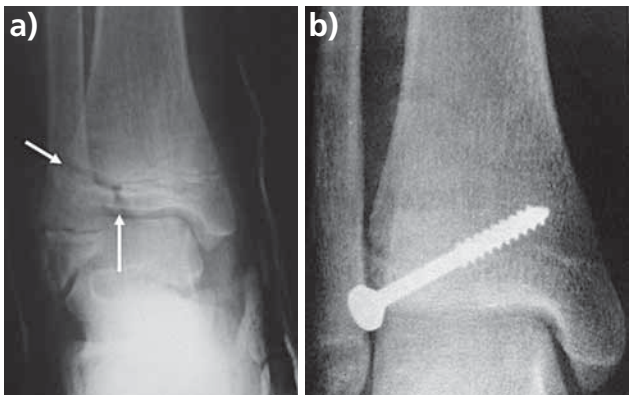


Figure 2.4.2.5 a) Epiphyseal injury Salter Type III of distal tibial epiphysis (Tillaux fracture). Line of separation of the fragment (arrows).
b) One year after anatomical reduction and fixation of the fragment.

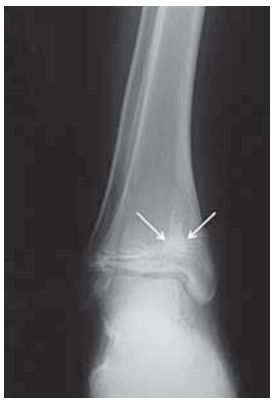


Figure 2.4.2.6 Bone bridge (arrows) and angular (varus) deformity of the distal tibia after injury of the growth plate Salter Type V in a boy aged 15 years.

Causes and mechanisms

When the growth zones of the distal patellar poles and the tibial tuberosity are overused (usually as a result of jump training, sprinting or repeated long football kicks or shots), they become irritated. The growth zone and the growth process will be disturbed. Eventually, continued overloading can cause fragmentation and sclerosis of the bone.

Symptoms and signs

The main symptom is pain when the extensor muscles are used. They occur mainly during the growth spurt, but symptoms can persist for a longer period if overloading is continued. Swelling eventually occurs. Pain is localised to the affected region; the posterior portion of the calcaneus in Sever's disease, the tip of the patella in Sinding-Larsen-Johansson disease and the tibial tuberosity in Osgood-Schlatter disease. Pain increases during training and may eventually force the player to stop. Pain can also persist for hours after training or matches. The area is usually tender to palpation, there may be slight swelling and the player may limp.

Examination and diagnosis

The diagnosis is clinical and is made based on tenderness to palpation and possibly mild swelling over the tendon insertion. If symptoms persist despite relative rest, the physician should take X-rays to exclude avulsion fractures or tumours. If the player has had this disorder for some time, an X-ray may show fragmentation and sclerosis of the affected apophysis.

Treatment

The treatment is relative rest and alternative training, i.e. to refrain from vigorous loading of the extensor apparatus (jumping, sprinting, kicking) for six weeks. Most players recover in that time, but a few must refrain from football for as long as six months. Other measures to reduce tendon load should also be tried such as proper shoes, training on soft ground and, in the case of Sever's disease, using a heel cup inside the shoe to unload the tendon. There is no relationship between apophysitis and avulsion fractures of the tuberosity, so strict restrictions on the child's activities are unnecessary. The physician should inform the player and the player's parents that this is a benign, self-limiting disorder and that it will resolve spontaneously. The player's activity is limited only by pain.

Prognosis

Every player recovers from apophysitis. Recovery occurs when the growth zones close – at the latest when the player is between 16 and 18 years old. There can be minor fragmentation of the tibial tuberosity in a few players with Osgood-Schlatter disease. This may cause symptoms when the player is fully grown and the small fragments of bone can be surgically removed at that time.

Avulsion fractures

Classification and grading

Avulsion fractures are mainly located in the growth zones of the pelvis and hip region; other anatomical locations are rare, like an avulsion of the triceps surae muscle on tuber calcanei or a sleeve fracture of the patella. They represent relatively frequent injuries in different sports with an incidence of three to seven per cent. In football, a number of case reports have been published. Concerning localisation, the avulsion of ischial tuberosity (hamstrings and adductor muscles) is common (Figure 2.4.2.7), followed by anterior superior iliac spine (sartorius muscle) and finally anterior inferior iliac spine with attachment of the rectus femoris muscle (Figure 2.4.2.8). Less common are avulsion fractures of the lesser trochanter (iliopsoas muscle, Figure 2.4.2.9) and iliac crest (abdominal muscles). The latter, however, should not be confused with a physiological Risser sign. Boys are more often affected then girls, and age at injury is between twelve and 16 years, just prior to the closure of the apophyses. Avulsion of the ischial tuberosity ossification nucleus also occurs in adults as the ischial growth plate remains open until the age of 25 years. The grading of the avulsion fractures may

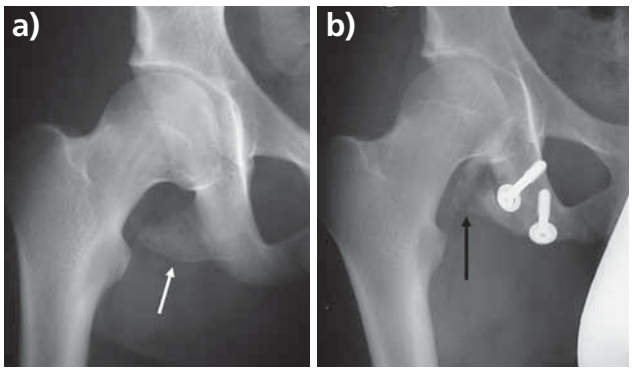


Figure 2.4.2.7 a) Avulsion fracture of the ischial tuber and its dislocation in a 15- year-old football player (white arrow). b) X-ray six months after surgical fixation of the avulsed fragment with screws. Post-traumatic ossification in the injured area (black arrow).

be according to the degree of displacement, namely: mild (a few millimetres) and major (1-2cm).

Causes and mechanism

The injury mechanism and causes are the sudden powerful contractions of the muscles, like explosive kicking, jumping, turning or stopping. Avulsion fractures in youth are the equivalent of acute muscle strains in adults. Chronic repetitive overload can also result in avulsion fractures. The muscles of the hip region are mostly affected, namely: hamstrings by sprints, rectus femoris by kicking or sprints, adductor muscles by "splitting" or by sliding techniques. The iliopsoas muscle is avulsed from the lesser trochanter by sudden flexion or by passive extension (kick from behind). Adequate warm-up plays an important role in the prevention of these injuries.

Symptoms and signs

The typical history is a sudden sharp pain after strenuous activity like kicking the ball or making a quick turn. The pain is localised to the area of injury and results in limitation of motion. Swelling and blood effusions are visible depending on the location.

Examination and diagnosis

Sudden sharp pain after powerful muscle contraction, rather localised to the area of injury. Restriction of movement and localised tenderness on palpation over the area of avulsion are obvious. The pain is aggravated by passive stretching of the avulsed muscle in the opposite direction, by its active contraction or by resistance to the contraction. Native X-ray mostly confirms the diagnosis when an avulsed dislocated fragment is clearly visible, like tuber ossis ischii or trochanter minor avulsions (Figures 2.4.2.7, 2.4.2.9). The fragments of both anterior iliac spines are more difficult to visualise because of their usually only mild displacement. Computer tomography or MRI are used mainly before surgical treatment. In some cases, X-rays may not confirm the diagnosis at the beginning, and diagnosis is confirmed only later by abundant post-traumatic bone formation (Figure 2.4.2.10)

On-field treatment

The player is immediately removed from the pitch. The extremity should be immobilised in a relaxed position and ice and compression applied to the injured area. Transportation to the hospital for further treatment is indicated.

Non-operative treatment

Avulsion injuries are mainly treated conservatively, provided that the dislocation of the fragment is mild. It includes rest in bed in a relaxed position of the muscle and non-weight bearing with crutches. After pain relief – usually within two to three weeks – and radiological evidence of healing, a functional rehabilitation programme may be started.

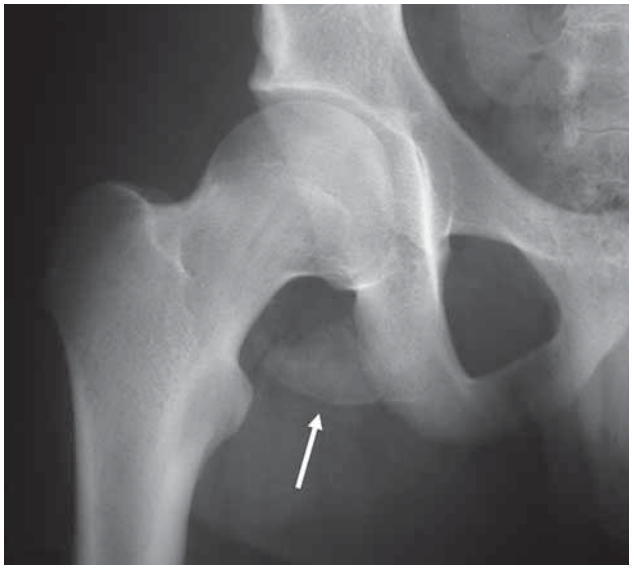


Figure 2.4.2.8 Avulsion fracture of the anterior inferior iliac spine (arrow) with attachment of the rectus femoris muscle in a 16-year-old football player.

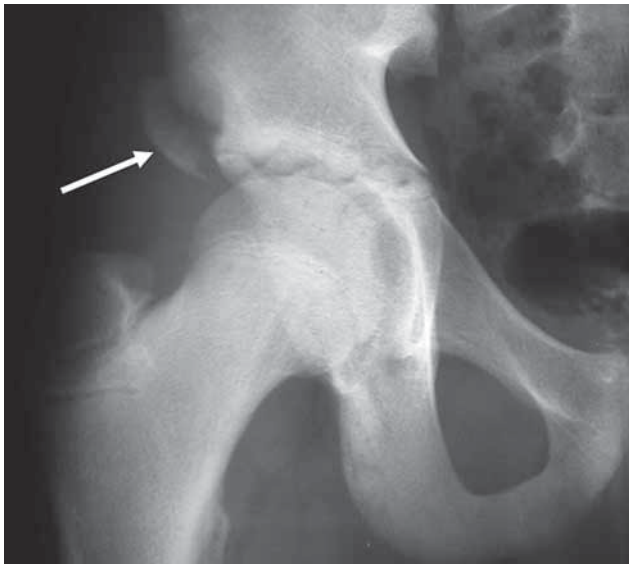


Figure 2.4.2.9 Avulsion of the trochanter minor with attachment of the iliopsas muscle (arrow) in a 15-year-old boy.

Operative treatment

Concerning surgical treatment, there are controversial opinions. Acute surgical reduction of avulsed fragment is rarely indicated, e.g. if the function of an important muscle is lost, like triceps surae muscle on tuber calcanei, and if there is major dislocation (more than 2cm), mainly in tuber ossis ischii (Figure 2.4.2.7), because it is prone to a high rate of non-union. Further more, indication for delayed surgery may be present with impingement of the hip (restricted movement and pain by activity) due to the avulsed fragment. In this case, the fragment is partially excided and reattached. Also, major post-traumatic ossification may lead to problems in daily and sporting activities. In these cases, excision of ossification may be indicated.

Rehabilitation programme

Functional rehabilitation programmes start after pain relief and radiological evidence of healing, and include increasing the range of motion, conditioning and gradually increased weight-bearing. When full range of motion without pain is achieved, strengthening exercises may start, followed by football-specific exercises later.

Prognosis and return to play

Prognosis is generally good, provided that the injury is treated properly. A return to training and football is possible when full range of motion and full strength without pain are achieved, mostly three to four months after injury. A meticulous medical examination before returning to play is mandatory. In cases without proper treatment, the dislocated fragment may lead to impingement of the hip (typical for avulsion of the rectus femoris muscle), or a formation of post-traumatic ossification may lead to chronic irritation (Figure 2.4.2.10). Excision of the ossification and reattachment of the muscles may be indicated in these cases. A return to training takes five to six months.

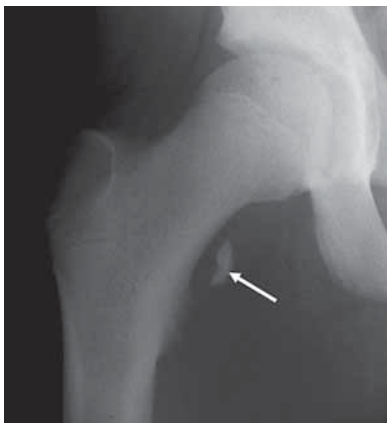


Figure 2.4.2.10 Post-traumatic large ossification (arrows) after avulsion of the anterior inferior iliac spine in a twelve year-old boy.

2.4.3 Referees

Introduction

The football referee, also considered the 23rd player in a football match, is supported by two assistant referees (Figure 2.4.3.1). Together, the three officials have to ensure that the players follow the Laws of the Game. An official survey ("Big Count") by FIFA revealed that, in 2006, there were, worldwide, and over all levels of football, more than 840,000 registered referees and assistant referees (about 94% male, 6% female). Compared with the first survey in 2000, this represented an increase of 17% in the total number of referees.

In the last ten years, several studies have examined referee profiles, both physiological and anthropometric, as well as the movement patterns and physiological load during actual match play. The physical match demands of football refereeing have recently been investigated in detail. Similar to the players, the referees show a mixed walking and running profile (low, medium, and high intensity) during a match. Therefore, high demands are put on the cardiovascular and musculoskeletal system of the referees, who are on average ten to fifteen years older than the players. But, in addition to being older than the players, the referee exhibits further unique characteristics: he is rarely a full-time professional, he does not play the ball and he normally cannot be substituted during the match (exception:

Figure 2.4.3.1 Referee and his two assistant referees

if an acute injury occurs). During a match, a referee may cover a mean distance of 11.5km (range: 9-14km) which is comparable to the distance covered by a midfielder. 16-17% of this distance is run at high intensity, while up to 12% of the total match distance is covered in a sprinting manner. Therefore, a referee needs to be prepared to perform at high intensity throughout the match. Elite referees may perform up to 1,270 activity changes and make more than 130 decisions during a match: this indicates the high level of physiological and cognitive demands experienced by the referee. Similarly, but with other movement patterns (combination of sideways shuffling and sprinting) and other cognitive demands (attention to offside situations, for example), the assistant referee is also highly stressed in today's game of football.

Injuries and musculoskeletal complaints

Some studies on the incidence of acute injuries and the prevalence of overuse injuries in football referees and assistant referees have recently been published. In a retrospective epidemiological survey, all 71 Swiss elite referees and assistant referees (mean age: 36 years) of the Super and Challenge Leagues were asked to answer a specific questionnaire concerning their experience in refereeing, their medical history, their previous injuries and their current musculoskeletal complaints. In addition, they were interviewed in order to establish or confirm potential diagnosis. In two further studies, the 123 male referees (mean age: 41 years), who were pre-selected for the 2006 FIFA World Cup™, and the 81 female referees and assistant referees (mean age: 35 years), who were pre-selected for the FIFA Women's World Cup 2007™ were examined using a similar approach. In addition, the injuries and musculoskeletal complaints they incurred during the respective World Cups were documented.

Over 40% of all involved referees stated that they had suffered from at least one *injury* during their career. In general, more injuries were incurred during training than during matches, which might be explained by the greater exposure time in training than in matches. The most common injuries in both groups were hamstring muscle strains and ankle joint sprains, followed by calf muscle strains, knee lesions (meniscus and cartilage injuries), and quadricep muscle strains. More than two-thirds of all involved referees had suffered from *musculoskeletal complaints* (caused by training and officiating) during their career. The most prevalent locations of these complaints were: lower back, hamstrings, quadriceps, knee, Achilles tendon and calf. This data indicates that overuse injuries *(musculoskeletal complaints)* are a far greater concern for the fitness of the referee and assistant referee than acute injuries.

Although no significant statistical differences were found, there were some trends differentiating the injuries/complaints of referees from those of assistant referees. Problems in the thigh and lower leg muscles were somewhat more typical for the referees, while problems in the groin/adductors and calf muscles were more pronounced in assistant referees. Possible explanations for these findings may be related to the different movement patterns that are typical of the two positions: the referee's job involves a considerable amount of changing direction, sprinting and backwards running (often with rotated trunk and head-neck), during which the dorsal muscle chains (lower back, hamstring) must control the eccentric load, and the knee and ankle joints have to master the rotational movements (Figure 2.4.3.2). The movements of the assistant referee are, in contrast, characterised by rapid lateral shuttle runs (often on their toes), which stress in particular the groin area, the calf and the knees (Figure 2.4.3.3).

In female referees, the incidence of injuries was higher than male referees, but the diagnoses were similar (same type of injuries and same locations of musculoskeletal complaints). About the same amount of match and training injuries were recorded in the female referees. Female assistant referees suffered from significantly more adductor muscle strains than referees.

Summarising these figures, the locations of injuries and musculoskeletal complaints were the same for both

Figure 2.4.3.2 Rotation

Figure 2.4.3.3 Lateral shuttle run

genders in training and matches: hamstring, calf (with the Achilles tendon), ankle, knee, quadriceps and lower back. These results show that there is a need to develop and implement specific injury prevention programmes for referees and assistant referees of both genders.

Basic injury prevention programme

The following basic injury prevention programme focuses on the common locations of injuries and musculoskeletal complaints of referees and assistant referees. The exercises are both evidence-based on various injury prevention studies and best-practice. The first part (exercises 1, 2, 3) focuses on the lower extremity, groin and hamstring; the second part (exercises 4, 5, 6) on the ankle, calf and Achilles tendon, and the third part (exercises 7, 8, 9) focuses on knee, groin and quadriceps. The programme can be performed in 15 minutes, and should be integrated in the warm-up before each training session. The programme presented here is currently being implemented in the FIFA selections for the 2010 FIFA World Cup™ and the FIFA Women's World Cup 2011™.

Instructions for referees

Exercise 1: stabilisation/strength of the core (A)
Description: bench position; keep whole body stable and aligned while lifting each foot in turn, hold lifted leg about one second.
Basic intensity: 15 repetitions each side; two to three sets (two-minute break between sets).

Exercise 2: stabilisation/strength of core (B)
Description: sideways bench position; raise and lower hip (about one second rhythm). keep whole body stable and aligned.
Basic intensity: 20 repetitions each side; two to three sets (two-minute break between sets).

Exercise 3: eccentric strength of hamstrings
Description: kneel with your body completely straight from head to knees, partner should hold the lower legs firmly to the ground, slowly lean forward by keeping the body aligned, control the movement for about 30-45 degrees, then use the hands to control the fall.
Basic intensity: 5-10 repetitions.

Exercise 1 Stabilisation/strength of the core (A)

Exercise 3 Eccentric strength of hamstrings

Exercise 2 Stabilisation/strength of core (B)

Exercise 4 Static stabilisation of ankle

Exercise 4: static stabilisation of ankle
Description: stand on one leg, keep foot-knee-hip aligned, move the non-weight-bearing leg in a half-circle while keeping stabilisation and balance.
Basic intensity: 20 seconds each side; two sets (30-second break between sets).

Exercise 5: dynamic stabilisation of ankle
Description: stable body position, perform small hops (front, back, diagonal) while maintaining stabilisation and balance.
Basic intensity: 15 hops each side; two sets (30-second break between sets).

Exercise 6: eccentric strength of calf/Achilles tendon
Description: stand on one leg, support yourself on a wall or post, perform a calf raise with a step-up movement, then slowly lower the heel to the ground.
Basic intensity: 15 repetitions each side; two sets (30-second break between sets).

Exercise 7: dynamic control of knee position
Description: front lunge position, keep core-pelvis stable and front knee over the foot, lower the body until the back knee touches the ground while maintaining stabilisation and balance.
Basic intensity: ten repetitions each side; two sets (one-minute break between sets).

Exercise 8: dynamic control of pelvis/groin
Description: perform side-to-side hops, stop after each hop while maintaining stable body position and balance for about two seconds.
Basic intensity: ten times landing on each foot; two to three sets (one-minute break between sets).

Exercise 5 Dynamic stabilisation of ankle

Exercise 7 Dynamic control of knee position

Exercise 6 Eccentric strength of calf/Achilles tendon

Exercise 8 Dynamic control of pelvis/groin

Exercise 9: strengthening of quadriceps
Description: stable body position, perform one-leg squat with foot-knee-hip aligned, lower the body by bending the knee (trunk inclined) while maintaining stabilisation and balance, then slowly return to the straight position.
Basic intensity: 15 repetitions each side; two to three sets per leg (one-minute break between sets).

Basic warm-up programme before a match

The following warm-up programme was originally developed by C. Castagna (University Tor Vergata, Sport Sciences, Rome, Italy), the head conditioning coach of elite Italian referees. This 15-minute programme should be performed as a routine warm-up before every match. The first part focuses on a general cardiovascular activation, the second part comprises some stretching and mobilisation exercises and the third part focuses on a specific sprinting drill (with differentiation between referee and assistant referees). As the players do their pre-match exercises in the two halves of the field, the three officials may use a ten-metre wide lane in the middle of the pitch.

Part I (distance of about 50-60 metres, 5 minutes)

a) Two runs at an easy pace
b) Three runs at a moderate pace (about 60 to 70% of individual maximum speed)

After each run, walk back to the starting point.

Exercise 9 Strengthening of quadriceps

Part II (in the centre circle, 5 minutes)

a) simple static stretching exercises for the following muscles: hamstring, rectus femoris, calf and adductors (standing position, each stretch of 10-15 seconds, twice left/right, total 2 minutes)
b) simple range of motion exercises for: trunk rotation, front lunges and lateral lunges (standing position, each exercise about 1 minute, left/right, total 3 minutes)

Part III for the referee (distance of about 50-60 metres, 5 minutes)

First run about 100 metres at moderate intensity, then:

a) 5-metre run, followed by progressive ten-metre sprint (3 repetitions)
b) 5-metre skip, followed by five-metre sprint/cut/changing directions (3 repetitions)
c) 10-metre sprint, followed by following deceleration after 4-5 metres (3 repetitions)
d) 10-metre sprint (4 repetitions) or 2-3 explosive vertical jumps

(after each run, easily jog back to the starting point)

Part III for assistant referee (distance of about 50-60 metres, 5 minutes)

First run about 100 metres at moderate intensity, then:

a) 5-metre lateral shuffle, followed by 10-metre progression (2 repetitions each side)
b) 5-metre lateral shuffle, followed by 5-metre sprint (2 repetitions each side)
c) 10-metre sprint, followed by deceleration after 4-5 metres (3 repetitions)
d) 10-metre sprint (4 repetitions) or 2-3 explosive vertical jumps

(after each run, easily jog back to the starting point)

2.5 Nutrition and fluids for football training and match play

Introduction

Diet can significantly affect performance in all sports, and a good diet is important if peak performance is to be achieved. Some aspects of sports nutrition are common to all sporting activities, but there are obviously differences between various sports. In football, however, it is less easy to quantify performance: measurement of team performance in terms of the number of matches won and lost tells little about the performance of individual players. The aim is to win matches, and while measurements of heart rate or distance covered tell something about the player's effort, they say little or nothing about performance.

Nutrition is no substitute for fitness or skill, but it can help players to make the most of their abilities. In most matches, the competition is structured so that there is little difference between the two teams, and when teams are evenly matched in terms of skill, fitness and determination, good nutrition can make the difference between winning and losing. It is therefore surprising that some players seem not to be concerned about their diet: there is often a lack of knowledge of basic nutrition and of awareness about the food choices that should be made.

Fitness is obviously important in allowing the player to last the full 90 minutes of a match, and improving fitness is an important part of the preparation for competition. However, the ability to play well and to employ footballing skills will be impaired – particularly late in the match – if the player's diet is inadequate. Conversely, performance may be improved by dietary manipulation, but we still have an incomplete understanding of how best to control diet to optimise sports performance.

Not all football players follow a diet that will allow them to sustain optimum performance in training and competition. The results set out in Tables 2.5.1 and 2.5.2 suggest that the average energy intake of the players is not exceptionally high compared with the intake of athletes in many other sports, implying that there is a need for careful attention to the composition of the diet to ensure an adequate intake of protein, vitamins and minerals. The mean values indicate that the composition of the diet is not very different from that of the general population, but there are some individuals within this group whose diet provides inadequate amounts of carbohydrate, too much fat or too much alcohol.

Two distinct aspects must be considered: the first is the diet in training, which must be consumed on a daily basis for a large part of the year, and the second is the diet in the immediate pre-match period and during the competition itself. It is also important to recognise that players may need help in translating their nutrition goals – the requirement for energy and specific nutrients – into an eating strategy that allows them to know what foods to choose, when to eat them and how much should be eaten.

2.5.1 Diet for training

The primary requirement of the diet of the player in training is to meet the nutritional demands of daily living and, in addition, to provide for any additional nutrient requirement imposed. Throughout the season, which now lasts for almost 12 months for top-class players, some training is carried out most days of the week, and the training load has a significant effect on energy balance. A typical 90-minute hard training session will require about the same energy expenditure as the average game, adding about 1,000 to 1,500 kcal (about 4-6 MJ) to the daily energy budget. If energy intake is inadequate, players will suffer from chronic fatigue and illness and will not adapt effectively to training.

There have been few studies on the dietary intakes of football players, and dietary surveys are generally prone to methodological flaws. However, the available data suggests that football players, even at professional level, have moderate energy requirements relative to those in some other sports, with intakes being typically about

2,000-4,000 kcal/day (8.5-17 MJ/d) for male players and 2,000-3,000 kcal/day (8.5-13 MJ/d) for female players. Players may have a positive energy balance (i.e., energy intake exceeds expenditure, leading to a tendency for body mass to increase) during the close season or when injury forces a break in training or match play. It is common during pre-season training for energy intake to be restricted and energy expenditure to be increased to achieve the optimum playing body mass and to reduce body fat content if this is too high. An energy deficit is necessary to achieve a progressive loss of body mass. Large energy deficits, however, lead to an inability to continue training at a high level, and an increased risk of injury, so regular monitoring of body mass and body fat content over the season is recommended to ensure that weight loss is gradual (no more than about 0.5-1kg per week).

While it is undoubtedly true that an inadequate amount of protein will lead to a loss of muscle tissue, excess dietary protein alone will not drive the system in favour of increasing muscle mass. The recommended daily allowance for protein is 0.8 grams per kg of body weight per day (g/kg/d), though the actual amount needed is about 0.6 g/kg/d on average. Athletes will need more than this, but there is no evidence that more than twice this amount (more than about 1.6 g/kg/d) is necessary even during very hard resistance training. Hard training does lead to an increase in the minimum daily protein requirement, but this will be met if a varied diet that contains items from all of the major food groups is eaten in sufficient amounts to meet the increased energy expenditure. A diet that is based largely on nutrient-rich foods, including vegetables, fruits, beans, legumes, cereals, lean meat, fish and dairy foods, should ensure that sufficient protein is consumed, as well as meeting needs for vitamins and minerals. There does not appear to be any evidence that excessive protein intake among athletes is in any way damaging to health, but it may not leave enough

	Team A (n=26)			**Team B (n=25)**		
	Mean	SD	Range	Mean	SD	Range
Energy (MJ)	11.0	2.6	5.2-16.5	12.8	2.20	8.5-16.2
Protein (g)	103	26	5.2-16.5	108	20	77-144
Fat (g)	93	33	41-195	118	24	74-179
Carbohydrate (g)	354	95	167-538	397	94	243-599
Alcohol (g)	9	10	0-38	13	9	0-28

Table 2.5.1 Daily energy and macronutrient intake of first team squad players from two Scottish Premier League clubs. Assessment was by seven-day weight intake. Data from Maughan (1997).

	Team A			**Team B**		
	Mean	SD	Range	Mean	SD	Range
Protein	16	3	12-24	14	2	11-19
Fat	32	5	19-41	35	4	24-43
Carbohydrate	50	8	35-65	48	4	38-58
Alcohol	2	3	0-11	3	2	0-7

Table 2.5.2 Percent contribution of macronutrients to energy intake for the same players as in Table 2.5.1.

room in the diet for the carbohydrate that is needed to fuel the working muscles.

The energy requirements of training are largely met by oxidation of fat and carbohydrate. The higher the intensity of exercise, the greater the reliance on carbohydrate as a fuel: at an exercise intensity corresponding to about 50% of an individual's maximum oxygen uptake (VO_2max), approximately two-thirds of the total energy requirement are met by fat oxidation, with carbohydrate oxidation supplying about one-third. If the exercise intensity is increased to about 75% of VO_2max, the total energy expenditure is increased and carbohydrate is now the major fuel. Both training and match play involve repeated short, intense sprints and these rely largely on carbohydrate as a fuel. Towards the end of a training session, when the carbohydrate (glycogen) content of the liver and the muscles is low, it becomes more difficult to maintain the training intensity. There is some evidence that increased rates of muscle injury are also associated with exercise performed in the fatigued state, but it is difficult to obtain clear evidence to confirm or deny this suggestion.

During each strenuous training session, substantial depletion of the glycogen stores in the exercising muscles and in the liver takes place. If this carbohydrate reserve is not replenished before the next training session, training intensity must be reduced, leading to corresponding decrements in the training response. If players consume a low-carbohydrate diet, consisting mostly of fat and protein, after a day's training they will find it difficult, if not impossible, to repeat the same training load on the following day. Suggested targets for carbohydrate intake are to ensure about 5-7 g/kg/d during periods of moderate intensity training, and to aim for 7-10 g/kg/d during recovery from hard training (such as in pre-season).

The primary requirement during periods of intensive training, therefore, is for the carbohydrate intake to be sufficient to enable the training load to be sustained at the high level necessary to improve fitness. The training diet should be high in carbohydrate to meet the high requirement; this suggestion conforms to recommendations that, in a healthy diet for the average individual, carbohydrates should make up at least 50% of the diet. The recommendation for carbohydrate intake has traditionally been made as a fraction of the total energy intake, but it seems more appropriate to think of an absolute requirement related to the total amount of carbohydrate used during training and other daily activities. For a typical 70kg player, a carbohydrate intake of 500-600 grams per day (which might correspond to less than 50% or more than 70% of the total energy intake) may be necessary to maximise muscle and liver glycogen stores between daily training sessions (or between matches that are close together). These high levels of intake may be difficult to achieve without increasing the frequency of meals and choosing compact forms of high carbohydrate food and drinks – this is especially true when training twice a day. Many players find it difficult to eat two or three hours before training and the appetite is likely to be suppressed for some time after exercise. Choosing foods that are practical and appealing is important in a busy lifestyle and where the player cannot rely on hunger to govern food intake. Of course, this will depend on individual preferences, and many different foods can be chosen to meet energy needs and to provide the essential nutrients. Players may need to seek professional advice from a qualified sports dietitian to ensure that they make the best choices.

If nutritious carbohydrate-rich foods are eaten in sufficient quantity to meet energy needs, the diet should supply more than adequate amounts of protein, minerals, vitamins and other dietary components to meet any increase in requirements resulting from the training load. Inadequate nutrient intake may occur when the energy intake is restricted by chronic or over-zealous weight loss programmes, or when the diet lacks variety. Some players do not eat a varied diet in a sufficient amount to meet their needs, and dietary deficiencies may occasionally be encountered. The club doctor, dietitian and coach must all be alert to this possibility. For many players, a regular routine is an important part of their lifestyle, and this may lead to repetitive food choices and meal plans. Fad diets, disordered eating habits, perfectionism about eating only "good foods", poor nutrition knowledge, a lack of food preparation skills and limited access to food may all restrict food variety.

Players should practise drinking during training: this helps them to cope with training, but also ensures that their individual hydration strategy for training and match play can be defined. The individual hydration strategy should consider the individual sweating rate of a player and their preferences for fluid intake and particular drinks. The general aim for all players should be to limit weight loss during match play to not more than about 2% of body mass.

How to estimate sweating rate:

1) Measure body weight (kg) both before and after at least one hour of exercise under conditions similar to competition or a hard practice.
2) Measure body weight wearing minimal clothing and while barefoot. Towel dry after exercise and obtain body weight as soon as is practical after exercise (e.g. less than 10 minutes).
3) Note volume of fluid consumed during exercise (litres).
4) Sweat loss (litres) = Body weight before exercise (kg) - Body weight after exercise (kg) + fluid consumed during exercise (litres).
5) To convert to a sweat rate per hour, divide by the exercise time in minutes and multiply by 60.

Note: 2.2 pounds equals 1.0kg and converts to a volume of 1.0 litre or 1,000ml or 34 ounces of water.

2.5.2 Diet for competition

The ability to perform prolonged exercise can be substantially modified by dietary intake in the pre-exercise period, and this becomes important for individual players aiming to produce peak performance on a specific day. The pre-competition period can conveniently be divided into two phases – the few days prior to the match, and the day of the match itself.

Dietary manipulation to increase muscle glycogen content in the few days prior to exercise has been extensively recommended for endurance athletes following observations that these procedures can increase endurance capacity in cycle ergometer exercise lasting about 1½-2 hours. There are few carefully controlled investigations into the effects of manipulating the diet on the ability to play football, but one Swedish experiment showed that players starting a football match with low muscle glycogen content did less running, and much less running at high speed, than those players who began the match with normal muscle glycogen content. To ensure that muscle glycogen stores are adequate, players should avoid hard training for the last two or three days before competition and should simultaneously increase their dietary carbohydrate intake. This is obviously a problem at times during the season when it is necessary to play two or more matches within a few days. At such times it is even more essential to ensure a high carbohydrate intake between matches to ensure that recovery of the muscle glycogen stores takes place. It is common for players to have one match mid-week as well as one at the weekend, and it is likely that full restoration of the muscle glycogen content will not occur between matches unless a conscious effort is made to achieve a high carbohydrate intake. The available evidence suggests that most players do not eat a high-carbohydrate diet and would benefit from some attention to this. Players should aim for 7-10 g/kg/d over the last two to three days before an important match.

Ideas for high-carbohydrate pre-event meals*

Breakfast menus

- Breakfast cereal and milk, fresh or canned fruit
- Toast and jam/honey
- Pancakes and syrup
- Fruit-flavoured yoghurt
- Baked beans or tinned spaghetti on toast
- Liquid meal supplement or fruit smoothie
- Fruit juice or sports drink

Lunch and dinner menus

- Rice dishes – risotto, fried rice, paella
- Pasta and light sauce
- Bread, including rolls and sandwiches
- Fruit and fruit-based desserts
- Rice pudding

* A low-fat or low-fibre menu may help to reduce the risk of gastrointestinal problems in susceptible athletes

Snacks for recovery after training or matches

- Each choice provides ~ 50g carbohydrate and a valuable source of protein and other nutrients
- 250-350ml fruit smoothie or liquid meal
- 60g (1-2 cups) breakfast cereal + milk + 1 piece of fruit
- 200g carton of yoghurt + cereal/breakfast bar
- 1 round of meat/cheese and salad sandwiches or roll + 250ml fruit juice
- 150g thick crust pizza – lean meat and vegetable toppings and easy on the cheese
- 60g sports bar + 250ml sports drink

During the match itself, players can take drinks during stoppages in play, and there is also an opportunity to take drinks at half time. The primary aims must be to ingest a source of energy, usually in the form of carbohydrate, and fluid for replacement of water lost as sweat. Sports drinks that contain about 4-6% carbohydrate, in the form of a mixture of sugars, including glucose, fructose and sucrose, might be best. High rates of sweat secretion are necessary during hard exercise in order to limit the rise in body temperature which would otherwise occur, but sweat rates vary greatly between individuals. If the exercise is prolonged, this leads to progressive dehydration and loss of electrolytes. Salt supplements are not generally useful, but players who regularly experience muscle cramps are often those with high salt losses in sweat, and drinks with high salt content may therefore benefit them. Some players who know that they have high salt losses choose to use oral rehydration solutions (ORS) which are normally given to children with diarrhoea.

The composition of drinks to be taken during exercise should be chosen to suit individual circumstances. Even in the cold, fluid replacement may be necessary as there is still a need to supply additional glucose to the exercising muscles. Consumption of a high-carbohydrate diet in the days prior to exercise should reduce the need for carbohydrate ingestion during exercise, but it is not always possible to achieve this. Most commercially available sports drinks contain about 6g of CHO per 100 ml, but more concentrated glucose drinks (containing at least 8-10g of CHO per 100ml) will supply more glucose, thus sparing the limited glycogen stores in the muscles and liver, without overloading the body with fluid. In hot weather, the player may need to drink to the limits dictated by opportunity and comfort in order to replace sweat losses, but players should never need to drink so much that intake exceeds sweat loss, i.e. that they gain weight during the course of a match. The team physician should ensure that strategies to maximise the availability of drinks during matches, such as having bottles around the perimeter of the field so that players can grab a drink during stoppages in play, are in place. Drinking plain water is better than nothing, but properly formulated sports drinks are better than water and will supply a good balance of carbohydrate and fluid.

In the post-exercise period, replacement of fluid and electrolytes can usually be achieved through the normal dietary intake. Sports drinks and snacks with a high glycaemic index (GI) at this time are a convenient way of ensuring that replacement of glycogen begins as soon as possible, and high-carbohydrate drinks (energy drinks, but those without excessive caffeine) may be beneficial. If there is a need to ensure adequate replacement before exercise is repeated, extra fluids should be taken and additional salt (sodium chloride) might usefully be added to food. The other major electrolytes, particularly potassium, magnesium and calcium, are present in abundance in normal foods, and judicious selection of these, including fruit, fruit juices, milk, etc. will ensure that losses are replaced without resort to specific supplementation.

2.5.3 Alcohol

Where there is a need for rapid recovery between matches that are close together, it is best to ensure that alcohol intake is restricted with regard to the time of intake and the amounts consumed. Excessive amounts of alcohol may prevent the player from paying attention to their fluid and carbohydrate needs and hence delay the recovery process. Drinks with an alcohol content of more than about 5% are not recommended, as these will stimulate urine output and may prevent adequate restoration of fluid balance. In addition, there is an increased risk of accidents, including motor vehicle accidents, which may have serious consequences for the player and for others. In general, however, alcohol is a normal part of the diet of many players, and does no harm when consumed in moderation.

2.5.4 Supplements

Supplementation may be part of a treatment plan for diagnosed deficiencies in a player, but modifications to food intake are the desirable long-term goal. Supplements may be useful to safeguard nutrient intake when energy intake is restricted (e.g. during weight loss) or when the player is travelling frequently with uncertain food supplies or limited access to nutritious foods. Occasionally, micronutrient supplementation may be helpful as part of dietary therapy to correct or prevent deficiencies, but diagnosis and management of any suspected nutrient deficiencies should be undertaken only under the guidance of an expert.

In general, there is not at present good evidence to support the use of vitamin and other nutritional supplements (including compounds such as bee pollen, ginseng, etc.) in football, although some of these have gained wide popularity. There is, however, a need for the team physician and dietician to be aware of the supplements that are in use in the sport so that players can be advised accordingly. Caffeine in small doses (1-3g/kg) is not harmful to health and can aid performance in some situations, but players should try this out in training before using it in a match situation. Creatine supplements are popular and can improve repeated sprint performance as well as increasing muscle strength and power, but should be used with care and only after thorough consideration of the potential costs and benefits.

Care should be taken with the use of any supplements, but in particular "energy boosting" and "muscle building" supplements that are widely sold to players, as many of these contain stimulants, anabolic-androgenic steroids or prohormones that would lead to a positive doping test. The team physician should explicitly warn players of these products and discourage their use.

2.5.5 Nutrition needs of the female player

The basic nutritional requirements are generally the same for male and female players, and will be determined largely by body size, training load and the total energy expenditure. In general, the energy requirement of female players is about 10-20% lower than that of male players. Some female players will be weight conscious (as will some male players, see also 2.4.1) and may deliberately restrict energy intake to control body weight. This will be a problem if the dietary carbohydrate intake is restricted, and there may be a need to encourage players to identify foods that are high in carbohydrate and low in fat if the dietary goals are to be achieved.

Despite the low energy intake of some female players, and therefore low intake of vitamins and minerals, there is no good evidence that deficiencies are more common than in the general population. As in the general population, however, some players have diets that are inadequate for their needs. Some attention to iron intake may be warranted, as low iron levels are not uncommon in female athletes. Dietary strategies involving good food choices are the first recommendation and the most effective long-term solution. Red meat and iron-fortified breakfast cereals are good choices: the haem iron in red meat is well absorbed, and the addition of vitamin C, in the form of fruit or fruit juices, to meals containing sources of non-haem iron (cereals, legumes, green leafy vegetables) will enhance absorption. Supplementation, on the advice of a physician, should be considered for players shown to be iron depleted; routine supplementation may do more harm than good, and assessment should include a full blood analysis. Calcium intake may also be low in many female players, particularly when a low-fat diet is followed. Low dietary calcium intake puts bone growth and remodelling at risk, and increased consumption of dairy products (at least three servings per day) should be encouraged. Low-fat products alleviate fears of increasing energy intake.

2.5.6 Nutrition needs of the child player

The dietary requirements of young players are generally similar to those of older players. For active children, the extra energy demand associated with normal growth is small in relation to the total daily energy turnover. Equally, the additional protein requirement of the growing child is small relative to total daily needs. There may be a special need for attention to fluid intake in warm weather because children are more prone to heat illness. Extra stoppages for drinks may be advisable, the players should be provided with pleasant-tasting drinks and should be encouraged to consume these at every opportunity.

2.5.7 Conclusion

There is clearly a need for the team physician to implement and supervise an educational programme for players and those who support them to increase awareness of what constitutes an appropriate diet and how this can be achieved. Specialist professional advice should be sought where necessary. To make effective food choices, players should have some understanding of their nutritional goals and of the types and amounts of foods that should be eaten at different times to achieve those goals. As well as educating players, clubs should take responsibility for making sure that those who purchase and provide meals for players are aware of their needs. This obviously includes staff at the club dining room, but also includes partners, the parents of young players who stay at home and other food providers. In most cases, these individuals, rather than the players themselves, determine the foods that are eaten, and yet they often have no knowledge of the physical demands of the match or the nutritional goals of the players.

Consensus statement: Nutrition for football: The FIFA/F-MARC consensus conference. J Sports Sci. 2006 Jul;24(7):663-4. (Supplement)

2.6 Enviromental factors

2.6.1 Extreme temperatures – heat and cold

Physiology

Body temperature is actively controlled by the energy balance between heat production and heat loss. The body core temperature (Tc) is related to the wellbeing and performance of a player.

The *energy balance* of the body is determined by:

1. Heat production:

- *Metabolic heat production:* resting metabolic rate is increased by exercise and stress. 70 to 80% of energy expenditure during exercise will be lost to heat production and will not be used to perform mechanical work
- *Environmental heat*

2. Heat loss from the skin and the airways:

- *Conduction:* direct contact with air, water and other materials. Air has poor heat conductivity
- *Convection:* motion of air or liquids across the heated skin
- *Radiation:* infrared rays transport heat
- *Evaporation/sweating:* as fluid evaporates, heat is lost

The *environmental factors* which contribute to the heat balance of the body are:

- ambient air temperature
- wind velocity
- relative air humidity
- mean radiant temperature (from radiation; sun, car, walls)

Commonly, the environmental influences are represented by measurement of air temperature. However, the net energy balance of the body is determined mainly by the heat exchange of the skin and by breathing, and therefore the other factors mentioned above have an important influence on heat exchange.

Wind velocity determines heat convection and heat loss through sweating. In cold climates, wind contributes mainly to heat losses. The effect of wind on heat loss is often referred to as the "wind chill factor".

The relative air humidity mainly determines the effects of sweating, as with high humidity the effect of sweating may be severely diminished. Conditions with high air humidity pose a high risk of impaired thermal regulation. Radiation is represented by mean radiant temperature; it is high when the weather is sunny and low in cloudy conditions. Radiation is also present in overheated cars and rooms.

Clothing

The clothing worn significantly influences heat balance. Important factors are the heat insulation of the fabric, the moisture permeability and how these change with wind or body motion or both. Heat insulation is high for wool due to its good moisture permeability. Modern sport synthetic fabrics have moderate heat insulation and provide good cooling as a result of high moisture permeability.

In wet conditions, heat insulation differs between fabrics and this has an effect in cool and windy conditions. Wet wool provides heat insulation and wind protection, wet sport synthetics provide very low heat insulation and no wind protection, therefore an additional wind shield liner is necessary in cold environments. Wet cotton fabrics are heavy and provide very low heat insulation.

Rain gear has low moisture permeability that significantly limits heat exchange and can be used as a wind shield because the body is insulated by the saturated air between skin and garment. If a rain garment enables some kind of air pumping over the skin during exercise, additional heat may be lost which is beneficial to heat exchange in warm climates. Some watertight fabrics with

semi-permeable membranes enable the exchange of some quantities of moisture and increase thermal comfort.

Regular cleaning and drying are important for the thermal function of clothing and for personal hygiene. Manufacturers' cleaning instructions have to be followed for function.

Heat and heat-related illnesses

The popularity of football in all parts of the world and increasing numbers of international competitions in warm and humid climates may expose players, officials and visitors to warm or even tropical climates they are not used to.

Such climates therefore pose a health risk to the travelling party, particularly when not acclimatised. Organisers and other persons with responsibility should be prepared to evaluate the potential risks and take precautions.

Body and skin temperature

Small increases in muscle temperature render the muscles' energy systems more efficient, and this is made use of by warming up. In exercising muscles, temperature may reach 42°C (107.6°F). Body core temperature may exceed 40°C (104°F) during exercise (Table 2.6.1.1). Performance decreases when body core temperature reaches 40°C or more. This may affect the nervous system and reduce the ability to unload excess heat. With increasing temperatures, central nervous symptoms and serious brain damage may rapidly occur. Temperatures above 44°C (111°F) cannot be sustained without serious damage.

With increasing heat production, more and more cardiac output has to be directed to the skin to ensure heat exchange. This means that maximum performance may be limited in hot climates due to temperature regulation mechanisms.

Percentage of maximal oxygen uptake (VO_2max)	~ Increase in body core temperature
50 %	1.2°C
70 %	2.0°C
80 %	2.5°C
90 %	3.2°C

Table 2.6.1.1 Increase in body core temperature in relation to oxygen uptake

In warm environments, the skin is particularly important for temperature regulation if circulation is maintained. When water losses lead to decreased body water content, this will result in paradoxically cold, wet skin due to peripheral vasoconstriction and reduced sweat production. This highlights the importance of regular replacement of water losses as a precaution. As a rule of thumb, a 5% decrease in body water content will increase body core temperature by 1 to 1.3°C, mainly by impairing skin blood flow.

In football matches, the intermittent character of the effort, with short bouts of sprinting and running in between periods of walking, limits energy expenditure and allows players to adjust their speed according to the thermal load. However, modern football is more athletic and therefore energy expenditure may increase as compared to previously.

Football stadiums impose a higher risk on players than unprotected playing fields because of the construction which shields the playing field from winds. Furthermore, the concrete wall may reflect radiation and there is additional thermal radiation from heated walls.

At high ambient temperatures, additional cooling with water is a very good strategy to increase the heat exchange capacity of the skin because water has a far better heat capacity than air.

Water balance and sweating in hot environments

The water losses during exercise at 60% of VO_2max at 21°C (70°F) air temperature can be estimated as 1 L/h by sweating and 0.1 - 0.2 L/h by breathing. Additionally, approximately 0.5 grams of sodium chloride and other minerals (potassium, calcium and magnesium) are lost with sweat and approximately 50mg of potassium are lost with urine. These water and salt losses are increased when ambient temperature increases, but will decrease with acclimatisation.

Weighing is a good and simple way to measure water losses. Urine osmolality can be estimated visually (light colour = low osmolality: no problem; dark = high osmolality: water deficit) and can be measured using urine sticks. A further simple indicator of water balance is urine volume, which should be more than two litres/day (but not more than four litres/day, see also Table 2.6.1.2).

Haematocrit may be used as a means of assessing hydration status. The haematocrit is the cell part of the blood and is estimated by centrifugation. The haematocrit

should be below 50%, and optimum levels are considered to lie between 42 and 48%. Haematocrit is influenced by body position during sampling; it requires capillary blood sampling and a centrifuge.

Prevention of heat illness

The most important prevention strategy is heat acclimatisation, which can improve performance quite dramatically so that acclimatised subjects can easily perform an exercise in the heat that earlier was difficult or impossible to achieve.

The minimum time for heat acclimatisation of unacclimatised players is three days; total heat acclimatisation is achieved after 14 to 21 days.

Method	Limits	Special remarks
Weighing		Weight loss after arrival in hot environments and travel indicates water loss
Urine volume	> 2 l/day < 4 l/day	Simple method, indicates de- and over-hydration
Urine colour	Light yellow	Simple, yet often misleading
Urine osmolality	< 1015	Simple, cost for urine sticks or measurement tbc
Haematocrit	< 50%	Centrifuge needed, capillary blood needed

Table 2.6.1.2 Simple measurements of hydration status

Drinking

Adequate hydration is one of the key factors of heat tolerance, and part of a successful acclimatisation strategy to warm climates. Although the body can deal with fluid losses fairly well, reaction to dehydration cannot be "trained" or learned, and if it occurs impairs work tolerance and is a potentially risky condition.

As a rule of thumb, in addition to the basic intake of two litres per day, every hour of training will demand an additional litre of fluid. For each 5°C increase in ambient temperature above 25°C (77°F), one litre may be added to the drinking schedule. For hydration in sports, only hypotonic (water, mineral water, tea, etc.) and isotonic fluids (sports drinks, diluted fruit juices) should be used.

It is recommended to replenish 0.5 g loss of salt per hour of sweating. This amount may be somewhat lower in well acclimatised players. Many so-called mineral waters have very low sodium chloride concentrations which makes them equal to plain water.

Hot weather risk evaluation (Table 2.6.1.3)

The risk of high thermal load on hot, dry days can be approximated by considering the ambient temperature. For communication and preparation of organisers, ambient dry temperature is easily understood and helpful. When available, "Wet Bulb Globe Temperature" (WBGT) should be used to evaluate environmental heat stress. FIFA recommends using WBGT measurements or calculation whenever safety concerns have to be raised, usually when ambient temperature exceeds 32°C (89.6°F).

For calculation of WBGT and more detailed risk evaluation, several methods are available, e.g. the *Heat Stress Adviser* (author: J. Coyle, Tulsa, US; based on a Sports Medicine Australia (SA Branch) checklist for planning sports events) may be downloaded from the site *http://www.zunis.org/sports_p.htm*.

Ambient dry temperature	WBGT	Risk of thermal injury
25°-31.9°C (77-89.4°F)	24.0-29.3°C (75-85°F)	Moderate
32°-38°C (89.6-100°F)	29.4-32.1°C (85-89.9°F)	High
38°C and above (>100°F)	32.2°C and above (> 90°F)	Extreme

Table 2.6.1.3 Risk evaluation related to ambient temperature or WBGT (Wet Bulb Globe Temperature)

In conditions of high thermal risk, training and playing schedules should be modified. In competition, additional safety measures have to be taken.

In conditions of extreme thermal risk, training should be postponed to morning and evening hours, and the playing field may be closed during hours of extreme risk. Playing schedules must be modified.

- Additional shading for coaches and substitutes (sun shades).
- Supply of crushed ice and water for the teams.
- Cooling mist at both sides of the playing field.

Heat-related disorders

Heat-related disorders involve heat cramps, headache, hyperventilation, heat exhaustion, dehydration and exertional heat stroke. The signs and symptoms and the respective treatment are summarised in Table 2.6.1.4.

