Football Traumatology

Current Concepts: from Prevention to Treatment

Piero Volpi (Ed.)

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Foreword by:
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Springer

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Foreword

This anthology of Football Traumatology edited by Dr. Piero Volpi comprises the collective experience of many of the leading surgeons in Europe whose practice includes footballers at all levels. By pooling this experience and clearly defining common problems and areas of current interest Professor Volpi has produced a very useful reference source for orthopaedic surgeons, sports physicians and physiotherapists. Future editions may expand on emerging areas in non-surgical fields such as dietary supplements and the effects of psychological factors on both injury itself as well as recovery. Topics in this first edition such as Epidemiology, Training Methods, Prevention, and Future Treatment are welcome adjuncts to the usual subjects and reflect recent areas of research.

Neil P. Thomas
President ESSKA 2000
Winchester
UK

With football becoming ever more relentless and intense, the role of the sports doctor has become in the last few years increasingly important in protecting footballers' health and their physical integrity. It is in this context that Dr. Volpi's in depth and wide ranging work must be viewed. His attention has focused specifically on two important aspects: the fight against doping and the prevention of sports traumas. Together with his scientific experience as a sports clinician and traumatologist, Dr. Volpi can also draw on his direct field experience as a football player, which adds considerable value to the editing of the current volume, intended as a service to football. With the Football World Cup just round the corner, this book produced in collaboration with both Italian and foreign experts is particularly topical: I am convinced that the spread of medical-scientific knowledge and innovations is the most efficient way to safeguard the health of athletes and encourage the growth of a "better" football.

Franco Carraro
President of the Italian Football Federation

This wide editorial initiative, conceived and realized by Dr. Piero Volpi, a precious and not replaceable AIC consultant, brings an important scientific contribution to medical questions connected to football.

It is noticeable in the work the importance of the Author's direct experiences, first as players and as sport and traumatic doctor then.

AIC has always been strongly engaged in safeguarding the players' health, real protagonists of the sport event, and has, between his leading aims, the fight against doping and the traumas prevention.

The information, the knowledge and the education are fundamental for players who want to successfully face the fight to defend their physical integrity and professional dignity.

This praiseworthy editorial effort goes exactly in the direction wished by AIC