Dehydration may lead to heat exhaustion:

- Characterised by high heart rate, dizziness, headache, loss of endurance/skill, confusion and nausea.
- The skin may still be cool/sweating, but there will be signs of developing vasoconstriction, e.g. pale colour.
- Urine volume is small and highly concentrated.

Severe dehydration may lead to heat stroke:

- Characterised by symptoms similar to heat exhaustion, but with dry skin, confusion and collapse.
- Heat stroke may arise in a player who has not been identified as suffering from heat exhaustion and has continued his activity.
- Heat stroke is a medical emergency.

Cold and hypothermia

Cold exposure facilitates the loss of body heat which can reduce body temperature. Heat loss can be mitigated by heat conservation and/or increased heat production. Both exercise and shivering increase metabolic heat production helping to offset body heat losses in the cold. Exercise also increases peripheral blood flow facilitating heat loss, an effect that can persist for some time after exercise ceases. Prolonged exhaustive exercise may lead to energy substrate depletion, which compromises the maintenance of thermal balance in the cold. Hypoglycaemia lowers metabolic rate and shivering by mainly central mediated mechanisms to protect the brain.

When body temperature cannot be maintained by metabolism, vasomotor and thermogenic responses are elicited, both of which are mediated by sympathetic nervous activation. The effect of exercise on the heat debt during cold exposure depends on the heat transfer coefficient of the environment, the mode of activity and exercise intensity. The heat transfer coefficient is mainly influenced by ambient temperature, wind chill and clothing (material, air-tightness, wetness). When vasoconstrictor responses to cold are impaired, body heat conservation declines.

Chronic overexertion over many weeks impairs temperature control. When heavy physical activity in the cold is coupled with underfeeding for prolonged periods, the resulting negative energy balance leads to loss of body mass and a reduction in tissue insulation. These effects may be seen in some players during the winter season and may be responsible for decreased performance and an increased rate of injuries and infections. However, more severe hypothermia is not common in football.

Preventive measures in cold conditions are: appropriate clothing with heat insulation and/or wind shield liner, wearing a (woollen) cap, changing wet clothes immediately after training, a hot shower, sauna or hot bath, and dry and warm clothing after training. Nutrition should ensure sufficient intake of carbohydrates (maltodextrine) and appropriate drinking.

In extreme conditions with temperatures below –15°C (5°F), training schedules should be modified and shortened, and indoor training should be considered. Natural wind shields or buildings should be used for protection. Breathing difficulties and coughing may indicate cold air-induced bronchospasm or asthma.

Heat stress disorder	Cause and problem	Signs and symptoms	Treatment
Heat cramps	– Failure to replace salt lost through sweating – Muscle problems – Wrong training time (e.g. noon) – Wrong training suit (e.g. no cotton, only lycra, only nylon)	– Muscle cramps	– Rehydration – Supplementation of sodium chloride – Supplementation of calcium and magnesium – Avoidance of pre-game caffeine supplementation
Hyperventilation	– Overbreathing – Low blood CO_2 level – Low training status – Age	– Dizziness – Tingling around lips – Carpopedal spasm – Fainting	– Slow, deep breathing – Rebreathing from paper bag – Players trained in breathing techniques
Heat exhaustion	– Excessive heat strain with inadequate water intake – Failure to replace water loss – Cardiovascular problems (inadequate venous return, filling time) – Reduced skin perfusion – Increased sweating – Orthostatic hypotension – Wrong training suit – Increased humidity	– Weakness – Unstable gait – Fatigue – Wet, clammy skin – Headache > nausea > collapse	– Rest in shade – Cooling with water, shower – Rehydration – Pre-training proper hydration – Supplementation of magnesium and electrolytes – Very light meal in one hour's time
Physical exhaustion and dehydration Heat exhaustion combined with physical exercise		– Excessive work in heat – High Tc > 40°C (104°F)	– Rest in shade – Cooling with water, cold shower – Rehydration – Wear a cap made of suitable material – Use of sports sun glasses – Adequate electrolyte and carbohydrate replacement after every training session
Exertional heatstroke *Heat stroke is a medical emergency* **Classic heatstroke (rare in sports)**	Damage to or dysfunction of multiple organ systems is frequent High Tc > 40°C (104°F) Elderly and patients with serious underlying diseases, closed rooms, chronic dehydration	– Shivering – Mental status changes – Irrational behaviour to delirium – Convulsions – Loss of consciousness	– Rapid, immediate cooling – Cold-water immersion, ice packs – wrap in wet sheets and fan – Continue until Tc is < 102°F. – Intravenous rehydration – Cardiopulmonary resuscitation

Table 2.6.1.4 Heat stress disorders: causes and problems, signs and symptoms, treatment

2.6.2 High altitude

Introduction

This chapter is based on a consensus statement that was agreed upon by an expert panel at the invitation of F-MARC after an in-depth discussion of issues relevant to training and performing at different altitudes at a consensus conference at the Home of FIFA in October 2007. The review papers presented by the authors at that meeting were published recently (Bärtsch et al. 2008). The interested reader is referred to that publication for further references dealing with this topic.

Football, unlike many other sports disciplines, is a complex activity. For 90 minutes, the players, depending on their position on the field, not only have to perform repeated sprints, but also cover a total running distance of ten to 14 kilometres. The anaerobic and aerobic activities and the football-specific performance require skills which are decisive for the outcome of the match. On the one hand, the skills depend on training; on the other hand, they depend on intact cognitive functions and coordination controlled by neuromuscular mechanisms.

An extensive search of the literature does not reveal systematic research on football players or football teams. Most of the published papers address individual athletes in specific sports which cannot always be generalised for team sports such as football.

When playing at different altitudes, as well as the absolute altitude level, the difference between the starting (living) level and the playing level is important. The change in altitude will have a different impact on a player who lives, trains and plays near sea level and is going to play at high altitude above 3,000 metres than on a player who lives at a higher altitude of up to 2,000 metres and is going to play at high altitude above 3,000 metres.

Based on the literature and personal experience, there is high individual variability in the adaptation process to different altitudes which has to be considered when analysing the performance of an entire football team. Nevertheless, there is no evidence for generally different reactions to high altitude exposure between men and women.

In addition, it is important to understand the influence of different environmental conditions such as heat, cold and different relative humidity on the general health status and performance of the players. Extremes of these environmental conditions should be avoided in order to protect their health and minimise possible adverse effects on the players. This is also the subject of F-MARC studies currently being undertaken.

Finally, due to the different air density at moderate high altitude, the aerodynamics of the ball may change considerably, which in itself not only influences the flow of the game, but also has an impact on football-specific performance.

Definitions of altitude

0-500m	**"Near sea level"**
500-2,000m	**"Low altitude"**: minor impairment of aerobic performance becomes detectable
2,001-3,000m	**"Moderate altitude"**: mountain sickness starts to occur and acclimatisation becomes increasingly important for optimal performance
3,001-5,500m	**"High altitude"**: mountain sickness and acclimatisation become clinically relevant, and performance is considerably impaired
above 5,500m	**"Extreme altitude"**: prolonged exposure leads to progressive deterioration

It has to be stressed that the effects of altitude on performance and health show considerable variability between individuals. Therefore, the above definitions of altitude zones are average values that can differ between individuals by some hundreds of metres of altitude.

Performance in football at different altitudes

Players living at sea level or low altitude:

- will have to cope with a reduction in their aerobic performance when playing at moderate and high altitudes, which continually increases with the level of altitude. Highly trained players suffer higher reductions in performance than untrained individuals.
- will experience minor impairment of performance starting at 500 metres and substantial impairment above 3,000 metres.
- will not suffer impairment of performance in single sprints with altitude, but might suffer impairment of performance in repeated sprints due to an increased recovery time at moderate and even more so at high altitude.

Players living at moderate to high altitude:

– will show some improvement in aerobic performance with descent when playing at sea level/low altitude, but this improvement is limited by a lower capacity for oxygen usage.
– might have a disadvantage due to empiric observations of a lower performance on the first days at low altitude, but this area has been poorly investigated.
– Environmental factors (temperature, humidity, etc.) and different handling of the ball due to the altered aerodynamics will negatively affect the performance of players living at sea level or low altitude when playing at moderate and high altitudes. The opposite applies for players living at moderate/high altitude when playing near sea level or at low altitude.

Recommendations for preparation

These recommendations apply for all football competitions at altitude but their impact will naturally be greater for a competition as compared to a single match.

Acclimatisation

– Players ascending from sea level to low altitude do not necessarily require specific preparation because the reduction in performance is minor. However, above 1,500 metres, a short acclimatisation period of three to five days may help to achieve peak performance.
– For players ascending from sea level to moderate altitude, an acclimatisation period of one to two weeks at the location of the match is recommendable to achieve peak performance.
– For players ascending from sea level to high altitude, an acclimatisation period of at least two weeks at the location of the match is required to achieve peak performance.

Training concepts

– The "sleep/live high – train low" training concept:
  • is not recommended when playing near sea level. While enhanced aerobic performance at sea level has been observed after exposure to moderate altitude for at least 12-14 hours/day for a minimum of three weeks, it is unclear whether this translates into improved football performance.
  • cannot be recommended when playing at moderate or high altitude as the potential of this method of acclimatisation and any benefits for playing football have not been evaluated.
– The "intermittent hypoxic exposure at rest" concept is not recommended for playing football at any altitude as it does not improve performance.
– The "live low – train high" training concept:
  • is not recommended when playing at or near sea level as it does not improve performance.
  • is not recommended when playing at moderate or high altitude as the effects on performance and acclimatisation have not been sufficiently evaluated in high-level athletes and it is not practical for team sports.

Additional measures

– Attention should be paid to adequate fluid intake because loss through respiration and diuresis may be increased, particularly at high altitude.
– Nutrition should be high in carbohydrates, particularly at high altitude.
– Periods of rest should be prolonged and overexertion avoided during the first days after arrival at moderate or high altitude.
– Training schedules and intensity need to be adapted to the altitude-related reduction in performance.

Acute altitude illnesses

– In healthy players living at moderate or high altitude, there is no increased risk to their general health when playing at low altitude or near sea level apart from the general risk of injury.
– In healthy players living near sea level or at low altitude, there is no increased risk to their general health when playing at moderate or high altitude, apart from the individually variable risk of acute high altitude illness.
– Acute high altitude illnesses such as **Acute Mountain Sickness (AMS), High Altitude Pulmonary Edema (HAPE) or High Altitude Cerebral Edema (HACE)** do not occur at low altitude since there is a threshold altitude of about 2,000 metres.
– At moderate altitude, the risk of AMS for players living at sea level or low altitude is low and the illness usually mild. In healthy individuals, HAPE and HACE hardly ever occur at moderate altitude.
– At high altitude, particularly near and above 4,000 metres, the risk of AMS is considerable for players living at sea level or low altitude and the illness can be more severe.

Without proper treatment, AMS may progress to HACE. HAPE may occur particularly above 4,000 metres. HAPE and HACE are life-threatening diseases that can, however, be easily treated by supplemental oxygen and descent.

- The risk of AMS for players living at sea level or low altitude depends on their individual susceptibility, the degree of acclimatisation, the rate of ascent and the intensity of exercise.
- Mild AMS symptoms such as mild headache, loss of appetite, impaired sleep or dizziness do not prevent a healthy player from playing, but might still impair performance.
- Severe AMS symptoms, HACE or HAPE will prevent a player from playing.

Recommendations for prevention and treatment of AMS

- No specific ascent modality is needed when ascending for matches or competitions from low to moderate altitude.
- Staged ascent will prevent severe high altitude illnesses in players from near sea level or low altitude when playing a match or competition at high altitude.
- The "fly-in/fly-out" approach, which means arriving a few hours before the match and leaving immediately thereafter, may help avoid more severe forms of altitude illness at the cost of an almost maximum altitude-related reduction in performance due to lack of acclimatisation.
- For every 300-500 metres above 2,000 metres, one day of acclimatisation should be spent at an intermediate moderate altitude.
- In susceptible individuals, AMS can be prevented by drugs such as acetazolamide or systemic glucocorticosteroids, but these are both on the WADA list of prohibited substances.
- Moderate AMS can be treated with rest and non steroidal anti-inflammatory drugs (NSAID). If no improvement occurs within one day, or in case of severe AMS and the onset of HACE or HAPE, supplemental oxygen and descent are mandatory.

Bärtsch P, Saltin B, Dvorak J
Consensus statement on playing football at different altitude. Scand J Med Sci Sports 18 (Suppl 1):96-99

2.6.3 Air travel and jet lag

Introduction

Football is played throughout the world. The competitive schedule includes international matches between football clubs and representatives of national sides. At the top professional level, players may have to compete for their clubs and countries in different parts of the world in the same week. Teams within the USA, Canada, Australia and Russia may have to travel vast distances to fulfil their domestic fixtures, sometimes travelling across multiple time zones. Clubs may also travel great distances to play friendly matches, especially over the pre-season training period, or participate in warm-weather training. Such difficult itineraries are also encountered at youth level during international youth matches and among amateur teams undertaking recreational trips. The travel schedule induces travel fatigue resulting from the journey itself and a syndrome known as jet lag when multiple meridians are traversed.

Travel fatigue refers to the feelings of tiredness and stiffness due to travelling for a long time. It can occur during air flight in northerly or southerly directions or after long road journeys by car, bus or coach. It can be quickly reversed through rest, combined with light exercise and a shower or a short nap. In contrast, jet lag refers to symptoms that accompany flights across multiple time zones. The condition is more robust than travel fatigue and is caused by a desynchronisation of circadian rhythms, in other words, a disturbance of the body clock.

The symptoms of jet lag include intermittent periods of fatigue, lack of ability to concentrate, irritability and unusual mood disturbances, gastrointestinal upsets, and difficulty in sleeping at the appropriate time (see Table 2.6.3.1).

The main difficulty after a flight to the east is getting up in the morning, compared to travelling westwards when staying asleep for the night is a problem. The disembarkation schedule influences the severity of the malaise and the length of time for which symptoms persist. Where a long time has passed since the last sleep, jet lag may be worsened by a fatigue effect similar to the homeostatic drive for sleep. The choice of departure and arrival times and any stop-over en route is therefore important.

Likewise, activity during the flight is relevant. Diuretics such as alcohol or caffeine are best avoided, the preference being for water or fruit juices to compensate for the extra

losses incurred in breathing dry cabin air. Otherwise, the ensuing dehydration would compound the symptoms of jet lag. Light exercises such as isometrics or stretching, or walking periodically to the back of the plane can safeguard against deep vein thrombosis, which can be incurred following a long period of sitting inactive. Such activity can help in avoiding stiffness during the journey.

A strategy for coping with jet lag and hastening adjustment to the new time zone must be based on knowledge of circadian rhythms and how the body clock works. With this understanding, a travel strategy can be planned and implemented.

- Tired in the new daytime, yet unable to sleep at night.
- Delayed onset of sleep after eastward flight; early awakening after westward flight
- Unable to focus attention, lapses in concentration
- Motivation below normal, subjective fatigue above normal
- Decreased mental and physical performance during the new daytime
- Irritable and experiencing 'head-buzz' (headaches, disorientation)
- Poor appetite: decreased interest in food and enjoyment of meals
- Bowel irregularities; change in frequency of defaecation and consistency of stools

Table 2.6.3.1 The main symptoms of jet lag

The body clock

The body clock controls physiological functions in a rhythmic manner, following a 24-hour cycle. The phenomena are known as circadian rhythms and have exogenous as well as endogenous components. The endogenous rhythms (the body clock) are determined by activity in the suprachiasmatic nucleus cells of the hypothalamus, whereas exogenous factors include environmental variables such as temperature and light as well as social and physical activities. These specialised hypothalamic cells have receptors for melatonin, a hormone produced by the pineal gland during darkness and inhibited by light at other times of the light-dark solar day. The suprachiasmatic nuclei receive information about light through the retinohypothalamic neural tract and about general excitement in the nervous system via the intergeniculate leaflet (see Table 2.6.3.2).

A number of clock genes have been identified in molecular studies, the cyclic interactions between clock genes and clock proteins recurring naturally over about 24.3 hours (circadian) in a negative-feedback loop of clock gene expression. The resultant rhythm is fine-tuned to an exact 24-hour period by external factors that include ambient temperature, activity and feeding.

The body clock is sited close to areas of the brain where temperature, the autonomic nervous system, hormone secretion, feeding cycles and the sleep-wakefulness cycle are regulated. Circadian rhythms are observed in the majority of physiological systems, the curve in core body temperature corresponding to a cosine wave being a prime example. As many functions closely follow the phase of the core temperature curve, body temperature is often used as a marker of the body clock. There is also evidence that many measures of human performance vary in a circadian fashion

Input pathways	Note
Retinohypothalamic tract (information about light)	Visual rods and cones Melanopsin
Intergeniculate leaflet (information about nervous excitement)	Influenced by exercise
Superior cervical ganglion (Receptors for melatonin secreted in the pineal gland are found in the suprachiasmatic nuclei)	Connects the pineal gland to the suprachiasmatic nuclei

Table 2.6.3.2 Neural connections with the suprachiasmatic nuclei, the anatomical site of the body clock.

that is close in phase with the body temperature rhythm (see Figure 2.6.3.1).

Such rhythmic variation has been found in muscle strength, anaerobic power, reaction time, range of motion, the pace of exercise, and in skills such as dribbling and chipping a football. Circadian rhythms are disrupted after travelling across multiple time zones so impairment in performance would also be expected to occur.

Symptoms of jet lag are due to the dissociation that occurs after travelling across time zones between body clock time and local time in the new environment. The symptoms are more severe and last longer with the more time zones traversed and are usually worse after flying eastwards than following a westward flight. The reason for this difference is that the extra time after travelling west is in line with the increased natural rhythm length of slightly greater than 24 hours. Symptoms are transient and partly dependent on time of day, being sensed most sharply when the urge to sleep during the new daytime is strongest.

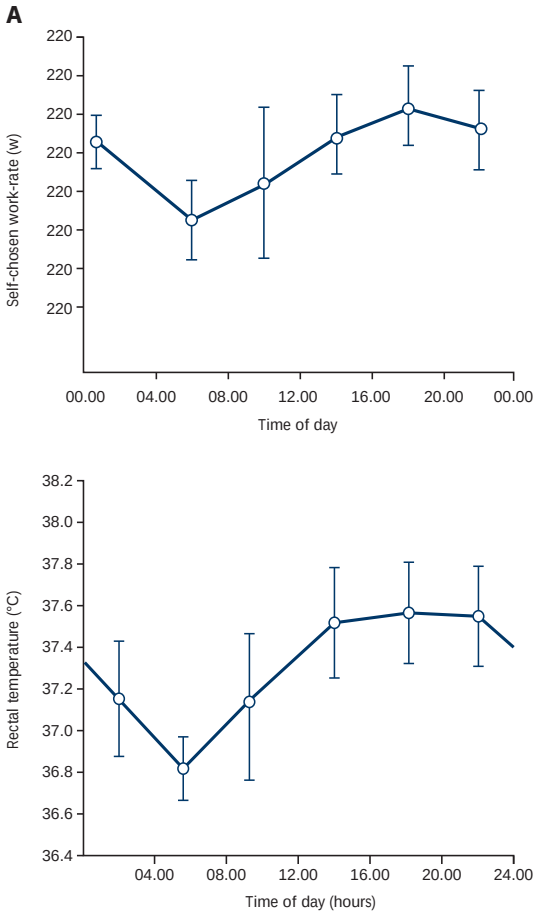


Figure 2.6.3.1 The circadian rhythm in the exercise pace chosen spontaneously at different times of day (A) and the rhythm in core body temperature (B). The temperature minimum normally occurs mid-sleep at around 05.00 hours. Mean values (± SD) are shown.

It is thought that deterioration of the body clock with age causes older people to suffer worse jet lag than younger counterparts. Experienced travellers tend to learn from their excursions and find individual means of improving their coping behaviour. Football officials, team support staff and players are all affected. Nevertheless, there are individual differences that affect tolerance of jet lag. "Morning type" individuals possess body clocks that tend to run faster than the population average, a potential advantage when a phase advance is required i.e. after flying eastwards. In contrast, "evening types" should cope best with a phase delay, needed after flying westwards. There is a genetic basis for individual differences, people with the 5/5 variant of the PER3 clock gene tending to suffer disproportionately from effects of sleep loss. Generally, nobody is immune to the effects of jet lag but an attitude of "mental toughness" can assist in minimising subjective effects.

As a rough guide, about one day is needed per time zone crossed for the body clock to adjust completely. Extraneous rhythms such as digestion and physiological measures influenced by external factors, e.g. heart rate, tend to adjust relatively quickly and before the normal sleeping pattern is restored. Jet lag is generally absent by the time the core temperature curve is restored to its normal rhythm. During the period when the body clock is adjusting to the new time zone, the individual is vulnerable to decrements in performance. During this time, light exercise rather than strenuous training or competitive matches is advised, in order to reduce the risk of injury due to impaired skills. Errors that occur may go undetected and mistakes in decision-making during play may not be recognised as such.

In their study of travellers between the United Kingdom and New Zealand, Reilly and Mellor demonstrated that the normal circadian rhythm in muscle strength was reversed for up to one week. Travellers to Australia from the United Kingdom showed symptoms of jet lag for eight to nine days after arrival, having crossed ten time zones. Similarly, Olympic Games athletes demonstrated disrupted rhythms in leg and back strength for five days (see Figure 2.6.3.2) after crossing five time zones in a westerly direction. Indeed, there is evidence of impaired team performance in American football when the time-zone transition is three hours, that is, from coast to coast in the USA Such transitions are common in association football matches, in both Asia and Europe for example.

Coping with jet lag

Adjust or not!

Where trips are over fewer than two to three time zones, the effects of jet lag are small but nevertheless call for attention. One solution is to behave as if on home time throughout the visit, that is, according to body clock time. Adjustments need to be made for waking time, the timing of meals and training sessions, and the time of retiring to bed. This strategy may be used by teams from the English Premier League playing in continental Europe in midweek and in the domestic league during the preceding and the following weekend.

A similar strategy may be adopted for brief visits of two to three days. It should be possible to organise daily activity over this period according to body-clock time. The task of staying on home time becomes much more difficult when multiple time zones, more than four, are crossed due to the problems in avoiding and seeking exposure to light at the appropriate times. In this instance, the solution is to arrive in good time for adjustment of the body clock to have occurred by the time of competition.

Pre-adjustment of the body clock in advance of travelling is not a viable option for competitive footballers. Such a phase shift of the normal circadian rhythm induces a malaise similar to that experienced by nocturnal shift workers. These symptoms could compromise training in the days before departure; furthermore, the manoeuvre leads to a partial adjustment only. Nevertheless, a shift of one to two hours for one to two days prior to long-haul flights could be of benefit when a large phase advance is required, by ensuring the physiological adjustment is initiated in the correct direction.

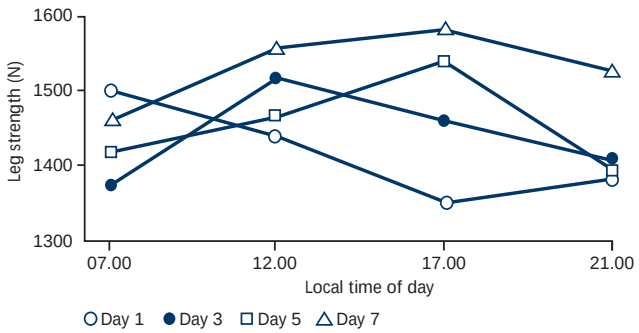


Figure 2.6.3.2 Change in leg strength (Newtons) on days 1, 3, 5 and 7 after a westward flight across five time zones. Time of day is shown according to local time in the new environment.

There is no physiological adaptation to repeated disturbances of the circadian rhythm, so individuals do not necessarily adjust any better (or worse) than in previous journeys. Airline staff as well as occasional travellers experience jet lag, although the long-term health consequences of frequent long-haul flights are unknown. Female flight attendants experience secondary amenorrhea, linked to interactions between female reproductive hormones and altered melatonin rhythms. Female football players are as vulnerable as their male counterparts to effects of jet lag but the syndrome has not been examined in detail in this group.

Pharmacological approaches to adjustment

Pharmacological agents have been proposed as a first line means of ameliorating the effects of jet lag. These substances include both stimulants and soporifics, although effectiveness depends on their phase-shifting properties i.e. whether the drug acts directly on the body clock as a chronobiotic. Extracts of melatonin and its synthetic analogues have gained in usage but as yet no single pill has been identified as an antidote to jet lag.

Some substances used in military contexts for countering jet lag might be considered for use by athletes. These drugs include treatments for narcolepsy, attention deficit disorder and other conditions with modafinil (banned in-competition for athletes), methylphenidate, pemoline, and related agents. Caffeine may help maintain daytime alertness but can cause unwanted effects in recovery sleep if taken too late in the evening.

Benzodiazepines are the drugs used in sleeping pills and known as the minor tranquillisers; they are helpful in initiating sleep but not necessarily maintaining it. Their use with athletes has been advised only for rare circumstances and under a physician's supervision. No benefit was noted for temazepam when administered to British athletes travelling across five time zones from London to Florida, USA. Benzodiazepines such as diazepam and loprazolam have longer half-lives than temazepam and can cause hangovers that impair performance the following morning. So-called non-benzodiazepines like zolpidem interfere less with homeostatic processes in the sleep-wake cycle than the hypnotics and are less likely to have hangover effects. Zolpiden, zopiclone and zalephon have short half-lives and affect short-term memory less than other benzodiazepines but were ineffective in reducing jet lag in travellers between

France and Martinique. Zopiclone appeared to have some benefit by hastening re-adjustment of the rest-activity rhythm and restoring the phase relationship between the rhythms of sleep and core temperature.

Melatonin has been suggested as the best chronobiotic therapy for jet lag due to the key role of the endogenous hormone in regulating circadian rhythms. Although not available in Europe except on prescription, it can be purchased off-the-shelf in some countries. Its effects are relatively rapid, having a half-life of 35-50 minutes. It has been found to be ineffective in travellers between the United Kingdom and Australia, the main difficulty being the timing of administration to suit its phase-response curve. Melatonin administered in the evening and before the fall in body temperature to its trough should advance the body clock whereas a phase delay follows ingestion after the body temperature has reached its nadir. The hormone causes drowsiness in preparation of the body for sleep but has no hangover consequences the following morning. The advice to athletes has been to refrain from using melatonin, unless the individual has already had positive experiences with it and knows its precise effects on him or her.

A behavioural approach to adjustment

A behavioural strategy is preferable to the use of drugs in helping to cope with jet lag. The effects of melatonin are opposed by light and so the timing of exposure to bright light is key in implementing a behavioural approach. Light demonstrates a phase-response curve, opposing the effects of melatonin. Exposure to natural or artificial light before the trough in core temperature promotes a phase delay whereas a phase advance is encouraged by light administered after this time, meaning "body-clock time". Exposure to light at 22.30 in Los Angeles following a flight from London (UK) would promote a phase advance on the first nights rather than the required phase delay, when administration occurs after the trough in core temperature. Where natural daylight cannot be exploited, artificial light from visors or light boxes can be effective for phase-shifting purposes. In contrast, dark glasses or dark rooms should be used at times when light should be avoided (see Figure 2.6.3.3). Exercise itself may act to re-synchronise the body clock but is likely to be more effective for inducing a delay than for an advance.

Behavioural strategies for coping with jet lag depend to some extent on the direction of flight. A flight westwards requires a phase delay, and after arrival, light training sessions can be undertaken at any time of the day. It is important to avoid long naps at midday since these rests will have a counter-productive effect of anchoring circadian rhythms to home time. In contrast, light exercise, exposure to daylight and social activities can allow external factors to aid the adjustment, the individual being encouraged to retire early to bed on the first evenings. Once the individual has adapted to the new time zone, an afternoon nap can be encouraged.

Following a long-haul flight eastwards, a short lie-in can be taken for the first one or two mornings. This extension of the sleep period is in line with the desired phase advance. Exercise and exposure to bright light should be avoided in the morning, since these stimuli could promote a phase delay rather than the required phase advance when administered before the trough in core body temperature. In a large group of travellers between the United Kingdom and Australia, more people adjusted by a 14-hour delay than by a ten-hour phase advance. It would be important for a travelling football party to have a common strategy so that adjustment is in a uniform direction for the group as a whole.

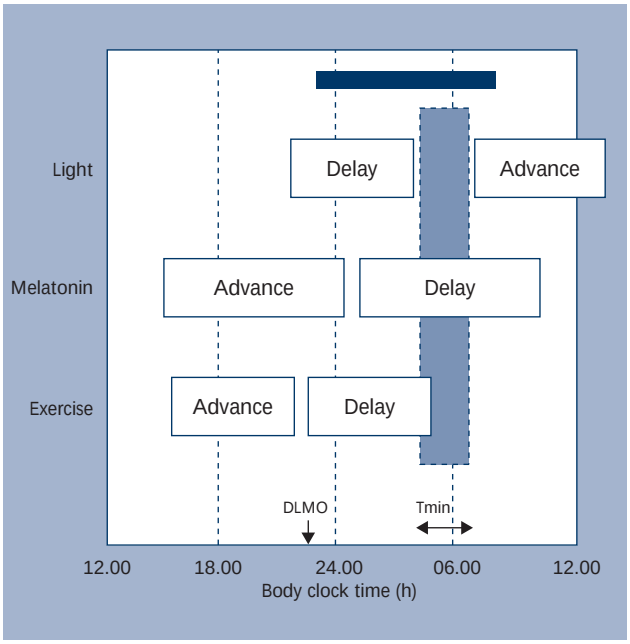


Figure 2.6.3.3 Phase shifts (advances and delays) of the body clock produced by light, melatonin ingestion and exercise at different times of the day. Body clock markers, dim-light melatonin onset (DLMO) and the minimum of core temperature (Tmin) are shown, and the shaded area shows the range of Tmin that is usually seen. The horizontal black bar indicates normal sleeping time.

The return journey

Once the competition is over, the immediate focus must be on the return journey and promoting recovery from competitive exertions. Attention is directed towards restoring hydration status and carbohydrate stores. The travel strategy is not simply a duplication of activities on the outward journey since the direction of flight and the new itinerary must be taken into account. The time the match ends and the distance to the nearest convenient airport are also relevant.

For matches that end late in the evening, the group may stay on overnight in its accommodation and participate in light training the following morning before returning home. Where it is feasible, some teams prefer to return home straightaway after the match. In this case, the players can compensate for sleep deprivation by "sleeping in" the next morning and training at a low to moderate intensity in the afternoon. This option is valid where no or only a few time zones are traversed. For long-haul flights across multiple meridians, sleep can be attempted on board the plane and a behavioural strategy adopted for re-adjustment of the body clock to the home country.

Overview

Travel across vast distances is now common among football teams for recreational, training or competitive purposes. International players are especially subject to arduous itineraries that entail playing matches in both the home country and the new environment. Knowledge of chronobiological principles can help to manage the fatigue due to travel stress and to cope with jet lag when journeys are across multiple time zones. Guidelines can be drawn up for individual journeys, in preference to generic advice, to help ameliorate the effects of jet lag and improve coping mechanisms. An education programme can assist in guiding individual players in cases of difficult journeys away from their clubs. It could include advice about preparing for the journey, behaviour on-board the flight and following arrival.

There is no single cure for jet lag and modifications of diurnal activity are likely to be more effective than using sedatives or hypnotics. Exposure to and avoidance of bright light at the appropriate times, coupled with the correct timing of training, are key elements of a coping strategy. Support personnel and officials should be included, as well as the players, when travel strategies are being drawn up. Attention to detail should help ensure that a successful journey is not marred by discomfort and performance impairment during the trip.

2.7. Prevention of doping

FIFA introduced regular doping controls in 1970 to ensure that the results of the matches in its international competitions are a fair reflection of the strength of the competitors. During the last fifteen years, prominent doping cases, particularly in individual sports, have alerted the public to the problem of doping. As suspicion grew of greater drug abuse at all levels, including amateurs and recreational sportsmen, doping increasingly became a concern for international and national sports organisations and governments alike. However, public discussion is not always characterised by expertise and insight into the complexity of doping in sports.

The team physician obviously plays a crucial role in the fight against doping and medication abuse in football. He educates players and coaches in nutrition, supplements and the allowed means of performance enhancement. He leads by example through his prescription attitude and knows about the health risks of doping substances and medications alike. He needs to be familiar with the latest Prohibited List International Standard and the TUE requirements for his players' level of play as well as with what constitutes an anti-doping rule violation.

2.7.1 Aims of anti-doping strategies in football

- Uphold and preserve the ethics of football
- Safeguard the physical health and mental integrity of the player
- Ensure that all competitors have an equal chance

It is the duty of each physician to protect players from harm and ensure that they can compete on a level playing field. The strategy of FIFA in the fight against doping as world football's governing body focuses on education and prevention. Any decisions and regulations are based on the specifics of the game, scientific evidence and the analysis of validated doping statistics.

2.7.2 Definition of doping

Doping is any attempt either by a player himself, or at the instigation of another person, such as his manager, coach, trainer, doctor, physiotherapist or masseur, to enhance mental and physical performance unphysiologically or to treat ailments or injury – when this is medically unjustified – for the sole purpose of taking part in a competition. This includes using (taking or injecting), administering or prescribing prohibited substances prior to or during a competition. These stipulations also apply for out-of-competition testing of anabolic steroids and peptide hormones, as well as for substances producing similar effects.

Other prohibited methods (e.g. blood doping) or manipulation of the samples collected are likewise classified as doping. The detailed definition as regards anti-doping rule violations is contained in the World-Anti Doping Code and the annually updated FIFA Anti-Doping Regulations.

2.7.3 The magnitude of doping in football

Any discussion of anti-doping strategies should be based on risk assessment and established facts. In this respect, FIFA developed its own database to keep track of records on samples being reported as positive to allow control of the management of these samples within the different confederations, member associations, national anti-doping agencies (NADOs) and within FIFA itself.

Since 1999, two players per team have been drawn by lots and tested at every match during FIFA competitions. No-advance-notice tests were broadly introduced at training camps prior to the 2002 FIFA World Cup™. Between 1994 and 2008, 6,384 doping tests were performed at FIFA competitions. Of these, three samples tested positive: one

each for ephedrine, cannabis and nandrolone, an incidence of 0.05%.

The total number of samples collected and analysed in football during the year allows the total incidence of positive samples (2004: 0.34%, 2005: 0.33%, 2006: 0.32%), the most commonly detected prohibited substances as well as the distribution of positive samples in the different confederations to be calculated. In 2007, 28,313 doping tests were performed in football worldwide. According to the FIFA doping control database, 91 samples (0.32%) tested positive and, of these, eleven samples (0.04%) were positive for anabolic steroids. Over the years, cannabis and cocaine have accounted for about 80% of positive test results, whilst in 2007 about 61% of positive samples were due to cannabis and cocaine (Figure 2.7.3.1).

The relatively low incidence of positive doping samples, particularly for true performance-enhancing drugs such as anabolic steroids (2004: 0.04%, 2005: 0.06%, 2006: 0.03%, 2007: 0.04%) and stimulants indicates that there is no evidence of systematic doping in football. However, this does not mean that doping does not exist in football. It is often speculated that team sports such as football are not as prone to abuse of performance-enhancing substances as individual sports for a number of reasons, and this seems to be affirmed by the high number of positive cases in individual sports at the Olympic Games as compared to team sports. It remains to be seen how the extension of out-of-competition testing will influence statistics, although the results of such testing, e.g. in the UEFA Champions League, are not so far indicative of a different trend.

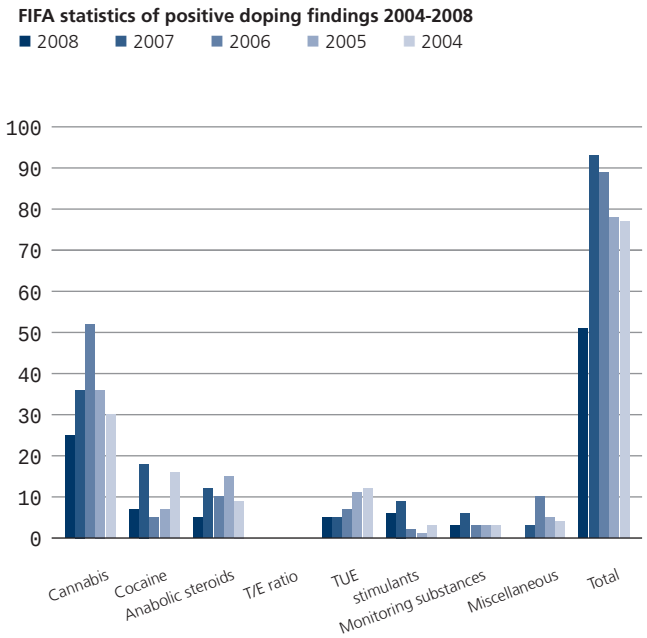


Figure 2.7.3.1 FIFA statistics of positive samples 2004-2008 (For 2008, analysis of data is not yet finalised; 66 pending cases, incl. T/E ratio)

2.7.4 Doping substances

Stimulants

These drugs stimulate the central nervous system to reduce tiredness while increasing alertness, competitiveness and aggressiveness. They can enhance explosive power and endurance performance because one can exercise more strenuously and be less sensitive to pain. The stimulants comprise several different groups of substances.

Amphetamines are used in sport to enhance endurance, sharpen reflexes and reduce tiredness. The health risks to the player are considerable, as evidenced by several amphetamine-linked deaths during strenuous effort. Amphetamine-induced heatstroke and cardiac arrest are two examples of the possible health risks.

Cocaine is the most potent naturally-occurring stimulant and probably the most addictive agent known. Its misuse is linked to its euphoric effects and a feeling of decreased fatigue. Its increasing use as a recreational drug is reflected by the fact that cocaine accounts for about one-third of all positive doping controls in football. The initial "rush" or sense of well-being, of having more energy and being more alert, quickly wears off, often leaving the user feeling more depressed than before. Contrary to popular belief, cocaine does not really enhance performance, whether on the job, in sport, at school or during sex.

Ephedra alkaloids are naturally-occurring stimulants obtained from ephedra plant species. Ephedrine could also be inadvertently ingested because of its wide availability in over-the-counter medications, mainly as a decongestant, and in numerous nutritional and dietary supplements as an energy stimulant and anorexic agent. Research shows that isolated use of ephedrine, pseudoephedrine and phenylpropanolamine at dosages typically found in medications has only an inconsistent, and probably

insignificant, ergogenic benefit in terms of power, endurance, strength and speed.

Anabolic-androgenic steroids

Since the middle 1950s when an artificial version of testosterone was being used to enhance weightlifting performance, sport has been prey to an ever-expanding array of anabolic-androgenic steroids (AAS), synthetic derivatives designed to enhance anabolic properties and minimise androgenic actions. AAS are effective in enhancing athletic performance due to their anabolic effects, provided the player also consumes adequate protein and trains intensely. The trade-off, however, is the myriad of adverse side-effects that jeopardise health, such as damage to the liver, heart and vessel diseases, hormonal disturbances (including infertility), behavioural changes, psychological problems as well as tendon lesions.

Testosterone and related hormones have their own category in the list of forbidden substances (S1). To prove AAS abuse in sport, the laboratory has to differentiate between endogenously-produced steroids and synthetic steroids. The ratio of testosterone to epitestosterone (T/E) is used to screen for possible abuse since the intake of testosterone causes characteristic changes in this ratio in the urine. If this ratio exceeds a certain value, further tests are conducted which may verify or exclude the intake of AAS. The finding of synthetic steroids in the urine is a severe doping offence.

Nandrolone, or 19-nortestosterone, is one of the most widely-used synthetic anabolic-androgenic steroids by athletes who need power and muscle strength. It is derived from testosterone, but has far more powerful anabolic properties. Athletes use nandrolone to accelerate muscle growth and increase lean body mass, strength and aggressiveness or to achieve faster recovery from intense training.

A potential doping offence involving nandrolone use is based on the detection of the two major metabolites, 19-norandrosterone (19-NA) and 19-noretiocholanolone (19-NE). These metabolites can be detected in urine for several days after oral ingestion or for months after injection into the muscles. Elimination is strongly dose-dependent and individual. Over the last decade, concerns about the origin of nandrolone metabolites found in the urine led to the identification of several factors that might influence a positive finding. For example, physical effort can have different, but certainly no systematic effects, on the excretion of metabolites.

Erythropoetin (EPO) and blood doping

Increasing oxygen transport capacity by stimulating the production of red blood cells in the body is the principle behind high-altitude training and also lies at the heart of the abuse of recombinant EPO (rhEPO), a synthetic version of the endogenous erythropoietin, or blood transfusions. Excessive red blood cells increase the risk of blood clots due to higher viscosity of the blood, even more so in combination with the training-induced slow heart rate and low blood pressure of endurance athletes. Furthermore, the blood, already thicker with the extra red blood cells, might become even thicker during heavy exercise at high temperatures as fluid is lost from the body. Therefore, these methods carry a considerable risk of death from a stroke or heart attack.

In the 1970s, blood transfusions were a common practice, but this method virtually disappeared when rhEPO became available on the market. Unfortunately, the doping test for EPO introduced in 2000 induced a return to the "ancient" method of blood transfusions. This method of doping is particularly targeted through longitudinal blood profiles.

Human growth hormone

Today, the use of HGH in sport is not only based on its anabolic properties, but also on its effects on carbohydrate and fat deposits in the body. The extent to which HGH actually improves performance is still under debate. In adults with HGH deficiency, injection of the hormone increases the muscle mass while decreasing the fat mass. It also shows favourable effects on exercise capacity as well as kidney and heart function. These positive effects are, however, less clear in athletes. There is a remarkable difference between the objective results from scientific studies and subjective reports by abusers. HGH is frequently used in combination with anabolic steroids or EPO.

The long-term risks of hGH abuse are not well known since there is no data describing the well-being of healthy sportsmen. Acromegaly, which has complex symptoms including swelling of the hands and feet, coarsened facial appearance, joint pain, fluid retention and excessive sweating is often cited as a major risk of excessive use. An abuser may also be at risk of diabetes, hypertension, heart muscle damage and osteoporosis. HGH belongs to category

S2 "Hormones and related substances" on the Prohibited List. While its detection in urine is unreliable and expensive, new blood tests may identify HGH abuse.

2.7.5 Inadvertent doping

There are two main reasons for inadvertent doping, of which a physician needs to be fully aware.

1. Use of nutritional supplements

The use of nutritional supplements carries a considerable risk of an inadvertent doping offence. Contamination of dietary supplements by substances that may cause a positive doping test is widespread – some surveys have suggested that as many as one in four supplements may result in a positive test. These prohibited compounds have not been declared on the label, so there is no way for the player to know that they are present. According to the World Anti-Doping Code, however, ignorance is not an acceptable excuse for a positive test result.

At the same time, whilst there is no evidence that prohormones such as androstenedione and norandrostenedione are effective in enhancing muscle mass or strength, these prohormones are promoted for use and are readily available in shops and via the internet. They will result in negative health consequences for the player as well as positive drug tests. Many herbal supplements are claimed to increase testosterone levels and hence have an anabolic action. However, these claims are based on experiments carried out in test tubes, and none has been shown to work in humans. The physician must caution all players against the use of supplements. At present, the purity of most commercially available supplements cannot be guaranteed. The only way to be sure is to avoid supplements altogether.

2. Prescription of prohibited substances for medical reasons without a therapeutic use exemption

In a player who has a medically confirmed pathological condition, drugs containing prohibited or partially prohibited substances could be permitted in exceptional cases if:

- The player's health would be impaired if the prohibited drug were withheld.
- No performance enhancement could result from the prohibited substance being administered as medically prescribed.
- No permitted or practical alternative drug is available in place of the prohibited substance.

In such a situation, the player requires a therapeutic use exemption (TUE) approval allowing him to use the substance. If such a request is not made, or the TUE has not been granted, the finding of the respective prohibited substance in a urine or blood sample is considered a doping offence.

2.7.6 Therapeutic use exemption

In the situation described above, a player or his physician must submit a formal application to the responsible granting body (see Table 2.7.6.1), requesting a TUE for the substance in question. A player may not apply to more than one anti-doping organisation for a TUE. The application must identify the player's affiliation, and the specific competition, if applicable, for which the application is being made. The application must list any previous and/or current requests for permission to use an otherwise prohibited substance or prohibited method.

A TUE application submitted to the FIFA TUE advisory group must include a comprehensive medical history and the results of all examinations, laboratory investigations and imaging studies relevant to the application. The arguments related to the diagnosis and treatment, as well as duration of validity, should follow the "WADA Medical Information to Support the Decisions of TUECs". The application must include a statement by the physician attesting to the necessity of the otherwise prohibited substance or prohibited method and describing why an alternative, permitted medication cannot, or could not, be used in the treatment of this condition. The dose, frequency, route and duration of administration must be specified. If anything changes, a new application should be submitted.

For treatment of asthma, the use of inhaled beta-2-agonists reflects current clinical practice. For players included in any of the FIFA testing pools and for any players participating in a FIFA competition, the use of these substances requires a prior TUE approved by FIFA. The use of these substances must also be declared on the FIFA doping control form 0-1 to be completed by the team physician at the time of testing. The use of inhaled glucocorticosteroids must also be declared on the TUE application form for asthma and on the FIFA doping control form 0-1 (see below).

The medical file to be used for a TUE application in the case of asthma and its clinical variants must include the following to reflect current best medical practice: 1) a complete medical history detailing the presence of symptoms typically related to asthma during and after exercise, e.g. fatigue, prolonged recovery and poor performance, as well as the onset and severity of symptoms related to exercise, notably the disappearance of symptoms after cessation of exercise, as well as any influencing factors (e.g. environmental conditions, respiratory tract infections); 2) a comprehensive report on the clinical examination with specific focus on the respiratory system; 3) a spirometry report with a forced expiratory volume in one second (FEV1) measurement at rest (peak expiratory flow measurements are not accepted); 4) if airway obstruction is present at rest, spirometry needs to be repeated after inhalation of a short acting beta-2 agonist to demonstrate the reversibility of bronchoconstriction; 5) in the absence of reversible airway obstruction at rest, a bronchial provocation test is required to establish the presence of airway hyperresponsiveness; this may be either in the form of an exercise test or a metacholine challenge.

Glucocorticosteroids used by non-systemic and non-inhaled routes, namely intraarticular, periarticular, peritendinous, epidural and intradermal injections, are frequently used to treat medical conditions encountered in football players. These substances, for which the route of administration is not prohibited, will require a declaration of use. A player (through his physician) must declare the use of the substance in question on the doping control form 0-1 at the time of testing.

Decisions of the FIFA TUE advisory group are completed within thirty days of receipt of all relevant documentation and will be conveyed in writing to the player by the FIFA Anti-Doping Unit. In the case of a TUE application made in reasonable time prior to a competition, the FIFA TUE advisory group use their best endeavours to complete the TUE process before the start of the competition. The requirements of the TUE application process are laid out in detail in the FIFA TUE policy and the FIFA Anti-Doping Regulations and are based on the WADA International Standard for Therapeutic Use Exemptions.

2.7.7 Doping control administration within football

At national level, the NADOs are the primary body for all doping matters. They are authorised to grant TUE exemptions to national level players and organise out-of-competition controls at national level in close coordination

Level of play	TUE application sent to	Application submitted by
National players participating in domestic competitions only	National anti-doping agency (NADO), or other authorised national body such as National Olympic Committee	Player and/or club physician
International players called up to compete in international team competitions and friendly matches at confederation level	Confederation	Player and/or national team physician
International players participating in international club competitions	Confederation	Player and/or club physician
International players participating in FIFA competitions (incl. FIFA World Cup™ qualifying matches) or part of FIFA testing pool	FIFA TUE granted by confederation recog-nised	Player and/or national team physician

Table 2.7.6.1 Granting bodies for TUEs

with the FIFA member associations. The confederations and/or member associations that come under FIFA's umbrella carry out their own doping controls at the competitions they stage. Urine and or blood samples collected must be analysed at WADA-accredited laboratories. These laboratories send reports on any "chemically positive" A samples – so-called Atypical Findings and Adverse Analytical Findings (AAFs) to the member associations, the FIFA Medical Office and WADA as appropriate.

The FIFA Medical Committee has overall responsibility for implementing doping control at all FIFA competitions and out-of-competition as well as for approving applications for therapeutic use exemptions (TUE). It delegates the organisation of doping tests to the FIFA Anti-Doping Unit, which coordinates the FIFA doping control officers. It delegates the evaluation and the approval of TUEs to the TUE advisory group.

The administrative management of sample collection sessions for doping controls is carried out by the FIFA Medical Office. The in- and out-of-competition testing schedule follows the FIFA Anti-Doping Strategy and the annual testing plan. Blood testing has not yet been implemented at competitions but is continuously evaluated.

Once the FIFA Anti-Doping Unit receives the result of a confirmed AAF in an A sample collected by another anti-doping organisation, follow-up information is required from the NADO, member association and/or confederation in question in order to learn of the results of the B sample and particularly the relevant disciplinary committee's decision. If the information is not provided, the FIFA Disciplinary Committee takes appropriate action.

2.7.8 FIFA network of doping control officers (DCOs)

FIFA requires a doping control officer to be a physician. Physicians play the key role in FIFA's long term strategy of prevention and education in the fight against doping, following their Hippocratic oath as well as their professional and ethical values. Over the years, F-MARC has developed a worldwide network of specialists who are involved in the educational process within the confederations and member associations but also in the practical performance of sample collection at international and FIFA competitions.

Recruitment

a) at FIFA competitions

The team physicians and the local organising committee (LOC) DCOs observe and attend a considerable number of doping controls at a competition, thus learning far more than could be taught in a course or workshop. The team physicians, LOC DCOs or LOC medical officers (MOs) can fill out a personal details form on which they can indicate whether they would be interested in working as a FIFA DCO. The relevant FIFA MO is asked to evaluate the individuals based upon his experience at the competition, which helps to identify the most suitable applicants.

b) at FUTURO III courses, other courses and workshops

Taking part in a half-day or day course can give an idea of the FIFA procedure, but never qualifies the participant to conduct doping controls. Course participants, if interested, can fill out the personal details form and would then require additional training.

c) Confederations

The FIFA Anti-Doping Unit contacts the confederations to see whether FIFA could use their DCOs for FIFA assignments.

Education and training

a) Doping Control Workshop

The aim of this workshop is to gather all the DCOs together, both experienced and less experienced, and ensure a common, "FIFA" approach to doping control. The workshop is dedicated to:

- informing DCOs of any changes in the FIFA Anti-Doping Regulations or procedure, the World Anti-Doping Code or the International Standards, as well as their practical implications
- presenting new scientific results and new analytical methods
- sharing personal experiences, in particular of out-of-competition no-advance-notice testing
- discussing problems, posing questions and proposing ideas for improvement of the procedure

This workshop takes place at least every two years in the major confederations. The basic layout of the workshop is the same in all confederations. The FIFA Anti-Doping Unit invites all DCOs to this course. Attendance of courses

is mandatory for every DCO, if not every year then every second year.

b) Gaining experience

For the newly-recruited DCOs from competitions, confederations or courses, it is important to gain a level of experience with regards to the FIFA procedure for doping control tests. Therefore, they undergo the following training:

- attendance of the annual or bi-annual FIFA doping control workshop;
- accompanying an experienced DCO to a control mission in order to observe the procedure;
- accompanying an experienced DCO for a second time to a doping control mission, but this time conducting the procedure themselves with the experienced DCO as an observer. He would then report back to the FIFA Anti-Doping Unit.

Upon successful completion of this programme, an applicant is accredited and certified as a FIFA DCO.

The way forward

Some experts advocate out-of-competition testing and longitudinal blood profiling with indirect proof instead of substance detection as the future of the fight against doping. Such an approach obviously involves considerable investment, which needs to be carefully weighed against the potential benefits. Thorough discussion and reflection on the subject of so-called "intelligent testing" is required by all those involved in sports and anti-doping, especially those with practical experience and knowledge of sports-specific issues. An individual approach based on sound risk assessment, statistics, background information and organisational and logistical aspects of the different sports is indispensable. Furthermore, the focus should also be directed away from testing itself and sanctioning the athlete towards other parties, such as those who provide and offer doping substances as well as those who administer prohibited substances to and use prohibited methods on athletes. FIFA strongly believes that physicians who engage in doping and put the health of players at risk also need to be heavily sanctioned.

2.8 Use and abuse of medication

The use of prohibited and non-prohibited substances is a reality of elite-level international sport. While the consumption of illegal substances such as anabolic steroids, growth hormones, erythropoietin and others receives considerable attention from the public and sports organisations alike, physicians primarily concerned about the health of their players are growing equally apprehensive about the extent to which legal substances such as beta-2 agonists, non-steroidal anti-inflammatory drugs (NSAIDs) or corticosteroids are prescribed in football. At the Olympic Games 2000 in Sydney, 80% of the athletes declared that they were using some sort of medication. More recent data on medication use by professional football players indicated a high intake of both supplements and NSAIDs.

Unlike usual anti-doping practice, since the 1998 FIFA World Cup France™, the FIFA Medical Committee has requested team physicians at FIFA doping controls to report not only any prohibited substances, but also any non-prohibited substances, whether prescribed or taken by the player himself as an over-the-counter product. This requirement not only applies to the players selected for doping control, but to all players on the official player list. Laid down in the FIFA Anti-Doping Regulations, this demand arose less from fears of doping than from a concern about the risk posed to players' health by non-prohibited, but nevertheless potentially harmful medications. To receive an accurate picture of actual medical practice in elite-level international football, F-MARC decided to analyse the doping control forms reporting on medication use at FIFA competitions.

The use of medication at FIFA World Cups™

As part of the FIFA doping control procedure, team physicians are required to supply information on any medication and nutritional supplement taken by all their players during the 72 hours prior to a match on a specific form. F-MARC analysed these doping control forms from a total of eight FIFA World Cups™ (2002 and 2006 FIFA World Cups™; FIFA Women's World Cups 2003™ and 2007™; FIFA U-20 World Cups 2005 and 2007; FIFA U-17 World Cups 2005 and 2007).

The average intake of medication was 0.51 substances per match for male youth (U-17 and U-20), 1.0 for senior female and 0.75 for senior male players. The highest number of medical substances used by a player prior to a match was nine.

Medications

Analgesics

Painkillers and anti-inflammatory drugs were the most prescribed medications, accounting for 45.5% of all reported substances. When comparing the use of painkillers with the injury reports provided by the team physicians after each match, F-MARC found that there was no correlation between their use and the reported injuries. Furthermore, there was no correlation between the final ranking of the team and the average number of medications taken per match.

a) Non-steroidal anti-inflammatory drugs

Every third female and male senior player was taking NSAIDs prior to each match, and nearly 70% of all players were taking NSAIDs at least once during the competition, regardless of whether they participated in match play

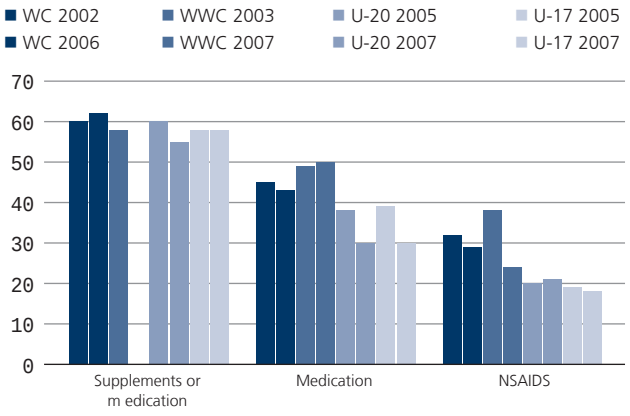


Figure 2.8.1 Comparison of the percentage of players taking either medication, nutritional supplements or both prior to a match at four different FIFA competitions

(see Figure 2.8.1). The intake reported for youth players was somewhat lower than in the senior teams. Every fifth player was taking NSAIDs prior to each match and 43.3% of the players took NSAIDs at least once during the competition.

For some players, team physicians reported up to four different NSAIDs at a time, and one team physician reported NSAID use in 22 out of 23 players prior to each single match of the competition. The only exception in this team was one player who "only" used NSAIDs prior to four out of five matches.

b) Local anaesthetics and injected corticosteroids

Local injections were reported three times more often in male senior players than in female or youth players. In men, 3.8% of all players were receiving an injection of either a local anaesthetic or corticosteroid prior to a match, and almost every tenth player had at least one injection during the course of the competition. Corticosteroid injections accounted for 43% of all local injections in men's, and 23% in women's and youth competitions.

Beta-2 agonists

Use of inhaled beta-2 agonists for treatment of asthma was reported significantly more often in female than in male senior and youth football players. In total, however, only 1.8% of football players were using inhaled beta-2 agonists during the FIFA competitions.

By contrast, up to 30% of endurance athletes have been reported to use such medication. Current medical opinion agrees that there is insufficient evidence for the ergogenic effect of the short-acting inhaled beta-2 agonists salbutamol, terbutalin and the longer-acting salmeterol and formoterol in non-asthmatic athletes. Among the beta-2 agonists, clenbuterol is considered an anabolic agent and is listed under S1. Urine samples containing higher concentrations than 1000ng/mL of salbutamol are, however, reported as an adverse analytical finding despite the granting of a therapeutic use exemption..

Nutritional supplements

The intake of nutritional supplements was reported as 1.02 for youth, 0.63 for senior female, and 1.01 for senior male players. This high consumption of nutritional supplements contrasts with very little scientific evidence regarding their beneficial effects in football. Most experts on sports nutrition are of the opinion that nutritional supplementation has very little effect on performance even in elite football players, and indiscriminate use of such supplements is strongly discouraged (see also 2.5). Despite the claim of producers that their supplements reduce body fat or build stronger muscles or speed recovery, the reality is that many products contain substances that are either on the prohibited list or may be harmful to a player's health, or both.

Conclusion

It had been postulated that there were four types of medication use in professional athletes: legitimate therapeutic use, performance preservation (treatment of sports injuries), recreational/social use, and performance enhancement. While the high intake of medication at the four FIFA World Cups™ as well as the four youth World Cups was alarming indeed, F-MARC could not scrutinise the underlying reasons for such use.

While there was no indication of beta-2 agonist overuse, the extensive use of NSAIDs in all groups examined was of particular concern as it was not supported by scientific evidence. While these drugs have an uncontested painkilling effect, the high prevalence of considerable side effects, particularly with extensive and long-term use, is well known. Negative influences of NSAIDs on renal function have been reported. Their influence on the healing process is controversial and alternative substances are available. The findings at FIFA competitions indicate that despite recommendations to restrict their use, the use of NSAIDs appears to have been broadened to almost any painful condition in football. It has to be recognised, however, that to date, there are no evidence-based guidelines for the use of painkilling agents in sports medicine literature.

The broad use of nutritional supplements reported by elite players is in sharp contrast to the lack of evidence for any positive effect on performance. In addition, the risk of a positive doping test when using supplements is considerable. Often, the critical ingredients that could lead to an adverse analytical finding are not declared on the product label.

There is considerable need for initiating a dialogue with team physicians to provide a better understanding of the issue of medication use in football. Furthermore, FIFA's educational efforts with regard to the risks and benefits of nutritional supplements have to be intensified in order to raise awareness among players and team support personnel.

3. Injuries

3.1 Ankle injuries

3.1.1 Incidence

The ankle is one of the most commonly injured joints in football. Ankle injuries constitute 12-23% of all injuries recorded during FIFA competitions. The average proportion of ankle injuries is 20% over the different skill and age levels, being higher (35%) among low-level amateur players and children and adolescents.

	Injuries	Complaints without injury
FIFA competition, male	12-23%	-
FIFA competition, female	17-22%	-
Annual prevalence	20%	22%

Table 3.1.1.1 Ankle injuries and complaints without injuries

3.1.2 Functional anatomy

Inversion trauma, which causes about 85% of all ankle injuries, usually results in damage to the lateral ligaments. Three anatomically and functionally separate units – the anterior talofibular, the calcaneofibular and the posterior talofibular ligaments – provide ligament support on the lateral side. Normally, the anterior talofibular ligament is torn first (about 50% of the injuries are isolated ruptures of the anterior ligament), then the calcaneofibular ligament is also torn, and only in rare cases (about 1%) are all three of the lateral ligaments torn. The proportion of patients with combined ruptures (i.e., ruptures of both the anterior talofibular and the calcaneofibular ligaments) is higher among patients who have been injured before.

Eversion trauma usually causes a ligament injury on the medial side to the deltoid ligament (a continuous ligamentous unit that runs along the entire medial malleolus). Medial ligament injuries occur with or without simultaneous syndesmosis injuries and fractures of the lateral malleolus. Isolated ligament injuries on the medial side are rare, totaling about 1% to 2% of the ligament injuries in the ankle. There are probably several reasons for this, including a movement pattern in which a natural landing occurs with the foot in plantar flexion and slight supination. A more critical factor, however, may be that the deltoid ligament has greater rupture strength than the lateral ligaments. For that reason, eversion injuries usually cause fractures or syndesmosis injuries in addition to the medial ligament injury.

3.1.3 Classification and grading

When a player reports to the physician or physiotherapist after suffering an "ankle sprain", the first objective is to determine which structures have been injured and to what extent. In the vast majority of cases, an ankle sprain results in injury to the lateral ankle ligaments. As mentioned above, the anterior ligament (the anterior talofibular ligament) is injured first, then the middle ligament (the calcaneofibular ligament). In some cases, other structures may be involved (Table 3.1.3.1). In some cases, injuries of the medial ligament (the deltoid ligament) may result and fractures are also seen on occasion. In children, growth plate injuries can result from this type of trauma, whereas older players are more likely to sustain a fracture of the lateral malleolus or at the base of the fifth metatarsal. Note that syndesmosis injuries can occur alone or in combination with ligament injuries or fractures.

Although a number of different injury classification systems exist for the most common injury type, lateral ligament injuries are usually classified according to the number of ligaments involved (Figure 3.1.3.1). Grade I can be defined as a partial rupture of the anterior talofibular ligament and/or the calcaneofibular ligament; grade II as a total rupture of the anterior talofibular ligament, but

with an intact calcaneofibular ligament; and grade III as a total rupture of the anterior talofibular ligament and the calcaneofibular ligament. Ankle fractures can be classified according to the location of the fracture line through the lateral malleolus (AO-classification). However, these classification systems are less important in the acute stage, where the main goal is to distinguish between a lateral ligament injury, a fracture or some other injury that may require early immobilisation or acute surgical treatment.

Common	Less common	Not to be missed
Lateral ligament injury	Medial ligament injury	Syndesmosis injury
	Anterior capsular injury (footballer's ankle)	Growth plate injury
	Fractures: - Lateral malleolus - Medial malleolus - Base of the fifth metatarsal - Talus - Calcaneus	
	Dislocated ankle	
	Tendon rupture/dislocation (tibialis posterior, peroneal, Achilles tendon)	

Table 3.1.3.1 Diagnoses to consider after an acute ankle sprain.

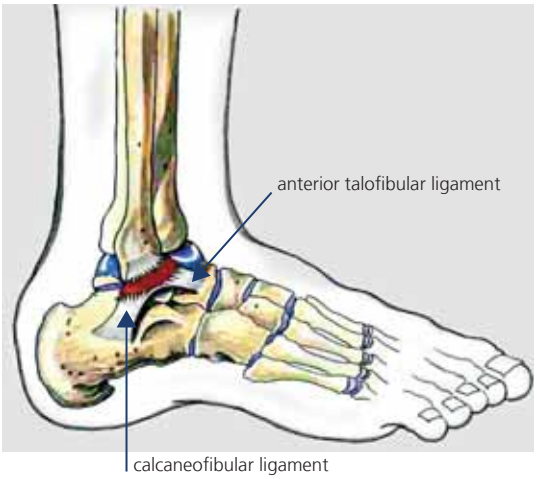


Figure 3.1.3.1 Grade III lateral ligament injury – combined injury to the anterior talofibular and calcaneofibular ligaments. Illustration reproduced with permission from Clinical Guide to Sports Injuries (Bahr and Maehlum, 2003) ©Gazette Bok/T. Bolic.

3.1.4 Causes and mechanisms

The injury mechanism is an important clue to diagnosis after an ankle sprain. The typical injury mechanism is landing with the foot in an inverted position, i.e. plantar flexed, internally rotated and supinated. With the foot in this position, the ankle joint is inherently unstable. The posterior talar plafond is narrower than the anterior portion, thereby reducing the bony stability of the ankle mortise when the foot is plantar flexed. Unless the dynamic musculotendinous ankle stabilisers can compensate for this reduced structural stability when the ankle joint is perturbed, the ligaments that statically stabilise the lateral ankle are acutely overloaded when the player puts weight on his inverted foot. In football, such perturbations often result from tackles, where the player receives a laterally directed hit on the medial side of the ankle or lower leg, whereupon landing in a supinated position leads to an inversion sprain (Figure 3.1.4.1).

As the foot inverts and the subtalar joint over-supinates, the ligaments fail in a predictable pattern, rupturing sequentially in an anterior to posterior direction. The forces involved determine both the extent and degree of ligament injury. In about half the cases, there is an isolated tear of the anterior talofibular ligament only, in about 25% there is a combined rupture of the anterior talofibular and

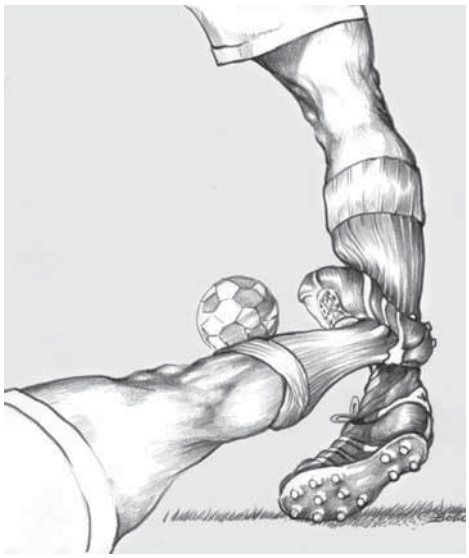


Figure 3.1.4.1 Typical mechanism for lateral ligament injury in football: Opponent contact to the medial side of the leg, causing the player to put weight on an inverted ankle. Illustration reproduced with permission. ©Oslo Sports Trauma Research Center/T. Bolic.

calcaneofibular ligaments, whereas additional rupture of the posterior talofibular ligament is rare (1%). If there is an eversion injury (pronation and external rotation), a medial ligament injury must be suspected, but this rarely occurs. Nevertheless, if the injury mechanism is atypical, the team physician or physiotherapist should maintain a heightened suspicion for injuries other than the typical lateral ligament injury.

An injury mechanism that seems to produce an injury type specific to football is forced plantar flexion of the ankle joint. This occurs when the player is blocked when attempting to shoot or clear the ball (Figure 3.1.4.2). Hitting the opponent's foot when kicking with the foot in a full equinus position can lead to injury of the anterior capsule of the ankle joint, eventually giving rise to capsular inflammation, loose bodies and osteophyte formation (footballer's ankle).

Figure 3.1.4.2 Likely mechanism for development of footballer's ankle. Illustration reproduced with permission. ©Oslo Sports Trauma Research Center/T. Bolic.

3.1.5 Risk factors

The most important risk factor that has been identified for ankle sprains is a previous ankle injury. In fact, research has shown that among senior players, four out of five ankle sprains occur in previously injured ankles (an observation made in other sports as well). Compared to an ankle with no prior injury, the risk of injury is five times greater for an ankle that has been sprained one or several times. Furthermore, the more recent the injury, the higher the risk. The injury rate during the first 6-12 months after an ankle sprain is five to ten times greater than for an ankle without previous injury. Therefore, adequate rehabilitation of an ankle sprain is critical to prevent re-injuries.

3.1.6 Symptoms and signs

In the case of a lateral ligament injury, the player will have felt something tear on the lateral aspect of the ankle. Patients experience swelling and tenderness around the lateral malleolus. Findings are usually distinctly localised over the ligaments of patients examined immediately after the injury occurs, and it is easy to distinguish between a ligament injury and a fracture. A localised haematoma may be evident as early as a few minutes after the injury. If untreated, within the next hours the size of the haematoma will increase gradually, and if the player does not seek medical attention until one or two days after the injury occurs, they often have significant swelling and ecchymosis in large sections of the lateral side of the ankle. In such cases, it may be difficult to distinguish between ligament injuries and fractures.

3.1.7 Examination and diagnosis

The goal of the initial physical examination is to decide whether the patient has a lateral ligament injury, and not a different injury requiring surgery or immobilisation. The diagnosis is based on clinical examination, where the site of pain on palpation is important. If there is no pain on palpation of the anterior talofibular ligament, there is no lateral ligament rupture. Palpation is reliable for the first few hours after injury, and even more accurate after four or five days, when swelling has subsided. Ankle X-rays are indicated only if there is bone tenderness according to the Ottawa ankle rules (Figure 3.1.7.1) or the patient is unable to bear weight both immediately following the injury and at the time of the subsequent clinical assessment. These guidelines, followed correctly, detect all clinically significant fractures (100% sensitivity).

Injury to the tibiofibular syndesmosis can be diagnosed by a number of specific tests (Figure 3.1.7.2). The "squeeze test" is performed by compressing the fibula against the tibia about halfway between the knee and ankle. If the syndesmosis is injured, this manoeuvre will produce local pain. The "external rotation test", performed by externally rotating the foot with the ankle in neutral degrees of flexion, is considered positive if the athlete complains of pain in the region of the syndesmosis. These tests are reasonably specific, i.e. they usually do not cause significant pain if only the lateral ankle ligaments have been injured. A positive test necessitates radiographic evaluation to rule out injury to the syndesmosis. Simultaneously, a Maisonneuve (caput fibulae) fracture has to be ruled out.

It is often claimed that the anterior drawer and talar tilt tests can be used to clinically evaluate whether the ankle is mechanically unstable after a significant lateral ligament injury. From a theoretical anatomic and biomechanical perspective, the anterior drawer test should be positive if the anterior talofibular ligament is torn, while the talar tilt test should be positive if the calcaneofibular ligament is also ruptured. However, studies have shown that these tests have limited diagnostic value in the acute phase of injury, as they do not enable the clinician to distinguish between total and partial ligament ruptures, or between isolated or combined lateral ligament injuries. Furthermore, the treatment of ankle sprains is not dependent on the degree of ankle instability demonstrated on stress radiographic views. Therefore, the talar tilt and anterior drawer tests and stress X-rays have no clinical relevance in the evaluation of ankle sprain injuries in the acute stage; however, they have their place in testing for chronic instability.

If signs indicate that a fracture may be present according to the Ottawa ankle rules, a routine X-ray investigation is indicated (images obtained should include anteroposterior, lateral and mortise views). Also, the same radiographic investigation is indicated if the physical examination has raised suspicion of a syndesmosis injury. Other imaging studies are usually not indicated in the acute phase.

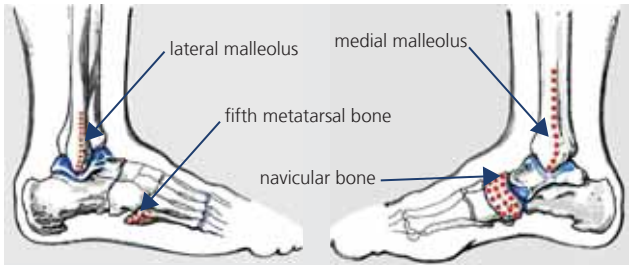


Figure 3.1.7.1 Ottawa ankle rules: Ankle or foot X-rays are only necessary if there is pain on palpation along the margins of the lateral or medial malleoli, the navicular bone or the head of the fifth metatarsal bone, as indicated by the red dots. If distinct tenderness cannot be detected in these locations, and the player can bear weight on the foot, X-ray examination is not necessary in the acute stage. Illustration reproduced with permission from Clinical Guide to Sports Injuries (Bahr and Maehlum, 2003) ©Gazette Bok/T. Bolic.

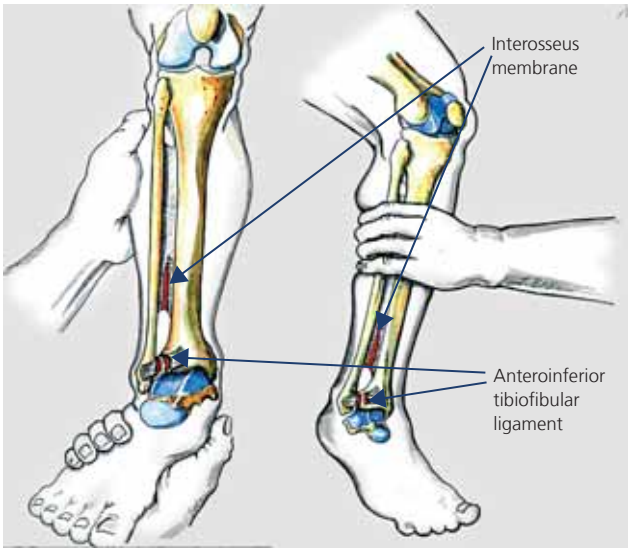


Figure 3.1.7.2 Tests for syndesmosis injury: (A) The external rotation test, and (B) the squeeze test. Illustration reproduced with permission from Clinical Guide to Sports Injuries (Bahr and Maehlum, 2003) ©Gazette Bok/T. Bolic.

3.1.8 On-field treatment

The goal of the on-field assessment is to determine if there is a significant ligament injury, and if so, to remove the player from the pitch for further assessment and on-field treatment. Whether a ligament injury has occurred can usually be established with reasonable certainty based on history ("What happened?") and quick palpation over the anterior talofibular ligament in front of and below the lateral malleolus ("Does this hurt?"). A ligament injury results in immediate bleeding from severed vessels in the ligament and surrounding joint capsule. The goal of the on-site treatment of acute ankle sprains is therefore to minimise bleeding and swelling. This may be accomplished by providing immediate protection, rest, ice, compression and elevation ("PRICE" treatment, see Figure 3.1.8.1). Of these early interventions, compression is

probably the most important to limit bleeding, while the main effect of cold therapy is to provide analgesia. Intermittent cold treatment provides effective pain relief and can be given for 20-30 minutes every two to three hours. Cold treatment can be given by simply using cold, running water or dedicated cold therapy equipment. If PRICE treatment is initiated immediately following the sprain injury and compression is provided continuously for the first 24-48 hours thereafter, the amount of bleeding or swelling following a ligament injury can be limited significantly.

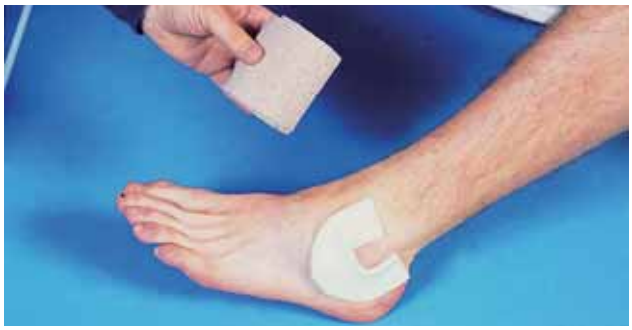
Do not bear weight on the ankle – no walking or testing. A quick examination to determine that there is lateral injury is all that is needed at this stage. Mix the contents of an ice bag by crushing the inner bag and shaking the bag carefully.

Place the ice bag with the centre over the tip of the lateral malleolus. Fasten the proximal end of the ice bag with an elastic wrapping. If ice is being used, it is advisable to put a wet cloth in between.

Fasten the distal end of the ice bag – continue fastening the ice bag with the elastic wrapping to apply firm compression using the ice bag as a compression tool.

Place the patient with the ankle elevated as much as possible and the cold/compression bandage on for at least 30 minutes.

Avoid weight-bearing when the patient needs to be moved – provide crutches if possible. Keep the cold/compression bandage on during transportation even after the cold effect has subsided.



A more complete examination can be done after the initial 30-minute PRICE treatment has been completed. Compression bandage treatment is continued for the first 48 hours using an elastic wrapping with a felt or paper filling around the malleolus to provide maximum pressure on the injured ligaments.

Figure 3.1.8.1 Initial on-site management of acute ankle sprains. Illustration reproduced with permission from Clinical Guide to Sports Injuries (Bahr and Maehlum, 2003) ©Gazette Bok/T. Bolic.

3.1.9 Non-operative and operative treatment

Conservative management of lateral ligament injuries is recommended, even if there is evidence of a combined injury to the anterior talofibular and calcaneofibular ligaments. Although there may be advantages to surgical repair, in most cases functional treatment provides the quickest recovery of full range of motion and return to play, does not compromise mechanical stability any more than other treatments, and is safer and less expensive. Surgery is therefore not indicated as primary treatment of ankle sprains. The goals of a functional treatment programme are to minimise initial injury, swelling and pain, to restore range of motion, muscle strength and neuromuscular control, and then graduate to a sport-specific exercise programme prior to return to competition. Analgesics can be used to provide pain relief, but acetylsalicylic acid (aspirin) can prolong bleeding and should therefore be avoided. Non-aspirin pain relievers and anti-inflammatory medication are good alternatives, and may also accelerate recovery by permitting earlier active range of motion, earlier weight-bearing and earlier return to play.

During the rehabilitation period, it is important to protect the ankle from new injury by taping or bracing the ankle. Tape or a semi-rigid brace should be worn during both everyday and sporting activities where there is an increased risk of re-injury (even walking on uneven terrain). The athlete should protect the injured ankle with a brace until a balance training programme has been completed.

3.1.10 Rehabilitation programme

After the initial bleeding phase is over, the goal of treatment is to regain normal, pain-free range of motion. Increased range of motion can be achieved through passive, active or active-assisted stretching exercises, and by submaximal exercise on a cycle ergometer. The exercise programme should progress (according to the improvement in function and degree of symptoms) from progressive linear movements – e.g. toe-raises, squats, jogging, jumping in place on two legs, then one, skip-rope jumping – to cutting movements – e.g. running figures of eight, sideways jumping, sideways hurdle jumps. The goal of these exercises is to gradually progress towards sport-specific exercises.

An important goal in the successful rehabilitation of an ankle sprain injury is to re-establish neuromuscular control of the ankle through a programme of balance exercises. Proprioceptive function is impaired in patients with residual functional instability after previous sprains, and can be improved by balance board exercises. Such programmes can reduce the risk of re-injury to the level of a previously uninjured ankle. Neuromuscular training should be carried out for six to ten weeks after an acute injury.

The programme is performed as balance exercises on one leg on a disk or foam mat (Figure 3.1.10.1). The original programme described by Tropp consists of ten minutes five times weekly. His evaluation suggests that in football players with functionally unstable ankles, the programme results in an improvement in sensorimotor control for six weeks, and a further (although smaller) improvement during the next four weeks, but no further protective effect was observed after that. Two studies have shown that early balance training after a primary ankle sprain provides protection from recurrent injuries. Thus, it appears reasonable to recommend six to ten weeks of balance board training to all players with an acute ankle sprain (see also 2.2.3).

3.1.11 Prognosis and return to play

The prognosis is good. There is usually complete function in six to eight weeks, but this can often be achieved much earlier with good PRICE treatment during the acute stage. However, the scar tissue takes much longer to

Figure 3.1.10.1 Example of one leg balance exercise on an ankle disk.

heal, maybe as long as a year. This means that return to play is possible before complete healing of the ligament has taken place. Consequently, protecting the ankle with bracing or taping is advocated during this period, at least until a balance training programme has been completed. In addition, the player should undergo a series of functional tests before being allowed back into football practice, and tested in football practice before playing games. Although most patients do well, as many as 10% to 20% of the patients have persisting problems after an acute ankle injury (see below). Therefore, the patient should be told to contact his physician if he has late symptoms.

3.1.12 Sequelae and their treatment

While most patients with a lateral ligament injury seem to do well following functional treatment, some players will develop residual symptoms and persistent complaints. The prevalence of chronic ankle problems following sprain injury has ranged between 18% and 78% in different studies. It is therefore important to instruct players during the acute phase of rehabilitation to follow up if they have persistent problems after completing a programme of functional rehabilitation.

Players with residual complaints after an ankle sprain can be broadly classified into two groups: those complaining of pain, stiffness and swelling, and those with recurrent sprains and episodes of ankle instability. The cause of pain, stiffness and residual swelling is often chondral or osteochondral injury of the ankle joint. Such lesions are more common after high-energy injuries, such as when landing after a maximal jump, and may therefore be expected to occur rather often in football players. Focal uptake on a bone scan may indicate that there is an osteochondral injury. CT or MR scans can be used to differentiate between subchondral fractures and chondral fractures with or without separation and/or displacement. Players with persistent symptoms and chondral injuries should be referred to an orthopaedic surgeon. Pain may also result from impingement of scar tissue, particularly in the anterolateral corner of the ankle joint.

Ankle instability may be described as either mechanical or functional in aetiology. Mechanical instability can occur after complete ligament tears if the scar tissue is lengthened and provides inadequate mechanical support, while functional instability results from inadequate sensorimotor control of the ankle joint. Some players can suffer from both mechanical instability and loss of sensorimotor control. Subtalar instability may also result from ankle sprains, and the sinus tarsi pain syndrome may occur as a sequela of a lateral ankle sprain injury.

The anterior drawer and talar tilt tests may be used to assess the mechanical stability of the ankle joint in such chronic cases, and stress X-rays are used by some clinicians to quantify and document the degree of instability. However, the large variability in talar tilt values in both injured and non-injured ankles precludes the routine use of these diagnostic tests. Several authors have shown that proprioceptive function is reduced in athletes who complain of a feeling of persistent instability following an ankle sprain. A simple functional balance test may be used to estimate sensorimotor control, although the predictive value of the test has not been properly documented. The player is instructed to stand on one leg for one minute with arms held across the chest, eyes fixed forward and the opposite leg straight down. The test is said to be normal if the player can complete one minute on one leg and during at least 45 seconds of this time not have to adjust balance other than at the ankle (i.e. using the knees, hips or shoulders to keep balance). The test result is supra-normal if the player can complete an additional 15 seconds with his eyes closed.

Players with persistent instability symptoms should complete at least ten weeks of intensive proprioceptive training. The affected ankle should be taped or braced to prevent re-injury during this period. If instability episodes persist even after an adequate sensorimotor training programme has been completed, the players should be referred to an orthopaedic surgeon for further evaluation and management. Surgical stabilisation of the lateral ligaments may be indicated in such cases.

3.2 Knee injuries

Incidence

Next to the ankle, the knee is the most frequently injured joint of players at FIFA competitions (Table 3.2.1).

	Injuries	Complaints without injuries
FIFA competition, male	9-23%	-
FIFA competition, female	0-10%	-
Annual incidence, male	18%	17%

Table 3.2.1 Knee injuries and complaints without injuries

Functional anatomy

Knee stability depends on the passive and active stability of the joint. Passive stability depends on the geometry of the articular surfaces, ligaments, meniscus and fibrous capsule (Figure 3.2.1). Active stability is exerted by the muscles surrounding the knee under contraction. The most important stabilising muscles are the quadriceps, hamstrings, sartorius, gracilis and gastrocnemius.

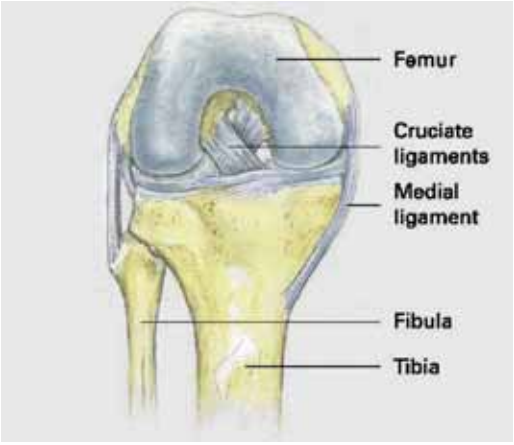


Figure 3.2.1 Knee joint, anterior view. Medial (collateral) ligament (MCL) and lateral collateral ligament (LCL) provide medial/lateral stability; medial (MM) and lateral meniscus (LM) add general stability; anterior cruciate ligament (ACL) and posterior cruciate ligament (PCL) provide anterior/posterior stability of the tibia in relation to the femur.

Active stability can be improved by neuromuscular training and enhancing muscular function. The passive stability of the knee cannot be improved by training.

Classification and grading

The different types of injuries include:

- Ligament injuries to the anterior cruciate ligament, posterior cruciate ligament, medial collateral ligament and lateral collateral ligament;
- Meniscus injuries to the medial and lateral meniscus;
- Articular cartilage injuries to the tibia, femur and patella;
- Fractures of the tibia, femur and patella.

The most common injuries concern the medial collateral ligament and meniscus. However, the most common serious injury concerns the anterior cruciate ligament (see Figure 3.2.2 and Chapter 3.2.1). Muscle tendon injuries are also common in the knee region.

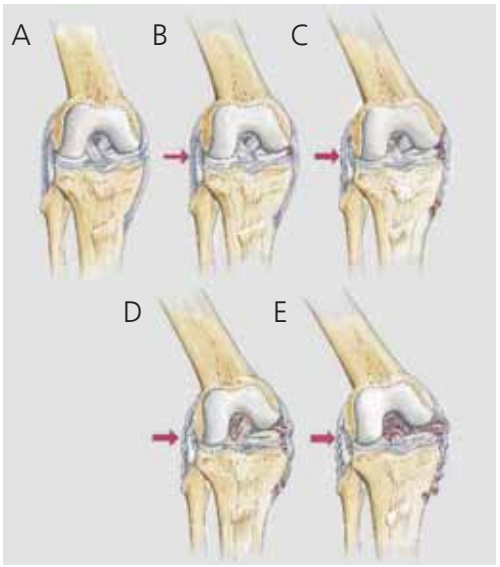


Figure 3.2.2 Normal knee joint (A) and injuries of increasing severity: deep (partial) MCL injury (B); complete MCL injury/tear (C); with additional ACL injury (D); with additional PCL injury (E).

Causes and mechanisms

Most knee injuries in football are caused by either body contact or direct impact, transferring extrinsic forces to the player, or by intrinsic forces without contact, generated by the player himself when running, accelerating, decelerating, "cutting", twisting and turning. The two most common causes occur during tackling: in the first one, the impact hits the lateral side of the knee, forcing the knee into valgus and the tibia into external rotation, causing a sequence of injuries with progressive severity. The same mechanism of sudden enforced valgus is found, for example, when two players hit the ball at the same time with the inside of their foot. The second important cause in tackling is an impact hitting the medial side of the knee, forcing the knee into varus and the tibia into internal rotation (Figure 3.2.3). The same mechanism of sudden enforced varus can be seen when a player is hit on the outside of the foot, forcing internal rotation of the lower leg. The same mechanism of injury is found when the foot is fixed on the ground and the player turns, resulting in intrinsic forces causing varus or valgus stress on the knee and external or internal rotation of the tibia.

Risk factors for knee injuries include joint laxity, muscle weakness and fatigue, inadequate rehabilitation after previous injury, poor fitness and foul play with tackle on the lateral or medial side of the knee leading to the mechanism as described above. In female players, inadequate landing after jumping may cause injuries of the anterior cruciate ligament.

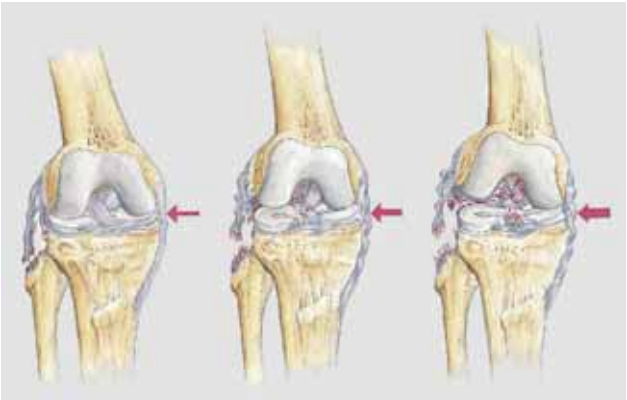


Figure 3.2.3 Stages in the development of injury caused by impact against the medial side of the knee. Left: LCL ruptures on moderate impact. Middle: with more violent impact, ACL also ruptures. Right: with extreme impact, PCL also ruptures.

On-field management

The aim of on-field assessment is to determine whether there is a significant injury to the knee – either skeletal, ligamentous or to the soft tissues. The assessment and decision as to whether or not to carry the player off the field has to be made by the responsible physician or other medical person. In order to evaluate whether a serious injury has occurred, one needs to find out exactly what has happened. Was there body contact or not? What was the character of the forces involved? What was the direction of these forces and at what speed did the impact take place? Was there deceleration or acceleration at the moment of injury? Was there a jump and landing followed by twisting of the knee? The answers to these questions will provide a preliminary appraisal of the seriousness and the location of the injury.

The next step is a quick examination of the knee. Is there pain felt along the bones, ligaments or other soft tissues? Does moving the knee elicit pain and, if so, where? Is the player able to contract the quadriceps muscle? Is the Lachman test negative or positive? Is there medial or lateral tenderness over the collateral ligaments? Is there a swelling of the joint indicating internal bleeding?

Once the initial assessment is complete, the physician must take the following decisions:

1. Is this a significant knee injury requiring the player to be removed from the field of play?
2. Is a secondary evaluation required off the field?

After secondary examination at the sideline or in the changing room, acute treatment should start as early as possible. Rest, ice, compression and elevation (RICE) will reduce swelling and pain. The RICE treatment should be continued for 24-48 hours. The nature of the further evaluation and treatment depends on the follow-up examination and assessment. More specific treatment is applied as soon as the definite diagnosis has been established.

3.2.1 Knee ligament injuries

Classification and grading

A ligament tear may affect only a few fibres or the entire ligament (Figure 3.2.1.1). In clinical work it is practical to distinguish between partial and complete tears, because

treatment and prognosis are different. Partial tears may be classified as a grade 1 tear, meaning disruption of a few fibres, or a minor grade 2 tear, meaning disruption of less than half of the fibres. In both grade 1 and minor grade 2 tears, the joint is stable. Complete tears with instability could include major grade 2 tears, corresponding to disruption of more than 50% of the fibres, and grade 3 tears, corresponding to disruption of all fibres, with varying instability.

Anterior cruciate ligament (ACL) injuries

Functional anatomy

The ACL is a complex structure and represents the second strongest ligament in the knee with a maximum load capability of around 2,200 N. The ACL is made up of an anteromedial bundle, which is tight with the knee in flexion and relaxed when the knee is in extension, and a posterolateral bundle, which is tight with the knee in extension and relaxed in knee flexion (Figure 3.2.1.2). A third bundle has been named the intermediate bundle and consists of interconnecting tissue between the anteromedial and posterolateral bundles. It is tensed throughout the whole range of motion. The ACL prevents anterior movement of the tibia in relation to the femur and, together with the posterior cruciate ligament, it resists and limits hyperextension, hyperflexion and internal rotation of the tibia.

Classification and injury types

Isolated ACL injuries occur in about 20-30% of players, while combinations with a meniscus injury represent about 50% of knee sprains in football. There are also combination injuries with other ligaments, e.g. the medial and lateral collateral ligaments and the posterior cruciate ligament. ACL injury can also be combined with articular cartilage injury and capsular injury.

ACL injuries can either be partial with preserved stability, or complete with instability. They can occur in the femoral insertion in the mid-substance or in the tibial insertion, sometimes with a bone fragment from the tibia (especially in adolescent players). Due to the destroyed circulation in the ligament substance caused by a tear, the remnants of the ruptured cruciate ligament will atrophy over time.

Causes, mechanisms and risk factors

Isolated injury of the ACL is caused by intrinsic forces with a twisting force either in internal rotation and hyperextension, or in external rotation and valgus with the foot fixed to the ground. Combination injuries with MCL, LCL and PCL may be caused by medial or lateral impact to the knee or to the medial side of the foot, as described above. An injury of the ACL in combination with the LCL could occur upon impact on the foot from the lateral side as described above. Combination injuries with the PCL can be the result of any lateral or medial impact to the knee as well as hyperextension and hyperflexion injuries. Most ACL injuries, however, result from non-contact mechanisms.

Risk factors are insufficient conditioning, muscular weakness, hyperlaxity and a narrow notch. Very deep studs causing a strong grip between the shoe and the pitch may also play a role in the mechanism causing ACL injuries. Inadequate landing after jumping is a risk in female players.

Symptoms and signs

An ACL injury should be suspected if there is a history of any kind of rotation, direct trauma or rapid acceleration/deceleration as well as after a twisting force caused by

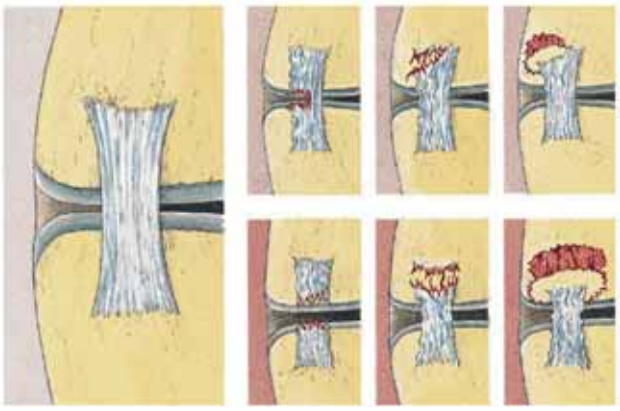


Figure 3.2.1.1 Ligament injuries: Left: intact ligament; Top right: 1. partial ligament tear; 2. partial ligament tear at insertion; 3. partial detachment of bony insertion of ligament; 4. complete tear of ligament; 5. complete tear at insertion of ligament; 6. complete detachment of bony insertion of ligament (avulsion).

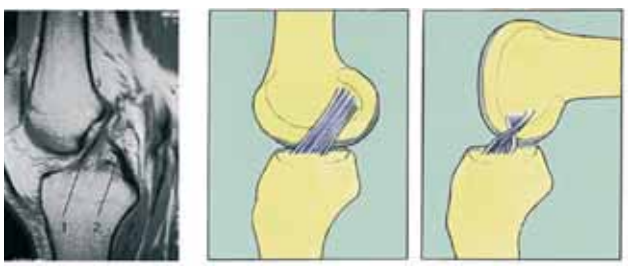


Figure 3.2.1.2 Anterior cruciate ligament (ACL): Left: MRI of normal ACL (1) and PCL (2). Middle: ACL in extension. Right: ACL in flexion

inadequate landing. The player may also have felt a sudden pain or experience or heard a "pop". In this situation the injury should be considered an ACL tear until proven otherwise.

If the player tries to go back into the match, he may feel a recurrent "giving way", which indicates a complete ACL injury. Swelling may develop within a few hours causing pain and discomfort. In 70% of cases, the swelling is caused by bleeding. Aspiration of blood from the joint indicates an ACL injury. Active and passive ranges of motion are limited, but increase in the days after trauma.

Examination and diagnosis

The anterior drawer test with the knee in 20-30° flexion and the tibia in neutral rotation (Lachman test, see 2.1.2) is positive. The test is performed by pulling the tibia forward in relation to the fixed femur (see Figure 3.2.1.3). A positive Lachman test is diagnostic for an ACL tear.

The anterior drawer test in 90° flexion with the tibia in neutral or internal rotation is also positive (see Figure 3.2.1.4). This test, however, is not as reliable as the Lachman test, because the hamstrings and the medial posterior horn of the meniscus can resist this motion.

The pivot shift test may be difficult to perform in the case of an acute injury. The foot and lower leg are internally rotated and the knee is flexed with a valgus stress to the knee (see Figure 3.2.1.5). A positive pivot shift may indicate the need for surgery in a player.

Valgus and varus stability should be tested with an extended knee and also with knee flexion of 20-30° to exclude MCL and LCL injuries. In addition, a posterior drawer test should be undertaken (see 2.1.2).

An X-ray should be performed to exclude any bony injury. An MRI is only needed in questionable cases, especially in combination injuries and bony involvement. Bone bruises are a common finding in ACL ruptures due to compression fractures in the subchondral bone of the lateral tibial and femoral condyles. Mostly, however, a reliable diagnosis can be made from history and clinical findings.

Arthroscopy will provide a definite diagnosis. Diagnostic arthroscopy is usually not necessary with a positive Lachman test as this test is diagnostic for an ACL tear.

Non-operative treatment

Partial tears of the ACL as well as complete tears in older and non-compliant players should be treated conservatively. Non-operative treatment includes acute management of the injury with control of swelling and pain using a compression bandage, ice and anti-inflammatory medication, bracing and gradual increase of range-of-motion exercises.

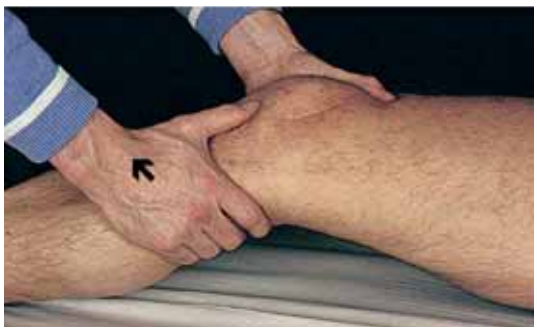


Figure 3.2.1.3 Lachman test (20-30° knee flexion) for anterior cruciate ligament (ACL) tear

Figure 3.2.1.4 Anterior drawer test (90° knee flexion) for anterior cruciate ligament (ACL) tear

Figure 3.2.1.5 Pivot shift test for anterior cruciate ligament (ACL) tear

Operative treatment

Players with complete tears of the ACL or ACL tears in combination with other ligament tears usually have to undergo an ACL reconstruction if they want to return to football. This is normally performed three to eight weeks after the injury. Prehabilitation with facilitated muscle function reduces swelling and pain and helps to achieve full range of motion prior to surgery, thereby enabling better results. This time should be used to inform the player about the surgery and to ensure that he has realistic expectations regarding the outcome (see below).

The standard ACL reconstruction uses the central third of the patellar tendon as a single bundle (see Figure 3.2.1.6). A quadrupled hamstring tendon graft offers the possibility of double bundle reconstruction. More techniques are available. In combination injuries with MCL or LCL ruptures, acute surgery may be required, especially if the instability is pronounced.

In some areas of the world, tendon allografts (Achilles and tibialis posterior tendons) have become popular for single and double bundle ACL reconstruction. Allografts may not give rise to any donor-site morbidity, but there is an increased risk of infections and other side effects. The double bundle technique mimics normal ACL anatomy better and improves rotation stability.

Rehabilitation programme

Rehabilitation after conservative treatment should be advanced as rapidly as tolerated and functional exercises, including activities such as cycling, swimming and straightforward jogging, should start as early as possible. These activities can start as soon as there is a full range of motion and no effusion in the joint. Football training or matches should be avoided for at least 12 weeks after an ACL tear.

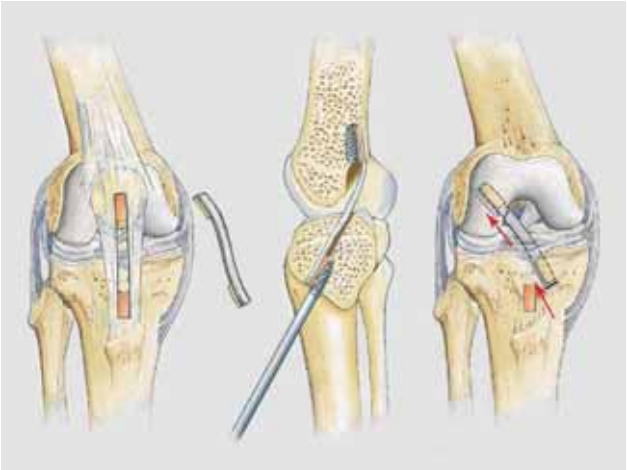


Figure 3.2.1.6 ACL reconstruction: Left: patellar tendon graft taken with bone fragments at both ends. Middle: Graft is passed through tunnel drilled into bone of the tibia and inserted into prepared tunnel in femur. Right: screws are inserted to fix the bone fragments in the tibial and femoral tunnel.

A brace may be used when the player returns to training and match play, but may not always prevent instability. If the patient has subjective and objective instability, giving way and a positive pivot shift test, surgery might become necessary to restore stability.

Rehabilitation following arthroscopic ACL reconstruction is critical to success. Early protected range-of-motion exercises decrease stiffness and make it possible to achieve a full range of motion. Weight-bearing within the pain threshold and increasing weight-bearing over time are encouraged. Besides systematic rehabilitation of the quadriceps and hamstring muscles, functional training should be started. Bracing may be used to provide some protection during the initial rehabilitation, but only soft braces are permitted during a competition under FIFA regulations. Optimum performance and results require restoration of strength, flexibility, proprioception and agility. Moreover, in ACL rehabilitation it is essential to establish an ACL prevention programme for the player's second knee.

Prognosis and return to play

The prognosis for a partial tear after conservative treatment is good. Conservative treatment of a complete tear may not allow a player to return to football. For surgically treated players, the period of prehabilitation prior to ACL reconstruction should be used to inform the player in detail about the procedure and to ensure that he has realistic expectations regarding the recovery of his knee function. It is important to stress that the normal biomechanical behaviour of the ACL cannot be mimicked by surgery. While about 90% may initially retrieve good subjective function, the functional result may deteriorate over the course of time. On average, most players return to football training and matches after four to seven months, but not all players will regain normal function and reach pre-injury activity level after ACL reconstruction. Chronic injuries may lengthen the rehabilitation period prior to return to play.

Sequelae and treatment

An early complication may be the development of an arthrofibrosis which means that the formation of fibrous scar tissue restricts the range of motion and brings the rehabilitation process to a halt. This may be addressed by

increased physiotherapy efforts, but sometimes arthroscopic debridement and mobilisation are needed to regain a full range of motion. Late sequelae are more likely to occur if the meniscus has been removed, articular cartilage lesions have been diagnosed, or if other ligament structures show insufficiency leading to instability or post-traumatic osteoarthritis. Secondary stretching out of the ACL graft with recurring laxity and even instability may occur especially after overly aggressive rehabilitation or a premature return to play. All grafts undergo initial necrosis due to avascularity followed by secondary revascularisation. Creeping substitution with the ingrowth of new functional tissue to replace the necrotic tissue is a process that takes about nine to 12 months, during which time the graft is liable to stretch out. For more information about the treatment of meniscus injuries and articular cartilage injuries, see 3.2.2 and 3.2.3.

Posterior cruciate ligament (PCL) injuries

Functional anatomy

The PCL is the strongest ligament of the knee. The PCL has a larger anterior band, which is tight in flexion and relaxed in extension, and a smaller posterior bundle, which is tight in extension and relaxed in flexion (see Figure 3.2.1.7). The distal insertion of the PCL into the tibia lies extra-articularly. The PCL prevents posterior movement of the tibia in relation to the femur. It limits hyperextension, internal rotation and hyperflexion.

Classification and injury types

PCL tears are not very common and constitute only 5-10% of all major knee ligament injuries. Complete tears with avulsion of a bony fragment from the tibia are seen mostly in young players. Partial tears are more common. Isolated PCL injury can occur either as a bony detachment of the tibial insertion, a distal ligament substance tear, or a periosteal avulsion from the femoral origin. While substance tears are rather uncommon, femoral avulsion tears occur more often. PCL injuries can be combined with ACL, MCL and LCL injuries. Concomitant meniscus and cartilage injuries are common as well.

Causes, mechanisms and risk factors

PCL injuries can be caused by body contact with an opponent player who hits the anterior proximal part of the tibia in a sliding tackle from the front. The PCL may also tear as a result of a fall on a flexed knee while the foot is in plantar flexion. Hyperextension of the knee may cause an isolated PCL injury. Combination injuries between PCL and ACL, MCL and LCL can occur and ought to be considered more seriously.

Risk factors include previous injuries to the knee, insufficient rehabilitation of a previous injury, insufficient conditioning, weakness of the thigh muscles, hyperlaxity, body contact and foul play. Goalkeepers in particular are at risk of PCL injuries.

Symptoms and signs

A hyperflexion or hyperextension trauma as well as impacts from the side or from the front can result in either an isolated or a combination injury. In acute isolated PCL injury there is only a mild haemarthrosis, if any. Generally, the swelling and pain are less pronounced than in acute ACL injuries. There is an increase in pain when flexion of more than 90° is attempted.

Examination and diagnosis

The diagnosis may be confirmed by inspection of both knees in 90° flexion. In PCL injury there may be a posterior

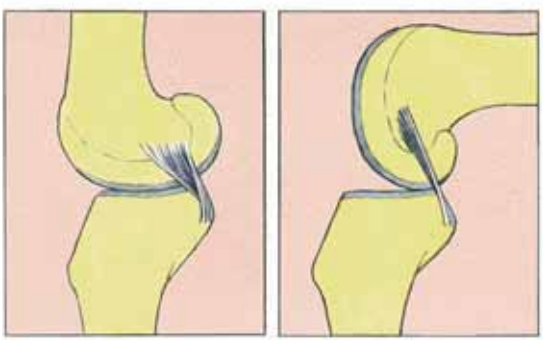


Figure 3.2.1.7 Posterior cruciate ligament (PCL): Left: PCL in extension. Right: PCL in flexion.

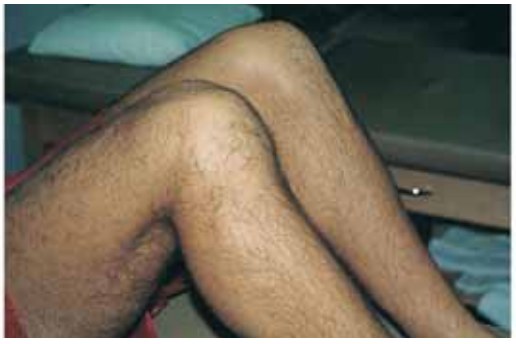


Figure 3.2.1.8 Posterior sag sign in PCL injury

sag sign due to a spontaneous posterior displacement of the tibia (Figure 3.2.1.8).

The posterior drawer test is a classic test to reveal straight posterior instability. The knee should be flexed to 90° and the tibia should be in a neutral rotation position. If there is about 3-10mm of increased excursion, this usually indicates a partial PCL tear. If the displacement is more than 10mm, a complete PCL tear is present.

The quadriceps active test is performed with the player's knee in 90° flexion. In this position the tibia subluxates posteriorly if there is a PCL tear or in chronic cases of insufficiency. By contracting the quadriceps muscle, which works agonistically to the PCL, the tibia moves anterior to its normal position – this is considered a positive test (Figure 3.2.1.9).

In the acute stage, it may be difficult to produce a posterior drawer sign. The initial test is positive in 31-76% of cases where a serious PCL tear is verified later. If a PCL tear is suspected but tests are negative, the player should be re-examined under anaesthesia to verify a PCL tear. Plain X-rays will exclude fractures and bony avulsions. MRI may be required if the diagnosis of PCL injury remains uncertain. Arthroscopy may give the definite answer about the injury, especially when the ligament is probed.

Treatment

PCL injury treatment recommendations depend on the spectrum of injury to the PCL and other stabilisers of the knee. PCL injuries with bony avulsions, especially in young players with a dislocated fragment, can be treated by open reduction and internal fixation with excellent results.

Isolated PCL injuries in the substance and partial ruptures can be treated with accelerated rehabilitation, bracing and early functional rehabilitation including range-of-motion training, quadriceps training, cycling, swimming, etc. The patient may remain clinically unstable but experience functional stability without any symptoms.

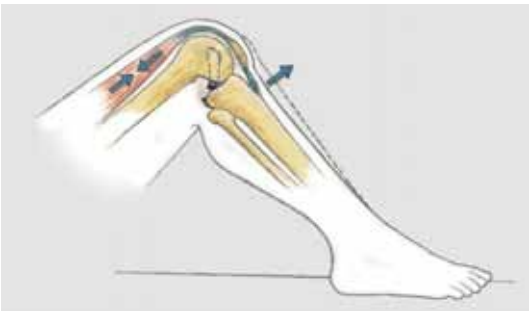


Figure 3.2.1.9 Quadriceps active test in PCL injury: In 90° flexion, tibia is in a subluxated posterior position. Fixation of the foot and contracting of the quadriceps muscle leads to anterior translation of the tibia towards its normal position.

Surgical treatment of acute PCL injuries is controversial. If there is severe posterior instability, especially if secondary stabilisers of the knee are also injured (such as MCL, LCL and ACL), indication for surgical treatment needs to be considered.

Rehabilitation programme

During the early phase of the rehabilitation programme, a brace which limits the posterior drawers should be used even during the functional period of training. Active motion can be started immediately and muscle training should be focused on the agonistic quadriceps muscle. Especially during the initial four to six weeks, the hamstrings should not be trained specifically. The main aim is to achieve full range of motion and 80% of quadricep strength.

Prognosis and return to play

The prognosis of an untreated PCL tear is not favourable, as it alters knee mechanics, resulting in abnormal motion with instability and finally in the development of post-traumatic osteoarthritis. Many players experience patello-femoral pain and may develop early patello-femoral osteoarthritis. With conservative treatment of isolated, partial or complete PCL tears, the return to football training and matches may be quite fast if range-of-motion and muscular strength are restored and can be attempted after eight to 12 weeks. However, each player must be evaluated individually and combination injuries in particular require close follow-up.

Persistent subjective and objective instability after a PCL injury is not uncommon and may, in the long term, result in osteoarthritis of the knee. Secondary lengthening of other ligamentous structures, such as ACL or LCL, may cause progression of instability over time. In reconstructive surgery of the PCL, attention should be paid to insufficiency of other ligamentous structures. Recently, more interest has been focused on the anatomy of the PCL with the anterior bundle tensed in flexion and the posterior bundle tensed in extension. As single bundle PCL reconstruction has not been successful in restoring posterior instability, there is a trend towards double bundle PCL reconstruction to improve stability. Concomitant meniscus and articular cartilage injuries require individual attention (see 3.2.2 and 3.2.3).

Medial collateral ligament (MCL) injuries

Functional anatomy

The MCL is the ligament most commonly injured in the knee. There are three parts to the MCL: the superficial, the deep and the posterior oblique ligament. The meniscus is attached to the deep part of the MCL, the oblique ligament and the medial capsule.

The MCL is the primary stabiliser for valgus stress and external rotation forces of the tibia in relation to the femur.

Classification and injury types

MCL injuries can either be partial with stability or complete with instability. The MCL can either be torn from the femoral origin, torn in the mid-substance or from the tibial insertion. An MCL injury can either be isolated or in combination with a meniscus injury or an ACL or PCL injury.

The grading of the injury is as follows:

- Partial tear: clinically stable: grade 1 and minor grade 2, medial joint opening up to 4mm.
- Complete tear: clinically unstable knee, major grade 2 and grade 3 up to 10-15mm joint opening.
- Always perform complete stability testing with Lachman, anterior drawer, pivot shift and posterior drawer tests, and compare the results to the uninjured knee.

Causes, mechanisms and risk factors

The history of MCL injuries often includes non-contact valgus external rotation trauma by intrinsic forces when turning, twisting or landing. More often a lateral blow or impact to the lower thigh or knee with direct contact may be the cause of valgus stress and external rotation by extrinsic forces.

Symptoms and signs

Pain occurs at the time of injury. Absence of severe pain, however, does not exclude an injury. Minor injuries may be more painful than more severe injuries. The ability to walk or run can be impaired after an MCL injury, especially in the unstable knee.

Swelling of the joint is not common and might indicate a more severe injury in the joint itself. Tenderness is usually present at the location of the injury. The most common location for tenderness is the medial femoral condyle.

Examination and diagnosis

Testing of instability with valgus stress is important, both with the knee in 20-30° flexion (Figure 3.2.1.10) and in full extension.

Perform X-rays to exclude fractures. An MRI may be helpful in uncertain cases and when associated meniscus or other ligament injuries are suspected.

Treatment

For grade 1 and minor grade 2 injuries (stable knee, partial tear) the rehabilitation programme with weight-bearing and early motion may start as early as possible. A brace is recommended to protect the initial healing of the ligament structures. If there is satisfactory progress leading to full extension, no effusion and decreased tenderness after two to three weeks, the player is advised to optimise his range of motion and muscular strength before returning to training and competition, which usually occurs within four to eight weeks.

The treatment of major grade 2 and grade 3 injuries (unstable knee, complete tears) depends on associated injuries. Complete rupture of the MCL is often associated with an ACL injury. In a combined MCL/ACL injury, ACL reconstruction should be carried out, as well as repair of the medial structures. Post-operatively, the healing process should be protected by a brace. Early motion and weight-bearing starts as soon as possible within the limits of pain.

Isolated complete MCL tears may be treated conservatively with early weight-bearing and motion with protective bracing. It may take up to six to eight weeks or longer before the player can return to football. In principle, stable and unstable isolated MCL injuries are treated

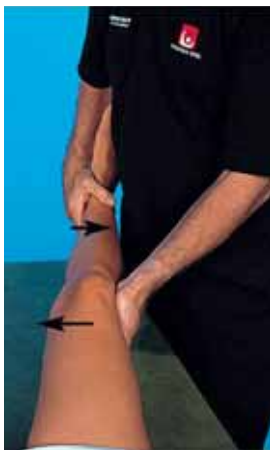


Figure 3.2.1.10 Valgus stress test in MCL injury in 20-30° flexion.

conservatively. MCL injuries in combination with cruciate ligament injuries may require surgical treatment.

Prognosis and return to play

Prognosis is good for stable MCL injuries and, given adequate treatment, also for unstable MCL injuries. Sometimes instability will persist despite adequate treatment and MCL reconstruction becomes necessary. Early reconstruction of chronic instability of the MCL has a better prognosis than delayed reconstruction. Sometimes stiffness can be a problem but is less frequent with early motion and weight-bearing.

Muscle exercises aimed at regaining at least 80% of the initial strength are of great importance for a safe return to football (Figure 3.2.1.11). A return to training and match play is permitted as soon as the player has recovered a full range of motion, adequate muscular strength and acceptable stability on testing. Stable knees may return within four to eight weeks, while unstable knees may require four to six months. Sometimes a brace is needed for a longer period of time.

Lateral collateral ligament (LCL) injuries

Functional anatomy

The LCL is the primary stabiliser against varus stress on the knee in the posterolateral corner (complex) of the knee. Other stabilising factors are the posterolateral fibrous capsule, the arcuate ligament, the popliteo-fibular ligament and the lateral gastrocnemius tendon and muscle. Posterolateral corner injuries often occur in combination with injuries to the ACL, PCL or both.

Causes, mechanisms and risk factors

The mechanism of injury might be a medial impact to the knee or an external rotation with the foot in a fixed position, causing internal rotation of the tibia and varus stress to the knee.

Symptoms and signs

A history of medial impact to the knee or an internal rotation of the tibia and varus stress to the knee with the foot fixed may indicate an LCL injury.

Examination and diagnosis

Varus instability when testing in extension and in 20-30° of knee flexion (see Figure 3.2.1.12) indicates at least a partial LCL rupture. A varus opening of less than 5mm indicates a partial rupture and can be treated conservatively with an early range of motion and weight-bearing, protective bracing and muscle strengthening.

Complete ruptures and injury to the posterolateral corner should be treated surgically to avoid late varus instability. Genu varum is an important factor to correct in severe posterolateral corner insufficiency and should be done by tibial osteotomy.

Figure 3.2.1.11 Special apparatus for dynamic eccentric isokinetic training

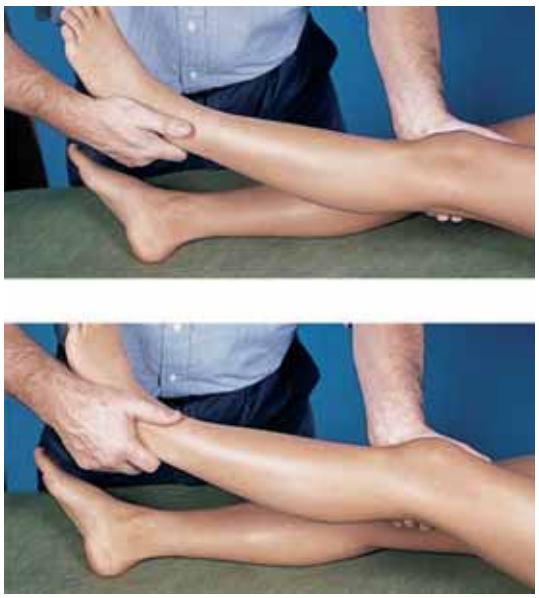


Figure 3.2.1.12 Varus stress test in LCL injury in extension (top) and 30° flexion (bottom).

Prognosis and return to play

If not diagnosed or treated adequately, LCL injury may result in significant problems. For partial ruptures with preserved stability, the prognosis is good and a return to football is usually possible within four to eight weeks. In a case of complete ruptures with instability, acute surgery is recommended and a return to football may not be possible before four to six months, depending on the individual recovery process. As for MCL injury, full range of motion, a stable knee on stability testing and 80% of the muscle strength should also be achieved prior to a return to football. A brace is recommended for the first phase. In cases where there is additional injury to the posterolateral corner of the knee, serious lateral insufficiency with considerable instability in the extended knee may occur and is treated surgically by LCL reconstruction and an advancement of the posterolateral structures.

3.2.2 Knee meniscus injuries

Incidence

Injuries to the medial and lateral menisci are the most common knee injuries in football. The incidence of meniscal injuries that result in meniscectomy has been shown to be 61 per 100,000 in a common U.S. population. Treatment of meniscal lesions with arthroscopy has become the most common orthopaedic surgical procedure in the majority of orthopaedic centres and constitutes 10-15% of all surgery. When a player is examined after a distortion trauma, a medial meniscus tear should always be suspected with medial symptoms and a lateral meniscus tear with lateral symptoms. Articular cartilage injuries may mimic meniscus injuries and may be present in about 40-45% of players with meniscus injuries. Furthermore, an anterior cruciate ligament injury or collateral ligament injury may mimic meniscus injuries. The most common diagnosis after a knee distortion is a medial meniscus, which occurs five times more often than injuries to the lateral meniscus. Meniscus tears occur in 50% of all ACL injuries. Medial meniscus injury is common in combination with medial collateral ligament injury.

Functional anatomy

In the past, the meniscus was thought to be a dispensable structure. The treatment of an injured meniscus therefore often resulted in its complete removal. Unfortunately, removal of the meniscus results in unphysiological loading of the articular cartilage, which will erode over the course of time and finally result in osteoarthritis. Nowadays, we consider the meniscus as having a vital function in the knee. The meniscus plays an important role in shock absorption for the knee joint, in dispersing the weight-bearing load as well as in the stabilisation of flexion-extension and rotational movements of the knee. The anterior and posterior horn of the medial meniscus is attached to the tibial plateau, the joint capsule and the medial collateral ligament (see Figure 3.2.2.1).

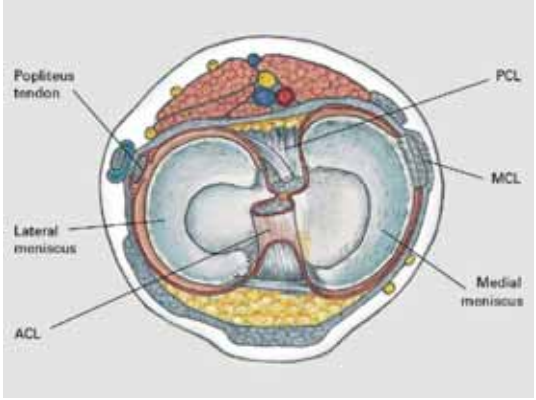


Figure 3.2.2.1 The medial and lateral meniscus

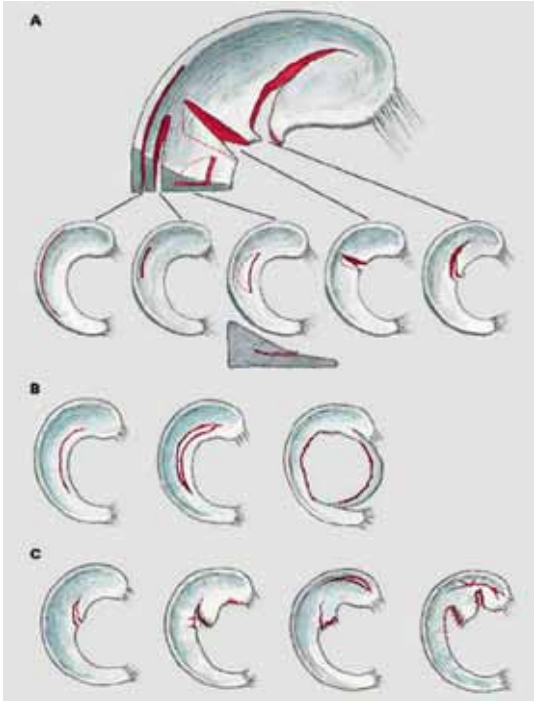


Figure 3.2.2.2 Different types of meniscal injuries (A). Development of meniscus "bucket handle" tear (B). Development of meniscus flap tear (C).

The lateral meniscus has an anterior and posterior attachment to the tibia but has no attachment to the lateral collateral ligament. The capsular attachment of the lateral meniscus is also less tight than that of the medial meniscus, which renders the lateral meniscus more flexible.

The peripheral third of the meniscus near its capsular attachment is richly vascularised. In the intermediate third, vascularisation decreases closer to the centre, while the inner third is not vascularised at all. This is of significance for the possibility of repairing a meniscus tear, which is possible only in the vascularised area.

Classification

Meniscus tears can be classified as peripheral tears, horizontal tears, radial tears, flap tears and "bucket handle" tears (see Figure 3.2.2.2).

Causes, injury mechanisms and risk factors

Meniscus injuries mostly occur with body contact, particularly when the medial meniscus is involved, often in combination with ligament injuries. This is partly because the medial meniscus is firmly attached to the medial collateral ligament and capsule, and partly because tackles occur more frequently against the lateral side of the knee, causing external rotation of the tibia. In external rotation of the lower leg in relation to the femur, the medial meniscus will tear, whereas in internal rotation of the lower leg the lateral meniscus will tear. Meniscus injuries can also occur with hyperextension and hyperflexion of the knee.

Previous knee injury, incomplete rehabilitation after an injury, tackling from the side, foul play, poor ball handling skills, "cutting", turning or twisting movements as well as joint laxity are all risk factors for meniscus injury.

Symptoms and signs

A meniscus injury should be suspected after a distortion of the knee, either by body contact or by twisting or "cutting". Meniscus injuries should be suspected when symptoms are localised to the medial or the lateral side, with pain on motion or weight-bearing or with twisting movements.

Examination and diagnosis

The diagnosis of meniscus injury is based on tenderness over the joint line, pain over the joint line during hyperextension of the knee, hyperflexion of the knee and external rotation of the lower leg for medial meniscus injury, and during internal rotation of the lower leg for lateral meniscus injury. Here, a weakened and reduced muscle volume of the quadriceps is present. If the diagnosis is uncertain, an MRI can verify the diagnosis, but arthroscopy is the diagnostic and therapeutic tool of choice.

Treatment

Acute "locking" or blocking with extension deficit of the knee requires arthroscopic surgery within a few days to reduce muscle wasting (atrophy). During arthroscopy, the surgeon will evaluate the meniscus tear. In young players with a tear in the vascularised zone of the meniscus, suturing of the meniscus is the best treatment and should be attempted whenever possible. If suturing is impossible, the damaged part of the meniscus is removed and the remaining part is trimmed to vital stable tissue. It is important to evaluate articular cartilage surfaces as well as to carry out stability testing of the knee and probing of the cruciate ligaments.

Rehabilitation programme

The patient is mobilised with crutches and weight-bearing within the limits of pain. Quadriceps and hamstring exercises with special attention to full extension must be included, particularly with a locked knee at the beginning.

Prognosis and return to play

The prognosis of partial meniscectomy is good in the short term. In the long term, osteoarthritis may occur but is more common after total meniscectomy. Osteoarthritis after meniscectomy is a very common cause of early retirement from football. The long-term results of meniscus repair are not yet available. In young players with an acute or fresh tear suitable for repair, this procedure is highly recommended, even if recovery and return to football lasts much longer and may take up to six to eight months.

After partial meniscectomy, a return to training and match play should not take place until nearly full mobility and full strength of the thigh muscles have been regained. Usually, this takes between two to six weeks after arthroscopic surgery, depending on the size and location of the tear and individual recovery. It is very important to continue the strength and endurance training of the quadriceps and hamstring muscles and to improve the player's general condition after a meniscus injury.

Sometimes a discoid meniscus will be diagnosed (see Figure 3.2.2.3). The symptoms are usually the same as for meniscus tears, but are usually present in young players. In a tear in the discoid meniscus, the inner central part of the discoid meniscus will be removed to create a normal-sized meniscus. If the discoid meniscus is unstable, re-fixation of the unstable part to the capsule should be attempted. Whenever possible, removal of the discoid meniscus should be avoided. Return to play after partial resection of a discoid meniscus usually takes as long as or a little longer than after conventional arthroscopic meniscus surgery.

Sequelae and their treatment

Whenever possible, the menisci should be preserved. If it is necessary to resect the damaged part of the meniscus, one should try to preserve as much vital tissue as possible. Total meniscectomy should generally be avoided, and suturing of the meniscus will reduce the risk of later post-traumatic osteoarthritis. After total or sub-total meniscectomy with articular cartilage lesion in the same compartment, meniscal transplantation using allograft is a possibility to restore the joint function. Partial substitution after partial meniscal resection with animal-collagen-based meniscus transplants is showing promising early results, but long-term follow up results are still missing. Repair of articular cartilage injuries should also be considered.

3.2.3 Knee cartilage injuries

With the advent of more active treatment of ligament injuries and the use of arthroscopy and MRI, the frequency of articular cartilage injuries has been found to be much higher than previously believed. Articular cartilage injuries down to the subchondral bone are found in more than 40% of acute and chronic injuries to the knee during surgical treatment. Articular cartilage injuries have been found in 20-70% of patients with acute and chronic anterior cruciate ligament ruptures. In MRIs taken shortly after an ACL injury, a high percentage of bone bruises are seen affecting the subchondral and trabecular bone below the cartilage which may either heal or progress into cartilage degradation over time. To prevent or minimise further deterioration, bone bruises should be treated as fractures of the trabecular bone or subchondral bone plate. Articular cartilage lesions are diagnosed in about 40-50% of meniscal tears.

Functional anatomy

Articular cartilage covers the end of joining bones and optimises joint function by reducing friction and increasing shock absorption. Cartilage consists of cells, the chondrocytes, and supportive tissue, the matrix. Chondrocytes comprise only 5% of the total volume of the cartilage and are organised in columns. They produce the surrounding supportive tissue, the matrix, consisting of collagen type II made of protein which armours the cartilage and provides tensile strength to the tissue. It also produces proteoglycans, a compound of protein and carbohydrates, which maintains the water content of cartilage at about 70% of the total volume. The water content contributes to the shock-absorbing function of cartilage (see Figure 3.2.3.1).

In the absence of weight-bearing, the water originating from the synovial fluid is absorbed by the proteoglycans in the matrix. During weight-bearing, the water content of the cartilage is reduced by pressing the

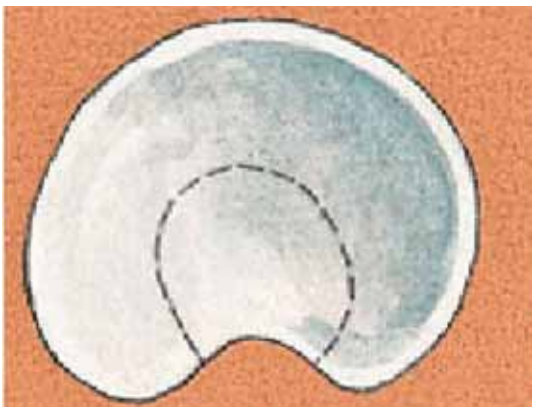


Figure 3.2.2.3 Discoid meniscus

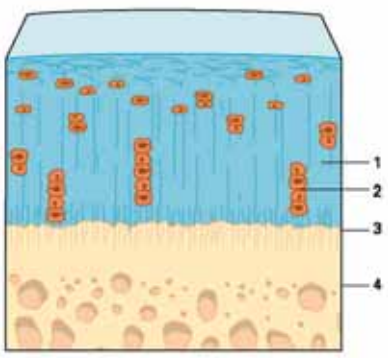


Figure 3.2.3.1 Structure of cartilage: 1. cartilage matrix 2. chondrocytes 3. tidemark 4. bone below cartilage (subchondral and trabecular)

water back into the joint. All nutrients and oxygen are supplied to the cartilage with the synovial fluid produced by the joint capsule. The metabolic turnover is slow. The synovial fluid also lubricates the articular cartilage surfaces, reducing the friction between them. The articular surface friction is lower than friction between ice surfaces.

Articular cartilage is a unique tissue as it lacks vascular, nerve and lymphatic supply. The lack of vascular supply means that cartilage cannot heal by inflammatory tissue repair and exclusively depends on the exchange of synovial fluid for nutrients and oxygen. The lack of nerve supply means that injuries to the cartilage will not cause pain unless the surrounding tissues, such as the synovial lining, the subchondral bone, or the periosteum, are affected.

Classification and grading

There are many classifications for articular cartilage injuries. However, two classifications are used more frequently. Articular cartilage injuries may be graded according to the Outerbridge classification (Figure 3.2.3.2) or the International Cartilage Repair Society (ICRS) classification system (Figure 3.2.3.3).

Articular cartilage damage may be either superficial (partial), deep (complete, full) or osteochondral. Superficial articular cartilage injuries extend into the upper half of the cartilage. These injuries will neither heal nor progress unless located in weight-bearing areas.

Deep or full-thickness articular cartilage injuries extend down to the subchondral bone, but not through it. They will not heal and will progress to osteoarthritis with time. Osteochondral bone and cartilage injuries extend through the subchondral bone into the trabecular bone and may heal by inflammation, allowing vascular ingrowth with fibroblast (fibrous cell) invasion that will produce fibrous tissue repair.

Injured cartilage may avulse from the bone and end up as flaps or loose bodies interfering with joint function. Articular cartilage has no capacity for self-healing.

Causes, injury mechanisms and risk factors

Articular cartilage injuries may be caused by distortion, dislocation or contusion of the knee joint. They are often caused in combination with ligament injuries (such as MCL and ACL) and meniscus tears. Articular cartilage injuries may

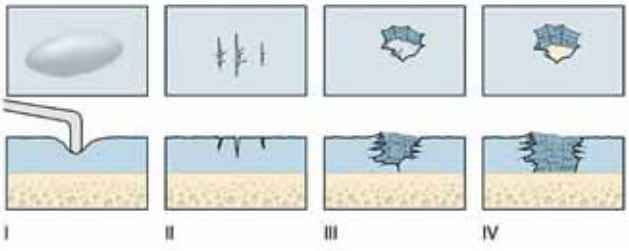


Figure 3.2.3.2 Outerbridge classification of cartilage injury: surface and cross-sectional views of cartilage injuries I-IV

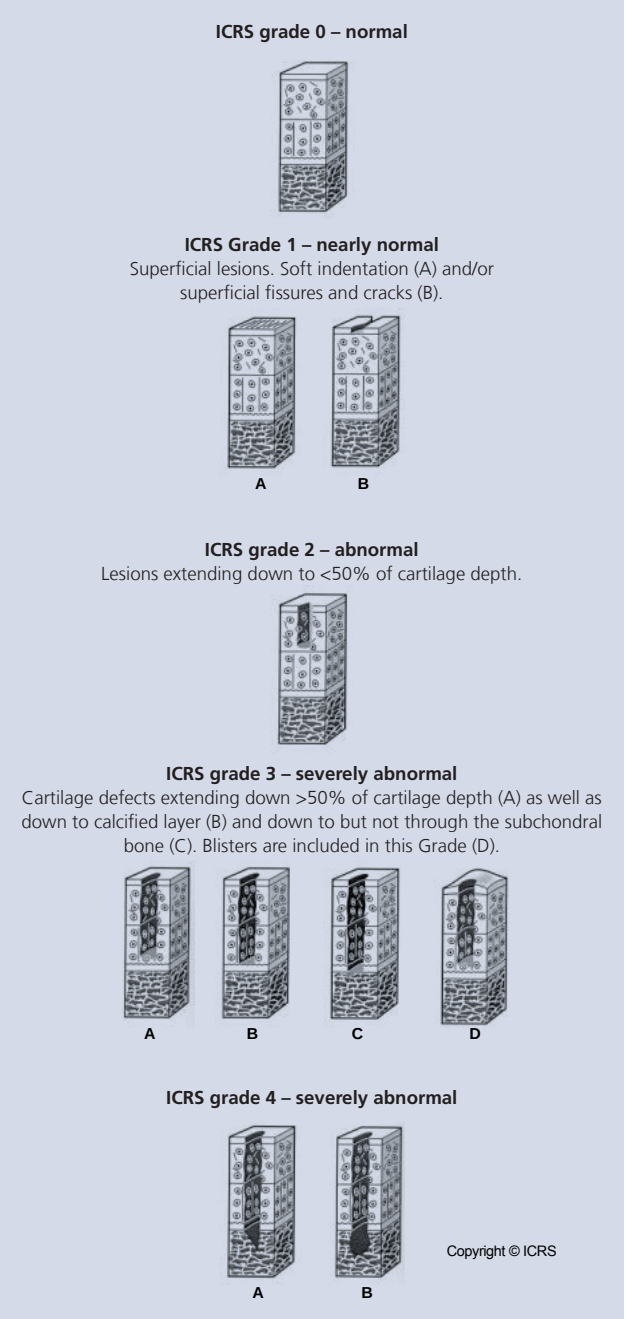


Figure 3.2.3.3 ICRS classification of cartilage injury 0-4

be caused by repetitive trauma with each incident resulting in only minimal or no symptoms, or by repeated overuse or overload.

Risk factors are previous injuries to the meniscus or knee ligaments, persistent instability or sequelae after partial or total meniscectomy, varus or valgus malalignment of the knee, patella malalignment or instability, tackling and foul play, falling on hard surfaces, insufficient muscle conditioning or muscle weakness.

Symptoms and signs

There may be a history of distortion, dislocation or contusion of the joint or a history of previous ligament or meniscus injury with subsequent treatment. Swelling and effusion may be present. Acutely, there may be intra-articular bleeding, especially if the subchondral bone is injured, or if ACL or other injuries are involved.

Pain occurs when the joint is moved or loaded. "Catching" or "locking" may occur during weight-bearing, running, "cutting" or twisting activities. Crepitation (creaking) is noted with joint motion, especially on weight-bearing. Joint line tenderness may be present. Ligament stability testing has to be performed.

Examination and diagnosis

Acute or chronic symptoms after a knee injury may mimic meniscus tears, ligament injuries, etc. For confirmation of the diagnosis and further assessment of articular cartilage injuries, examination with MRI (with or without contrast) is useful for evaluating bone involvement or other additional pathology. In about 20-70% of articular cartilage injuries, other injuries are present, such as meniscus tears, ACL-PCL injuries or collateral ligament injuries. Arthroscopy needs to be performed. Weight-bearing X-rays to determine varus or valgus deformity are also important.

If the articular cartilage injury has already been diagnosed by previous investigations, arthroscopy or surgery, it is very important to examine the stability of the joint as well as varus or valgus deformity, patello-femoral malalignment or instability.

An osteochondral injury may also cause similar symptoms to traumatic osteochondral fractures or osteochondritis dissecans (see Figure 3.2.3.4 and 3.2.3.5). When these injuries also occur they cause a spectrum of pain, stiffness and inability to perform.

Conservative and surgical treatment

Conservative treatment includes reducing or totally stopping football training and/or matches. Muscle strength, endurance and proprioception should be improved by physiotherapy. Symptomatic treatment of inflammation with anti-inflammatory medication.

Conservative treatment is rarely successful in players who want to return to play. Surgical treatment involves debridement of damaged cartilage and cartilage flaps interfering with joint function by causing catching or locking. However, this only removes the symptoms and may temporarily stop the progress, but initiates no repair.

Procedures to stimulate bone marrow cell include drilling, microfracturing and abrasion arthroplasty.

- Drilling creates multiple holes through the subchondral bone of the defect into the trabecular bone.
- Microfracturing or "picking" is the creation of small fractures in the subchondral bone to encourage bleeding into the defect, creating fibrous repair.

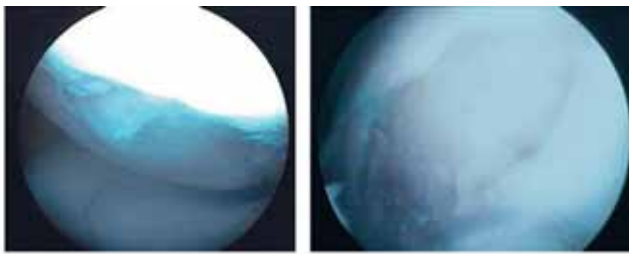


Figure 3.2.3.4 Arthroscopy of articular cartilage injury of medial femoral condyle (left) and of the patella (right)

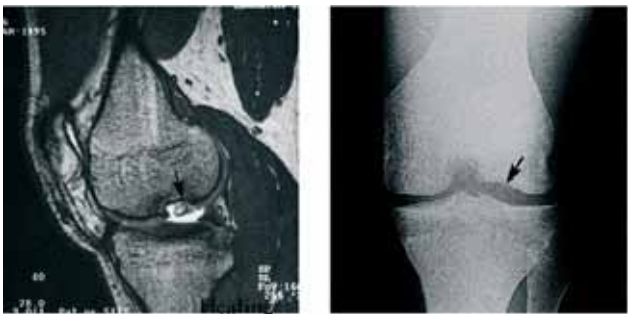


Figure 3.2.3.5 MRI (lateral view) of osteochondritis dissecans of the femoral condyle (left) and X-ray showing osteochondritis dissecans of the lateral femoral condyle (right)

– Abrasion arthroplasty is the superficial abrading of the subchondral bone surface to create bleeding from capillaries in the subchondral bone allowing fibrous repair of the defect.

Periosteal and perichondral grafting is performed by transferring periosteum or perichondrium from the tibia or from the ribs to the injured area which has been prepared by abrasion. The long-term results have not been satisfactory, however.

Autologous chondrocyte transplantation (ACT) is suitable for localised cartilage injuries (size 2-12 cm^2) to the articular surface of the knee (Figure 3.2.3.6). At arthroscopic evaluation of the injury, cartilage slices are harvested for cell culturing. The patient is operated on two weeks later, with excision of the chondral injury and injection of the cultured cells into the defect, which has been covered with periosteum (Figure 3.2.3.6). The long-term results are promising. Injured players may return to football but this may take between 12 and 18 months.

Osteochondral grafting of cylinders of bone and cartilage has been used for minor injuries with promising results. It may be possible to return to play in the case of smaller injuries (see Figure 3.2.3.7).

Rehabilitation programme

Early range-of-motion exercises are of utmost importance for the restoration of joint function. Muscle strength training and a progressive increase in weight-bearing of the joint with partial weight-bearing for six to eight weeks, gradually increased to full weight-bearing in a period of eight to 12 weeks after surgery is initiated. Functional training (bike riding, swimming, long-distance walking) or intermediate functional training may be started and hase to be assessed on an individual basis. Before returning to play, every player needs to be evaluated either clinically and arthroscopically or by MRI.

Prognosis and return to play

Conservatively treated or untreated articular cartilage injuries will progress into osteoarthritis in the course of time. Exclusive debridement will not show good long-term results. Techniques to stimulate bone marrow cells may result in temporary healing and may allow for return to play. However, re-injuries or destruction of the repair tissue are likely over the course of time. Perichondral- and periosteal-grafting seems to produce fibrous tissue repair but may deteriorate if football play is continued. Expert panels have considered ACT as the operation of choice after failed bone-marrow-stimulating procedures or other treatments.

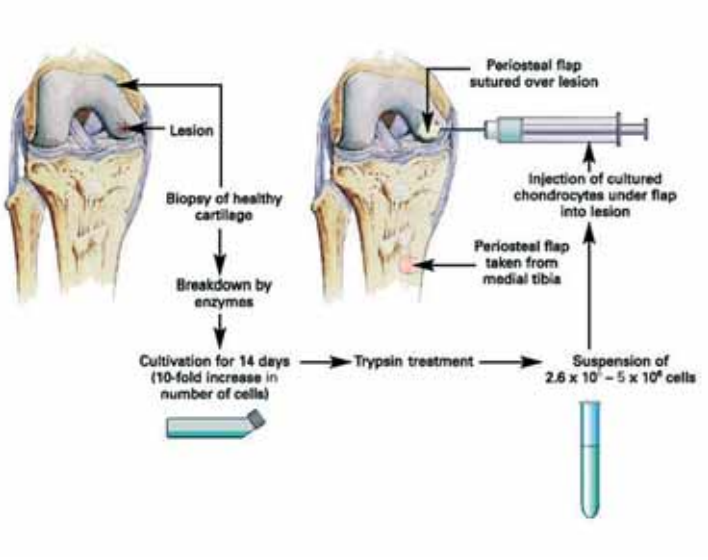


Figure 3.2.3.6 Autologous chondrocyte transplantation: biopsy, cartilage cell culture and re-injection

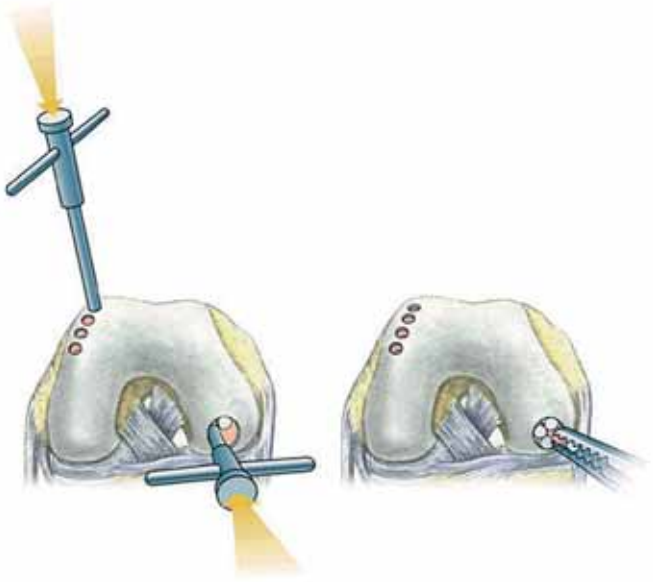


Figure 3.2.3.7 Osteochondral grafting: bone and cartilage cylinders are from areas with minor weight-bearing of the joint and transplanted into holes drilled into the damaged area.

3.3 Thigh muscle injuries

3.3.1 Incidence

Thigh muscle injuries occur frequently as contusion injuries in contact sports, and as strains in sports involving maximal sprints and acceleration. Because football combines maximal sprints with frequent player-to-player contact, it is not surprising that up to 30% of all football injuries are thigh muscle injuries. In fact, results from the elite leagues in England, Iceland and Norway show that hamstring strains are the most common type of injury in male football, accounting for between 13% and 17% of all acute injuries. Other studies have shown that muscle contusion injuries to the thigh account for up to 16% of all acute football injuries at an elite level. The F-MARC studies have revealed similar results.

	Injuries	Complaints without injury
FIFA competition, male	8-23%	-
FIFA competition, female	8-22%	-
Annual prevalence, males	15%	19%

Table 3.3.1.1 Thigh muscle injuries and complaints without injuries

3.3.2 Functional anatomy

The hamstrings muscle group consists of three muscles of the posterior thigh, the biceps femoris, semitendinosus and semimembranosus muscles. Except for the short head of the biceps, these are two-joint muscles, i.e. they extend beyond both the knee and hip joint. Their action is to extend the hip and flex the knee and this may partly explain why they are prone to strain injury. When there is simultaneous motion across the two joints, both the force and rate of force development can become very high. All three muscles are supplied by the sciatic nerve.

3.3.3 Classification and grading

Muscles are injured by two different mechanisms, through direct contact or by muscle strain. The quadriceps muscles are the muscle group most susceptible to contusion injuries because they are located ventrally and laterally on the thigh. The hamstrings are the muscle group at the posterior thigh, and injury to these muscles typically occurs when they are acutely stretched beyond the limits of tolerance during maximal sprints. Since, in the vast majority of cases, the hamstrings are injured through strains and the quadriceps muscles through contusions, it is customary to use the terms hamstring strains and quadriceps contusion to describe the most frequent injury types. However, hamstring contusions and quadriceps strains also occur, although they are much less common (Table 3.3.3.1).

Muscle cramps are also common in football players, and it can be difficult to distinguish cramps (where there is no structural damage to the muscle) from a mild strain or a mild contusion in a case where the player does not remember any trauma. Muscle soreness is a troublesome but generally harmless symptom that occurs after training to which the player is unaccustomed, usually after eccentric training. Major trauma to the thigh can also, on rare occasions, result in fractures. This occurs more often among children than among adults.

Most common	Less common	Not to be missed
Quadriceps contusion	Adductor strain	Acute compartment syndrome
Hamstring strain	Avulsion or total ruptures of the hamstrings and quadriceps	Fractured femur
Hamstring cramps		

Table 3.3.3.1 Diagnoses to consider after an acute thigh muscle injury

Whether muscle injury is caused by indirect (strain) or direct trauma, both of these injury mechanisms result in the destruction of muscle fibres. Muscle fibres are surrounded by capillaries to ensure maximum supply of the musculature with oxygen and nutrients. Moreover, local blood flow in the muscle is usually high during exercise. These two factors, the abundant capillary supply and the elevated flow, combine to result in considerable internal bleeding, reduced function and pain immediately after injury, regardless of the injury mechanism. The principles for the assessment, acute treatment and rehabilitation are therefore very similar for both injury types.

Classification systems exist to classify muscle injuries according to the amount of tissue damage and bleeding seen. One aspect of particular importance, at least for the prognosis, is whether the haematoma is intermuscular or intramuscular. Bleeding is classified as either intramuscular, where there is no injury to the muscle fascia, or intermuscular, where the blood escapes from the muscle compartments through a defect in the muscle fascia (Figure 3.3.3.1). In general, healing time is significantly longer with intramuscular bleeding than it is with intermuscular bleeding.

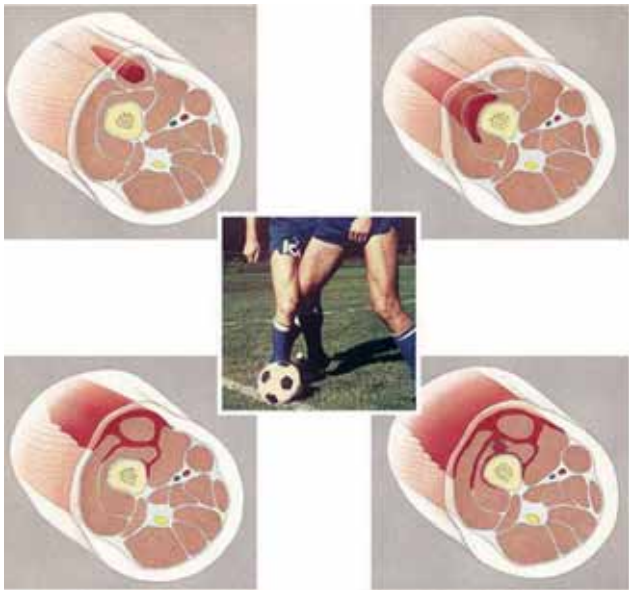


Figure 3.3.3.1 Muscle injuries can be classified as intermuscular or intramuscular, based on whether the haematoma is able to escape the muscle compartment or not. Illustration reproduced with permission from Sports Injuries (Peterson & Renström, 2001) ©Martin Dunitz Ltd.

3.3.4 Causes and mechanisms

As mentioned above, there are mainly two different mechanisms involved in thigh injuries: direct (contusion) and indirect (distension or strain) injuries. The contusion mechanism is straightforward: the player is injured by a direct blow from an opponent, usually the knee hitting the lateral thigh in a tackle (also known as a "charley horse" or a "cork thigh"). The muscle is thereby crushed between the opponent's kneecap and the player's own femur (Figure 3.3.4.1).

The injury mechanism and risk factors for hamstring strains are less well understood. The hamstrings muscle group is composed of three muscles – semimembranosus, semitendinosus and biceps femoris. All of them (except the short head of biceps) have their origin at the tuber ischii on the pelvis and they insert at the medial and lateral of the lower leg, right below the knee. This means that they overlap two joints – they straighten the hip joint and bend the knee joint. Muscle strains usually occur in the interface between the muscle and its tendon (the myotendinous junction), but avulsion injuries from the tuber ischii are also seen (Figure 3.3.4.2).

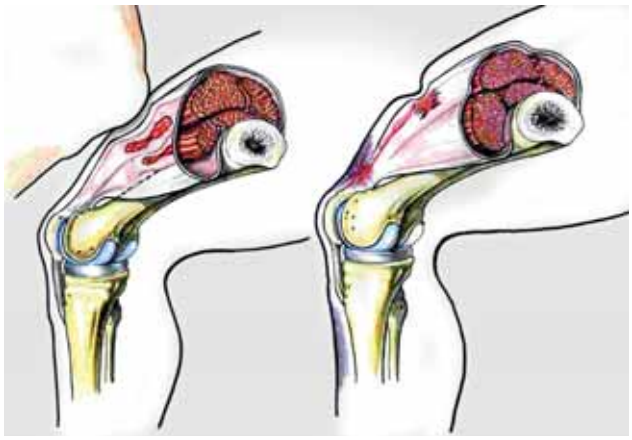


Figure 3.3.4.1 Typical injury mechanism for quadriceps contusions – crushing of the muscle belly between the femur and the opponent's patella resulting in (A) intramuscular haematoma or (B) intermuscular haematoma. Illustration reproduced with permission from Clinical Guide to Sports Injuries (Bahr & Maehlum, 2003) ©Gazette Bok/T. Bolic.

Figure 3.3.4.2 Muscle strain injuries can occur in the myotendinous junction anywhere along the muscle belly of the hamstring muscles (illustrated here for the biceps femoris). Avulsions from the ischial tuberosity can also occur. Illustration reproduced with permission from Clinical Guide to Sports Injuries (Bahr & Maehlum, 2003) ©Gazette Bok/T. Bolic.

Hamstring strains occur most often during maximal sprints. No research exists to show exactly at what time during the gait cycle injuries occur. However, since the net moments developed by the hamstrings are thought to be greatest in the late swing phase, right before heel strike, and at push-off, these are thought to be the vulnerable positions. In these phases, the hamstrings work eccentrically.

A number of candidate risk factors have been proposed for hamstring strains, the most prominent being the following three internal factors: previous injury, reduced range of motion (ROM), and poor hamstring strength. In theory, limited ROM for hip flexion could mean that muscle tension is at its maximum when the muscle is vulnerable and close to maximum muscle length. However, this hypothesis has yet to be confirmed. In fact, studies on football players suggest that hamstring flexibility is not a risk factor for strains. Low hamstring strength would mean that the forces necessary to resist knee flexion and start hip extension during maximal sprints could surpass the tolerance of the muscle-tendon unit. Hamstring strength relative to quadriceps strength is often expressed as the hamstring: quadriceps strength ratio, since it is the relationship between the ability of the quadriceps to generate speed and the capacity of the hamstrings to resist the resulting forces that is believed to be critical. In contrast to flexibility, several studies show that players with low hamstring strength or a low hamstring:quadriceps strength ratio are at increased risk of injury. A previous injury can cause scar tissue to form in the musculature, resulting in a less compliant area with increased risk of injury. A previous injury can also lead to reduced ROM or reduced strength, thereby indirectly affecting injury risk. Football players with a previous hamstring injury have a seven times higher risk of injury than players with no injury history; among players with no history of previous strains, approx. 2% can expect to suffer a new injury during a season, while the corresponding figure for players with a history of previous strains is 13%.

Other risk factors, which have been suggested but not yet critically examined, include improper running technique, high running speed, low back pain, and increased or changed training programmes (particularly intense periods of training).

3.3.5 Symptoms and signs

History is in most cases sufficient to diagnose an acute thigh muscle injury, with the player reporting having "pulled" his hamstrings during sprint running or receiving a blow from an opponent, followed by acute onset of localised pain and immediate reduction of function. If a significant contusion injury has occurred, the thigh will look thicker than normal. Measuring the circumference of the thigh with a tape measure and comparing it with the healthy side can help quantifying the degree of swelling though other factors have to be considered such as the frequent differences in thigh circumference in players (dominant leg etc.). A major contusion will leave a mark on the thigh

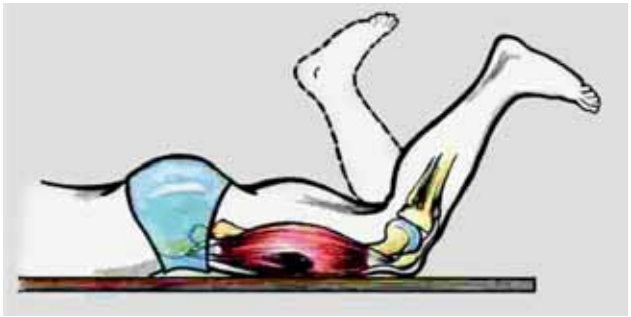


Figure 3.3.5.1 Functional test to distinguish between intramuscular (where the knee can be flexed less than 90°) and intermuscular haematomas (where the knee can be flexed more than 90°) after a quadriceps contusion. Illustration reproduced with permission from Clinical Guide to Sports Injuries (Bahr & Maehlum, 2003) ©Gazette Bok/T. Bolic.

so that there is no doubt about whether or not the player sustained a high-energy trauma. If the player has suffered a muscle or tendon rupture in the hamstrings, a depression will initially be visible at the site of the injury, and distinct changes in skin color will gradually become visible distal to the location of the rupture. Bruises usually become visible on the skin (often distal to the location of the injury) a couple of days after the patient has sustained an intermuscular injury, but not after an intramuscular injury.

The degree of reduced flexion in the knee joint may also help to distinguish between an intramuscular and an intermuscular injury. If the compartments are intact, bleeding in the thigh is restricted. This leads to increased intramuscular pressure, which reduces ROM, namely flexion in the knee joint (Figure 3.3.5.1). It is often easier to conduct the functional tests and make an assessment two to three days after the injury occurred. Less than 90° flexion in the knee joint indicates intramuscular bleeding and a longer rehabilitation period. More than 90° flexion indicates intermuscular bleeding, with a shorter rehabilitation period.

Functional muscle tests must be performed to distinguish between partial and total tendon or muscle ruptures. Both the quadriceps and the hamstring apparatus can be tested isometrically and dynamically – including resistance to eccentric force – to obtain information about the degree of the injury.

3.3.6 Examination and diagnosis

Acute moderate and severe muscle injuries are usually not difficult to diagnose with a typical history, localised pain, tenderness to palpation, and loss of function. Minor injuries are more difficult to distinguish from cramps, where there is no structural damage to the muscle. If the technology is available, ultrasound or MRI examinations can help when making the diagnosis. However, although the average time before return to play is higher when the hamstring muscle injury is confirmed by MRI or ultrasound (a mean of 27 days) than in cases where no hamstring injury can be detected (a mean of 16 days), there is considerable variation within each of these groups.

Unless there is clinical evidence to suspect a femur fracture, further investigations are not necessary at the acute stage. If the clinical findings cannot exclude a fracture of the femur or an avulsion fracture of the ischial tuberosity, a plain X-ray should be taken. Also, MRI is an accurate examination for determining whether or not there is a total rupture, particularly for hamstring and quadricep injuries, or an avulsion of the ischial tuberosity (an avulsion will usually also be visible on a plain X-ray).

Later in the rehabilitation period, calcium deposits will gradually become visible after intramuscular bleeding if the patient develops myositis ossificans (see below), and X-rays should be obtained if recovery is slower than expected.

3.3.7 On-field treatment

The key issue when assessing an acutely injured player on the pitch is whether he should be removed from play or not. Intramuscular bleeding starts immediately and is maximal during the first minutes after the injury occurs. Thus, the sooner optimal PRICE therapy is started (protection, rest, ice, compression, elevation), the more the bleeding can be limited. Immediate PRICE therapy is therefore key to confining the recovery period.

As mentioned, hamstring strains occur during a maximal sprint, and are often not even noticed by anyone other than the player himself. However, it will often be obvious to the player that he cannot perform because of reduced sprinting performance or even limping, and he will ask to be removed from the field of play. Even for smaller injuries this is often the case, and the decision to remove them from play is easy.

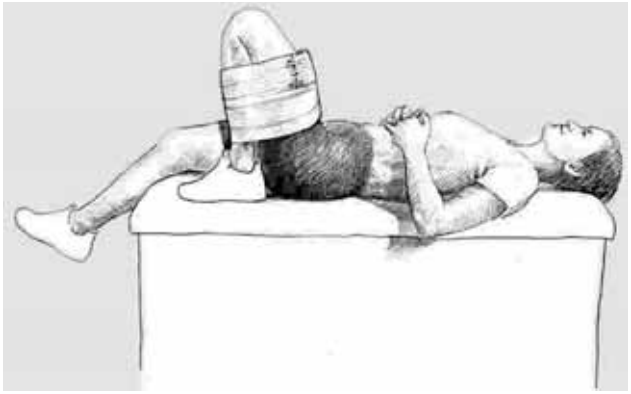


Figure 3.3.7.1 An effective way to provide PRICE therapy immediately after a contusion injury to the thigh is with the knee in maximal flexion. Illustration reproduced with permission from Clinical Guide to Sports Injuries (Bahr & Maehlum, 2003) ©Gazette Bok/T. Bolic.

In the case of contusions, the decision is often more difficult. The typical scenario is a player going down after having received a blow to the thigh, and medical personnel being called to the pitch to assess and assist. In this case, it is not easy to distinguish between a minor subcutaneous contusion and a muscle injury. The decision must be made based on the force of the trauma involved. A high-force blow by the kneecap almost always results in significant bleeding, and the player should be removed for further assessment and functional testing on the sideline.

If a decision is made to remove the player from play, the key is to establish maximal compression, preferably also with an ice pack, as soon as possible. Ice sprays have no effect. For a contusion injury, the best way to limit bleeding in the first hour after injury is to apply compression with the knee in maximal flexion (Figure 3.3.7.1). This increases compartment pressure and significantly limits blood flow to the muscle.

3.3.8 Non-operative and operative treatment

Intramuscular bleeding will gradually subside, but after significant injuries there will be some bleeding for as long as 48 hours after the injury. The main objective – whether treating a contusion injury or a muscle strain – is to control haemorrhage – through rest and compression with a compression bandage. Massage is contraindicated during this period. If the patient has a major injury, it may be necessary to wait four or five days before beginning active exercises, but rehabilitation of minor injuries should begin two or three days after the injury.

Whether to use nonsteroidal anti-inflammatory drugs (NSAIDs) or COX-II inhibitors in the acute phase is debatable. Animal studies have shown that NSAIDs increase the rate of force recovery in the first week after injury, but that torque production is still reduced four weeks after injury. Because they do not inhibit blood platelet function and therefore do not increase the risk of bleeding, COX-II inhibitors may be preferable to regular NSAIDs, if used at all. Corticosteroid injections are contraindicated in the acute stage. Although they may provide some advantages in the short term by accelerating recovery, they may make the player more susceptible to re-injury in the long term. Also, since corticosteroids are usually given mixed with a local anaesthetic, it is not known which of these substances act to provide pain relief in the short term.

If an acute tendon rupture from the ischial tuberosity has occurred, surgical fixation achieves good results. Surgery should however be performed within two weeks after the injury.

3.3.9 Rehabilitation programme

The goals of the subsequent rehabilitation phases are, first, to restore pain-free range of motion and, second, to reach a performance level that allows return to play through a functional rehabilitation programme. Progression from one stage to the next is guided by function, not time. Massage may be used at this stage to improve circulation.

The rehabilitation of most minor thigh injuries should begin with active mobilisation. The player should start with gentle motion exercises of the relevant joints and should allow the pain to control how long to move. Pressure should not be applied during the start phase. Use of a cycle ergometer is a gentle and effective method of increasing mobility. The seat should be adjusted so that it is high, and the foot should be placed further forward on the pedal than normal. This position reduces the demand on knee flexion and makes it easy to pedal when cycling. If mobility is reduced to the extent that the player cannot pedal, then the player should oscillate gently forth and back as far as possible on the bicycle. As mentioned above, for major muscle strain injuries and contusions, it is advisable to allow the area to relax for up to five days before beginning active mobilisation.

Exercises are of primary importance during the rehabilitation phase, but other physical therapy may be useful in removing bleeding residue and avoiding scar tissue formation in the injured area. Massage, stretching and various types of electrotherapy may be indicated.

The exercise programme should include various stretching exercises, strength exercises, neuromuscular exercises and functional exercises aimed at a return to football. The progression of the exercises is individually controlled by pain and function. In general, numerous repetitions and low loads (up to a total of 100 repetitions split into four to five series) are emphasised early in the rehabilitation phase. Then load is gradually increased, and the number of repetitions decreased. Easy exercises are

used during the start-up phase, later the tempo is gradually increased.

Football players with muscle strain injuries must not run at their maximum pace during training until the injury is completely healed. It often takes as long as six to eight weeks before the musculature will tolerate maximum spurts or turns. Light running training in a relaxed manner may begin as soon as the pain allows. An insulating neoprene sleeve is useful during the retraining phase to keep the muscles warm. A good warm-up, including gentle stretching, is critical before more vigorous exercises are undertaken.

Building maximal strength, especially eccentric strength, is key to avoiding re-injuries. One such exercise, Nordic hamstring lowers (cf. 2.2.3, The 11+), has been shown to be much more effective than regular hamstring curls in improving eccentric strength, and has also been shown to reduce the risk of hamstring strains in footballers.

3.3.10 Prognosis and return to play

Healing often takes as long as six to twelve weeks after intramuscular bleeding. However, an injury with intermuscular bleeding can heal within a couple of weeks. If the injury is minor, after a week the muscles will regain strength and the ability to contract. About 50% of the strength is regained within 24 hours. After seven days, 90% of the strength returns. The player should be able to train without symptoms at the intensity level of a competition, and be tested thoroughly before participating in matches or competitions. Returning to explosive sports such as football too early may however cause a re-injury.

The prognosis is generally good if the above-mentioned considerations and rehabilitation guidelines are duly considered, and the return to play has been sensibly evaluated.

3.3.11 Sequelae and their treatment

Acute compartment syndrome is rare, but may occur in football players after quadriceps contusion injuries. Whereas normal pressure in a muscle compartment is less than 20mm Hg, pressure in the compartment of the rectus femoris muscle may increase up to between 80 and 100 mmHg if there is a large muscle injury with intramuscular bleeding. The main symptom is gradually increasing and eventually intolerable pain with low to no response even to powerful analgesics such as morphine derivates, and the muscle is rigid on palpation. The player can be monitored by repeated measuring of the circumference of the thigh and by comparing it with the opposite side. If the circumference of the thigh increases gradually, the patient should be admitted to hospital and closely monitored using direct pressure monitoring by qualified personnel. In rare cases, it may become necessary to open the fascia surgically to relieve pressure.

Occasionally after a muscle injury in the thigh, the haematoma calcifies. This condition is known as myositis ossificans and can usually be seen on a plain X-ray no earlier than 3-4 weeks after the injury. Myositis ossificans is a potential consequence of major bleeding in the deep thigh muscles. At the beginning of the calcification process, the area is still swollen and often very tender (warm phase). Pain and swelling result in long-term reduced movement, which usually restricts knee joint flexion. This diagnosis should be considered and X-rays or an MRI obtained if progression is slower than expected with persistently reduced ROM. The calcification eventually stabilises (cold phase). Treatment consists of stretching and exercise therapy to such extent as the pain allows. Usually, the calcium and haematoma are resorbed without surgical treatment, but if after more than six months there are no signs of resorption, the player should be referred to a surgeon for evaluation of a possible need for surgery. The prognosis is good, but it often takes a long time before the calcification is totally resorbed.

3.4. Groin and hip injuries

3.4.1 Incidence

Groin and hip injuries in football are common, yet often difficult to diagnose and treat. Whereas the differential diagnoses previously consisted of more than 90% of tendinopathies and muscle weakness/ hernias, the new knowledge of the hip joint anatomy and impingement symptoms in the young player has necessitated a shift in diagnostic focus and clinical skills during the last decade.

Groin injuries are among the four most common types of injury in football. They account for 7-11% of all injuries in some Olympic sports, including ice hockey, speed skating, football, swimming and athletics, and 9% of injuries in Norwegian elite football. Groin strain injuries have been cited as accounting for 20% of all muscle strain injuries at elite levels of football. However, to date, there is little knowledge about the prevalence and incidence of femuroacetabular impingement (FAI) in football.

Groin injuries may be acute but often become chronic in nature. The most common location (>50%) of groin pain reported in athletes in general is the adductor muscle tendon region. Acute onset pain in this region is commonly attributable to an adductor longus muscle enthesopathy but may also be related to the iliopsoas and/or the abdominal muscles. The differential diagnoses for groin pain in football players are multiple and outlined in Tables

Sport	Competition incidence[1]	Training incidence[1]	Rank[2]	Comments
Team sports				
Ice hockey	2-5	0.5	2-4 (10%)	Prospective data
Football	4	1	2-4 (10-20%)	Prospective data
Individual sports				
Speed skating	NA	NA	5 (10%)	Retrospective data
Swimming	NA	NA	NA	Retrospective data suggests 25/100 elite swimmers per year experience groin pain
Athletics	NA	NA	4-6 (7-9%)	Prospective data

Table 3.4.1.1 Risk of groin injury in different sports. The numbers reported are average estimates based on the studies available.

1) Incidence is reported for adult, competitive athletes as the number of injuries per 1,000 hours of training and competition.

2) Rank indicates the relative rank of this injury type within the sport in question, as well as the proportion as a percentage of the total number of injuries within the sport.

NA: Data not available.

Most common	Less common	Must not be overlooked
Adductor muscles Muscle strain Tendinopathy	Iliopsoas strain Rectus femoris strain Rectus abdominis strain Avulsion of m. sartorius Avulsion of rectus femoris	Slipped capital epiphysis
Hip joint Synovitis labral tear Chondral lesion	Stress fracture - Neck of femur - Pubic ramus	Intra-abdominal abnormality
Direct hernia	Referred pain Sacroiliac joint Lumbar spine	Prostatitis Gynaecological conditions Urinary tract infections

Table 3.4.1.2 Overview of current differential diagnoses of acute groin and hip pain

Most common	Less common	Must not be overlooked
Adductor-related tendopathies Iliopsoas-related tendinopathy Rectus-abdominis-related tendo-pathy	Hip joint Femuro acetabular impingement (FAI) Osteoarthritis Labral tear Chondral lesions	Slipped capital epiphysis Coxitis Legg-Calvé-Perthe's disease Avascular necrosis of the head of the femur
Abdominal-wall-related (footballer's hernia) - Posterior wall weakness Tear of the external oblique aponeurosis	Stress fracture - Femoral neck - Pubic bone Osteoid osteoma	Intraabdominal abnormality Prostatitis Urinary tract infections Gynecological conditions
Pubic bone "osteitis"	Nerve entrapment - Obturator - Ilioinguinal - Genitofemoral	Spondyloarthropathies Ankylosing spondylitis
	Referred pain - Lumbar spine - Sacroiliac joint	Tumors
	Apophysitis	

Table 3.4.1.3 Overview of current differential diagnoses of chronic thigh and hip pain

3.4.1.2 and 3.4.1.3 separately for acute injuries and for chronic painful conditions. There is a considerable overlap between the two since the majority of these problems are not presented to the physician or physiotherapist until considerable time has elapsed and acute injuries have turned into chronic conditions.

3.4.2 Functional anatomy

Three major areas of anatomy require attention to and knowledge of the groin and hip area. The adductor group consisting of adductor longus, magnus, brevis and gracilis

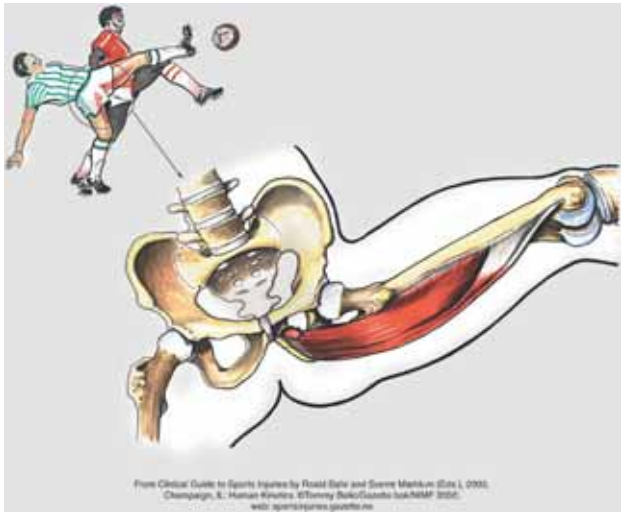


Figure 3.4.2.1 Knowledge of functional anatomy is necessary to understand injuries to the hip and groin

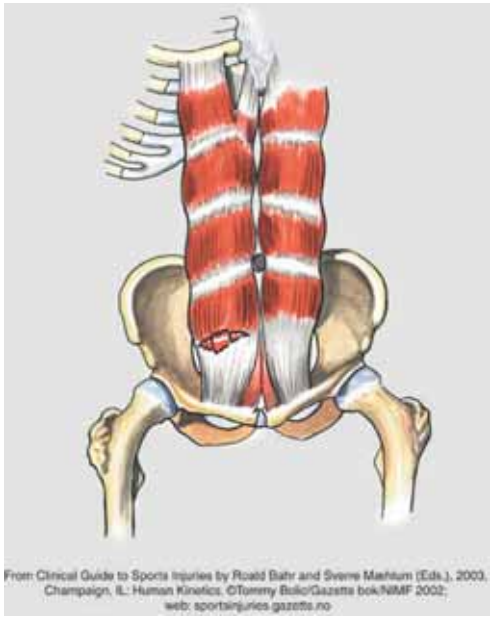


Figure 3.4.4.1 Strain of the rectus abdominis

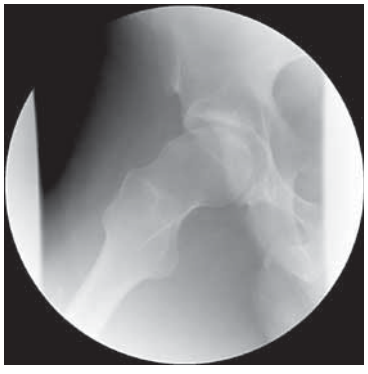


Figure 3.4.4.2 An example of cam impingement of the hip on X-ray using the Lauenstein technique: there is an extra "bump" (pistol-grip formation) of the proximal femoral neck leading to impingement against the acetabulum when the hip is flexed and internally rotated.

and the rectus femoris and pectineus, the pelvis groin group consisting of tranversus abdominis, obliquus externus and internus and rectus abdominis, and the anatomic structures of the hip (femur, acetabulum), the labrum and the rotating muscles and tendon of the hip all require the attention of the clinician in a footballer with pain in the groin or the hip (Figure 3.4.2.1).

3.4.3 Classification and grading

Since there are multiple differential diagnoses of injury and pain in the groin area, only tendon strains and ruptures have a classification, in which grade III is a total rupture, grade I a light strain in the tendon that usually does not change the ultrasound or MRI image, and grade II a partial tear.

3.4.4 Causes and mechanisms

Groin injuries have two typical histories:

1. 40% of players experienced an acutely painful incident during training or competition, and
2. 60% experienced gradually developing pain in the groin region, which is typical of an overuse injury where the exact onset of symptoms or an initial event can be hard to establish.

An acute strain usually involves one or more muscles and happens during forceful action. In most cases, the lesion lies within the musculotendinous junction, but in some cases the site of the injury is the tendon itself or the entheses where the tendon inserts into the bone. Adductor muscles are often acutely strained during an eccentric contraction (e.g. in a forced abduction), when this muscle is at its weakest and as such more prone to injury. This could be the sudden resistance of an opponent's foot in an attempt to reach a ball or a sliding tackle (Figure 3.4.2.1). In many cases, some degree of hip joint rotation is involved. Another mechanism is forceful concentric adduction, for instance during a kick for a ball in the air, or after direct blunt trauma by an opponent.

The iliopsoas muscle, being a very important and strong hip flexor, can be acutely strained if forceful hip flexion is suddenly interrupted, e.g. when the player is tackled during running, skating, jumping or kicking, or when

he accidentally kicks into the ground instead of the ball. The ilipsoas can also be injured when the thigh is suddenly forced into extension and the iliopsoas tries to decelerate the movement by instantaneous eccentric contraction.

An acute strain of the lower abdominal muscles usually involves either the conjoint tendon of the transverse and the internal oblique abdominal muscle inserting at the pubic tubercle, or the rectus abdominis muscle at its enthesis at the pubic bone or in its distal musculotendinous junction. The typical mechanism is over-stretching of the groin and lower abdomen as in a forceful sliding tackle, or an uncontrolled fall backwards where the hip is extended and the abdominal muscles try to stop the fall by contracting with great speed and force, often eccentrically (Figure 3.4.4.1).

The more rotation involved in the fall, be it either in the hip joint or in the torso, the more likely the conjoint tendon will sustain the lesion. In this situation, the hip flexors (iliopsoas, rectus femoris and tensor facia latae) are also at risk of sustaining an injury.

Dysplasia of the hip joint can be found in a number of variations and many of these include abnormalities in the range of motion (ROM) of the hip joints, in most cases reduced ROM. The impingement problems of the hip joint mentioned above include groin pain and in most cases some degree of decreased ROM of the joint. In the case of the two most common types, bony abnormalities potentially lead to further structural damage to the hip joint. Cam impingement (more frequent in males) is due to a lack of offset of the femoral neck in relation to the femoral head (Figure 3.4.4.2), whereas pincer impingement (more frequent in females) is usually caused by a retroverted acetabulum where the labrum is squeezed between the bony labrum and the femoral neck (Figure 3.4.4.3). Both forms often occur together. They can be found in players without groin or hip pain but decreased ROM of the hip joints and are most probably a risk factor for developing groin pain. If the impingement problem is symptomatic of hip joint and groin pain, damage to the acetabular cartilage as well as to the labrum of the hip joint is often found.

Lesions to the lower abdominal muscles and other structures associated with the inguinal canal can lead to a condition showing some similarities to a hernia, and the terms "sports hernia" and "incipient hernia" are commonly used for this condition, which is particularly common in football players. It refers to pain located at the lower part of the abdomen and the upper and medial part of the groin region, often radiating to the medial thigh and across the pubic symphysis. A predisposition for hernia development might be present, but the primary mechanism of injury is probably an acute trauma as described above, or a period of overuse as a result of a misbalance of the muscles acting on the pelvis plus intense strenuous activity often involving sprinting, jumping, kicking and sudden changes of direction. The nature of the lesion is not always clear. It may be a strain or tear, inflammation or degeneration due to excessive stress, an avulsion, haemorrhage or oedema. It is probably the result of a structural lesion of the muscles and/or tendons involving weakness of the posterior inguinal wall without a clinically obvious hernia (Figure 3.4.4.4).

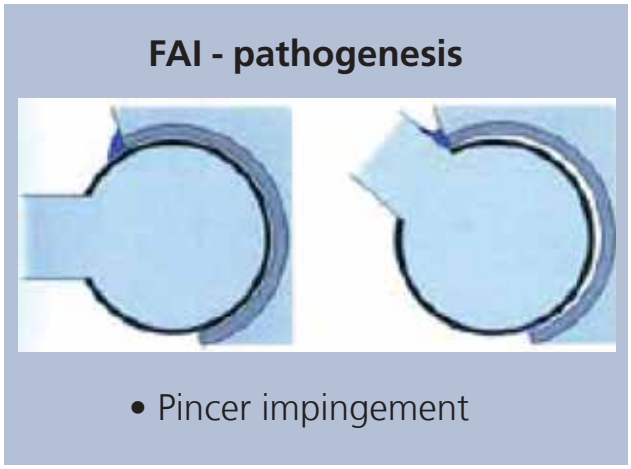


Figure 3.4.4.3 Pincer impingement of the femoral head against the acetabulum due to a retroverted acetabulum

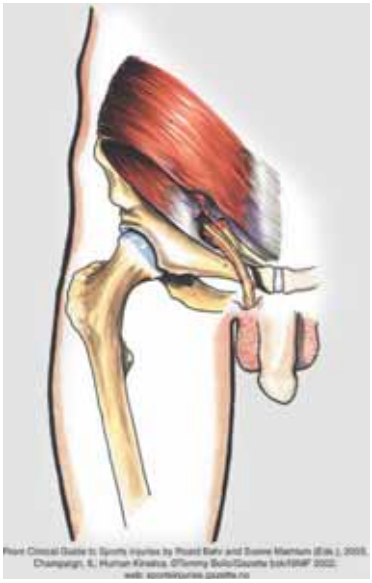


Figure 3.4.4.4 Classical site of weakness of the abdominal wall

Because of the insidious onset and non-specific nature of the symptoms, there is often a prolonged course of disease prior to diagnosis.

3.4.5 Symptoms and signs

As mentioned above, acute strains occur as a result of forceful action and the players are usually well aware that something has happened: they feel pain and the function of the limb is affected. In most cases, the player will have to stop training or playing and leave the pitch. In some cases the player describes a snapping feeling in the groin/hip, occasionally even accompanied by a sound. If not attended to appropriately, these injuries might develop into a more long-standing and sometimes chronic injury. Local signs are usually present such as swelling or discoloration of the skin in the case of a haematoma, a palpable gap in the tendon in the case of a partial or total rupture, or provoked pain when testing the various muscle groups, nerves or the hip joint.

The typical overuse pattern of groin injury is initially indicated by pain after activity, stiffness of the respective muscle group and a decreased range of motion of the hip joint, later progressing to pain with specific movements. If the player does not receive appropriate treatment and continues to play, the pain-free periods will become shorter until finally all football activity will cause pain, and even everyday activities might become a problem. Frequently, there is no recollection of a single incident causing the pain, but often there has been an increase or change in activity level, technique or likewise. In football players with hip pain (FAI), the gradual onset may have been preceded by an incident where the hip was overstretched, usually in flexion and internal rotation or in repetitive external rotation or hyperextension.

3.4.6 Examination and diagnosis

The key to a good, thorough clinical examination is to be familiar with the epidemiology, the nature of football as a sport, the anatomy and the various validated examination techniques. The examination includes a systematic hip examination (be aware of pain in internal rotation and flexion = anterior labrum tear), a lumbar and sacral spine examination, distinct palpation of the major tendon insertion sites and muscles and finally a regular hernia examination. Depending on the history, palpation of the prostate or a gynaecological examination may be necessary, as may urine culture and microscopy.

In some players, impaired pelvic stability puts increased stress on the symphysis joint, leading to a stress reaction which, combined with the bony changes seen with the enthesopathy of the adductor longus insertion, leads to an increased signal when examined with bone scan and with MRI, and to irregularities on X-ray. This is sometimes called "osteitis-pubis-like changes" as it looks like the changes seen with infection of the joint. These changes are not diagnostic of groin injury since they are primarily related to the amount of play.

An ultrasound or an MRI will help in establishing a tendinopathy diagnosis, but the choice of treatment must primarily be based on a good clinical examination. A sport hernia is an exclusion diagnosis and, so far, little additional information can be obtained from an MRI and ultrasound except that they enable differential diagnoses to be ruled out. In the case of FAI, radiography with Lauenstein projections and MRI arthrography are needed to confirm the clinical diagnosis. The clinical diagnosis is challenging, but the majority of these patients will have pain on hyperflexion and internal rotation of the hip joint.

3.4.7 Non-operative and operative treatment

Table 3.4.7.1 presents a general treatment approach in cases of acute injury. If little improvement is seen within six weeks of well-designed training, the possibility of a sport hernia or FAI should be discussed and adequate tests (see above) made. Surgery is rarely required for acute muscle tendon injuries in the groin.

Decreased ROM of the hip joint related to bony abnormalities is a difficult problem to treat. Osseous problems in impingement syndromes mostly need to be treated surgically if they are symptomatic, followed by a closely monitored rehabilitation programme to re-establish muscle strength and coordination as related to the pelvis. Currently, impingement surgery of the hip seems to enable the majority of players (60-80%) to return to football. How these players should be advised regarding further sports activities is not clear and is currently a subject of discussion and ongoing scientific studies.

In intractable adductor tendinopathy, surgical tenotomies can be carried out. The surgery is minor, the football player is usually out for eight weeks and the return rate to full play is high. In the case of abdominal wall weakness, success has been obtained with conventional hernia surgical techniques as well as with laparoscopic techniques. Many football players return to play within six weeks. The long-term disability has not been established for these problems. Occasionally, nerve impingement has to be treated with surgery. Recently, there has been a large increase in the number of hip arthroscopies for FAI and for treatment of labral tears. Surgical techniques have been developed for repair of the labrum and for removing impinging bone from the acetabulum or from the femoral neck. The indication for this type of surgery is still evolving and the long-term results are not known at this time. It seems that players will return to play three to four months after impingement surgery, whereas labral repair usually requires a break of at least four months.

3.4.8 Rehabilitation programme

Previous groin injury is one of the most well-established risk factors for sustaining a new groin injury. Consequently, rehabilitation needs to be completed prior to the return to play (i.e. pain-free and functional ROM completely regained, full strength on resistance testing, concentric and eccentric muscle-specific function fully retrained). The treatment needs to be specifically geared to the individual injury, and must be functional for the demands of the player, i.e. including football activities. It is not enough to treat a damaged tendon insertion with steroid injections and temporarily remove the player from play, allowing him to return once the pain is gone. Likewise, "core stability" exercises to stimulate the abdominal and low back muscles alone and rehabilitation of a damaged tendon insertion are not sufficient treatment if the aim is to prevent recurrence of the groin injury.

To successfully prevent the recurrence of groin injuries, treatment aimed at healing the structural damage should be combined with correction of the initial dysfunction causing the pain. Otherwise there is a risk that the factors that originally led to the structural damage will again "take over" and, combined with fatigue due to lack of sport-specific rehabilitation, may eventually lead to groin re-injury and increased risk of other injuries as well. This is achieved by subjecting the player to an exercise programme to re-establish pelvic stability and a rehabilitation programme that gradually includes the demands and skills needed to play football before he or she is allowed to return to play. The treatment protocols for groin pain (adductor injuries) have been used with success since they were published in 1999, but the therapist must be aware that non-operative treatment will take a long time and may at times be rather frustrating for the player.

3.4.9 Prognosis and return to play

In acute cases, healing will take place within two to four weeks and training is begun and gradually increased if the player is asymptomatic. Return to play is usually possible within one to three months but may take longer in chronic cases. When rehabilitation fails over more than six months, surgical treatment may need to be considered, with the time required for return to play as indicated above.

	Goals	Measures
Acute phase	Minimise or reduce swelling.	PRICE principle with emphasis on good compression.
Rehabilitation phase	Normalise movement and reduce pain so that the patient can achieve normal function.	Exercises, massage, stretching.
Training phase	As a minimum, achieve previous strength and flexibility. Reduce the risk of re-injury.	Functional exercises – sport-specific training. Recover fully before [engaging in] maximum activity.

Table 3.4.7.1 Goals and measures for rehabilitation of acute groin and hip injuries

3.5. Tendon and overuse injuries

3.5.1 Incidence

Overuse injuries are the result of constant overloading by repetitive forces and wearing down of a tendon, muscle, bone or joint. In football, overuse injuries account for 9-34% of all injuries and range from mild tendinopathy to stress fractures. This chapter focuses on tendon overuse injuries, in particular of the Achilles and patellar tendon.

3.5.2 Functional anatomy

Tendons are made up of tightly-packed bundles of collagen, which gives them a white, glistening appearance. Tendons are surrounded by either a loose connective tissue lining known as the paratendon, or by a sheath, which houses the blood supply and allows the tendon to glide. The tendon transmits force from muscle to bone, and also stores and releases elastic energy. Tendons are exposed both to low-level cyclic loading (for instance during running), and to peak loads such as when jumping. The Achilles tendon withstands forces of up to six times body weight when landing from a jump.

Collagen continually undergoes a low level of microtrauma, not only during training and matches, but during daily life as well. Tendon is therefore populated by cells (tenocytes) whose job is to repair or absorb old collagen and assemble new collagen. Tendons can gain volume and become stronger in response to suitable training by increasing the amount of collagen produced by tenocytes. Alternatively, tendons can lose mass during periods of disuse and inflammation.

Tendon that is chronically painful shows a breakdown of the normal anatomy (Figure 3.5.2.1). The tendon becomes dull brown or grey, and is grossly thickened. There are frequently deposits of fibrin or fibronectin in the tendon or its linings, indicating the presence of accumulated tissue injury. There may be adhesions (scarring) between the tendon and its connective tissue linings or sheaths, which prevents normal gliding. The normally sparse blood and nerve supply is increased, with ingrowth of nerves and vessels into the tendon. The collagen of the tendon becomes thin and disorganised, having lost its tightly packed arrangement.

3.5.3 Classification and grading

Tendon overuse injuries (tendinopathies) are a chronic problem and should be distinguished from acute tendon ruptures. Unlike acute injuries, overuse injuries have a gradual onset. Only 10% of patients with tendinopathy

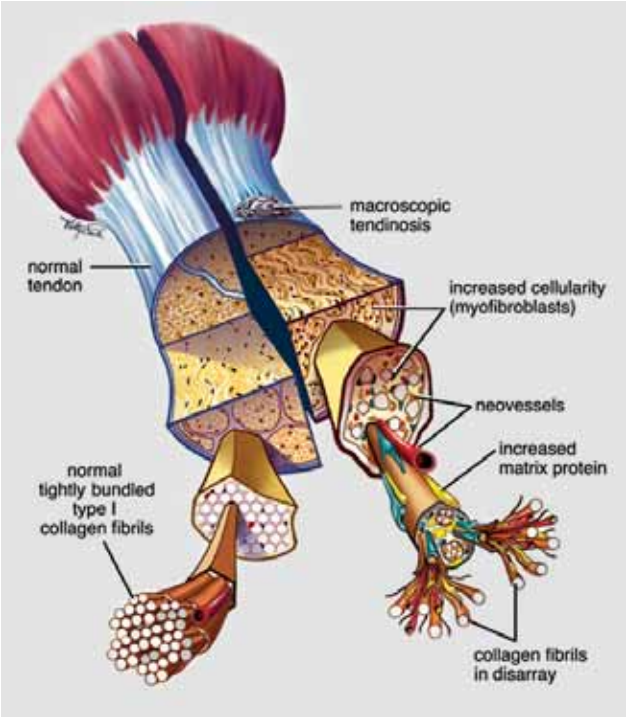


Figure 3.5.2.1 Normal and abnormal Achilles tendons. Abnormal tendons show collagen disarray, increased extracellular matrix (glycosaminoglycans), neovessels and increased number of fibroblasts.

can recall an acute onset. Thus, tendon rupture and tendinopathy are considered distinct clinical entities.

In football players, Achilles, patellar, hip and groin tendinopathies (adductors, iliopsoas, rectus abdominis, obliques, gluteal) are common, with Achilles and patellar tendinopathies being most prevalent. Tendinopathies may be insertional or involve the midsubstance of the tendon. Distinguishing involvement of the paratendon versus the tendon proper is difficult clinically, but this distinction does not usually influence treatment decisions.

Functional scores, VISA (Victorian Institute of Sport Assessment) and VISA-A (Achilles) have been validated for patellar and Achilles tendinopathies. These scores may be used when grading the severity of symptoms and response to treatment, or for pre-season screening of tendon function.

Ultrasound imaging, with or without colour Doppler, has been used as a means to identify asymptomatic, at-risk tendons at the start of season. However, ultrasound is highly user-dependent, and ultrasound-based predictions of tendon prognosis are not yet accurate enough to warrant routine use.

3.5.4 Causes and mechanisms

The major risk factors for tendinopathies involve tendon loading on the one hand, and factors which may be influencing tissue repair and pain on the other hand. Tendon behaviour under tensile loading follows the "stress strain curve". As loading progresses over time, tissue strains increase. As strains progress, a load which was initially safe may begin to cause damage. Thus, prolonged loading is a key factor in the development of tendinopathy. Other factors which increase local tendon strains and predispose to tendinopathy include reduced joint range of motion, muscle-tendon shortening or imbalance, or lower extremity malalignments.

The other important underlying cause of tendinopathy is an imbalance between the accumulation of damage to collagen fibres and the rate of repair of the tenocytes. Over time, this leads to a progression of injury and the insidious development of pain as nerves begin responding to the changes in the tendon. The tendon becomes dull brown or grey, and is grossly thickened and softened.

The histology underlying tendinopathy is tendinosis, a state of failed tissue repair. The source of pain appears to be an irritation of the local sensory nerves that supply the tendon and proliferate along with vessels in areas of injury, causing pain. In some cases, tendon tissue is replaced by fibrocartilage or bone, leading to a chronic irritation of the surrounding tendon. Associated innervated tissues (e.g. bursae, fat pads) may also contribute to tendon pain if they become irritated.

Many factors affect tissue repair. Advancing age, smoking, diabetes, infection and high BMI are all factors which impair tissue healing and increase the risk of tendinopathy. Corticosteroids inhibit collagen synthesis, and have been linked in some studies to impaired tendon healing or the recurrence and/or exacerbation of tendinopathy. The role of nonsteroidal anti-inflammatory drugs in tissue repair is a matter of controversy and currently under research. The prevention of tendinopathy in football players should include, where appropriate, weight-reduction, smoking cessation, and avoidance of glucocorticosteroids where possible. The latter is of particular importance as the application of local glucocorticosteroids is widespread in football.

3.5.5 Symptoms and signs

Patients with tendinopathy usually experience pain some time after exercise or, more frequently, the following morning upon rising. The tendon can be painful even at rest, and sometimes becomes less painful with use. Players can "run through" the pain or the pain may disappear with warm-up, only to return after exercise when they cool down. The player is able to continue to train fully in the early stages of the condition; indeed, this ongoing loading may interfere with the body's own healing process.

3.5.6 Examination and diagnosis

Physical examination is usually sufficient to establish a diagnosis of lower extremity tendinopathy. Local tenderness on palpation and macroscopic thickening are the hallmarks of tendinopathy. Frank swelling and crepitus are occasionally present. These may be due to associated paratendonitis, or due to the water-attracting nature of tendinosis tissue.

Imaging (colour Doppler ultrasound) is also helpful to visualise the extent of hypoechogenicity and

neovascularisation (Figure 3.5.6.1). Tenderness on palpation and pain during unilateral tendon loading are universally present.

3.5.7 On-field treatment

Occasionally, a chronic tendinopathy may rupture fully or partially during competitive play or training. Thompson's test may be used to identify an acute Achilles tendon rupture (Figure 3.5.7.1), but the possibility of a partial tear should also be kept in mind. Testing plantar flexion alone will lead to false negative results, since long flexor muscles of the toes (and tibialis posterior muscle to some extent) take over, even when standing on both feet. Acute tendon injuries require immediate PRICE treatment (protection, rest, ice, compression and elevation for at least 30 minutes). Transportation to a medical facility should be requested as soon as practicable.

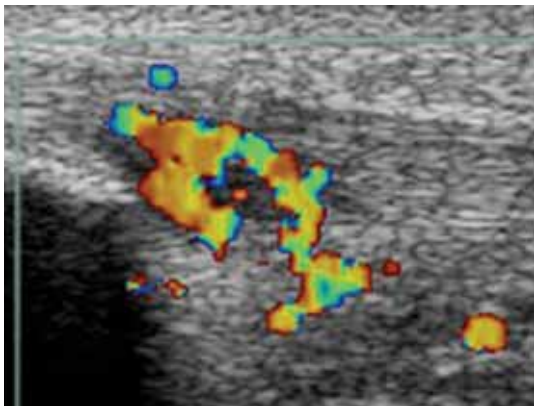


Figure 3.5.6.1 Colour Doppler ultrasound appearance showing increased blood flow in symptomatic tendinopathy.

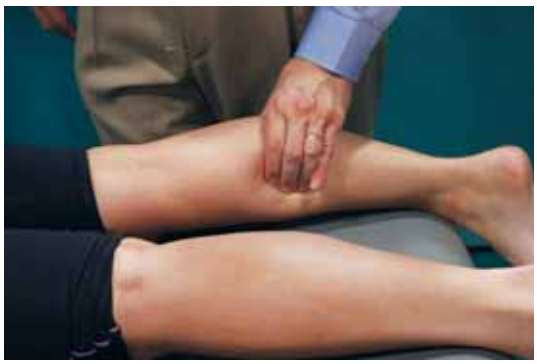


Figure 3.5.7.1 Thompson's test – the calf is squeezed. A lack of plantarflexion indicates a complete Achilles tendon rupture.

3.5.8 Non-operative and operative treatment

Chronic tendinopathies often take a long time to resolve, particularly if symptoms have been present for some months before presentation. Evidence-based treatments include exercise therapy (see below) and nitric oxide patches. There is emerging evidence for sclerosing therapy and prolotherapy, which are novel treatments. Other commonly used treatments include relative rest, electrotherapeutic modalities and soft tissue therapy.

Nitric oxide patches for tendinopathy are considered an "off-label" use for tendinopathy. They appear to act by directly stimulating collagen gene expression in tenocytes. A standard angina patch is divided into quarters and placed directly over the relevant tendon 24 hours a day. The skin should be monitored for signs of irritation, and the patient should be aware that dizziness and headaches are potential side effects. Nitric oxide has shown efficacy in one clinical trial of Achilles tendinopathy, but did not demonstrate efficacy in a second trial.

Sclerosing therapy is a relatively new technique in which a vessel-ablating substance (polidocanol) is injected under colour Doppler ultrasound to the regions with neovascularisation. Rather than injecting into the tendon itself, the injections are targeted to the point where vessels (and nerves) enter the tendon. This results, in some cases, in a rapid reduction of pain and return to play. By contrast, prolotherapy involves the intratendinous injection of glucose or platelet-rich plasma (PRP). The goal is to stimulate a repair response in the tendon. Both sclerosing therapy and prolotherapy should be considered adjuncts to exercise-based rehabilitation (see below). There is currently insufficient evidence to recommend the use of prolotherapy.

Operative treatment should be reserved for cases that do not resolve with appropriate rehabilitation (see below). A variety of operative treatments have been advocated. Traditional open procedures have a success rate of approximately 50%. Several techniques have been used, including opening of the paratendon and stripping of any adhesions. Any macroscopically degenerated tendon is debrided. Sometimes, longitudinal incisions are made in the tendon, with the aim of promoting a repair response.

Recently, minimally invasive surgical procedures have been developed. For the patellar tendon, an arthroscopic

procedure may be performed by debriding the area of neovascularisation on the posterior surface of the tendon until flow in the neovessels is no longer seen. For midportion Achilles tendinopathy, a similar approach involves stripping or debriding the neurovascular supply of the anterior surface of the tendon. The advantage of minimally invasive procedures is that the tendon itself is not disturbed, so lengthy rehabilitation may be avoided. Only the nerve and blood supply to the tendon built in the process of the disease is interfered with.

3.5.9 Rehabilitation programme

Conservative rehabilitation is the cornerstone of treatment for tendinopathy. It should focus on progressive, heavy loading of the tendon. The goal is to simulate tissue remodelling by introducing increasingly heavy loads in a slow, controlled manner.

Initial studies demonstrated that eccentric loading is superior to concentric loading. More recent studies that incorporate both concentric and eccentric loading have reported similar responses. Successful rehabilitation regimes appear to share the key features of heavy loads applied in a slow, controlled fashion. This loading promotes tissue remodelling, including collagen synthesis and alignment, and the reduction of neovascularisation.

For midsubstance Achilles tendinopathy, loading may be accomplished by performing slow heel drops (15 seconds duration) over a step, double legged progressing to single legged (Figure 3.5.9.1). For insertional Achilles tendinopathy, the same programme is followed, but the heel drop is only performed into neutral position (i.e. not into full dorsiflexion). Prescriptions should be tailored, but three sets of 15 heel drops twice per day is an evidence-based guideline. When this exercise becomes easy and pain free, weight may be gradually added in a backpack. Pain is acceptable during the exercise to the players's tolerance, as long as pain or weakness does not persist after the exercise. Pain or weakness is usually a result of performing the movement too quickly. This programme often leads to a reduction of tendon pain and a successful return to play.

For patellar tendinopathy, eccentric loading is performed by squatting on a decline board (Figure 3.5.9.2). The use of this board maximises the load on the patellar tendon. The patient squats to 80-90 degrees of knee flexion

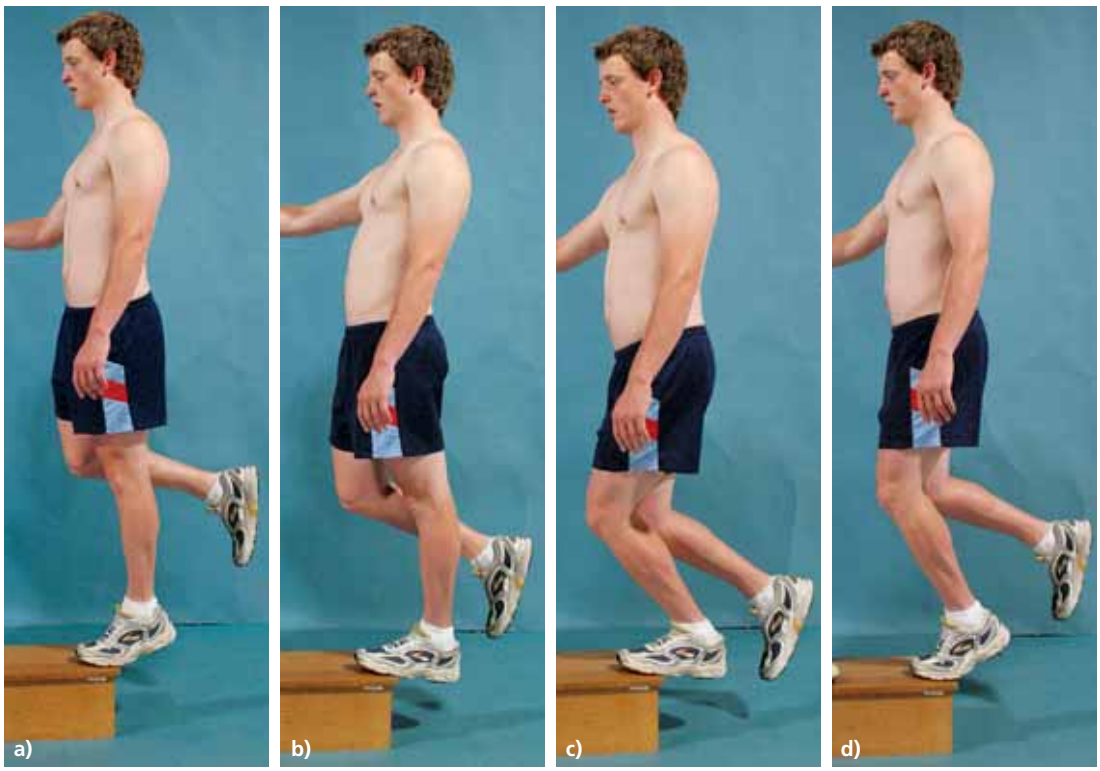


Figure 3.5.9.1 Exercise training for Achilles tendinopathy involves two key exercises – calf drops with straight knee (a, b) to maximise activity of gastrocnemius, and with bent knee (c, d) to maximise activity of soleus. Please see text for description.

with the trunk upright or leaning slightly forward, double legged progressing to single legged. The same progression of repetitions and loading is followed as for Achilles tendinopathy. Eccentric exercises are best performed during the off-season to minimise the volume of loading during the competitive season.

3.5.10 Prognosis and return to play

Tendon remodelling takes place over months, making the rehabilitation a long process requiring patience. The majority of tendinopathies resolve with conservative treatment involving eccentric exercise. For tendons that fail to recover after twelve months of conservative treatment, a surgical referral may be indicated. If surgery is unsuccessful, then tendinopathy can become a career-ending injury, so it deserves as careful management as the more spectacular football injuries. The decision to return to play is based on the player's pain and imaging profile, functional status, and the response to rehabilitation and training. The volume of high-velocity activities like jumping or sprinting should be reduced during the early rehabilitation phase. Some pain during rehabilitation is to be expected, but pain that persists beyond 24 hours, or other signs of exacerbation such as increased swelling or stiffness indicate that some aspect of the load was excessive. A gradual return to play should be coordinated and monitored by the physiotherapist, physician, trainer and manager.

Figure 3.5.9.2 Eccentric training for patellar tendinopathy is performed using a decline board.

3.5.11 Sequelae and their treatment

Tendinopathies frequently involve abnormalities elsewhere in the kinetic chain. Muscle tone around the hip, lower back, thigh or calf may be locally increased – these areas should be identified by palpation and treated with heat, massage and stretching. Muscle imbalance may also develop, so a lower-extremity biomechanical assessment should be performed to identify areas in need of lengthening or strengthening.

Poor core stability can lead to excessive movement of the pelvis, leading to abnormal strains in weight-bearing tendons. Thus, core stability should be assessed and a programme considered, if necessary.

Finally, tendon pain results in altered central nervous system processing, which can lead to increased blood flow and hyperalgesia on the contralateral tendon. Practitioners should be aware that tendon pain is not a direct indicator of tissue injury, but is ultimately an indicator of altered activity in the nervous system.

3.6 Head and brain injuries

Concerns have been raised about potential brain damage due to repeated ball heading as well as head injuries that occur in football. Fortunately, brain injuries that result in structural damage are extremely rare and usually occur only after severe ground-to-head, head-to-head or elbow-to-head collisions in the facial or parietal region (Figure 3.6.1). Most head injuries are minor (e.g. brain concussion), but more severe injuries can occasionally occur (e.g. subdural haematoma or intracerebral haemorrhage).

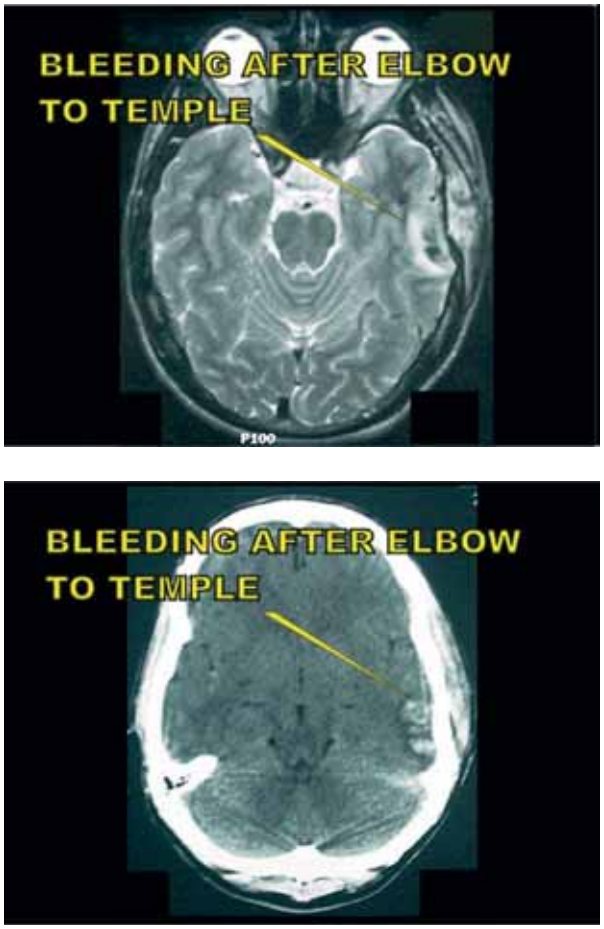


Figure 3.6.1 Intracerebral bleeding after direct elbow hit to the temperal reagion left in a young football player (courtesy of B. Mandelbaum, MD)

3.6.1 Incidence

There is little prospective scientific literature available concerning head injuries in football and F-MARC has relied mainly on descriptive surveys conducted at FIFA competitions, starting with the 1998 FIFA World Cup™ and including events for both male and female players as well as different age groups. A total of 13,992 playing hours in 424 matches from 14 competitions were taken into account. Head and neck injuries (165 in total) during matches accounted for approximately 14% of all injuries seen, but only as few as 23 concussions (2% of all injuries; 13.9% of all head and neck injuries; 1.6 per 1,000 playing hours) were noted. Concussions were due to contact with another player and not because of heading the ball. Comparing the head injury incidence from the FIFA competitions with previous F-MARC studies on amateur football players, the frequency of head injuries increases as the level of competition increases, with head injuries occurring up to four times more often in high-profile competitions (see Table 3.6.1.1).

	Tournament	Amateurs & professionals	Amateurs
Study group	424 matches	398 players	194 players
Skill-level	High	High-low	High-low
Age	>=U17	14-41 years	14-18 years
Injuries per 1,000 hours total	82	20	20
Concussion per 1,000 match hours	1.6	0.3	0.4
No. of concussions (with subs. absence)	23 (63%)	4 (0%)	2 (50%)

Table 3.6.1.1 Incidence of head injuries

Prospective video analysis of head injuries in the elite Norwegian *Tippeligaen* demonstrated an overall incidence of 1.7 per 1,000 player hours, with a concussion incidence of 0.5 per 1,000 player hours.

3.6.2 Causes of acute head injury – injury mechanism and risk factors

An assessment of tackle mechanisms has identified a deliberate or accidental clash of heads between players as having the highest probability of leading to an injury to either or both players compared with all other tackle mechanisms. The frequency of this type of tackle is relatively low compared with other tackle mechanisms during competition.

A clash of heads frequently occurred when players jumped to challenge for the ball in the penalty area during crosses or corners and in the centre of the pitch following clearances by goalkeepers or defenders. In the penalty area, a clash of heads was more likely to involve face-to-face contact whilst a midfield clash of heads was more likely to involve face-to-back-of-head contact. The second most common cause of head/neck injuries involved the use of an arm or hand by one player impacting another player's head.

In the Norwegian studies on head injury, the most common playing action accounting for injury was a heading duel (60% of injuries), with 41% of cases due to head contact with the elbow or hand and 32% due to head-to-head contact. The findings of this and similar studies led to recommendations from the FIFA Medical Committee and F-MARC to the International Football Association Board (IFAB) to ban deliberate elbow-to-head contact during heading duels. This rule change was instituted prior to the 2006 FIFA World Cup™ in Germany and resulted in a reduction in the incidence of concussion as compared to the previous FIFA World Cup™.

3.6.3 The potential for chronic traumatic brain injury

Epidemiological studies from sports other than football have suggested an association between repeated sports concussions during a career and later-life cognitive impairment. Similarly, case reports have noted anecdotal cases where neuropathological evidence of chronic traumatic encephalopathy was observed in retired American football players. At this stage, there is no convincing evidence that such anecdotal observations are a consequence of either repeated concussion or sports participation. Physicians need to be mindful, however, of the potential for long-term problems in the management of all players.

The precise threshold at which brain injury occurs is not known, but in moderate to severe traumatic brain injury (TBI), marked neuropathological change occurs. Mild TBI, particularly the subset of concussive injury, is not associated with structural brain injury. It has been proposed that the effects of repeated mild brain injury may be cumulative, but severe methodological flaws make this literature inconclusive.

In addition, biochemical serum and cerebral spinal fluid markers of brain injury (including S-100B, neuron-specific enolase (NSE), myelin basic protein (MBP), GFAP, tau, etc.) have been proposed as means by which cellular damage may be detected if present. S-100B is found in high concentrations in astroglial and Schwann cells – the support cells of the central nervous system. If these cells are damaged, S-100B is released and leaks into the cerebrospinal fluid and across the blood-brain-barrier into the circulation. Whether high local extracellular concentrations of S-100B have detrimental effects such as the enhancement of apoptotic cell death is still under debate. Where radiological lesions are demonstrated on CT scanning, increased serum levels of the neuroprotein S-100B seem to be a reliable indicator of brain injury. However, in concussion and in controlled studies of football heading, levels of S-100B or other biomarkers do not reach levels seen in TBI.

3.6.4 Types of brain injury

In sports medicine, physicians recognise and manage a spectrum of traumatic brain injury, ranging from concussion through to structural brain injury. All persons involved in player care need to have a thorough understanding of first aid principles, particularly the early management of a concussed player as well as knowledge of the potential sequelae of the injury.

There are numerous ways of classifying traumatic head injury and numerous books and reviews have been published in this regard. Readers are directed to the IOC Sports Medicine Manual (2000) that summarises the information on severe sports-related brain injury and its management. A simple classification for the purposes of common injuries seen in football is as follows:

1. Structural (e.g extra-dural haematoma)
2. Non-structural (e.g. concussion)

Concussion

The most common form of sports-related brain injury is concussion. Although this condition is a subset of mild traumatic brain injury, the terms should not be used interchangeably as they refer to different injury constructs.

Over the past decade, the Concussion in Sports Group (CISG) has held a series of international meetings and consensus conferences to help define this injury and determine best management practice. The section below is based on the current CISG consensus recommendations (McCrory et al 2009).

Definition of concussion

The CISG has developed the following definition of concussion:

"Concussion" is defined as a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces. Several common features that incorporate clinical, pathological and biomechanical injury constructs that may be used in defining the nature of a concussive head injury are set out below:

- Concussion may be caused by a direct blow to the head, face, neck, or elsewhere on the body with an "impulsive" force transmitted to the head.
- Concussion typically results in the rapid onset of short-lived impairment of neurological function that resolves spontaneously.
- Concussion may result in neuropathological changes but the acute clinical symptoms largely reflect a functional disturbance rather than structural injury.
- Concussion results in a graded set of clinical syndromes that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course.
- No abnormality on standard structural neuroimaging studies is seen in concussion.

3.6.5 Concussion classification and grading

The CISG recognised the strengths and weaknesses of the numerous published concussion grading scales that attempt to characterise injury severity, but no single system was endorsed. It was the recommendation of the CISG that combined measures of recovery should be used to assess injury severity (and/or prognosis) that individually guide decisions on return to play.

In the absence of scientifically validated return to play guidelines, a clinical construct is recommended using an assessment of injury recovery and graded return to play. The protocol outlined below is adapted from the 2000 guidelines of the Canadian Academy of Sport Medicine (CASM). Sideline evaluation includes clinical evaluation of signs and symptoms, ideally using a standardised scale of post-concussion symptoms for comparison purposes and acute injury testing as described below under neuropsychological testing.

3.6.6 Symptoms and signs of acute concussion

The diagnosis of acute concussion usually involves the assessment of a range of domains including clinical symptoms, physical signs, behaviour, balance, sleep and cognition. Furthermore, a detailed concussion history is an important part of the evaluation both in the injured player and when conducting a pre-participation examination. An example of a detailed clinical assessment of concussion is outlined in the SCAT2 forms (Figures 3.6.11.1–3.6.11.4, page 202–205).

The suspected diagnosis of concussion can include one or more of the following clinical domains:

- Symptoms – somatic (e.g. headache), cognitive (e.g. feeling like in a fog) and/or emotional (e.g. lability)
- Physical signs (e.g. loss of consciousness, amnesia)
- Behavioural changes (e.g. irritablity)
- Cognitive impairment (e.g. slowed reaction times)
- Sleep disturbance (e.g. drowsiness)

If any one or more of these components is present, concussion should be suspected and the appropriate management strategy instituted.

On-field or sideline evaluation of acute concussion

When a player shows ANY features of a concussion:

- The player should be medically evaluated on-site using standard emergency management principles and particular attention should be given to excluding a cervical spine injury.
- The appropriate disposition of the player must be determined by the treating healthcare provider in a timely manner. If no healthcare provider is available, the player should be safely removed from practice or play and urgent referral to a physician arranged.
- Once the first aid issues are addressed, an assessment of the concussive injury should be made using the SCAT2 or another similar tool.
- The player should not be left alone following the injury and serial monitoring for deterioration is essential over the initial few hours following injury.

Sufficient time for assessment and adequate facilities should be provided for appropriate medical assessment both on and off the field for all injured players. In football, this requires an off-field medical assessment without affecting the flow of the game or unduly penalising the injured player's team.

Sideline evaluation of cognitive function is an essential component in the assessment of this injury. Brief neuropsychological test batteries that assess attention and memory function have been shown to be practical and effective. Such tests include the Maddocks questions and the Standardized Assessment of Concussion (SAC). It is worth noting that standard orientation questions (e.g. time, place, person) have been shown to be unreliable in the sporting situation when compared with memory assessment. It is important to note that abbreviated testing paradigms are designed for rapid concussion screening on the sidelines and are not meant to replace comprehensive neuropsychological testing, which is sensitive to detect subtle deficits that may exist beyond the acute episode; nor should they be used as a stand-alone tool for the ongoing management of football concussions.

It should also be recognised that the appearance of symptoms might be delayed several hours following a concussive episode.

Evaluation in emergency room or office by medical personnel

A player with concussion may be evaluated in the emergency room or doctor's office as a point of first contact following injury or may have been referred from another care provider. In addition to the points outlined above, the key features of this exam should encompass:

- A medical assessment including a comprehensive history and a detailed neurological examination including a thorough assessment of mental status, cognitive functioning and gait and balance.
- A determination of the clinical status of the player including whether there has been improvement or deterioration since the time of injury. This may involve seeking additional information from parents, coaches, team-mates and eyewitnesses to the injury.
- A determination of the need for emergency neuroimaging in order to exclude a more severe brain injury involving a structural abnormality.

Most of, the above points are included in the SCAT2 assessment.

Concussion investigations

A range of additional investigations may be utilised to assist in the diagnosis and/or exclusion of injury. These include:

Neuroimaging

Conventional structural neuroimaging is typically normal in concussive injury. Given that caveat, brain CT scanning (or where available MR brain scanning) contributes little to concussion evaluation but should be employed whenever suspicion of an intra-cerebral structural lesion exists. Examples of such situations may include prolonged disturbance of conscious state, focal neurological deficit or worsening symptoms.

Newer structural MRI modalities including gradient echo, perfusion and diffusion imaging have greater sensitivity for structural abnormalities. However, the lack of published studies as well as absent pre-injury neuroimaging data at the present time limits the usefulness of this approach in clinical management. In addition, the predictive value of various MR abnormalities that may be incidentally discovered has not yet been established.

Objective balance assessment

Published studies, using both sophisticated force plate technology and less sophisticated clinical balance tests (e.g. Balance Error Scoring System (BESS)), have identified postural stability deficits lasting approximately 72 hours following sports-related concussion. It appears that postural stability testing provides a useful tool for objectively assessing motor functioning, and should be considered a reliable and valid addition to the assessment of players suffering from concussion, particularly where symptoms or signs indicate a balance component.

Neuropsychological assessment

The application of neuropsychological (NP) testing in concussion has been shown to be of clinical value and continues to contribute significant information in concussion evaluation. Although in most cases cognitive recovery largely overlaps with the time course of symptom recovery, it has been demonstrated that cognitive recovery may occasionally precede or more commonly follow clinical symptom resolution, suggesting that the assessment of cognitive function should be an important component in any return-to-play protocol. It must be emphasised, however, that NP assessment should not be the sole basis of management decisions, rather it should be seen as an aid to the clinical decision-making process in conjunction with a range of clinical domains and investigational results.

Neuropsychologists are in the best position to interpret NP tests by virtue of their background and training. However, there may be situations where neuropsychologists are not available and other medical professionals may perform or interpret NP screening tests. The ultimate return-to-play decision should remain a medical one in which a multidisciplinary approach, when possible, has been taken. In the absence of NP and other (e.g. formal balance assessment) testing, a more conservative return-to-play approach may be appropriate.

In the majority of cases, NP testing will be used to assist return-to-play decisions and will not be done until the patient is symptom free. There may be situations (e.g. child and adolescent players) where testing may be performed early whilst the player is still symptomatic to assist in determining management. This will normally be best determined in consultation with a trained neuropsychologist.

3.6.7 Concussion management

The cornerstone of concussion management is physical and cognitive rest until symptoms resolve and then a graded programme of exertion prior to medical clearance and return to play. The recovery and outcome of this injury may be modified by a number of factors that may require more sophisticated management strategies.

As described above, the majority of injuries will recover spontaneously over several days. In these situations, it is expected that a player will proceed progressively through a gradual return-to-play strategy. During this period of recovery following an injury, it is important to emphasise to the athlete that physical AND cognitive rest is required. Activities that require concentration and attention (e.g. scholastic work, videogames, text messaging, etc.) may exacerbate symptoms and possibly delay recovery. In such cases, apart from limiting relevant physical and cognitive activities (and other risk-taking opportunities for re-injury) while symptomatic, no further intervention is required during the period of recovery and the player typically resumes the game without further problem.

Graduated return-to-play protocol

Return-to-play (RTP) protocol after a concussion follows a gradual process as outlined in Table 3.6.7.1.

With this gradual progression, the player should continue to proceed to the next level if asymptomatic at the current level. Generally, each step should take 24 hours so that a player would take approximately one week to proceed through the full rehabilitation protocol once they are asymptomatic at rest and with provocative exercise. If any post-concussion symptoms occur while in the gradual programme, the player should drop back to the previous asymptomatic level and try to progress again after a further 24-hour period of rest has passed.

Psychological management and mental health issues

In addition, psychological approaches may have potential application in this injury, particularly with the modifiers listed below. Care givers are also encouraged to evaluate the concussed player for affective symptoms such as depression, as these symptoms may be common in concussed players.

3.6.8 The role of pharmacological therapy

Pharmacological therapy in sports concussion may be applied in two distinct situations. The first of these situations is the management of specific prolonged symptoms (e.g. sleep disturbance, anxiety, etc.). The second situation is where drug therapy is used to modify the underlying pathophysiology of the condition with the aim of shortening the duration of the concussion symptoms. In broad terms, this approach to management should only be considered by clinicians experienced in concussion management.

An important consideration in RTP is that concussed players should not only be symptom-free but also should not be taking any pharmacological agents/medications that may mask or modify the symptoms of concussion. Where antidepressant therapy may be commenced during the management of a concussion, the decision to return to play while still on such medication must be considered carefully by the treating physician.

The role of pre-participation concussion evaluation

Recognising the importance of a concussion history, and appreciating the fact that many athletes will not recognise all the concussions they may have suffered in the past, a detailed concussion history is of value. Such a history may pre-identify players that fit into a high-risk category and provides an opportunity for the healthcare provider to educate the player about the significance of concussive injury. A structured concussion history should include specific questions as to previous symptoms of a concussion – not just the perceived number of past concussions. It is also worth noting that dependence upon the recall of concussive injuries by team-mates or coaches has been demonstrated to be unreliable. The clinical history should also include information about all previous head,

Rehabilitation stage	Functional exercise at each stage of rehabilitation	Objective of each stage
1. No activity	Complete physical and cognitive rest	Recovery
2. Light aerobic exercise	Walking, swimming or stationary cycling keeping intensity < 70% maximum heart rate (MHR). No resistance training.	Increase heart rate
3. Sport-specific exercise	Skating drills in ice hockey, running drills in football. No head impact activities.	Add movement
4. Non-contact training drills	Progression to more complex training drills, e.g. passing drills in football and ice hockey. May start progressive resistance training.	Exercise, coordination and cognitive load
5. Full-contact practice	Following medical clearance participate in normal training activities.	Restore confidence and assess functional skills by coaching staff
6. Return to play	Normal game play	

Table 3.6.7.1 Graduated return to play protocol

face or cervical spine injuries as these may also have clinical relevance. It is worth emphasising that in the context of maxillofacial and cervical spine injuries, co-existent concussive injuries may be missed unless specifically assessed. Questions pertaining to disproportionate impact versus symptom severity matching may alert the physician to a progressively increasing vulnerability to injury. As part of the clinical history it is advised that details regarding protective equipment employed at the time of injury be sought, both for recent and remote injuries. A comprehensive pre-participation concussion evaluation allows for modification and optimisation of protective behaviour and provides an opportunity for education.

Modifying factors in concussion management

A range of "modifying" factors may influence the investigation and management of concussion and, in some cases, may predict the potential for prolonged or persistent symptoms. These modifiers would also be important to consider in a detailed concussion history and are outlined in Table 3.6.8.1.

In this context, there may be additional management considerations beyond simple RTP advice. There may be a more important role for additional investigations, including formal NP testing, balance assessment and neuroimaging. It is envisioned that players with such modifying features would be managed in a multidisciplinary manner coordinated by a physician with specific expertise in the management of concussive injury.

The role of female gender as a possible modifier in the management of concussion is not yet clear, but gender may be a risk factor for injury and/or influence injury severity.

The significance of loss of consciousness (LOC)

In the overall management of moderate to severe TBI, duration of LOC is an acknowledged predictor of outcome. Whilst published findings on concussion describe LOC

Factors	Modifier
Symptoms	Number
	Duration (> 10 days)
	Severity
Signs	Prolonged loss of consciousness (> 1 min.), amnesia
Sequelae	Concussive convulsions
Temporal	Frequency – repeated concussions over time
	Timing – injuries close together in time
	"Recency" – recent concussion or traumatic brain injury
Threshold	Repeated concussions occurring with progressively less impact force or slower recovery after each successive concussion
Age	Child and adolescent (< 18 years old)
Co- and pre-morbidities	Migraine, depression or other mental health disorders, attention deficit hyperactivity disorder (ADHD), learning disabilities (LD), sleep disorders
Medication	Psychoactive drugs, anticoagulants
Behaviour	Dangerous style of play
Sport	High-risk activity, contact and collision sport, high sporting level

Table 3.6.8.1 Concussion modifiers

associated with specific early cognitive deficits, it has not been noted as a measure of injury severity. Prolonged LOC (duration > 1 minute) would be considered a factor that may modify management.

The significance of amnesia and other symptoms

Published evidence suggests that the nature, burden and duration of the clinical post-concussive symptoms may be more important than the presence or duration of amnesia alone. Further, it must be noted that retrograde amnesia varies with the time of measurement post-injury and hence is poorly reflective of injury severity.

Motor and convulsive phenomena

A variety of immediate motor phenomena (e.g. tonic posturing) or convulsive movements may accompany a concussion. Although dramatic, these clinical features are generally benign and require no specific management beyond the standard treatment of the underlying concussive injury.

Depression

Mental health issues (such as depression) have been reported as a long-term consequence of TBI, including sports-related concussion. Neuroimaging studies using MRI suggest that a depressed mood following concussion may reflect an underlying pathophysiological abnormality consistent with a limbic-frontal model of depression.

3.6.9 Special populations

The child and adolescent player

The CISG agreed that the adult recommendations could be applied to children and adolescents down to the age of ten. Below that age, children report concussion symptoms different from adults and would require age-appropriate symptom checklists as a component of assessment. An additional consideration in assessing the child or adolescent player with a concussion is that in the clinical evaluation by the healthcare professional there may be the need to include both patient and parent input as well as teacher and school input when appropriate.

The decision to use NP testing is broadly the same as the adult assessment paradigm. However, timing of testing may differ in order to assist planning in school and home management (and may be performed while the player is still symptomatic). If cognitive testing is performed, it must be developmentally sensitive until late teen years due to the ongoing cognitive maturation that occurs during this period which, in turn, makes the utility of comparison to either the person's own baseline performance or to population norms limited. In this age group, it is more important to consider the use of trained neuropsychologists to interpret assessment data, particularly in children with learning disorders and/or ADHD who may need more sophisticated assessment strategies.

The CISG strongly endorsed the view that children should not be returned to practice or play until clinically completely symptom-free, which may require a longer time frame than for adults. In addition, the concept of "cognitive rest" was highlighted with special reference to a child's need to limit exertion with activities of daily living and to limit scholastic and other cognitive stressors (e.g text messaging, videogames, etc.) while symptomatic. School attendance and activities may also need to be modified to avoid provocation of symptoms.

Because of the different physiological response and longer recovery after concussion and specific risks (e.g. diffuse cerebral swelling) related to head impact during childhood and adolescence, a more conservative RTP approach is recommended. It is appropriate to extend the amount of time of asymptomatic rest and/or the length of the graded exertion in children and adolescents. It is not appropriate for a child or adolescent athlete with concussion to return to play on the same day as the injury, regardless of the level of athletic performance. Concussion modifiers apply even more to this population than to adults and may necessitate more cautious RTP advice.

Elite versus non-elite players

The CISG unanimously agreed that all players, regardless of level of participation, should be managed using the same treatment and RTP paradigm. A more useful construct was agreed whereby the available resources and expertise in concussion evaluation were of more importance in determining management than a separation between elite and non-elite player management. It is recommended that in all organised high-risk sports consideration be given to having this cognitive evaluation regardless of the age or level of performance.

3.6.10 Concussion prevention

Protective equipment – mouthguards and helmets

There is no good clinical evidence that currently available protective equipment will prevent concussion, although mouthguards have a definite role in preventing dental and oro-facial injury. Biomechanical studies have shown a reduction in impact forces to the brain with the use of headgear and helmets, but these findings have not been translated to show a reduction in the incidence of concussion. For skiing and snowboarding there are a number of studies to suggest that helmets provide protection against head and facial injury and hence should be recommended for participants in alpine sports. In specific sports such as cycling, motor and equestrian sports, protective helmets may prevent other forms of head injury (e.g. skull fracture) that are related to falling on hard road surfaces and these may be an important injury prevention issue for those sports.

Rule change

Consideration of rule changes to reduce the incidence or severity of head injuries may be appropriate where a clear-cut mechanism is implicated in a particular sport. In football, F-MARC research studies have demonstrated that upper limb to head contact in heading contests accounted for approximately 50% of concussions. It is important to note that rule enforcement may be a critical aspect of modifying injury risk in these settings and referees play an important role in this regard.

Risk compensation

An important consideration in the use of protective equipment is the concept of risk compensation. This is where the use of protective equipment results in behavioural change such as the adoption of more dangerous playing techniques, which can result in a paradoxical increase in injury rates. This may be a particular concern in child and adolescent players where head injury rates are often higher than in adult players.

Aggression versus violence in football

The competitive/aggressive nature of football-which makes it fun to play and watch-should not be discouraged. However, addressing violence that may increase concussion risk is important. Fair play and respect should be supported as key elements of the game.

Knowledge transfer

As the ability to treat or reduce the effects of concussive injury after the event is minimal, education of players, colleagues and the general public is essential for progress in this field. Players, referees, administrators, parents, coaches and healthcare providers must be educated regarding the detection of concussion, its clinical features, assessment techniques and principles of safe return to play. Methods to improve education, including web-based resources, educational videos and international outreach programmes, are important in delivering the message. Fair play and respect for opponents are ethical values that should be encouraged in football. Similarly, coaches, parents and managers play an important part in ensuring these values are implemented on the field of play.

3.6.11 Sport Concussion Assessment Tool

The Sport Concussion Assessment Tool (SCAT) was developed as part of the Summary and Agreement Statement of the second conference on concussion in sports in Prague in 2004. This tool represents a standardised method of evaluating people after concussion in sport. This tool was renamed SCAT2 following the Zurich consensus meeting in 2008 and has two forms – a sideline assessment form and a more detailed medical evaluation form.

McCrory P, Meeuwisse W, Johnston K M, Dvorak J, Aubry M, Molloy M, and Cantu R
Consensus Statement on Concussion in Sport: the 3rd International - Conference on Concussion in Sport held in Zurich, November 2008. Br J Sports Med 2009;43 i76-i84

SCAT2

Sport Concussion Assessment Tool 2

Name

Sport/team

Date/time of injury

Date/time of assessment

Age Gender n M n F

Years of education completed

Examiner

What is the SCAT2?[1]

This tool represents a standardised method of evaluating injured athletes for concussion and can be used in athletes aged 10 years and older. It supersedes the original SCAT published in 2005[2]. This tool also enables the calculation of the Standardized Assessment of Concussion (SAC)[3,4] score and the Maddocks questions[5] for sideline concussion assessment.

Instructions for using the SCAT2

The SCAT2 is designed for the use of medical and health professionals. Pre-season baseline testing with the SCAT2 can be helpful for interpreting post-injury test scores. Words in italics throughout the SCAT2 are the instructions given to the athlete by the tester.

This tool may be freely copied for distribtion to individuals, teams, groups and organisations.

What is a concussion?

A concussion is a disturbance in brain function caused by a direct or indirect force to the head. It results in a variety of non-specific symptoms (like those listed below) and often does not involve loss of consciousness. Concussion should be suspected in the presence of **any one or more** of the following:

- Symptoms (such as headache), or
- Physical signs (such as unsteadiness), or
- Impaired brain function (e.g. confusion) or
- Abnormal behaviour.

Any athlete with a suspected concussion should be REMOVED FROM PLAY, medically assessed, monitored for deterioration (i.e., should not be left alone) and should not drive a motor vehicle.

Symptom evaluation

How do you feel?

You should score yourself on the following symptoms, based on how you feel now.

	none	mild		moderate		severe	
Headache	0	1	2	3	4	5	6
"Pressure in head"	0	1	2	3	4	5	6
Neck pain	0	1	2	3	4	5	6
Nausea or vomiting	0	1	2	3	4	5	6
Dizziness	0	1	2	3	4	5	6
Blurred vision	0	1	2	3	4	5	6
Balance problems	0	1	2	3	4	5	6
Sensitivity to light	0	1	2	3	4	5	6
Sensitivity to noise	0	1	2	3	4	5	6
Feeling slowed down	0	1	2	3	4	5	6
Feeling like "in a fog"	0	1	2	3	4	5	6
"Don't feel right"	0	1	2	3	4	5	6
Difficulty concentrating	0	1	2	3	4	5	6
Difficulty remembering	0	1	2	3	4	5	6
Fatigue or low energy	0	1	2	3	4	5	6
Confusion	0	1	2	3	4	5	6
Drowsiness	0	1	2	3	4	5	6
Trouble falling asleep (if applicable)	0	1	2	3	4	5	6
More emotional	0	1	2	3	4	5	6
Irritability	0	1	2	3	4	5	6
Sadness	0	1	2	3	4	5	6
Nervous or anxious	0	1	2	3	4	5	6

Total number of symptoms (maximum possible 22)

Symptom severity score
(add all scores in table, maximum possible: 22 x 6 = 132)

Do the symptoms get worse with physical activity? Y N
Do the symptoms get worse with mental activity? Y N

Overall rating

If you know the athlete well prior to the injury, how different is the athlete acting compared to his/her usual self? Please circle one response.

no different	very different	unsure

Figure 3.6.11.1 SCAT2 form

Cognitive & physical evaluation

1 Symptom score (from page 1)

22 **minus** number of symptoms ____ of 22

2 Physical signs score

Was there loss of consciousness or unresponsiveness? ☐ Y ☐ N
If yes, how long? ____ minutes
Was there a balance problem/unsteadiness? ☐ Y ☐ N

Physical signs score (1 point for each negative response) ____ of 2

3 Glasgow Coma Scale (GCS)

Best eye response (E)

No eye opening	1
Eye opening in response to pain	2
Eye opening to speech	3
Eyes opening spontaneously	4

Best verbal response (V)

No verbal response	1
Incomprehensible sounds	2
Inappropriate words	3
Confused	4
Oriented	5

Best motor response (M)

No motor response	1
Extension to pain	2
Abnormal flexion to pain	3
Flexion/ withdrawal to pain	4
Localises to pain	5
Obeys commands	6

Glasgow Coma score (E + V + M) ____ of 15

GCS should be recorded for all athletes in case of subsequent deterioration.

4 Sideline assessment – maddocks score

"I am going to ask you a few questions, please listen carefully and give your best effort."

Modified Maddocks questions (1 point for each correct answer)

What venue are we at today?	0	1
Which half is it now?	0	1
Who scored last in this match?	0	1
What team did you play last week/game?	0	1
Did your team win the last game?	0	1

Maddocks score ____ of 5

Maddocks score is validated for sideline diagnosis of concussion only and is not included in SCAT 2 summary score for serial testing.

5 Cognitive assessment

Standardised Assessment of Concussion (SAC)

Orientation (1 point for each correct answer)

What month is it?	0	1
What is the date today?	0	1
What is the day of the week?	0	1
What year is it?	0	1
What time is it right now? (within 1 hour)	0	1

Orientation score ____ of 5

Immediate memory

"I am going to test your memory. I will read you a list of words and when I am done, repeat back as many words as you can remember, in any order."

Trials 2 & 3:

"I am going to repeat the same list again. Repeat back as many words as you can remember in any order, even if you said the word before."

Complete all three trials regardless of score on trial 1 & 2. Read the words at a rate of one per second. Score 1 pt for each correct response. Total score equals sum across all three trials. Do not inform the athlete that delayed recall will be tested.

List	Trial 1	Trial 2	Trial 3	Alternative word list		
elbow	0 1	0 1	0 1	candle	baby	finger
apple	0 1	0 1	0 1	paper	monkey	penny
carpet	0 1	0 1	0 1	sugar	perfume	blanket
saddle	0 1	0 1	0 1	sandwich	sunset	lemon
bubble	0 1	0 1	0 1	wagon	iron	insect
Total						

Immediate memory score ____ of 15

Concentration

Digits backward:

"I am going to read you a string of numbers and when I am done, you repeat them back to me backwards, in reverse order of how I read them to you. For example, if I say 7-1-9, you would say 9-1-7."

If correct, go to next string length. If incorrect, read trial 2. One point possible for each string length. Stop after incorrect on both trials. The digits should be read at the rate of one per second.

		Alternative digit lists		
4-9-3	0 1	6-2-9	5-2-6	4-1-5
3-8-1-4	0 1	3-2-7-9	1-7-9-5	4-9-6-8
6-2-9-7-1	0 1	1-5-2-8-6	3-8-5-2-7	6-1-8-4-3
7-1-8-4-6-2	0 1	5-3-9-1-4-8	8-3-1-9-6-4	7-2-4-8-5-6

Months in reverse order:

"Now tell me the months of the year in reverse order. Start with the last month and go backward. So you'll say December, November ... Go ahead."

1 pt for entire sequence correct

Dec-Nov-Oct-Sept-Aug-Jul-Jun-May-Apr-Mar-Feb-Jan 0 1

Concentration score ____ of 5

[1] This tool has been developed by a group of international experts at the 3rd International Consensus meeting on Concussion in Sport held in Zurich, Switzerland in November 2008. The full details of the conference outcomes and the authors of the tool are published in the British Journal of Sports Medicine, 2009, volume 43, supplement 1.
The outcome paper will also be simultaneously co-published in the May 2009 issues of the Clinical Journal of Sports Medicine, Physical Medicine & Rehabilitation, Journal of Athletic Training, Journal of Clinical Neuroscience, Journal of Science & Medicine in Sport, Neurosurgery, Scandinavian Journal of Science & Medicine in Sport and the Journal of Clinical Sports Medicine.

[2] McCrory P et al. Summary and agreement statement of the 2nd International Conference on Concussion in Sport, Prague 2004. British Journal of Sports Medicine. 2005; 39: 196-204

[3] McCrea M. Standardized mental status testing of acute concussion. Clinical Journal of Sports Medicine. 2001; 11: 176-181

[4] McCrea M, Randolph C, Kelly J. Standardized Assessment of Concussion: Manual for administration, scoring and interpretation. Waukesha, Wisconsin, USA.

[5] Maddocks, DL; Dicker, GD; Saling, MM. The assessment of orientation following concussion in athletes. Clin J Sport Med. 1995;5(1):32–3

[6] Guskiewicz KM. Assessment of postural stability following sport-related concussion. Current Sports Medicine Reports. 2003; 2: 24-30

SCAT2 SPORT CONCUSSION ASSESSMENT TOOL 2 | **PAGE 2**

Figure 3.6.11.2 SCAT2 form

6 Balance examination

This balance testing is based on a modified version of the Balance Error Scoring System (BESS)[6]. A stopwatch or watch with a second hand is required for this testing.

Balance testing

"I am now going to test your balance. Please take your shoes off, roll up your pant legs above ankle (if applicable), and remove any ankle taping (if applicable). This test will consist of three twenty second tests with different stances."

(a) Double leg stance:
"The first stance is standing with your feet together, your hands on your hips and your eyes closed. You should try to maintain stability in that position for 20 seconds. I will be counting the number of times you move out of this position. I will start timing when you are set and have closed your eyes."

(b) Single leg stance:
"If you were to kick a ball, which foot would you use? [This will be the dominant foot.] *Now stand on your non-dominant foot. The dominant leg should be held in approximately 30 degrees of hip flexion and 45 degrees of knee flexion. Again, you should try to maintain stability for 20 seconds with your hands on your hips and your eyes closed. I will be counting the number of times you move out of this position. If you stumble out of this position, open your eyes, return to the start position and continue balancing. I will start timing when you are set and have closed your eyes."*

(c) Tandem stance:
*"Now stand heel-to-toe with your **non-dominant foot** at the back. Your weight should be evenly distributed across both feet. Again, you should try to maintain stability for 20 seconds with your hands on your hips and your eyes closed. I will be counting the number of times you move out of this position. If you stumble out of this position, open your eyes, return to the start position and continue balancing. I will start timing when you are set and have closed your eyes."*

Balance testing – types of errors
1. Hands lifted off iliac crest
2. Opening eyes
3. Step, stumble, or fall
4. Moving hip into > 30 degrees abduction
5. Lifting forefoot or heel
6. Remaining out of test position > 5 sec.

Each of the 20-second trials is scored by counting the errors, or deviations from the proper stance, accumulated by the athlete. The examiner will begin counting errors only after the athlete has assumed the proper start position. **The modified BESS is calculated by adding one error point for each error during the three 20-second tests. The maximum total number of errors for any single condition is 10.** If an athlete commits multiple errors simultaneously, only one error is recorded but the athlete should quickly return to the testing position, and counting should resume once the subject is set. Subjects that are unable to maintain the testing procedure for a minimum of **five seconds** at the start are assigned the highest possible score, ten, for that testing condition.

Which foot was tested: ☐ Left ☐ Right
(i.e. which is the **non-dominant** foot)

Condition	Total errors
Double leg stance (feet together)	of 10
Single leg stance (non-dominant foot)	of 10
Tandem stance (non-dominant foot at back)	of 10
Balance examination score (30 **minus** total errors)	of 30

7 Coordination examination

Upper limb coordination

Finger-to-nose (FTN) task: *"I am going to test your coordination now. Please sit comfortably on the chair with your eyes open and your arm (either right or left) outstretched (shoulder flexed to 90 degrees and elbow and fingers extended). When I give a start signal, I would like you to perform five successive finger to nose repetitions using your index finger to touch the tip of the nose as quickly and as accurately as possible."*

Which arm was tested: ☐ Left ☐ Right

Scoring: 5 correct repetitions in < 4 seconds = 1

Note for testers: Athletes fail the test if they do not touch their nose, do not fully extend their elbow or do not perform five repetitions. Failure should be scored as 0.

Coordination score of 1

8 Cognitive assessment

Standardized Assessment of Concussion (SAC)

Delayed recall

"Do you remember that list of words I read a few times earlier? Tell me as many words from the list as you can remember in any order."

Circle each word correctly recalled. Total score equals number of words recalled.

List	Alternative word list		
elbow	candle	baby	finger
apple	paper	monkey	penny
carpet	sugar	perfume	blanket
saddle	sandwich	sunset	lemon
bubble	wagon	iron	insect

Delayed recall score of 5

Overall score

Test domain	Score
Symptom score	of 22
Physical signs score	of 2
Glasgow Coma score (E + V + M)	of 15
Balance examination score	of 30
Coordination score	of 1
Subtotal	**of 70**
Orientation score	of 5
Immediate memory score	of 5
Concentration score	of 15
Delayed recall score	of 5
SAC subtotal	**of 30**
SCAT2 total	**of 100**
Maddocks Score	**of 5**

Definitive normative data for a SCAT2 "cut-off" score is not available at this time and will be developed in prospective studies. The SAC score, which is embedded within the SCAT2, can be utilised separately in concussion management. The scoring system also takes on particular clinical significance during serial assessment where it can be used to document either a decline or an improvement in neurological functioning.

Scoring data from the SCAT2 or SAC should not be used as a stand-alone method to diagnose concussion, measure recovery or make decisions about an athlete's readiness to return to competition after concussion.

Figure 3.6.11.3 SCAT2 form

Athlete information

Any athlete suspected of having a concussion should be removed from play, and then seek medical evaluation.

Signs to watch for

Problems could arise over the first 24-48 hours. You should not be left alone and must go to a hospital at once if you:

- Have a headache that gets worse
- Are very drowsy or cannot be awakened (woken up)
- Cannot recognise people or places
- Have repeated vomiting
- Behave unusually or seem confused; are very irritable
- Have seizures (arms and legs jerk uncontrollably)
- Have weak or numb arms or legs
- Are unsteady on your feet; have slurred speech

Remember, it is better to be safe.
Consult your doctor after a suspected concussion.

Return to play

Athletes should not be returned to play the same day of injury. When returning athletes to play, they should follow a gradual symptom-limited program, with stages of progression. For example:

1. Rest until asymptomatic (physical and mental rest)
2. Light aerobic exercise (e.g. stationary cycle)
3. Sport-specific exercise
4. Non-contact training drills (start light resistance training)
5. Full contact training after medical clearance
6. Return to competition (game play)

There should be approximately 24 hours (or longer) for each stage and the athlete should return to stage 1 if symptoms recur. Resistance training should only be added in the later stages.
Medical clearance should be given before return to play.

Tool	Test domain	Time	Score							
		Date tested								
		Days post injury								
SCAT2	Symptom score									
	Physical signs score									
	Glasgow Coma score (E + V + M)									
	Balance examination score									
	Coordination score									
SAC	Orientation score									
	Immediate memory score									
	Concentration score									
	Delayed recall score									
	SAC Score									
Total	**SCAT2**									
Symptom severity score (max possible 132)										
Return to play			n Y	n N	n Y	n N	n Y	n N	n Y	n N

Additional comments

Concussion injury advice (to be given to concussed athlete)

This patient has received an injury to the head. A careful medical examination has been carried out and no sign of any serious complications has been found. It is expected that recovery will be rapid, but the patient will need monitoring for a further period by a responsible adult. Your treating physician will provide guidance as to this timeframe.

If you notice any change in behaviour, vomiting, dizziness, worsening headache, double vision or excessive drowsiness, please telephone the clinic or the nearest hospital emergency department immediately.

Other important points:

- **Rest and avoid strenuous activity for at least 24 hours**
- **No alcohol**
- **No sleeping tablets**
- **Use paracetamol or codeine for headache. Do not use aspirin or anti-inflammatory medication**
- **Do not drive until medically cleared**
- **Do not train or play sport until medically cleared**

Clinic phone number

Patient's name

Date/time of injury

Date/time of medical review

Treating physician

Contact details or stamp

SCAT2 SPORT CONCUSSION ASSESSMENT TOOL 2 | **PAGE 4**

Figure 3.6.11.4 SCAT2 form

3.7 Spinal injuries

3.7.1 Incidence

Although there is no data available that would allow a precise comparison, it may well be true that real spinal injuries occur considerably less frequently among footballers than they do among other sportsmen and women, such as ice-hockey players or skiers. On the other hand, though, spinal problems generally are more common among footballers than they are among other sportsmen and women.

The reason behind the greater incidence of spinal problems among footballers might be that football training often concentrates on building up or exercising the lower body musculature, while the stomach, back, shoulder and neck muscles tend to be neglected. However, it is precisely these muscle groups that play a decisive part in stabilising and ensuring trouble-free upper body mobility. Mass screening among football players often detects muscular imbalance of the above-mentioned muscle groups, which can be the cause of longer-lasting spinal complaints, including those occurring in the pelvic and hip regions, such as recurrent groin pain.

An F-MARC study investigated back injuries and other complaints among footballers. All the injuries and complaints sustained by a total of 264 footballers from different age and ability groups were recorded on a weekly basis over a one-year period. The weekly examinations of the players conducted by specially designated doctors ensured that complete documentation and records were kept; these even included minor complaints and injuries. Over the course of the year, some 37% of the players in the study reported suffering from lumbar spine complaints, while 33% complained of problems in the neck and head region (Figure 3.7.1.1).

Lumbar spine injuries are, however, much less common in football than injuries to other parts of the body. Only 6% of the players examined had sustained lumbar spine injuries, and in most cases these injuries had been of a minor nature. Throughout the year of the study, no cervical spine injuries were recorded; 3.6% of the players did, however, complain of head injuries (Figure 3.7.1.2).

3.7.2 Clinical biomechanics

The major axial support of the body consists of the spinal column and the surrounding soft tissues (ligaments and musculature), which, among other functions, provide protection for the spinal cord and the nerves that emerge from the spinal cord. The osseous spinal column has two

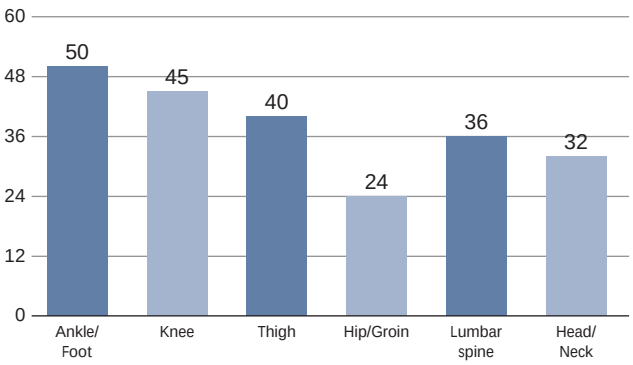


Figure 3.7.1.1 Lumbar spine complaints
Comparison of complaints unrelated to injuries of football players during one year observation (from Peterson et al. 2000)

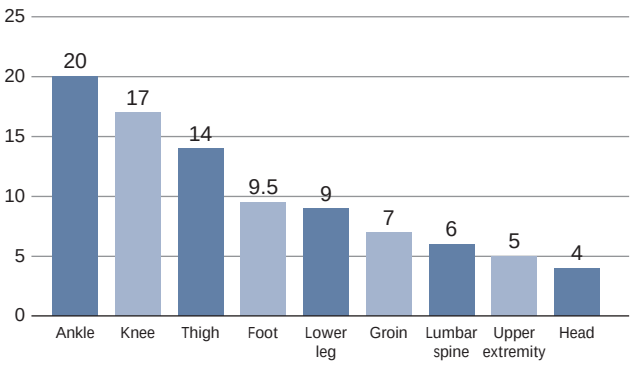


Figure 3.7.1.2 Lumbar spine injuries
Frequency of lumbar spine and head/neck injuries in comparison with other injuries in amateur football players (from Peterson et al. 2000)

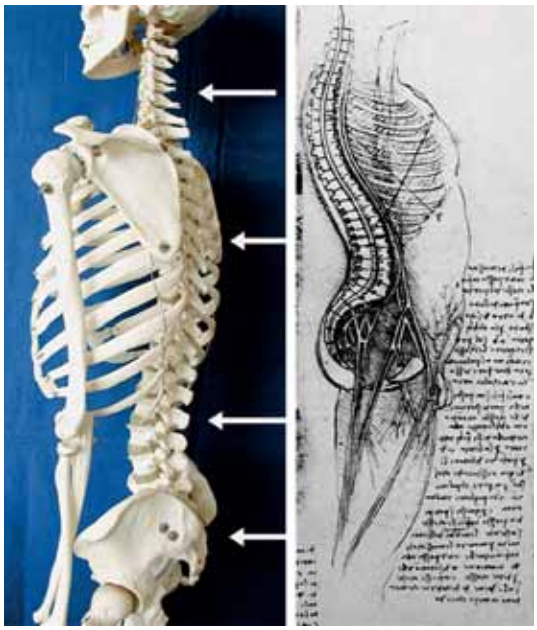


Figure 3.7.2.1 Spine with torso, arrow points at cervical, thoracic, lumbar spine and pelvic girdle

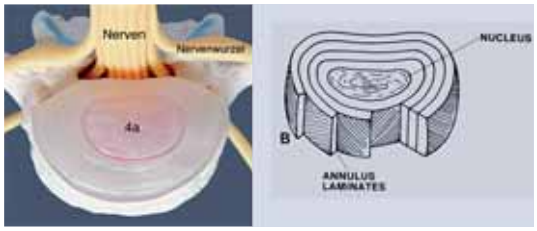


Figure 3.7.2.2 Intervertebral disc

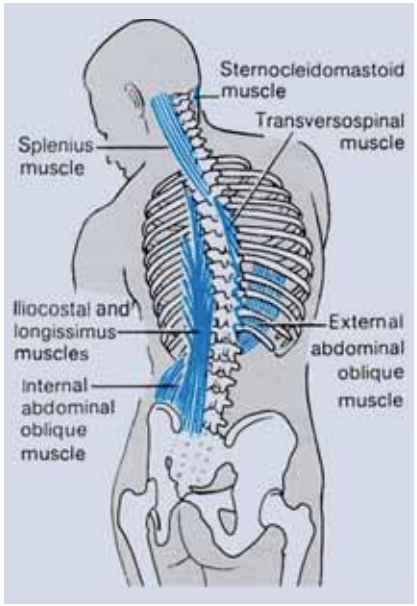


Figure 3.7.2.3 The most important spinal muscle groups

natural curves – in the neck and lumbar regions – and consists of seven cervical vertebrae in the neck, 12 thoracic vertebrae in the upper back, five lumbar vertebrae in the lower back, as well as the articulation between the sacrum and innominate bones (Figure 3.7.2.1).

The individual vertebrae are linked to one another by an intervertebral disc. The intervertebral disc is composed of an inner gelatinous nucleus, enclosed in fibrocartilage, arranged like the skin of an onion (Figure 3.7.2.2). The intervertebral disc balances the axial load between the vertebrae but also limits the range of motion, particularly in the lumbar spine.

Incorrect or excessive loading of the spine, or even injuries brought about by extreme, uncoordinated movements or a direct blow, can cause the intervertebral disc to tear and the gelatinous nucleus pulposus to herniate. The herniated nucleus pulposus is known as a "slipped disc" or "disc hernia". This can lead to pain in the back (lumbago) or in the leg (sciatica).

The stability of the otherwise very delicately balanced spinal column is guaranteed by the efficient functioning of the ligaments and, most importantly, by the surrounding musculature, which ultimately holds the spinal column erect. To ensure stability, balance between the dorsal paravertebral and ventral abdominal muscle groups of the spinal column is crucial (Figure 3.7.2.3).

3.7.3 Cervical spine

The cervical spine is the most mobile segment of the spine and is predisposed to complaints brought about by wear and tear phenomena. Scientific studies show that around one third of the adult population suffers from neck problems even though these problems do not always require treatment. A normal young adult can turn his/her head

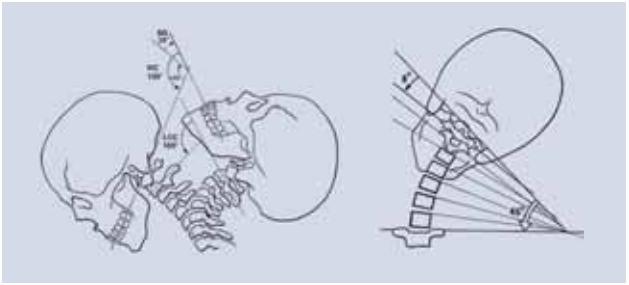
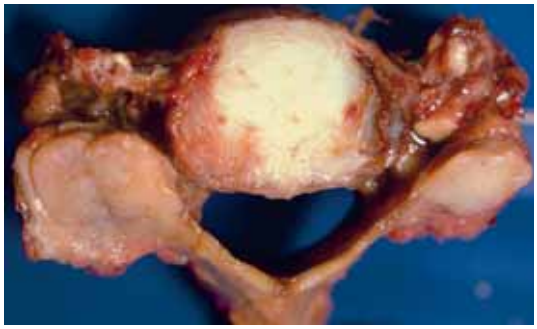


Figure 3.7.3.1 The range of motion for the cervical spine (from Kapandji, 1974)

to either side approximately 90°, incline it forwards at an angle of around 45° and bend it backwards and forwards at an angle totalling 130° (Figure 3.7.3.1). The weight of the head, which averages somewhere between six and eight kilograms, thus has a detrimental lever-arm effect on the facet joints if used inappropriately.

The comparatively large range of motion of the cervical spine and the orientation of the zygapophyseal joints (intervertebral joints) can lead to overuse or, as a result of repetitive minor injuries, to acceleration of degenerative changes/osteoarthritis. In the baseline examination, players with suspected hyperlaxicity will present an increased range of motion mainly for axial rotation. A careful assessment to plan the player's career is essential.

It is established that the intervertebral discs in the cervical spine show degenerative changes in the third life decade which might lead to the restriction of range of motion and, more importantly, motion-induced pain. In particular, for football players who might be inappropriately heading the ball, an asymmetrical load to the cervical spine could also result in degenerative changes in the intervertebal joints (Figure 3.7.3.2).



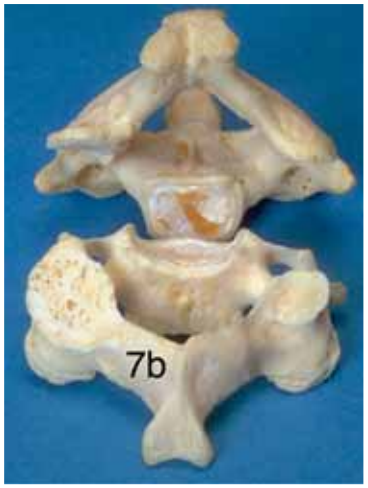


Figure 3.7.3.2 Osteoarthritis of the zygaphophyseal joint of the fourth cervical vertebra

3.7.4 Bony and ligamentous injuries

Incidence

Unknown, rare.

Injury types and mechanisms

In the case of serious injuries associated with the flexion-rotation mechanism and occurring with axial compression, not only the neck vertebrae but also the facet joints can sustain fractures. This constitutes a serious danger for the spinal cord and the spinal nerves. The rupture of the ligaments of the posterior elements is usually associated with rupture of the intervertebral disc (Figure 3.7.4.1).

Axial trauma to the cervical spine combined with flexion/extension, following a direct headbutt might cause a stable fracture of the vertebral endplate and the concordant articular process (Figure 3.7.4.2).

Symptoms

Stable injuries of the cervical spine do not necessarily cause pain when resting in a neutral position, but pain

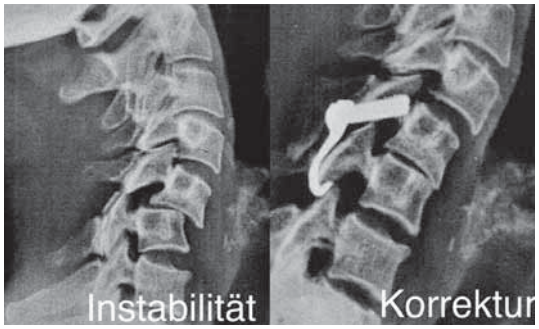


Figure 3.7.4.1 A major instability after forceful flexion of the cervical spine with complete rupture of posterior ligaments and the intervertebral discs C5/6: surgical intervention with fusion was necessary

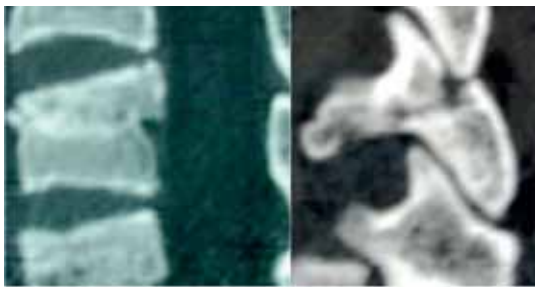


Figure 3.7.4.2 Endplate fracture of fifth cervical endplate with concordant stable fracture of articular process, treated conservatively with soft collar and consequent muscle stabilising exercises in a 22-year-old male athlete

increases during motion. Unstable injuries with or without fracture of the bony structures are also painful while resting, and movement of the cervical spine is restricted. The symptoms occur immediately after injury or with a latency of minutes.

Unstable injuries are commonly followed by neurological symptoms such as paraesthesia in the arm or fingers, or weakness of segmentally innervated muscles. If a player complains of neurological symptoms in the lower extremities, such as L'hermitte's sign or pins and needles in the foot, damage to the spinal cord should be suspected.

Signs and diagnosis

The clinical examination of the cervical spine often shows a reduced range of motion for axial rotation being accompanied by motion-induced pain. To differentiate the restriction in the upper or lower cervical spine, passive flexion should be performed to exclude the motion in the lower cervical spine. Rotation out of flexion can occur only in the upper cervical spine, mainly in the atlanto-axial joint, while rotation out of extension mainly occurs in the segments below the second cervical vertebra. The functional examination is followed by palpation of the soft tissue including the paravertebral muscles and intravertebral joints. Localised painful spots called "zones of irritation" might be an expression of an unstable or functionally disturbed segment. The aim of the clinical examination is to identify the regional and/or segmental dysfunction.

All patients who have sustained an injury of the cervical spine should undergo a neurological investigation with an assessment of muscle-tendon reflexes indicating a possible nerve root deficit and/or increased reflexes as a sign of cervical myelopathy. The examination of the sensory qualities according to the dermatomal distribution is helpful in locating or raising suspicion of a nerve root lesion. Long trunk signs (pyramidal signs), such as absent abdominal skin reflexes or present Babinsky signs, are expressions of cervical myelopathy.

According to the clinical symptoms and signs, after cervical spine injury, additional examinations are indicated, such as neurophysiological testing (motor- and sensory-evoked potentials, electromyography) conventional AP, and lateral and oblique X-rays. Functional X-rays are performed to document and/or exclude segmental instability. If bony lesions are suspected, CT tomography is the appropriate tool. If structural lesions are suspected, such as nerve root and/or spinal cord, an MRI will document the lesion.

On-field treatment

According to the clinical symptoms and signs, every cervical spine injury should be treated with extreme caution, particularly if neurological symptoms and signs are present, as an unstable situation has to be suspected unless the contrary is proven. Therefore, it is recommended to take the player out of the game. Should a player complain of neurological symptoms immediately after the injury, he should be removed on a stretcher with extreme caution after applying a cervical orthosis. The cervical orthosis, which is usually not available at the football stadium, should be applied with the aid of available material such as a towel and tape. The head should be held during the stabilising procedure by accompanying persons in a neutral position, as indicated in Figure 3.7.4.3. If neurological symptoms and/or signs are present, the injured player should be transferred to a regional spinal centre in a supine position.

Players who do not present neurological symptoms and/or signs but have motion-induced pain should be taken out of the game and require additional medical assessment, including X-rays.

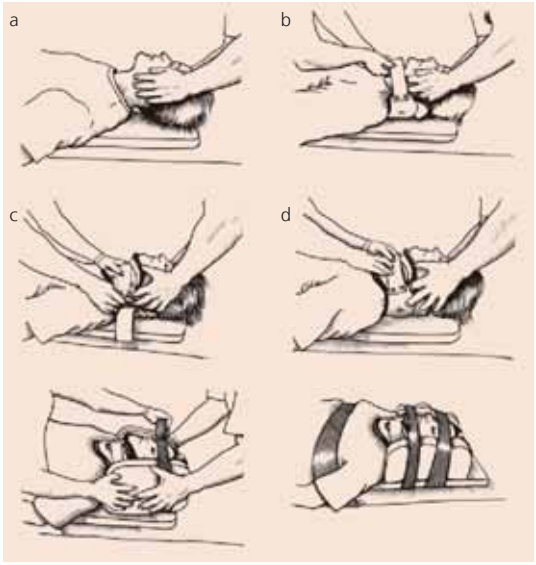


Figure 3.7.4.3 Stabilisation of patients with a suspected unstable fracture

- Stabilise the head and neck (a)
- Put on the back part of the collar (b)
- Put on the front part of the collar (c)
- Secure the collar (d)
- Roll the patiient onto a stiff board or stretcher
- Centre the patient on the board
- Place two blanket rolls or foam blocks on the board
- Anchor the blocks around the patient's head (e)
- Anchor the blocks to the board (f)

Treatment

An unstable situation of the cervical spine normally requires surgical intervention according to the pathology. Such operations should be performed in a specialised spinal centre.

In a stable situation, spinal specialists will decide after additional examinations whether a conservative approach with cervical collars and muscular rehabilitation (stabilising exercises) is justified.

3.7.5 Soft tissue injuries of the cervical spine

Incidence

The true incidence of soft tissue injuries of the cervical spine is unknown; however, considering the frequency of neck pain and headache, soft tissue injuries might be more common than expected.

Injury mechanism and risk factors

There are a number of situations during the football game when soft tissue injury of the cervical spine can occur as an indirect trauma.

The clash of heads, elbow to head contacts and simple falls with direct head trauma could all cause an indirect trauma to the cervical spine.

Symptoms and signs

The indirect trauma (soft tissue injury) to the cervical spine can present a wide variety of clinical symptoms such as neck pain, headache, vertigo, asystematic dizziness, nausea, blurred vision and others. The most frequent symptom is motion-induced pain locally and radiating into the shoulder region.

Less frequently, neurological symptoms are accompanied by paraesthesia in the arm or fingers and rare muscular weakness (most probably pain-induced motor inhibition).

During the clinical investigation, a full range of motion is normally observed, with pain at the end of the range of axial rotation, flexion/extension and side bending. Typically, there are painful tender points above the zygapophyseal joints, accompanied by muscle tenderness of the paraspinal (mainly posterior) muscles. The neurological examination does not normally reveal signs of any deficit.

Diagnosis

Soft tissue injury of the cervical spine is a clinical diagnosis. Radiological and imaging findings are normal and neurophysiological investigation seldom identifies pathological findings.

On-field treatment

Should the player present clinical symptoms and signs of soft tissue injury, he/she should be taken out of the game if symptoms besides pain are presented. If the clinical and neurological investigations reveal no sign of deficit, then a rest period (internal stabilisation by the neck muscles) is indicated until the symptoms resolve. Additional application of analgesics or non-steroidal antirheumatic drugs is seldom indicated.

Treatment and rehabilitation programme

After the initial symptoms are resolved, appropriate physiotherapy treatment with muscular rehabilitation is indicated. Should a segmental dysfunction be diagnosed by special manual diagnostics, appropriate manual treatment by specially trained physicians and/or a specially trained physiotherapist might be helpful if contraindications are excluded.

Prognosis and return to play

In general, the prognosis is good, with symptoms being resolved within two to four weeks in the majority of cases.

Should symptoms remain, extensive investigation is indicated after four weeks, with functional X-rays of the cervical spine and neuropsychological assessment to document potential deficits of cognitive function.

3.7.6 Thoracic spine

The thoracic spine is the least mobile segment of the spinal column, by virtue of being attached to the ribs and to the sternum. The channel surrounding the spinal column in the area of the thoracic spine is relatively narrow. This means that when vertebrae are broken the risk of spinal cord injury is relatively high. However, it is extremely rare for footballers who suffer a spinal cord injury to be subsequently paralysed in the lower part of the body (paraplegia).

3.7.7 Lumbar spine/pelvic girdle

The lumbar spine is a mobile section of the spine, its main movement being forward (flexion) and backward (extension) bending (approx. 70-80°) and side bending (lateral flexion) (approx. 40-50°). Rotatory movements are rendered largely impossible by virtue of the steeply-oriented facet joints and this acts as a protection for the intervertebral disc (Figure 3.7.7.1).

On the other hand, however, the joints are subjected to considerable strain and, as is the case with the cervical spine, this can lead to wear and tear phenomena (osteoarthrosis) with the corresponding movement-related symptoms of pain.

3.7.8 Acute low back pain (LBP)

Incidence

The true incidence of acute low back pain in the population of active football players is not known, but the data from the F-MARC studies indicates that almost half of the football players experienced acute low back pain during the year of examination.

Injury mechanism and risk factors

Acute LBP can occur without any prodromal symptoms and without any special event or injury. Taking a detailed medical history can help identify some inducing mechanisms.

Symptoms and signs

The players will usually report the onset of low back pain as being located in a small region of the lumbar spine in the acute stage, seldom followed by referred pain to the thoracolumbar or lumbosacral junction or into the buttock.

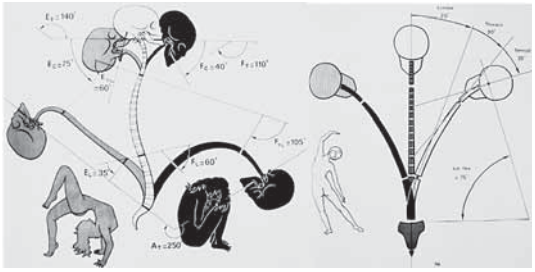


Figure 3.7.7.1 Drawing of entire and regional range of motion of the spine (from Kapandji 1974)

The pain, localised and aggravated during motion, can be followed by muscle soreness and tenderness.

During the functional examination of the lumbar spine, finger to toe distance is increased and the range of motion is decreased, particularly for side bending. Whilst in a prone position, the presence of palpable bands in the paravertebral muscles is verified. The manual diagnostics will reveal local tenderness and painful spots in the transverse process region as well as at the level of the intervertebral joints.

Radiological investigation is not necessary for acute low back pain.

Diagnosis

The diagnosis is purely clinical and described as a functional disorder of the lumbar spine.

On-field treatment

If there is only local tenderness and a palpable muscle band, continuation of the training session and/or game can be justified, particularly if neurological signs are not present.

The treatment of choice by a physician and/or physiotherapist trained in manual medicine would be manual therapy followed by muscular rehabilitation according to the muscle status and the balance between the postural and phasic muscles.

Prognosis and return to play

In the majority of cases, acute low back pain will resolve without any specific therapy to reduce recurrences. Ongoing muscular rehabilitation with strengthening of the paravertebral and abdominal muscles is the best preventive measure.

3.7.9 Recurrent or chronic low back pain

Incidence

Unknown but not very common.

Risk factors

The major risk factor for the development of chronic low back pain is a muscular imbalance between the phasic and tonic muscle groups surrounding the spine. Footballers who concentrate primarily on the development of the lower extremity muscles are prone to developing such a muscular imbalance with segmental and/or regional dysfunction of the lumbar spine.

Symptoms and signs

The symptoms are similar to those of acute LBP, but the duration is more than three months. Also, the clinical signs during the examination are similarly enhanced by more prominent palpable and painful muscle bands commonly described as myotendonotic changes. The pain in these muscles can be elicited while palpating across the fibre orientation and normally accompanies painful insertions of the muscle on both sides of the bone, while palpating in the direction of inserting fibres.

In addition to local pain, the referred pain is not only towards the thoracolambar and thoracic spine, but is also observed into the buttock. In cases where the origin of the low back pain could be identified in the sacroiliac joints, referred pain into the buttock and thigh is typical.

For players who suffer chronic low back pain, additional examination should be performed, such as X-rays in AP, lateral and oblique view to exclude spondylolysis or other structural changes of the bone, i.e. primary bony tumours. CT and MRI are seldom indicated.

Diagnosis

The diagnosis is clinical based on the functional and manual examination and commonly described as chronic segmental and/or regional dysfunction of the lumbar spine or sacroiliac joint.

The treatment should be focused on consequent muscular rehabilitation aimed at restoring the balance between the phasic and tonic muscle groups, focusing on strengthening the postural muscles. Manual therapy can be introduced as an adjuvant treatment.

Prognosis

In young football players, if the cause of chronic LBP is diagnosed, appropriate treatment leads to a good prognosis.

3.7.10 Trauma-induced disc herniation

Incidence

Unknown but rare.

Injury mechanism and risk factors

Repetitive strains due to rotatary movement of the lumbar spine might cause micro-injuries to the annulus fibrosus of the intervertebral disc. Despite the complex biochemical process inside the intervertebral disc, the annulus fibrosus can be ruptured by forceful movements, such as movement involving side-bending rotation combined with increased axial loading (stress) to the lumbar spine, e.g. when a player runs into the opponent in such a position.

Symptoms and signs

The symptoms of acute disc herniations are impressive due to the immediate onset of pain primarily in the leg and in the lower back. The pain radiates towards the knee in patients with disc herniation in segment L2/3 and L3/4 and towards the foot in herniations of segment L4/5 and L5/S1. The disabling pain is accompanied by a stiff lumbar and thoracic spine in a slightly tilted position (antalgic position). Coughing, sneezing or pressing will aggravate the pain. In the case of nerve root compression, the pain is followed by the development of sensory symptoms (hyposthesia, anaesthesia) distributed according to the dermatomal innervation and also the weakness of either the quadriceps, hamstrings, gluteal or anterior tibial or gastrocnemius muscles.

The clinical examination will include a forward bending test, walking on tip toes and heels, as well as stepping on a chair with one leg (quadriceps). The neurological examination is normally performed in the supine position and will include the straight leg raising test (Lasegue Phenomenon), the assessment of muscle tendon reflexes as well as the examination of sensory deficit.

If the clinical signs are suspicious for nerve root compression due to disc herniation, an MRI examination is the next step to secure a diagnosis.

Diagnosis

If there are clinical neuroradiological signs (MRI or CT scans) in the case of motor deficit, a neurophysiological assessment is indicated (electromyography, H-reflex, motor- and sensory evoked potentials).

Treatment

As long as the neurological deficit is not apparent and the motor weakness is not present or is insignificant, conservative therapy could be justified with a few days of bed rest in an appropriate antalgic position and systemic steroids. The application of local steroids by means of epidural injections via hiatus canalis sacralis (sacral block) with 80 mg Triamcort and 20 ml 0.5% Lidocaine is preferable. Such

an injection can be repeated twice or three times over the course of one week. Regular monitoring of the clinical signs is essential and any increase in the sensory or motor deficit requires a reassessment of conservative treatment.

Physical therapy in acute nerve root compression is less valuable, but becomes more important during rehabilitation.

Should sensory and motor deficits increase and the clinical findings substantiate the symptoms, surgical decompression (discectomy) has to be discussed. A consultation by spinal specialists is required at an early stage.

Rehabilitation programme

In both cases, after successful conservative and/or surgical treatment of disc herniations, a consequent rehabilitation programme is required similar to the one in the case of chronic low back pain.

Prognosis and return to play

Disc herniation that has been successfully treated conservatively and/or operatively has a good prognosis. After the appropriate period of rehabilitation (two to three months), the player can gradually increase his training load and return to play. The rehabilitation has to be monitored by specialists in this field.

3.7.11 Traumatised spondylolysis

Incidence

Injury mechanism and risk factors: often congenital, but also acquired, spondylolysis becomes symptomatic after rotational trauma. Weak lumbar spine muscles are a major risk factor for becoming symptomatic. Repetitive rotational micro-traumas could facilitate development.

Symptoms and signs

Local and referred pain is similar to that found in players with chronic low back pain. Motion-induced pain, particularly during axial rotation, is seldom accompanied by nerve root symptoms or signs. During the manual examination, provocation of the pain during lateral bending and rotation is positive at the affected level, mostly L5. Rare neurological deficits are seen except when additional disc herniations occur at the same level or during the development of spondylolisthesis with encroachment of the nerve root.

Diagnosis

The diagnosis of traumatic spondylolysis is clinical and radiological with oblique X-rays, preferably followed by functional flexion extension X-rays of the lumbar spine, verified by the results of MRI and/or CT scan with 3D reconstruction (Figure 3.7.11.1).

Treatment

At the first occurrence of the symptoms of traumatised spondylolysis, conservative treatment is indicated with injections of local anaesthetics in combination with physiotherapy and manual treatment. In a recurrent situation, surgical fusion has to be carefully considered, especially if the adjacent intervertebral discs show signs of degeneration.

If a surgical procedure is an option, the direct fusion of the lytic zone with screws or intersegmental fusion by translaminous screws or posterior fixation should be considered.

Rehabilitation and prognosis

In general, the prognosis of grade I and II spondylolysis is good with appropriate therapy.

A post-operative rehabilitation programme is mandatory after the healing period has been completed (six to eight weeks). A gradual increase of muscular rehabilitation with stabilising exercises and later combined with stretching, under the supervision of specially trained physiotherapists, will get the player back to the field of play.

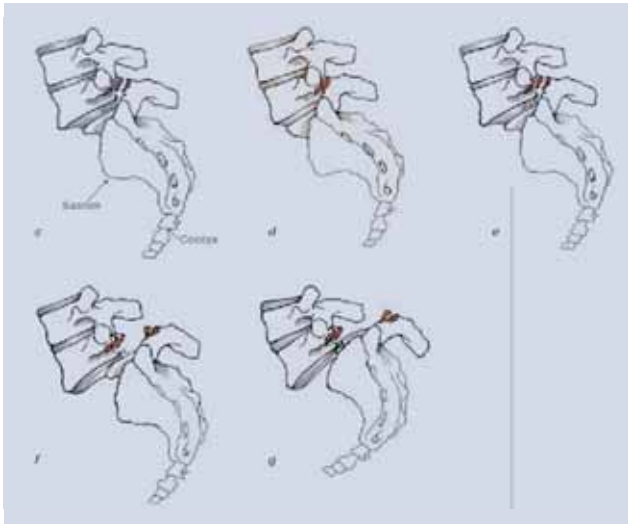


Figure 3.7.11.1 The drawing shows the grading of spondylolisthesis I-IV

4. Diseases

4.1 Exercise-induced asthma

The relationship between exercise and asthma has been recognised for centuries. In his landmark "A Treatise of the Asthma", Sir John Floyer wrote in 1717 that "… all violent exercise makes the asthmatic to breathe short …".

Exercise-induced asthma (EIA) is defined as the acute transient airway narrowing that occurs during, but more often and more distinctively after, exercise; often arbitrarily defined as a fall in Forced Expiratory Volume in one second (FEV_1) of >10%.

This definition incorporates the objective fall in lung function associated with exercise that occurs in:

- persons with known pre-existing asthma; the phenomenon of exercise as a "trigger" for exacerbations of asthma;
- otherwise asymptomatic persons, often athletes, without a known history of asthma. This is often termed exercise-induced bronchoconstriction. However, this is a possible misnomer as the term suggests airway narrowing is due solely to airway smooth muscle constriction. Vascular congestion and airway wall oedema may contribute to the airflow obstruction.

In the context of this chapter, the term "EIA" will be used to cover both of these phenomena although the relationship between these two entities remains contentious: different pathophysiologic processes may be involved, and responses to interventions may differ.

In up to 90% of people with known asthma, exercise is a trigger. In some, EIA may be the only manifestation of asthma. While the prevalence of EIA amongst athletes will vary according to the group under study, the definition of EIA used, the type of challenge test used, and the FEV_1 cut-off, the prevalence of EIA is comparatively high in elite athletes. The prevalence of asthma in the general population varies from 5-20%, while that in athletes has been reported as being from 10-70%.

Bearing in mind that one would intuitively expect a lower prevalence of asthma amongst elite athletes, it is remarkable that 11% of the 1984 and 17% of the 1996 US Summer Olympic Team either had a diagnosis of asthma and/or were using asthma medication. Prevalence appears highest in those engaged in endurance sports, particularly winter sports. According to the therapeutic use exemption (TUE) applications for beta-2-agonists at the Winter Olympic Games 2002, 5.2.% of all participating athletes inhaled these agents, while at the Summer Olympic Games in 2004 it had been 4.2% of all athletes. When comparing TUE applications for asthma treatment at the previous three Summer Olympic Games, the prevalence of asthma in football players seems rather low (1.9%) compared to other sports such as cycling (15.4%) and swimming (11.3%).

4.1.1 Pathogenesis

While the precise mechanism remains controversial, it is generally regarded as involving the loss of water by evaporation from the surface of the airways causing cooling and/or dehydration of the airway surface.

When exercise of moderate intensity is undertaken, the normal warming and hydrating effect of the upper respiratory tract is by-passed and conditioning of the inspired air occurs in the proximal lower airways. As the volume of the airway surface lining (ASL) fluid for these airways is in the order of 1ml, loss of a very small volume of water can lead to a transient increase in the osmolarity of the ASL fluid and impairment of mucociliary clearance. With more intense exercise and/or exercise in cold/dry air, more distal airways are recruited to condition the inspired air, with a resultant increase in the airway surface area that is dehydrated and hyperosmolar. This results in the release of bronchoactive mediators, predominantly from mast cells but also from other resident and recruited cells. Submucosal oedema developing in response to dehydration injury amplifies the airway narrowing caused by airway smooth muscle contraction (Figure 4.1.1.1).

Repetitive drying and cooling of the airway mucosa causes epithelial/mucosal injury. Such mucosal damage may not only allow greater access of allergens and expose airway smooth muscle to products that alter their contractible properties but may also stimulate neural pathways. It may also reduce the epithelial cell production of bronchodilator products such as PG E2. This repetitive damage may also be responsible for neutrophilic airway inflammation reported in elite athletes with exercise-induced airflow obstruction.

In (atopic) asthma, eosinophilic airway inflammation is typical, although in recent years it has been recognised that a small but significant proportion of asthmatics have neutrophilic inflammation. It is possible that the pathologic processes in the airways of elite athletes differ from those which occur in most clinical asthma. It is not known whether the repeated injury and inflammation associated with long-term participation in high-level sport is responsible for the high prevalence of EIA in athletes. However, this contention is supported by the progressive development of airway hyper-responsiveness in elite athletes followed longitudinally and the apparent resolution after participation in high-level sport has ceased. This progression from airway inflammation and airway hyper-responsiveness to clinical asthma in athletes is consistent with epidemiologic evidence where a substantial proportion of those with asymptomatic airway hyper-responsiveness develop clinical asthma during follow-up.

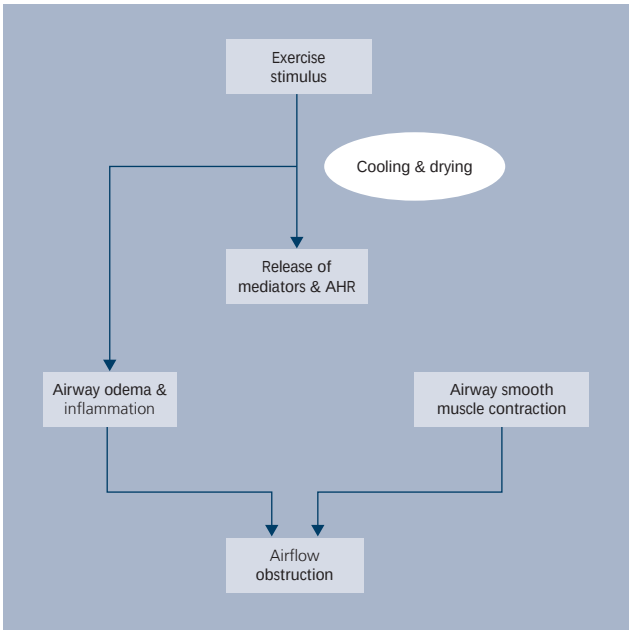


Figure 4.1.1.1 Mechanism of exercise-induced asthma

However, there is also a relationship between atopy, eosinophilic airway inflammation and exercise-induced symptoms. EIA is more frequent and severe in those who are atopic and asymptomatic atopic persons often have evidence of eosinophilic airway inflammation.

Other factors may also be relevant in the development of EIA in athletes. These include exposures to allergens and irritants, as well as autonomic deregulation associated with high intensity and prolonged physical training. Also to be considered is the fact that persistent rhinitis reduces the conditioning properties of the upper airway and subjects the lower airway to greater cooling and drying injury at a lower level of exercise.

4.1.2 Symptoms and signs

Symptoms such as shortness of breath, wheezing, chest pain with coughing, and chest tightness are those most suggestive of EIA. Typically, symptoms occur within five to ten minutes after starting vigorous exercise, but maximal airway narrowing occurs only after cessation of the exercise.

4.1.3 Influence on performance

As a result of impaired pulmonary function (reflected in reduced FEV_1 and reduced flows at mid and low lung volumes, there is a reduction in ventilatory capacity and increased (resistive) work of breathing, which manifests as dyspnoea. Flow limitation may exist at rest, when there is often associated static hyperinflation, or during exercise in association with dynamic hyperinflation. Hyperinflation has significant mechanical and sensory consequences including inspiratory muscle loading, restricted tidal volume increase during exercise, and increased (elastic) work of breathing. The reduction in inspiratory capacity is correlated with the sensation of dyspnoea. Apart from poor exercise performance, fatigue and prolonged recovery after exercise may occur.

Another aspect to be considered in this regard is a performance benefit players with EIA may gain from taking asthma medications, in particular those on the World Anti-Doping Agency's (WADA) Prohibited List. To date, the data on the performance effects of inhaled glucocorticosteroids in healthy athletes is rather limited, whereas there is convincing

evidence that inhaled beta-2-agonists do not improve performance in healthy athletes. Today, most authorities agree that there is little or no ergogenic potential of beta-2-agonists in athletes when used at doses required to treat or prevent EIA.

4.1.4 Diagnosis

The report of recurrent symptoms of bronchial obstruction provoked by different stimuli, and in particular by exercise, may suggest the diagnosis of EIA. The use of self-reported symptoms alone, however, leads to unacceptable false-positive and false-negative rates. Non-specific symptoms are often reported, and in highly trained individuals, exercised-induced respiratory symptoms are poor predictors of EIA. Clinical examination and baseline pulmonary function tests are almost invariably normal in athletes and are therefore poorly predictive of EIA.

The most direct method of establishing a diagnosis and therefore the gold standard would be to perform an exercise challenge and to demonstrate a fall in lung function in association with exercise. FEV_1 is the respiratory parameter most often used because of its ease of measurement, its repeatability and the relative effort-independence of the measure. Because it is a highly effort-dependent test, peak flow measurement is not recommended. All lung function testing should be conducted in line with ERS/ATS standards whenever possible. Although inexpensive portable spirometers exist, consideration needs to be given to the robustness of the device, the need for calibration, its accuracy, reliability and reproducibility, the inclusion of quality control criteria and the ability to display flow-volume loops for scrutiny.

Maximum fall in FEV_1 occurs five to ten minutes ***after*** a two-minute period of exercise but is also dependent on the intensity of exercise, the type of exercise and the conditions (temperature and humidity) in which the exercise takes place. There is no absolute FEV_1 cut-off that can be used for the diagnosis of EIA and the response to exercise is a continuum. Arbitrarily, a fall in FEV_1 of >10% (of baseline FEV_1) is required to make a diagnosis of EIA.

Challenge testing

No simple, efficient diagnostic exercise challenge exists for adults and expensive exercise-testing equipment is required. If such tests are to be performed, it is recommended that they are performed in a cold (20-25°C) laboratory with a relative humidity of <50% using either a treadmill or cycle ergometer. Workload needs to be rapidly increased to reach >80% of the player's maximal heart rate within two to three minutes. Exercise at this level should be performed for at least five minutes. Spirometry should be performed one, three, five, ten and fifteen minutes after cessation of exercise.

Eucapnic voluntary hyperventilation (EVH) is regarded as a surrogate challenge which correlates well with EIA in trained athletes. While EVH was designated the preferred tool for diagnosis of EIA by the International Olympic Committee Medical Commission, testing requires specialised equipment and players other than elite players may have difficulty maintaining the required level of ventilation.

Other challenge tests to demonstrate airway hyper-responsiveness can be classified into:

- **Indirect challenges**, including hypertonic saline, AMP and mannitol.
  These substances do not act directly on airway smooth muscle but cause the release of bronchoactive mediators. Whilst these challenges tend to correlate well with each other, they correlate less well with the results of direct challenges and different populations with airway hyper-responsiveness may be identified. (Direct and indirect challenges may give the same prevalence of airway hyper-responsiveness but identify different individuals. This is the rationale for preferring indirect challenges over direct ones.)
  The results of the hypertonic saline challenge correlate closely with those of exercise and EVH challenges.
  The hypertonic saline challenge is better standardised, less complex and no expensive equipment is required.
  As the challenge is progressive, excessive falls in lung function are avoided and a continuous measure of airway responsiveness can be obtained. In elite athletes, the mannitol challenge had a 96% sensitivity and specificity of 92% to identify a positive response to EVH.
- **Direct challenges**, in which the substance acts directly on airway smooth muscle to cause contraction. These include methacholine and histamine. These are regarded as inferior tests for the diagnosis of EIA although some investigations have found these tests to be more sensitive

than field testing. They do however require a high standard of equipment and technique.

When assessing the athlete with exercise-induced symptoms, the clinician needs to consider alternate diagnoses such as exercise-induced laryngeal dysfunction (vocal cord prolapse, laryngeal prolapse, functional laryngeal spasm), exercise-induced hyperventilation, exercise-induced anaphylaxis, gastro-oesophageal reflux, cardiac arrhythmias, other cardiac lesions, mitochondrial diseases of muscle, as well as a variety of other respiratory disorders.

4.1.5 Prophylaxis and management of EIA

To the extent to which it is practical and possible, allergen/trigger avoidance should be practised. It should be borne in mind that a significant proportion of asthmatics are likely to be sensitive to aspirin. Exacerbations due to NSAIDs are likely to be more relevant in football players due to their frequent use. On a theoretical basis, NSAIDs might also inhibit the development of the "refractory period" (by inhibiting the production of bronchodilator prostanoids such as PG E2).

Tachyphylaxis to exercise challenges occurs in up to half of athletes with EIA. During the resultant two-hour "refractory period", the exercise stimulus causes considerably less airway narrowing and this is thought to be due to the release of inhibitory mediators during exercise. However, this "refractory period" can be induced by warm-up exercise which does not in itself cause airway narrowing; prolonged (30 min.) periods of sub-maximal exercise and repeated short sprints have been demonstrated to induce the "refractory period". However this phenomenon does not occur in all asthmatics, the airway narrowing due to exercise is not completely abolished and it has not been well-documented in athletes without prior asthma. Nonetheless, recommendation of a warm-up period in players with EIA is appropriate.

4.1.6 Treatment

Many asthmatics have co-existent rhinitis and the loss of the conditioning effect of the upper airway may exaggerate the drying/cooling effect of exercise on the lower airway. Furthermore, poorly controlled upper airway disease can adversely effect the control of lower respiratory tract disease by a number of possible mechanisms. All this serves to emphasise the importance of adequately treating associated upper respiratory tract disease in players with EIA. Allergic rhinitis is best treated with topical intranasal steroids,

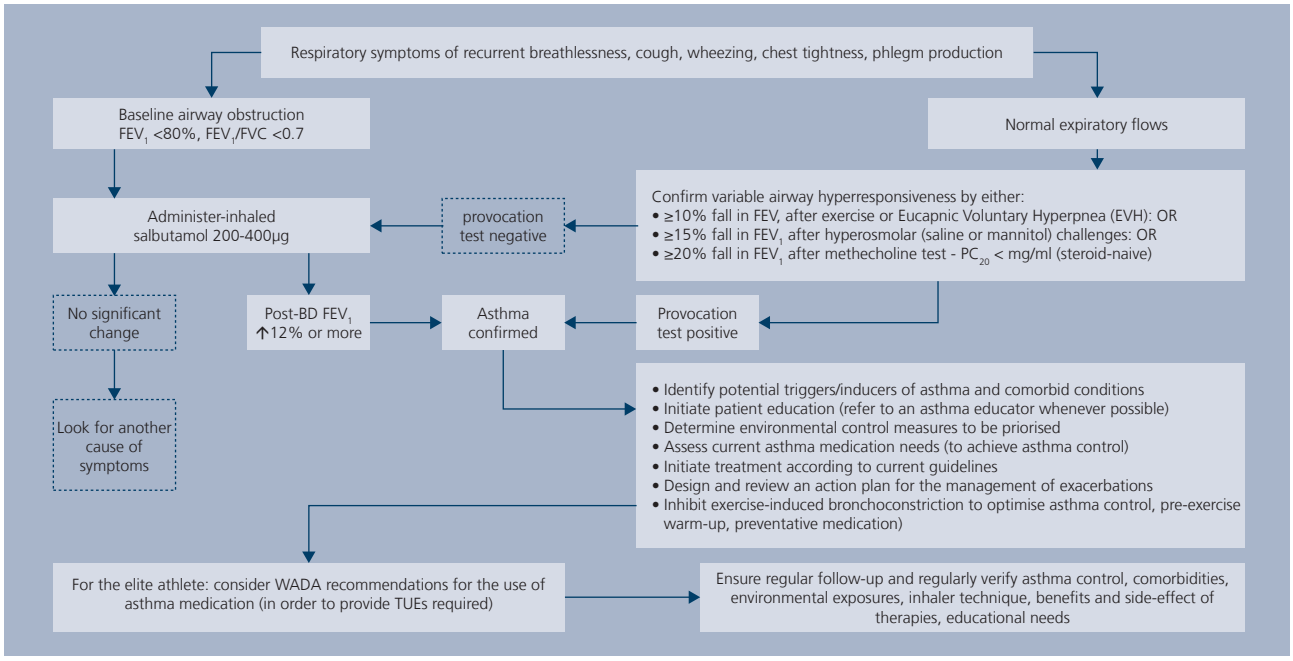


Figure 4.1.4.1 Diagnostic and therapeutic algorithm for asthma in football players. Source: Fitch K et al. "Asthma and the elite athlete: Summary of the IOC Consensus Conference, Lausanne, Switzerland, 22-24 January, 2008" Journal Allergy & Clinical Immunology Volume 122, Number 2, August 2008, p. 257.

possibly in association with oral anti-histamines, especially if the player has other manifestations of allergic disease.

In players with known asthma, treatment should be along conventional lines using a therapeutic escalator consisting of, as required, the use of short-acting beta-2-agonists (SABAs), low-dose inhaled corticosteroids, long-acting beta-2-agonists (LABAs), and higher-dose inhaled corticosteroids. This fundamental strategy is sometimes overlooked in the management of players.

Most players with EIA will require pharmacologic therapy, and the mainstay of this therapy is beta-2-agonists. SABAs are the most effective therapy and have advantages in EIA that include baseline bronchodilation, greater prevention of exercise-induced airway narrowing than occurs during the refractory period and the effects are additive to those of warm-up exercise. However, the overuse of SABAs has been associated with worsening of baseline lung function and of EIA. Although the clinical relevance of this phenomenon is not clear, it does serve to remind clinicians that high use of SABAs alone may not be in the players' best interest.

Long-acting beta-2-agonists provide a similar level of protection against EIA but are of much longer duration of action. Because of its more rapid onset of action, formoterol may have advantages over salmeterol.

Many athletes for whom SABAs or LABAs are recommended will not have previously used inhaled medications. It is the responsibility of the prescriber to ensure that the patient can use the device effectively and that this is checked on a number of occasions. Although pressurised meter dose inhalers (PMDIs) are generally preferred, the use of a large volume spacer or a dry powder preparation should be considered. Just because the patient is a talented player does not mean he can use a PMDI effectively!

Inhaled corticosteroids should be used in players with known asthma and exercise-induced symptoms, and there is evidence of benefit from long-term use. However, the effectiveness of inhaled corticosteroids in players without a diagnosis of asthma but exercise-induced airway narrowing is not known.

So called "mast cell stabilisers", such as cromolyn and nedocromil, reduce the degree of narrowing associated with exercise but are less effective than SABAs. However, they do not improve baseline lung function, have a shorter duration of action and are more expensive.

Similarly, leukotriene modifiers are effective at reducing EIA and have the advantage of once-daily dosing and an oral formulation. However they do not completely block EIA, do not produce bronchodilation and are more expensive than SABAs. Selective histamine agonists are ineffective in EIA.

There are few studies to define the most appropriate combination of agents for the management of EIA and it is likely that the physician will extrapolate the findings from asthma generally and undertake trials of therapy on individual players.

The reported effects of other agents such as vitamin C, frusemide and heparin are of interest because of the insights they may provide into pathophysiologic mechanisms rather than being alternate therapies to be seriously considered.

Players in whom adequate control of EIA cannot be achieved by the above measures should be referred to a respiratory specialist.

The above recommendations are in line with those of the Joint Task Force of European Respiratory Society and European Academy of Allergy and Clinical Immunology:

- EIA without other clinical manifestations of asthma may be best controlled by the use of short-acting inhaled beta-2-agonists taken ten to 15 minutes before exercise.
- EIA combined with other asthma symptoms may be best controlled by anti-inflammatory treatment either alone or in combination with reliever treatment. Inhaled corticosteroids in low to moderate doses are the preferred treatment.

4.1.7 Doping issues

Asthmatic football players should receive both optimal prophylaxis and treatment for their asthma. Of the medications commonly used for treatment of asthma, beta-2-agonists and glucocorticosteroids have been on the prohibited lists of first the IOC and later WADA for more than 15 years. As mentioned above, when inhaled, a performance-enhancing effect of these substances is at least to be doubted in the case of glucocorticosteroids and generally refuted by most in the case of beta-2-agonists.

However, the health of the player is a major issue when restricting the use of substances. To ensure that these agents are used appropriately and because

physician-diagnosed asthma was not always based on objective criteria, the International Olympic Committee's Medical Commission has established criteria for the diagnosis of asthma that are followed by other sports organisations and also recently by WADA. The International Standard for Therapeutic Use Exemptions (IS TUE) and WADA's medical information on asthma list several requirements for the establishment of the diagnosis, but according to the latest versions of these documents, the exact criteria to be met for a player allowing him to use prescribed asthma therapy depend mainly on his level of play.

For the use of beta-2-agonists, the national anti-doping organisations decide for national-level players if a standard TUE, retroactive TUE or medical file only is required. For players at international level, FIFA, as the international federation, demands a standard TUE for all players within its testing pools and also for all players participating at FIFA competitions. The FIFA TUE application form for asthma corresponds to the online submission form in ADAMS, WADA's database system. Full application details are laid out in the FIFA TUE policy.

For inhaled glucocorticosteroids, the 2009 WADA regulations require a declaration of use only, which has to be made on the doping control form at the time of testing. According to the IST, it "should" also be made in ADAMS, and thus requires registration of the player and/or his physician in the system beforehand. FIFA in general asks team physicians to list all medication taken by their players prior to a match where in-competition control is undertaken, or in out-of-competition controls at training camps. FIFA also requires declaration of the additional use of glucocorticosteroids on the application for the use of beta-2-agonists. The other asthma medications mentioned above do not require a TUE.

4.2 Diabetes

Diabetes mellitus (DM) is a chronic endocrine disorder characterised by hyperglycaemia caused by either decreased insulin secretion, decreased insulin action, or both. While the health benefits of physical activity are numerous, before beginning an exercise programme, diabetic players should be evaluated for conditions that might be associated with increased likelihood of cardiovascular disease or that might contraindicate certain types of exercise (e.g. those requiring valsalva manoeuvres or those performed at high altitudes) or have a particularly high risk of injury. Such conditions include uncontrolled hypertension, severe autonomic or peripheral neuropathy, pre-proliferative or proliferative retinopathy, and macular oedema.

4.2.1 Types of DM

Type 1 DM accounts for 10 to 15% of all cases of diabetes. It is characterised by a destruction of beta cells, leading to a deficiency of insulin production but normal insulin sensitivity. Type 2 DM accounts for the vast majority of the other 85-90% of other cases of DM. It is characterised by variable abnormalities in insulin secretion and insulin sensitivity. Both types of diabetes, in the uncontrolled state, will have increased hepatic glucose output and decreased glucose uptake in muscle and adipose tissue. However, in general, only patients with type 1 DM are at risk of severe lipolysis leading to diabetic ketoacidosis (DKA). Patients with type 2 DM are at risk for hyperosmolar non-ketotic state (HNS). This is somewhat similar to DKA, but the presence of some insulin in individuals with type 2 inhibits lipolysis and therefore ketone production.

Other secondary causes of DM have been characterised and classified and the categories of DM are expanding as we learn more about the genetic and environmental predisposing factors. It is most important for the football medicine physician to know that players with any form of DM will require special attention to their glucose regulation. Knowing whether the player's glucose metabolism involves more impairment in insulin secretion (as in type 1 DM) or impairment in insulin sensitivity (as in type 2 DM) is quite helpful in management. Also knowing the athlete's general diabetes control is important in anticipating his/her response to athletic activity and medical treatment. Thus, in this chapter, variations of DM will only be referred to under the global categories of "type 1" and "type 2".

4.2.2 Types of DM treatment

The primary goal of diabetes management is to keep blood glucose as close to the normal range without causing hypoglycaemia. Various baseline therapies exist. For some individuals with type 2 DM, this may include a form of insulin. For those players with type 1 DM, at least one form of insulin is always indicated. Types of insulin are listed in Table 4.2.2.1. Preferences and availability differ worldwide, however premixed combination insulins are falling out of favour and are suboptimal in athletes whose insulin needs will fluctuate dramatically throughout the day depending on activity level.

Oral hypoglycaemic agents and non-insulin injectables are listed in Table 4.2.2.2. It is important to know if a player on oral agents is on a drug that has the ability to contribute to hypoglycaemia, such as sulfonylureas and meglitinides.

4.2.3 Hypoglycaemia

Hypoglycaemia (blood glucose level < 3.6mmol/L or <65mg/dL) is the major concern among players with type 1 diabetes. There are many factors that need to be considered in understanding the development of hypoglycaemia during exercise. These include the intensity and duration of exercise, blood glucose concentrations before initiating exercise, the

time relation of exercise to meals, basal/bolus insulin doses, the individual's physical fitness level, his/her insulin sensitivity and the adequacy of his/her counterregulatory responses to exercise.

In players without DM, insulin levels decrease during exercise, but in players with DM who take exogenous insulin, levels do not decrease with activity. Increased insulin impairs hepatic glucose production and can induce hypoglycaemia 30 to 60 minutes after exercise begins. Counterregulatory hormones (e.g. glucagon, catecholamines, growth hormone, glucocorticoids) may be impaired in players with neuropathy or frequent hypoglycaemic episodes. In addition, exercise improves insulin sensitivity in skeletal muscle, leading to post-exercise, late-onset hypoglycaemia, often at night when the player is sleeping. In fact, in patients with type 1 DM, exercise increases the risk of severe hypoglycaemia up to 31 hours after cessation of activity.

In general, signs and symptoms of hypoglycaemia occur when blood glucose drops below 70mg/dl (3.9 mmol/l), but this number varies among individuals. Sweating, tachycardia, palpitations, hunger, nervousness, trembling, headaches and dizziness are early autonomic symptoms. Unfortunately, these are often hard to differentiate from responses experienced during vigorous exercise. If the blood glucose level continues to fall, neuroglycopenic symptoms may be observed and include fatigue, blurry vision, impaired cognition, loss of coordination, aggression, confusion, seizures and loss of consciousness. Thus, the rate of blood glucose decrease can be very important. Each individual has his/her own specific response to hypoglycaemia, and ideally the team physician should be familiar with these prior to the athlete's participationin sport. Table 4.2.3.1 shows an action plan for hypoglycaemia and initiating or returning to play.

Human insulin and insulin analogue	Time of onset of action	Peak of action	Duration of action
Rapid-acting			
aspart, glulisine, lispro	10-15 min.	1-2 hours	3-5 hours
Short-acting			
regular	0.5-1 hour	2-4 hours	4-8 hours
Intermediate-acting			
NPH	1-3 hours	4-10 hours	10-18 hours
Long-acting			
detemir	1 hour	none	up to 24 hours
glargine	2-3 hours	none	24+ hours
Pre-mixed insulin			
aspart mix 70/30 (70% aspart protamine + 30% aspart)	10-20 min.	1-4 hours	10-16 hours
lispro mix 75/25 (75% lispro protamine + 25% lispro)	10-15 min.	1-3 hours	10-16 hours
lispro mix 50/50 (50% lispro protamine + 50% lispro)	10-15 min.	1-3 hours	10-16 hours
70/30 (70% NPH + 30% regular)	0.5-1 hour	2-10 hours	10-18 hours
50/50 (50% NPH + 50% regular)	0.5-1 hour	2-10 hours	10-18 hours

Table 4.2.2.1 Types of insulin

Drug	Mechanism of action	Peak of action	Duration of action	Risk of hypoglycemia
Sulfonylureas	stimulate insulin release			**Yes**
glimepiride		2-3 hours	24 hours	
glipizide		1-3 hours	12-24 hours	
glipizide-GITS		6-12 hours	24 hours	
glyburide		4 hours	12-24 hours	
micronised glyburide		2 hours	12-24 hours	
Meglitinides	stimulate insulin release			**Yes**
nateglinide		20 min.	4 hours	
repaglidine		1 hour	4-6 hours	
Biguanides	decrease hepatic glucose production			**No**
metformin		2-3 hours	12-18 hours	
metformin extended release		4-8 hours	24 hours	
α-Glucosidase inhibitors	slow gut carbohydrate absorption	N/A	2-3 hours	**No**
acarbose		N/A	2-3 hours	
miglitol		N/A	2-3 hours	
Thiazolidinediones	increase peripheral insulin sensitivity			**No**
pioglitazone		N/A	days-weeks	
rosiglitazone		N/A	days-weeks	
DPP-IV inhibitors	increase glucose-dependent insulin release and suppress glucagon release			**No**
sitagliptin		1-4 hours	24+ hours	
Incretin mimetics	increase glucose-dependent insulin release and suppress glucagon release			**No**
exenatide*		1-2 hours	6-8	
Amylin analogue	suppress glucagon release, delay gastric emptying and promote satiety			**No**
pramlintide*		20 minutes	3 hours	

* Injectable medication

Table 4.2.2.2 Types of oral hypoglycaemic agents and non-insulin injectables

Mild hypoglycaemia (athlete is conscious and can follow commands)	**Severe hypoglycaemia (athlete is unconscious, unable to follow commands, and/or unable to swallow)**	**Post-exercise, late-onset hypoglycaemia**
Check blood glucose.	**Check blood glucose if possible to rule out other causes of change in mental status.**	**Check blood glucose.**
Based on blood glucose: • If glucose > 50mg/dl, administer 15g of fast-acting solid or liquid carbohydrate*. • If glucose ≤ 50mg/dl, administer 25-30g of fast-acting solid or liquid carbohydrate*.	Activate emergency medical system and mix and administer glucagon injection from glucagon kit# or administer 50% dextrose IV.	Based on blood glucose: • If glucose > 50mg/dl, administer 15g of fast-acting solid or liquid. carbohydrate* • If glucose ≤ 50mg/dl, administer 25-30g of fast-acting solid or liquid carbohydrate.*
	Assess response. • If athlete does not respond to glucagon or 50% dextrose, transfer to medical facility. • If athlete has regained consciousness and is able to swallow, have athlete eat complex carbohydrate¥.	
Recheck blood glucose in 15 minutes.	Continue to monitor.	Recheck blood glucose in 15 minutes.
If glucose still low, re-administer another 15g of fast-acting carbohydrate* and add 15g of complex carbohydrate¥ to sustain effect.	Do not allow same-day return to play and assess cause (e.g. medication dosing) before allowing resumption of physical activity in future.	If glucose still low, re-administer another 15g of fast-acting carbohydrate* and add 15g of complex carbohydrate¥ to sustain effect.
Recheck blood glucose in 15 minutes • If glucose in normal range, athlete is NOT on a long-acting glucose lowering medication and is feeling better, monitor for 15 to 30 minutes and then consider allowing return to play. • If athlete is on a long-acting glucose lowering drug, continue to monitor and do not allow return to play until after peak of medication has passed.		Continue to monitor blood sugar periodically. Note time of post-activity hypoglycaemia to plan snack or decrease in medication after future activity (e.g. if hypoglycaemia during night, decrease bedtime insulin and consider bedtime snack).

* Examples: glucose tablets or gel, hard candy, juice, honey

Because glucagon may cause nausea and/or vomiting, make sure athlete is on his/her side to prevent aspiration.

¥ Examples: crackers, bagel

Table 4.2.3.1 Action plan for hypoglycaemia

4.2.4 Hyperglycaemia

Exercise may cause hyperglycaemia in players with DM. In athletes who are under-insulinised and/or have poor baseline control, exercise (particularly high-intensity exercise) leads to increases in blood glucose levels and may eventually lead to DKA. High-intensity exercise is associated with increases in catecholamines, free fatty acids and ketones, all of which decrease muscle glucose utilisation and increase blood glucose. In the well-controlled athlete, these changes may be transient (decreasing in 30 to 60 minutes), but poor insulin balance along with stress regarding sports performance may increase counterregulatory hormones and perpetuate the hyperglycaemia. In the short-term, this may affect the athlete's ability to concentrate and may make him/her feel ill. In the long-term, such poor glycemic control can lead to end-organ damage. Table 4.2.4.1 shows an action plan for hyperglycaemia and initiating or returning to play.

4.2.5 Prevention

Each player with DM should have a care plan for training as well as matches/competitions. These should include the elements listed in Table 4.2.5.1 and should be familiar and available to the coach and team physician.

Fasting glucose should be checked, following the guidelines in Table 4.2.4.1. The pre-exercise meal should be one to three hours before the activity and should consist of low glycaemic-index foods to avoid too fast, but ensure continuous absorption of glucose. Immediately prior to the activity, the player should check his/her blood sugar again, with an ideal goal of 120-180 mg/dl. The player should continue to monitor his/her blood glucose throughout the sporting activity, supplementing with carbohydrate when needed, and continue to hydrate and reassess. Those using insulin should inject in areas away from exercising muscles approximately one hour before activity, as exercise, massage

Type of individual with DM	Fasting glucose (blood glucose ≥ 4 hours after eating a meal)	Comments
Adult or child with type 1 DM	≥ 250mg/dl (13.9mmol/l)	Check for ketones, and if positive, do not exercise. Treat DKA.
	251-300mg/dl (14-16.7mmol/l)	Check for ketones, and if negative, ok to perform physical activity.*
	> 300mg/dl (16.7mmol/l)	Check for ketones, and if negative, may exercise with extreme caution.*
Child with type 1 DM	≥ 250mg/dl (13.9mmol/l)	Check for ketones, and if positive, do not exercise. Treat DKA.
	251-350mg/dl (14-19.5mmol/l)	Check for ketones, and if negative, ok to perform physical activity.*
	> 350mg/dl (19.5mmol/l)	Do not exercise. Hydrate and adjust insulin/dietary regimen.
Adult or child with type 2 DM	≤ 350mg/dl (19.5mmol/l)	May exercise.*
	> 350mg/dl (19.5mmol/l)	Do not exercise. Hydrate and adjust medication/ dietary regimen.

* Continue to monitor blood glucose during activity. If blood glucose ≥ renal glucose threshold of 180mg/dl (10mmol/l), have athlete increase consumption of non-carbohydrate beverage to avoid dehydration.

Table 4.2.4.1 Action plan for hyperglycaemia

and heat can increase the rate of absorption. If possible, the player should try to anticipate the intensity and duration of activity to better adjust insulin dosing and carbohydrate intake. See Table 4.2.5.2 for a guide.

4.2.6 Insulin pumps

Individuals with DM using insulin pumps receive insulin through a continuous subcutaneous infusion. They may have basal insulin settings that are consistent throughout the day (e.g. 1.0 unit/hour for 24 hours) or vary (e.g. 0.5 units/hour from midnight to 6am, 1.2 units/hour from 6am to 12pm, 1.0 unit/hour from 12pm to 5pm, 1.4 units/hour from 5pm to midnight, etc.). In addition, pump users have bolus insulin doses administered prior to meals. Insulin pump settings may be adjusted prior to activity based on trial and error, similar to the injectable insulin guidelines in Table 4.2.5.2.

Depending on the activity and the player's preference, pump users may discontinue the pump during sport and switch to injectable insulin or may wear the pump during activity. If the pump is removed before physical activity, hyperglycaemia is more of a risk and a long-acting injectable insulin should be used appropriately prior to exercise. If the pump is continued during activity, pump malfunction or detachment (e.g. from contact or sweat causing tubing detachment) leading to hyperglycaemia and over-insulinisation leading to hypoglycaemia are both risks. Players who experience hyper- or hypoglycaemia and are wearing insulin pumps should always have their pump addressed at the beginning of the player assessment.

Blood glucose monitoring	- Working glucometer, lancets, test strips - Pre-exercise exclusion values - Recommendations for monitoring frequency
Medication list	- Insulin: type, dose, frequency. Insulin adjustment plan based on activity and correction doses for hyperglycaemia. - Other medication used by athlete
Hypoglycaemia guidelines	- Symptoms and typical blood glucose level of symptomatic hypoglycaemia - Plan for hypoglycaemia: glucose source, amount of glucose, and instructions for glucagon (glucagon injection kit should be readily available)
Hyperglycaemia guidelines	- Symptoms and typical blood glucose level of symptomatic hyperglycaemia - Plan for hyperglycaemia correction - Ketone test strips if type 1
Emergency contact information	- Family member and physician contact information as well as consent for medical treatment
Medical alert tag	- With athlete at all times

Table 4.2.5.1 Diabetes care plan

Activity	Duration	Peaking insulin adjustment
Low, moderate or high intensity	< 30 min.	No adjustment
Low intensity	30 to > 60 min.	Decrease by 5%
Moderate intensity	30 to 60 min.	Decrease by 10%
	> 60 min.	Decrease by 20%
High intensity	30 to 60 min.	Decrease by 20%
	> 60 min.	Decrease by 30%

Table 4.2.5.2 Injectable insulin adjustment guidelines (individual needs vary!)

4.2.7 Daily care of the diabetic football player

For players with type 1 DM, frequent glucose testing is a must. This includes pre-meal monitoring as well as pre-exercise and often during exercise checks. As players become more familiar with their own bodies' responses to exercise, diet and other stressors, glucose checks during exercise may become less frequent. However, as exercise conditions change, such as time of match, duration, intensity and climate, it is important for the athlete to be willing to check glucose levels more frequently. While players with type 2 may not need to check as often as those with type 1 DM, any individual requiring insulin or in newly modified exercise conditions needs to be aware of his/her glucose levels.

Conclusion

General guidelines may be helpful for treating a player with DM, but the most important is the individual player's diabetes information and care plan. The player should keep a log of glucose levels, insulin and other medication dosing, dietary intake and exercise to better understand his/her individual dietary and medication needs with various activities. Such knowledge, with ongoing adjustments, will improve glucose control and help to prevent hypo/hyperglycaemia. In the event of hypo- or hyperglycaemia, the severity and cause of such an episode must be kept in mind when making return-to-play decisions.

Infection	Clinical feature	Complication
Pharyngitis	Inflamed and edematous pharynx, scratchy or sore throat, myalgias, fever	Laryngitis, peritonsillar abscess
Laryngitis	Hoarseness	Epiglottitis, dysphonia, laryngotracheitis (croup), stridor
Rhinitis	Nasal dripping and nasal congestion, often accompanied by sinusitis and pharyngitis	
Sinusitis	Purulent nasal or postnasal drainage, nasal congestion, sinus pain or pressure	Chronic bacterial sinusitis, subperiosteal abscess
Otitis media	Ear pain, fever, decreased hearing acuity, red and bulging tympanic membrane	Mastoiditis, osteomyelitis
Otitis externa	Pruritic and painful ear, canal appears swollen and red	Chronic otitis externa, otitis media

Table 4.3.1.1 Clinical features and complications of upper respiratory tract infections

4.3 Infectious diseases

4.3.1 Management of respiratory tract infections

Infections of the upper respiratory tract

Infections of the upper respiratory tract include some of the most common infectious diseases like pharyngitis, laryngitis, rhinitis, sinusitis, otitis externa and otitis media (Table 4.3.1.1). On the one hand, these infections are often mild and can be treated on an outpatient basis, but on the other, they can cause serious complications, such as peritonsillar abscess from pharyngitis, subperiosteal abscess from sinusitis or osteomyelitis from otitis media. It is therefore important to take these infections seriously, to treat them well, and to allow the body enough time for recovery.

Pathogenesis

Pharyngitis: Most cases of pharyngitis are viral and occur as part of common colds caused by rhinovirus, coronavirus or parainfluenza virus. In 15% of all cases, there is a bacterial cause of pharyngitis (Staphylococcus aureus) that can cause significant complications.

Laryngitis: Acute laryngitis is mostly caused by viruses, but may also be associated with bacteria like Streptococcus species and Moraxella catarrhalis.

Rhinitis: Infection of the mucosal surface of the nose is most commonly due to respiratory viruses like rhinovirus and presents as acute rhinitis.

Sinusitis: Sinusitis is a common problem. The most common precursor is a viral infection of the upper respiratory tract. In only about 1% of cases of acute sinusitis there is a bacterial cause. Other causes of acute or chronic sinusitis can be allergies, dental infections and barotrauma from diving or airplane travel.

Otitis media: Acute otitis media is mainly caused by bacteria like Streptococcus pneumoniae and Haemophilus influenzae. Viruses, either alone or with bacteria, are found in one quarter of cases.

Otitis externa: Acute otitis externa, or swimmer's ear, occurs mostly in the summer and is due to bacterial overgrowth that results in a pruritic and painful ear.

Infections of the lower respiratory tract

Infections of the lower respiratory tract include bronchitis, bronchiolitis and pneumonia.

Pathogenesis

Most cases of bronchitis and bronchiolitis are caused by viruses. Pneumonia is caused by various bacterial species, viruses, fungi and parasites. Thus, pneumonia is not a single disease but a group of specific infections, each with a different epidemiology, pathogenesis and clinical presentation. Most cases of community-acquired pneumonia are caused by the following microbial pathogens: Mycoplasma pneumoniae, Streptococcus pneumoniae, Haemophilus influenzae, Chlamydia pneumoniae, Legionella pneumophila, Moraxella catarrhalis, Staphylococcus aureus, viruses or fungi and oral anaerobes.

These microbial pathogens enter the lungs by one of several routes:

- *Aspiration of organisms that colonise the oropharynx:* Most pulmonary pathogens enter by aspiration from the oropharynx. This happens very often during sleep (50% of healthy adults) and occurs more frequently in individuals with an impaired level of consciousness (alcohol, drugs, strokes).
- *Inhalation of infectious aerosols:* Typically acquired by inhalation of infectious aerosols are tuberculosis, influenza, legionellosis and Q-fever.
- *Hematogenous dissemination from extrapumonal locations:* Infections with Staphylococcus aureus or other pathogens can disseminate to the lungs.

Symptoms and signs

Lower respiratory infections are usually characterised by cough, sputum production, shortness of breath, fever and in some cases pleuritic chest pain. Players with pneumonia often suffer from non-respiratory symptoms like headache and myalgia. Pneumonia is a severe infection.

Prevention of respiratory tract infections

The evidence on the interrelation of exercise and respiratory tract infections is conflicting. It seems that vigorous exercise increases the incidence and severity of upper respiratory tract infections. Other research suggests that regular, moderate exercise reduces the incidence for optimum health.

Preventing respiratory infections means preventing the transmission of potentially contagious agents through good rules of hygiene and some standard behaviour rules. Immunisation against Haemophilus influenzae and influenza can be discussed. The following list summarises some preventive measures:

- Update players' vaccinations when at home and when travelling.
- Check air conditioning/ventilation system for potential contagious material if feasible.
- Minimise players' contact with infected people (coughing, sneezing).
- Players should wash their hands regularly.
- Players should use disposal paper towels and limit hand to mouth/nose contact when suffering from infections.
- Quickly isolate infected team members.
- Players should not use other peoples' drinking bottles or cups.
- Players should wear proper outdoor clothing and avoid getting cold and wet after exercise.
- Practise good recovery routines, including proper nutrition and hydration. There is evidence that carbohydrate ingestion during intense and prolonged exercise reduces stress on the immune system.

Examination and diagnosis

Medical history and physical examination are key. Additional examinations such as bacterial and viral cultures of throat swab specimens, nasal discharge or sputum, leukocyte differential count, C-reactive protein and chest X-rays might be required to confirm diagnosis or initiate appropriate treatment.

Treatment

Viral infections of the upper respiratory tract are treated symptomatically. Reducing the intensity of training or even resting for a couple of days gives the body time to recover. Nasal washes with sodium chloride, nasal decongestants and pain relievers like paracetamol help to reduce the symptoms and facilitate drainage of exudates from the mucosa of the upper airways.

Bacterial infections such as purulent sinusitis should be treated with antibiotics in accordance with the result of a good clinical evaluation and microbiological diagnosis. Epiglottitis that results in major breathing problems (stridor) must be treated immediately with proper medication, preferably in a hospital, since intubation may become necessary.

Viral bronchitis can be treated symptomatically. Cough-reducing medications should be restricted to cases of dry, non-productive coughing. Bacterial bronchitis and pneumonia are treated with antibiotics.

Doping issues

When treating players with respiratory tract infections, the current Prohibited List must always be taken into consideration, as in particular combinations cold medications may contain prohibited substances.

Examples of allowed substances according to the 2009 Prohibited List:

- Caffeine, codeine, diphenyhydramine, guaifenesin, hydrocodone, oxymetazoline, paracetamol, phenylephrine, phenylpropanolamine, pholcodine, pseudoephedrine, steam and menthol inhalations, xylometazoline.

  All antibiotics are allowed.

  Prohibited substances according to the 2009 Prohibited List:

- Stimulants (found in many cold and flu remedies) are included in the list of prohibited substances published by WADA but are only prohibited in competition. Samples taken out of competition are not tested for stimulants. However, when prescribing cold and flu remedies that contain stimulants in the run-up to a competition, it should be noted that these substances remain in the body following use of the product, and a player may therefore still test positive at the competition. Ephedrine and methylephedrine are prohibited when the urine value exceeds a certain threshold.

Return to play

In general, if symptoms of viral illness are generalised, players should not train intensely or play a match. If symptoms are confined to one area (e.g. runny nose, sore throat), they can train moderately.

Guidelines for players on exercise during respiratory tract infections (RTI):

First day of illness:

– Drop strenuous exercise or competitions when experiencing RTI symptoms such as a sore throat, coughing and a runny or congested nose.
– Drop all exercise when experiencing additional RTI symptoms such as muscle/joint pain, headache, fever and a general feeling of malaise.
– Drink plenty of fluids, avoid getting wet and cold and minimise stress.
– Consider the use of topical therapy with nasal drainage, decongestants and analgesics if you have a fever.
– Report illness to a team physician or healthcare personnel and keep away from other players if you are part of a team training or travelling together.

Second day:

– If you have a fever (temp >37.5-38°C) or coughing increases: do not train.
– If no fever or malaise and no worsening of "above the collar" symptoms: light exercise (pulse < 120bpm) for 30-45 minutes, indoors during winter and by yourself.

Third day:

– If fever and RTI symptoms persist: consult your (team) physician at home or at the office.
– If you have no fever or malaise and no worsening of initial symptoms: moderate exercise (pulse < 150bpm) for 45-60 minutes, preferably indoors and by yourself.

Fourth day:

– If you have experienced no symptom relief: do not try to exercise but visit your doctor.
– If this is the first day of improved condition, follow the "return to exercise" guidelines below.

Guidelines for players regarding return to play/ exercise after respiratory tract infections:

1. Make sure that you have one day free from fever and with improved RTI symptoms before returning to exercise.
2. Observe the body's reaction to your first exercise session before starting on a new session the next day.
3. Stop physical exercise and consult your physician if:
   – you experience a new episode with fever or a worsening of initial symptoms;
   – you experience persistent coughing and exercise-induced breathing problems.
4. Use the same number of days to step up to normal training as spent off normal training because of illness.
5. Closely observe your tolerance to increased exercise intensity and take an extra day off if recovery is not satisfactory.
6. Use proper outdoor clothing and specific cold-air protection for airways when exercising in temperatures below –10°C the first week after RTI.

4.3.2 Acute diarrhoea

Diarrhoea is empirically defined as the passage of three or more loose stools per day, which usually is clearly more than average for the individual player. Often this is accompanied by additional symptoms, such as abdominal cramps, faecal urgency, fever, nausea and vomiting.

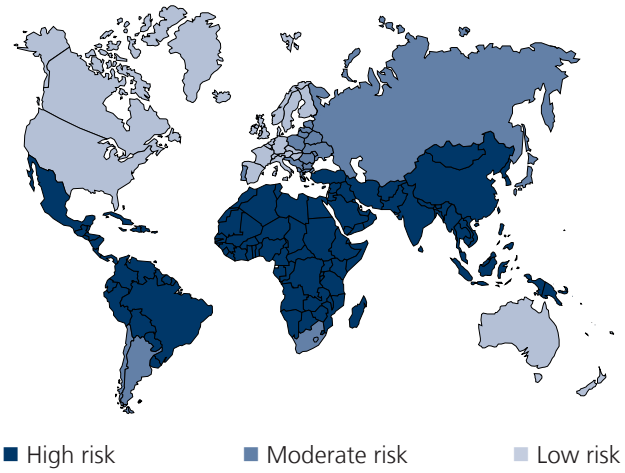


Figure 4.3.2.1 CDC Health Information for International Travel 2008 on diarrhoea

This is quite common, especially among travellers, such as travelling football teams (Figure 4.3.2.1). If a player or players are affected, this may disrupt final team preparations and selections for competitions.

The most common causes of acute diarrhoea are infectious agents or ingested toxins. Diarrhoea is usually caused by faecal-oral transmission of contaminated food or beverages. In contrast, food poisoning is less frequent and due to toxins, e.g. originating from pus in an infected finger.

Food or water may be contaminated by:

- toxins produced by bacteriae before consumption (*Staphylococcus aureus*, *Clostridium botulinum and Bacillus cereus*, scombroid fish poisoning, etc.);
- bacteria, viruses or parasites that have a direct influence on the gastrointestinal tract (*Escherichia coli*, *Salmonella ssp.*, Shigella ssp., Campylobacter ssp, viral gastroenteropathy, hepatitis A and E, and *Vibrio;*
- toxins produced by harmful algae, especially in fish and seafoods (shellfish intoxication).

Incubation period

The occurrence of illness is within a usually short but variable period of time (usually one to two days, but can vary from a few hours to a few weeks) after the consumption of the contaminated food or water. The period is shortest with toxin contaminations. The illness tends to affect a group of individuals who have consumed foods or drinks together and therefore may easily occur in teams dining together.

Prevention guidance for players:

Essentially, the old rule "Boil it, cook it, peel it, or forget it" is still valid.

- Be mindful of the sources of food, milk and other beverages (water!) consumed.
- Avoid ice cream, ice cubes.
- Avoid cold buffets and salads.
- Eat only freshly peeled fruits.
- Eat adequately cooked and preferably hot food (served above 60°C).
- Avoid food from street vendors.
- Wash hands repeatedly.

Treatment

If no rapid cure is needed, fluid and electrolyte replacement are usually sufficient. Dehydration should be limited to a minimum. If the player can eat, tea with sugar, bananas, salted crisps, rice or clear soups are recommended. Alcohol should be strictly avoided.

If rapid cure is paramount, anti-diarrhoeal medication such as loperamide (not be given as only medication to players with bloody diarrhoea) combined with antibiotic treatment offers the fastest relief. Chinolones or azithromycin in regions with widespread resistances (South and South-East Asia) are recommended, as they are effective against most bacterial agents responsible for diarrhoea.

Doping issues

The drugs, if any, that are normally prescribed in the case of diarrhoea (antibiotics, anticholinergic drugs) are not on the Prohibited List.

4.3.3 Malaria

Malaria is the main parasitic disease in humans, affecting more than 500 million people and causing more than one million deaths each year. About 40% of the world's population are at risk of malaria, mostly those living in the poorest countries (Figure 4.3.3.1). In football, due to the wide and rapid travel required by international match schedules, teams from malaria-free areas are exposed to infection. All teams travelling to malaria-endemic countries, even for short periods, may be at risk of becoming infected with malaria.

Infection and transmission

Malaria is a tropical disease which can be transmitted to people of all ages. It is caused by parasites of the

■ Areas where malaria transmission occurs
■ Areas with limited risk of malaria transmission
☐ No malaria

Figure 4.3.3.1 Malaria distribution worldwide

Plasmodium species. This parasite is spread through the bites of infective female *Anopheles* mosquitoes that are active between sunset and dawn. There are four species of *Plasmodium* that affect humans:

- *Plasmodium falciparum* (most likely to result in severe illness and death, Malaria tropica)
- *Plasmodium vivax* (Malaria tertiana)
- *Plasmodium ovale* (Malaria tertiana)
- *Plasmodium malariae* (Malaria quartana)

Incubation period

The minimum period between getting an infective bite and becoming ill with malaria is at least six days. Malaria symptoms can develop as late as several months after departure from a malarious area.

Symptoms and signs

Malaria is characterised by fever and flu-like symptoms, including bouts of feeling cold and shivery, profuse sweating, headaches, joint and body pains, loss of appetite and malaise. These symptoms can occur at intervals. Malaria may be associated with anaemia, and *Plasmodium falciparum* infections can cause seizures, mental confusions, kidney failure, coma and death. If malaria symptoms occur in a player, specialised medical attention should be sought immediately.

Diagnosis

Diagnosis is confirmed by examination of thick or thin blood smears for the presence of malaria parasites. A negative result does not exclude malaria and the test should be repeated if clinically indicated.

Prevention

No effective vaccine is currently available.

- *Protection against mosquito bites.* For effective protection, it is necessary to use a combination of the following measures. Long-sleeved clothing, long trousers and socks should be worn when outdoors after sunset. Light colours are less attractive to mosquitoes. Impregnate clothing with a pyrethroid-containing spray. This will prevent the mosquitoes from biting through the fabric. Apply an insect repellent to exposed skin (hands, face, ankles, etc.). In bedrooms with no air conditioning, a pyrethroid-impregnated mosquito net around the bed should be used and rooms sprayed with knockdown insecticide before the evening to kill any mosquitoes that may have entered the room during the day.
- *Anti-malaria prophylaxis.* This approach is recommended for high-risk areas and it involves the regular, continuous intake of anti-malarial pills. Drug sensitivity and resistance varies from region to region. Team physicians should obtain expert advice before leaving their home country. Options: Mefloquine (Lariam®), Atovaquone + Proguanil (Malarone®), and Doxycycline (Supracyclin®). Start taking prophylaxis one to ten days before visiting a malaria-endemic region and continue for one to four weeks after leaving the malaria area. This allows for the observation and management of adverse reactions, as well as to achieve protective blood levels of the drug by the time of arrival at the destination. Failures arise if drugs are not taken as prescribed. Seek expert medical attention if a player develops a fever while still on prophylactic treatment.
- *Emergency standby self-treatment.* This approach is recommended for areas with a low risk of malaria transmission. If malaria symptoms occur, expert medical attention should be sought immediately. If this is not possible within 24 hours, a player should receive emergency (standby) treatment (anti-malarial drugs, recommended regimes are regularly updated) as a life-saving measure. It is still essential, post-treatment, to have a medical check as soon as possible. Emergency drugs should not be used unless a player has been exposed to malaria for at least six days as this is the minimum period between getting an infective bite and becoming ill with malaria. Often, malaria symptoms occur only after returning home and this demands prompt medical attention. Each player needs to be advised to see a doctor immediately.

Treatment

Specific treatment with anti-malarial drugs is available and should be started within 24 hours of the first symptoms appearing.

Early diagnosis and effective treatment of malaria will shorten its duration and prevent the development of complications and the great majority of deaths. Plasmodium falciparum malaria in particular can be fatal in the absence of prompt recognition of the disease and urgent appropriate patient management. Respiratory, cardiovascular, renal and central nervous system complications occur in severe malaria cases. They are often fatal. The situation is complicated by the increasing occurrence of drug-resistant strains to chloroquine and other anti-malarial drugs.

4.3.4 Hepatitis

Infectious hepatitis is caused by different viruses. It is a systemic infection predominantly affecting the liver. Almost all cases of acute viral hepatitis are caused by one of the following five viral agents which have different means of transmission: hepatitis A virus, hepatitis B virus, hepatitis C virus, hepatitis E virus and hepatitis D virus which is associated with hepatitis B infection. Hepatitis B or C infection may turn, after an acute phase, into a chronic liver disease with cirrhosis and even hepatocellular carcinoma.

Figure 4.3.4.1
Hepatitis A in 2008 wordwide

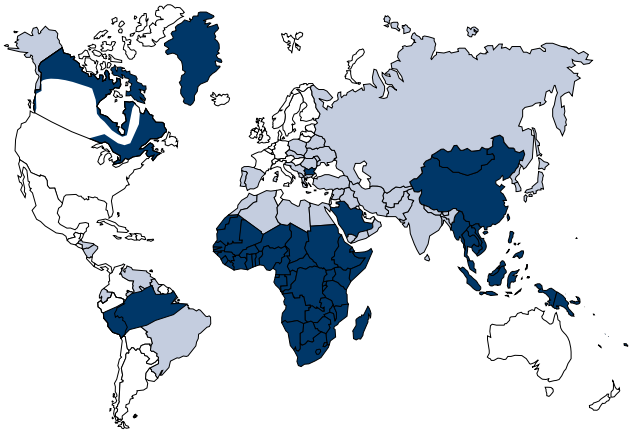


Table 4.3.4.2 Hepatitis B – prevalence of chronic infection with hepatitis B virus 2006

Vaccine	Number of shots and intervals
Hepatitis A	2 injections: first on day 0 and second 6-12 months later
Hepatitis B	3 injections: first on day 0, second 1 month and third 6-12 months later
Hepatitis A+B	3 injections: first on day 0, second 1 month and third 6-12 months later

Table 4.3.4.3 Vaccination scheme hepatitis

Pathogenesis, incubation periods, symptoms and signs

Hepatitis A: This agent is transmitted by the faecal-oral route. Person-to-person transmission is possible as a result of poor personal hygiene or unprotected sex between men, but the main causes of infection are contaminated food, water, milk and shellfish. The incubation period is approximately four weeks (two to six weeks). Infection starts with systemic symptoms like headache, fever, nausea, vomiting and anorexia and often leads to visible jaundice within one to two weeks. A hepatitis A infection never turns into chronic disease and complete clinical recovery is to be expected within one to two months (Figure 4.3.4.1).

Hepatitis B: Hepatitis B is transmitted through contact with the blood and body fluids (saliva, tears, seminal fluid, urine) of infected people. Therefore direct contact with blood or other body fluids, particularly sexual contact, is the main risk factor for infection. The incubation period is about eight to 12 weeks. A hepatitis B infection starts with an acute phase that is very often clinically asymptomatic. If symptoms develop into acute disease, they are the same as those of hepatitis A. Progression to chronic hepatitis B depends on the age at the time of infection. If adults are infected, 5-10% develop chronic disease and die prematurely from cirrhosis or liver cancer (Figure 4.3.4.2).

Hepatitis C: The main means of transmission is contact with blood and blood products. Hepatitis C can be transmitted through transfusions but also through other percutaneous routes like tattooing and piercing. Sexual transmission is possible, but less frequent than in hepatitis B. Very often the exact route of infection is unknown. The incubation period is two to ten weeks.

Hepatitis E: The virus is spread mainly through faecal contamination of water supplies or food; person-to-person transmission is uncommon. It is prevalent in most developing countries with low sanitary standards, and is common in

many countries with a hot climate. In its clinical course, hepatitis E is comparable to hepatitis A, as it never turns into a chronic disease, though hepatitis E occasionally develops into an acute severe liver disease.

Treatment

Acute hepatitis can only be treated symptomatically. Chronic disease needs special treatment with drugs like interferon and antiviral drugs such as telbivudine for hepatitis B or pegylated interferon alfa and ribavarin for hepatitis C.

Prevention

- Good personal and food hygiene ("cook it, boil it, peal it or leave it!").
- Safer sex.
- Vaccination against hepatitis A and B for players/teams and delegations that travel frequently, and risk groups. There is no vaccine available against hepatitis C, D or E (Table 4.3.4.3).

Links:
www.cdc.gov/STD/treatment/2006/hepatitis-a.htm
www.cdc.gov/STD/treatment/2006/hepatitis-b.htm
www.cdc.gov/std/Treatment/2006/hepatitis-c.htm

4.3.5 Tuberculosis

Overall, one third of the world's population is currently infected with tuberculosis (TB). The disease is caused by bacteria *(Mycobacterium tuberculosis)* and usually affects the lungs *(pulmonary tuberculosis)*, although in up to one third of cases other organs are involved (*extrapulmonary tuberculosis*, *e.g.* the central nervous system, the lymphatic system, the genitourinary system, the gastrointestinal system, bones, joints and skin). If properly treated, tuberculosis caused by drug-susceptible bacteria is curable in virtually all cases. The World Health Organization estimates that the largest number of new tuberculosis cases in 2005 occurred in South-East Asia (Figure 4.3.5.1). However, the estimated incidence rate in sub-Saharan Africa is nearly twice that of South-East Asia, at 300 or more cases per 100,000 of the population.

Transmission

Tuberculosis is a contagious disease. Like the common cold, infection is usually by direct airborne transmission from an infected person to an uninfected person. Bovine tuberculosis is transmitted by consuming raw milk from infected cattle.

Only patients who have open lung tuberculosis are infectious, particularly if they cough, sneeze, talk or spit. As long as players do not have open tuberculosis, they are not contagious. If a player is contagious, he has to be temporarily removed from the team and treated until it has been demonstrated that he is no longer contagious. If a team has been in prolonged contact with a contagious player, contact tracing must be performed.

From infection to disease

The incubation period varies from months to years. People of all ages can contract TB, but the risk of developing TB is highest in children younger than three years old and in older people. Those infected will not necessarily become sick with the disease. The initial infection usually remains unnoticed. Some 5-10% of people who are infected become sick at some time during their life. If the immune system is weakened, the chances of developing the disease are higher. Clinical illness directly following infection is named primary tuberculosis and is common among children. Although this form is often severe, it is usually not transmissible. Mostly the immune system "walls off" the bacteria, which can lie dormant for years before being reactivated to produce secondary tuberculosis, which is often infectious.

Clinical manifestations

- *Pulmonary tuberculosis:* early in the course of the disease, the symptoms are often non-specific. They consist mainly of fever, night sweats, weight loss, anorexia, general malaise, weakness and, in the majority of cases, a chronic

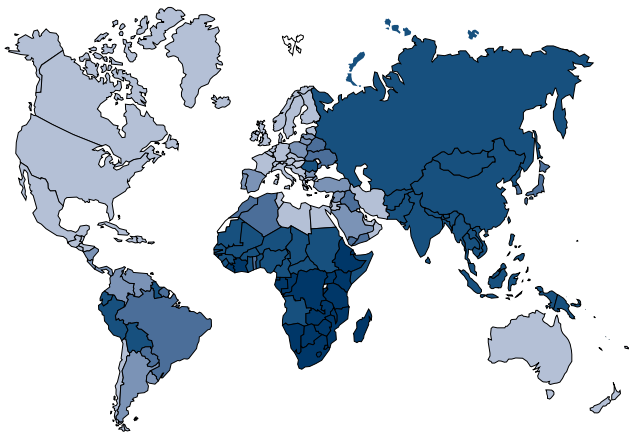


Figure 4.3.5.1 Tuberculosis worldwide in 2005 according to WHO (www.who.int/ith/maps/tuberculosis_2008.jpg)

cough develops. Bloody expectorations must always be considered as infectious.

– *Extrapulmonary tuberculosis:* this most commonly involves organs like the lymph nodes, the pleura, the genitourinary tract, bones, joints and the meninges.

Treatment

The standard treatment is a six-month course of combined drugs. It is important that the medicines are taken regularly during the required period. Inconsistent or partial treatment increases the risk of drug-resistant tuberculosis developing.

MDR-TB (multi-drug-resistant TB) describes strains of tuberculosis bacteria that are resistant to at least the two main first-line TB drugs – isoniazid and rifampicin. XDR-TB, or extensive-drug-resistant TB (also referred to as extreme drug resistance) is MDR-TB that is also resistant to three or more of the six classes of second-line drugs.

Prevention

By far the best way to prevent tuberculosis is through rapid diagnosis of infectious cases and appropriate treatment until cure. Avoid exposure to known open tuberculosis patients in crowded environments (e.g. hospitals or prisons). Additional strategies include BCG vaccination and preventive treatment of persons with latent tuberculosis and a high risk of active disease.

BCG vaccine is recommended for routine use at birth in countries with a high risk of TB, i.e. in most developing countries. In other countries, the vaccination is no longer routinely recommended.

This vaccine has a documented protective effect only in small children against meningitis and disseminated TB. It neither prevents primary infection, nor, more importantly, reactivates latent pulmonary infection, the principal source of bacillary spread in the community.

Links: www.who.int/immunization/topics/tuberculosis/en/index.html
www.who.int/immunization_monitoring/diseases/tuberculosis/en/index.html
www.who.int/mediacentre/news/notes/2006/np23/en/index.html

4.3.6 Sexually transmitted infections (STIs)

More than 20 infectious organisms can be transmitted through sexual activity. STIs are among the most common infections, with an estimated 340 million infections annually worldwide. STIs can often result in serious long-term complications. Infections caused by Chlamydia trachomatis are the most frequent sexually transmitted diseases, but no single STI can be regarded as an isolated problem because the presence of one STI denotes high-risk sexual behaviour that is often associated with other infections. Most STIs are rarely, if ever, transmitted by food or casual contact. At least one sexual partner is always infected.

Cause, transmission, symptoms and signs

Infection occurs during unprotected sexual intercourse. Many infected people are asymptomatic or have mild symptoms and transmit the infection. Hepatitis B, HIV and syphilis may also be transmitted in contaminated blood and blood products (Table 4.3.6.1).

Treatment

Bacterial infections like syphilis or gonorrhoea can be treated with antibiotics. It is therefore important to identify all the sexual partners of the infected person to see whether they are also infected. This is important from a therapeutic but also from an epidemiological perspective.

Infections caused by viruses like HIV or herpes can only be treated symptomatically. There is no causal therapy to eliminate the infectious agent although there are effective antiviral therapies nowadays.

ST agent	Clinical feature/syndrome
Chlamydia trachomatis	Urethritis/cystitis, epididymitis, pelvic inflammatory disease, ulcerative lesions of the genitalia, proctitis, arthritis
Neisseria gonorrhoeae	Urethritis, cystitis, epididymitis, pelvic inflammatory disease, proctitis, arthritis
Treponema pallidum (Lues/ Syphilis)	Ulcerative painful lesions of the genitalia, proctitis
Trichomonas vaginalis	Urethritis (male), vulvovaginitis (female)
Haemophilus ducreyi	Ulcerative painless lesions of the genitalia
Herpes simplex viruses	Urethritis/cystitis, proctitis, ulcerative lesions
Human papillomavirus	Genital and anal warts, dysplasia and cancer of the cervix, anus, vagina or penis
HIV	AIDS

Table 4.3.6.1 Clinical features of STIS

Prognosis

STIs can often result in serious long-term complications if left untreated. Viral infections result in chronic disease that can only be treated symptomatically.

Prevention

Prevention and control of STIs is best done by reducing the average risk of sexual exposure.

Advice to players

- Reduction of the average rate of partner change. Avoid multiple sexual partners.
- Reduction of transmission through safer sexual practices such as correct and consistent use of condoms.
- Pre-exposure vaccination against hepatitis A and B. A vaccine against human papillomavirus (HPV) is available for females only. No vaccine exists for any of the other STIs.
- Shortening of the duration of infectivity of STIs through early detection and effective treatment.

Medical injections using unsterilised equipment are also a possible source of infection. If an injection is essential, the traveller should try to ensure that the needles and syringes come from a sterile package or have been sterilised properly. For players under medical care who need frequent injections, the physician should carry sufficient sterile needles and syringes and a doctor's authorisation for their use. Unsterile dental and surgical instruments, needles used in acupuncture and tattooing, ear-piercing devices and other skin-piercing instruments can likewise transmit infection and should be avoided.

Annotation

Some countries have adopted entry and visa restrictions for people with HIV/AIDS.

4.3.7. HIV/AIDS and football

Introduction

The prevalence of the human immunodeficiency virus (HIV) and acquired immune deficiency syndrome (AIDS) is increasing rapidly worldwide, without distinction as to race, age, gender or socio-economic status. The socio-economic effects of this epidemic have caused the reversal of human development worldwide. The majority of cases are in sub-Saharan Africa, followed by South-East Asia and Eastern Europe (Figure 4.3.7.1). The HIV/AIDS epidemic has had the most profound impact on the most important source of

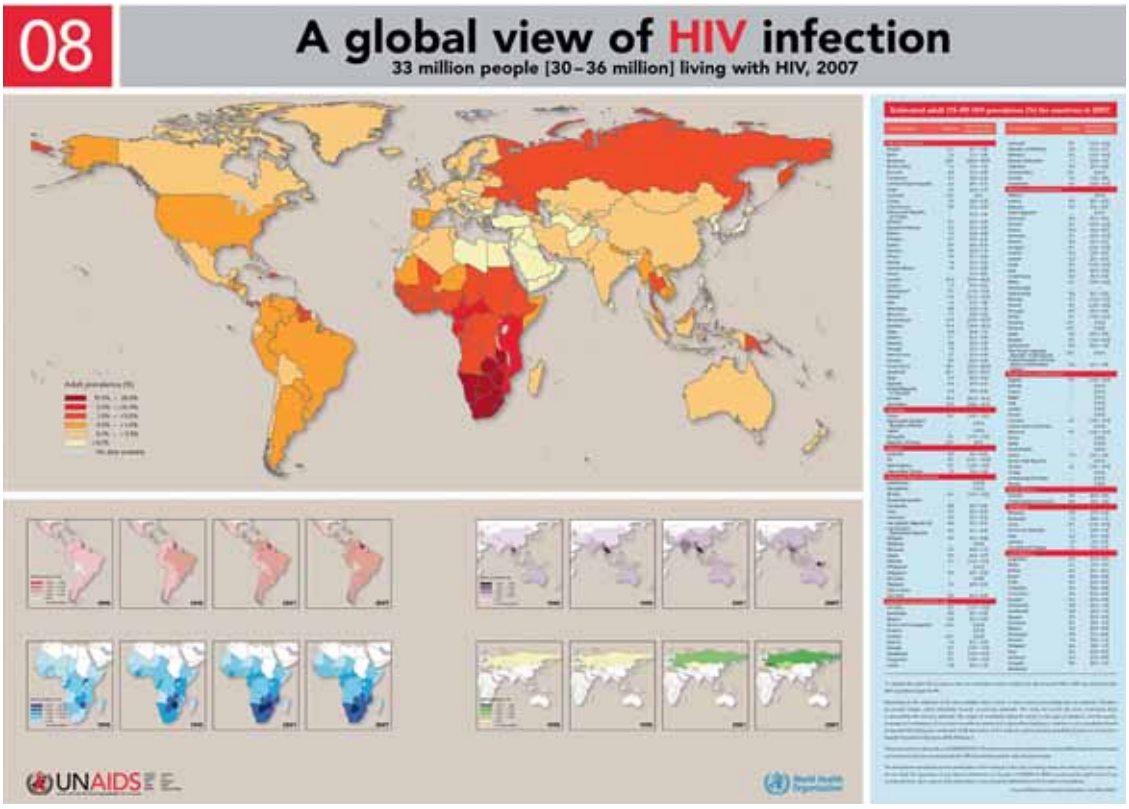


Figure 4.3.7.1 The global distribution of HIV infection

football manpower – the 15-49 age group. HIV/AIDS has caused the illness and death of football players, referees, coaches, managers, administrators and fans, and it threatens the development of football.

Pathogenesis

There are two strains of human immunodeficiency virus (HIV): HIV-Type1, the more virulent strain, and HIV-Type 2. HIV is transmitted through the exchange of contaminated body fluids from an infected to an uninfected person through unprotected sex, sharing intravenous needles (to self-inject vitamins, hormones and other performance-enhancing drugs, for example), mother's breast milk, and exposure to blood. The body fluid must contain a sufficient virus load for the infection to occur. Fluids that contain a threshold virus load include blood, semen and vaginal fluids. Sweat, the most commonly exchanged body fluid during participation in football, saliva, tears and urine do not pose a risk of HIV infection. HIV is not transmitted through mosquito bites, hugging, touching, toilet seats or sharing food or drinks.

The interval between initial exposure to HIV and the detection of antibodies to HIV in the blood is known as the "window period". The infected individual is capable of transmitting the virus to others through high-risk behaviour during this period, though he will test negative to the HIV antibody test (false negative). The body responds to HIV infection by producing antibodies to the virus. Blood-borne antibodies to HIV reach detectable levels in about one to three months following infection, although this period may extend to six months. The diagnosis is made by testing an individual's blood for the presence of antibodies to HIV.

Symptoms and signs

Individuals infected with HIV can live with the virus for years without having any symptoms. However, once the virus multiplies and spreads in the body, an infected person may, among many other symptoms, suffer from high fevers, skin rashes, become easily exhausted, and exhibit neurological and psychiatric manifestations.

HIV eventually weakens the immune system to such an extent that the body cannot protect itself against infection. The infected person develops multiple opportunistic infections and sometimes cancers. This fatal stage of repeated multiple infections and possibly cancer is known as the acquired immune deficiency syndrome (AIDS). In order to have AIDS, a person's T-cell count must be under 200.

Infected persons do not die from HIV but from the opportunistic infections and cancers.

Influence on performance

During the early stages of HIV infection, an individual is well enough to undertake football activities, and there is no reason for them to be excluded from participating in football. Regular moderate exercise has proven to be beneficial to people infected with HIV by delaying the onset of AIDS. Moderate football exercise boosts the body's immunity, provides infected persons with psychosocial support and renders them more capable of fighting the disease.

After the virus has sufficiently multiplied in the body, individuals start developing frequent illnesses that make it impossible for them to routinely continue with football activities. Frequent absenteeism of players and essential technical staff from training and competition due to their illness leads to inadequate preparation of teams, resulting in poor competitive performance.

It is common, particularly in areas with a high HIV prevalence, to find HIV-infected individuals participating in all forms of football activities without knowing that they have the virus. When a player begins to fall sick more often than before, missing training sessions. and his form goes on a steady decline, team management should counsel him and include HIV infection as one of the conditions to investigate as part of his evaluation.

Undertaking frequent strenuous exercise may lead to faster progression of the infection to AIDS in HIV-infected persons. At the stage of AIDS, individuals are too ill to play football.

HIV transmission in a football environment

This is an issue of great concern to all football personnel. According to available literature, there is no definite case of HIV transmission occurring within a football setting. To date, HIV-infected football players have contracted the virus outside the football setting, mostly as a result of their lifestyle, particularly through unprotected sex. However, the available literature has been compiled from ideal football settings.

In much of the developing world, however, football is practiced under conditions which are far from ideal. Playing surfaces are poor and are a source of bleeding injuries, there is inadequate supply of medical equipment and supplies, and the number of adequately trained medical personnel to attend to players during training and competition is insufficient. Poor personal and medical hygiene is rampant. Added to the prevailing high rate of HIV infection in the population, this poses a real threat of HIV transmission in a football setting.

Whatever the level of risk of HIV infection in football, deliberate efforts must be made to minimise it based on determining the situations in which HIV is most likely to be transmitted. It is the responsibility of football managers to ensure that conditions for playing football are safe and that appropriate personnel and medical supplies are available to adequately manage open football injuries in both training and competition sessions.

Prophylaxis

HIV infection can be prevented in different ways and at different levels.

Voluntary counselling and testing

HIV is transmitted from an infected to an uninfected person. The ideal approach to preventing HIV transmission is for everyone to know his/her HIV sero-status. Confidential voluntary counselling and testing (VCT) for HIV is encouraged for all members of the football community. HIV-negative players should be encouraged to remain negative by observing the known HIV infection prevention methods and practices. HIV-positive individuals should be encouraged to live positively and avoid infecting others.

ABC strategy

The simple (ABC) strategy for the prevention of HIV transmission – **A**bstinence from sex, **B**e faithful to one sexual partner or use a **C**ondom – has been effective in reducing the number of new HIV infections in several countries. The practice of safe sex using condoms limits the spread of the HIV virus between sexual partners through semen and vaginal fluids.

Prompt and proper treatment of sexually transmitted infections

Sexually transmitted infections cause small wounds in the skin of the perineum. The broken skin makes it easy for the HIV virus in vaginal fluids to enter the body. Prompt and proper treatment of sexually transmitted infections preserves the integrity of the skin in this area, making it difficult for HIV to enter the body.

Proper medical hygiene/observance of universal precautions

Proper medical hygiene and the observance of universal precautions during and outside football activities reduces the risk of HIV infection through contaminated medical instruments and open wounds. Any visible body fluid, particularly blood, should be assumed to be infected with HIV and treated as such. Adequate stocks of medical supplies, especially protective gloves, dressings and disinfectants, ensure that proper medical care can be provided.

- Pre-dress any existing skin wound before football activity.
- Clean any bleeding wound sustained during sports activity with appropriate antiseptic and bandage.
- Change any uniforms saturated with blood.
- Decontaminate (using appropriate disinfectants) surfaces and equipment on which blood/body fluids/excretions/secretions have been spilled.
- Discontinue any bleeding player from play until bleeding has been stopped and the wound properly dressed.
- Properly dispose of used instruments and dressings.

Male circumcision

Male circumcision performed by well-trained medical professionals is now recognised as an efficacious intervention for HIV prevention, reducing the risk of acquiring HIV by approximately 60%. Men must not, however, develop a false sense of security and engage in high-risk sexual behaviour which could undermine the partial protection provided by male circumcision. Promoting male circumcision is an important additional strategy for the prevention of heterosexually acquired HIV in men.

Post-exposure prophylaxis (PEP)

Post-exposure prophylaxis is given to prevent HIV infection after accidental exposure to contaminated body fluids. PEP is an antiretroviral drug treatment that is started immediately after someone is exposed to HIV. The aim is to prevent HIV from becoming established in the body while allowing the person's immune system a chance to provide protection against the virus.

In the case of accidental exposure, for example bleeding wounds following a head collision between two players, first aid should be given immediately after the injury, cleaning the wounds with disinfectant and washing skin sites exposed to blood or body fluids with soap and water. The exposure should be evaluated for potential to transmit HIV infection (based on body substance and severity of exposure). Confidential testing of the involved players for HIV antibodies (HIVAb) should only occur after obtaining informed consent, and should include appropriate counselling and care referral.

There is a high risk of HIV transmission if the body fluid in question is blood and one of the players (the source) is HIV positive. In this case, PEP is recommended for the exposed player. The medication needs to be taken as soon as possible, definitely within 72 hours of exposure to HIV. If left any longer, it is thought that the effectiveness of the treatment is severely diminished.

PEP usually consists of a month-long course of two or three different types of antiretroviral drugs. As with most antiretrovirals, these can cause side effects such as diarrhoea, headaches, nausea/vomiting and fatigue. Some of these side effects can be quite severe and it is estimated that one in five people give up PEP treatment before completion. It is advisable to carefully consider the toxicity of PEP against its efficacy before administering it.

PEP is considered the very last resort in HIV prevention and should only be used when all other methods of HIV prevention have failed. Prevention of exposure to HIV remains the most effective measure to reduce the risk of HIV transmission. To date no effective vaccine against HIV has been developed.

Treatment

The use of antiretroviral (ARV) therapy in combinations of three or more as HIV treatment has dramatically improved the quality of life of people with HIV and prevented them from dying early.

Currently available drugs do not cure HIV infection but they prevent the development of AIDS. Antiretroviral drugs interfere with the way HIV makes copies of itself and the way it spreads from cell to cell. This stops the virus from damaging the immune system, but these drugs cannot eliminate HIV from the body. Hence, HIV-infected people need to continuously take ARVs. Combination ARV therapy, i.e. taking at least three medicines with different methods of action at the same time, helps the body's immune cells – most notably the CD4 cells – to live longer and provide the body protection from opportunistic infections.

With good and continued adherence to ARV treatment, the progression of HIV in the body can be slowed down and almost halted. Increasingly, people living with HIV are kept well and productive for very extended periods, even in low-income countries.

Comprehensive management of an infected football player

HIV infected players should have their sero-status treated confidentially. They should not be restricted from participating in football activities or in any other way.

HIV negative players should enjoy football activities without fear of the risk of HIV infection from HIV infected players. Coaches and football players should be educated on how to prevent the spread of HIV by practising safe sport. In environments where there is a high prevalence of HIV infection, procedures and guidelines to prevent accidental exposure to HIV infection should be reinforced.

Comprehensive care for a player living with HIV/AIDS includes, but is not limited to:

- Provision of antiretrovirals
- Available and accessible voluntary counselling and testing services
- Prevention and treatment of TB and other infections
- Prevention and treatment of HIV-related illnesses
- Prevention and treatment of sexually transmitted infections
- Good nutrition and adequate physical activity
- Social, spiritual, and psychological peer support
- Reduction of stigma and discrimination associated with HIV/AIDS

Travelling football parties

High-risk behaviour that includes excessive alcohol consumption, unprotected sex, violent behaviour and reckless driving can expose the travelling party to the risk of HIV infection. Travelling teams should seek information and expert advice on the prevalence of HIV in the population at their destination. That information will warn them about the high chances of HIV infection through any high-risk behaviour. The team should be reminded to carry condoms and to use them whenever necessary.

Excessive alcohol consumption usually leads to impaired responsibility and judgement. In that state people can easily forget all they know about precautions against HIV infection. Travelling parties need to look our for one another so that they can save an irresponsible member from dangerous temptation. Travelling with well-equipped first aid kits can save the day when a bleeding injury occurs while far away from adequate medical facilities.

The role of football in the global fight against HIV/AIDS

Popular football events are organised at all levels from local leagues to the FIFA World Cup™, and involve people across gender, age, and socio-economic strata. There are many great players who can make excellent role models for promoting good practices for preventing HIV infection. Football enjoys wide electronic and print media coverage through which behaviour change messages for HIV prevention can be delivered to the billions of football players and fans across the world. With all these resources at its disposal, football can make a great impact in reducing new HIV infections and arresting or reversing the global HIV/AIDS epidemic.

4.3.8 Mycotic foot infections

Mycotic skin diseases are caused by dermatophytes. Dermatophytes are fungi that cause infections of the skin, hair and nails. They can be divided into three main genera:

- Trichophyton
- Microsporum
- Epidermophyton

Infection most commonly affects the feet and is often chronic. The groin is the next most commonly involved area in mycotic infections. Dermatophyte infection of the scalp is rare. Males are affected more frequently than females.

Cause and clinical features

Among athletes and players, mycotic infection of the foot (tinea pedis, athlete's foot) is most common. It is characterised by variable erythema, scaling, pruritus and occasionally vesiculation, especially in the space between the fourth and fifth toes. The infection may extend all over the foot. Infection of the nail (tinea unguium) occurs in many patients with tinea pedis and is characterised by opacified, thickened nails and subungual debris.

Athlete's foot is a communicable disease. Transmission occurs via contact with the mycotic agent, either through direct contact with skin lesions of infected people or, as is more often the case, indirectly via contaminated floors (showers, changing rooms), uniforms or towels. Infections can easily spread from one member of a team to another, especially in situations where uniforms are shared or there is poor personal hygiene.

Treatment

Both topical and systemic therapies may be used to treat dermatophyte infection. Treatment depends on the site involved and the type of infection. Topical therapy is generally effective in uncomplicated tinea pedis. A powder or cream is applied to the affected body part. Topical preparations include Ketoconazol, Miconazol and Clotrimazole. Systemic treatment is needed for tinea unguium. Treatment should be continued until clinical examination and culture results confirm that the patient is free of infection, as otherwise there is a high relapse rate. Prevention is another most important key to avoiding further infections.

Advice to players: prevention of mycosis

- Clip your toenails regularly and wear well fitting shoes.
- Avoid chronic pressure marks.
- Keep your feet clean and dry them well after showering.
- Change your socks regularly.
- Wash towels, socks and uniforms in hot water (at least 60°C) especially when you have a mycotic infection.
- Do not share uniforms, socks or shoes.
- Observe strict personal hygiene, e.g. wear flip-flops in the shower.
- Ensure general cleanliness of dressing rooms and showers.

5. Football for Health

5. Football for Health

Health is our most precious possession and one of the fundamental rights of every human being. Nevertheless, whilst in some countries of the world the average life expectancy is over 80 years, in other countries, notably those in Africa, South America, Oceania and Asia, life expectancy may be below 40 years with every second person dying as a child.

For more than a decade, F-MARC has contributed an ever-growing body of knowledge on the numerous ways in which science and medicine contribute to developing the game of football. This Football Medicine Manual represents a synopsis of this knowledge that is practically applicable to football players worldwide and is intended not only to improve standards of care but also to establish effective prevention of injuries at all levels of play.

Over the years it has spent conducting research into football, F-MARC had come to realise that delivering health education in a football environment has several unique advantages. First, interest in football transcends, for example, social status, gender, religion, culture, and language. Second, football has minimal equipment requirements, so there are no barriers to participation. Third, football teams can operate independently of governmental and non-governmental organisations and resources. Fourth, football is full of metaphors and analogies that can make learning about health much more memorable. Finally, football unites people from all backgrounds in an activity that they trust, respect and enjoy; this creates an ideal environment in which to help people learn to keep healthy as individuals and communities.

F-MARC, GrassrootSoccer and coaches of pilot study "The 11 for Health in Africa"

Above that, playing football as a leisure activity could be an extremely effective disease prevention concept on its own. The positive effect of physical exercise such as jogging, walking, cycling and strength training in reducing risk factors for many diseases has been proven extensively, offering an enormous preventive potential in public health. It has further been shown that exercise can in fact be prescribed as therapy for the treatment of diseases such as obesity, diabetes, hypertension, heart and pulmonary diseases, muscle, bone and joint diseases, cancer and depression.

But, different from most interventions, and therefore of potentially far higher value, football is fun, and not a tedious and strenuous obligation. Promotion and motivation will therefore be much easier to achieve than with many other healthy lifestyle campaigns. And, as another vitally important point with a view to universal applicability, football infrastructure and organisations exist in virtually all countries in the world. Furthermore, the game can be played everywhere with very little equipment requirements. Both these factors offer a unique perspective with regard to the costs of education and training in prevention.

Both aspects of football, either as an educational tool or a health-promoting exercise, have been explored to some extent by a number of non-governmental organisations and also governmental institutions. However, a systematic approach including an assessment of the actual outcome of such measures is missing so far. World football's governing body FIFA is in an ideal position to carry out such a task.

F-MARC therefore decided to comprehensively explore how football can contribute to promote health, particularly public health, worldwide on a broader scale. All of the above-mentioned issues are to be addressed in a joint research approach with the final aim of developing a universally applicable and comprehensive "Football for Health" programme, the effects of which have been evaluated according to scientific standards. It is clear that such a universal programme will have a different focus in different regions of the world.

Children of the pilot study "The 11 for Health in Africa"

5.1. Assessment and reduction of risk factors for non-communicable diseases

Football as a regular physical activity has shown very promising results with regard to body composition, fitness and health in male players. So far, no studies have examined the health effects of football in persons with cardiovascular risk factors. Therefore, a set of studies addresses the effects of regularly playing football on both men and women with known risk factors for cardiovascular disease and overweight children in Europe. The results of these studies will provide important information on the motivational effect of football as a specific preventive approach and on how much effort is required from players who not only want to have fun, but also improve their health by practising their favourite sport, including the influence of football on cardiovascular risk factors.

However, non-communicable diseases such as diabetes, hypertension and cardiovascular diseases are continuously on the rise in developing countries too. The increasing prevalence of cardiovascular risk factors in sub-Saharan Africa is becoming a real threat to economic investment, adversely impacting on all the previous gains made in combating infectious diseases. F-MARC therefore supports a baseline survey of the main risk factors for the major non-communicable diseases in Uganda. The results of this study will provide important information on the true extent and risk of non-communicable disease in Uganda

and will pave the way for targeted prevention programmes for those most in need.

5.2 "The 11 for Health" in Africa

The major focus of F-MARC's Football for Health initiative however is Africa. Extreme poverty, hunger and infections are striking at the continent's very foundation. The "Big Three", namely HIV/AIDS, malaria and tuberculosis, kill three million people in Africa every year. However, most infectious diseases and conditions in Africa are preventable, treatable or both. The Football for Health programme delivers health education to the youth population of Africa in a football environment in order to improve their knowledge, attitudes, skills and behaviour towards all diseases, and consequently reduce the future incidence of disease. Each of the eleven simple messages on prevention is underpinned by a strong theoretical background. The messages are intended to be spread and delivered in a manner that is age- and gender-appropriate, culturally sensitive and, most importantly, engaging and sustainable.

To evaluate the acceptance of the educational material and the feasibility of the project, a pilot study is being conducted in South Africa.

"The 11 for Health" messages are:

1. Play football
2. Respect girls and women
3. Protect yourself from HIV
4. Avoid drugs and alcohol
5. Use treated bed nets
6. Wash your hands
7. Drink clean water
8. Eat a balanced diet
9. Get vaccinations for yourself and your family
10. Take your prescribed medication
11. Fair play

If proven to be effective in this study, "The 11 for Health" will be promoted during the 2010 FIFA World Cup South Africa™ and then implemented in all African countries.

Launch of "The 11 for Health in Africa" pilot study at a school in Kayelitsha, Cape Town

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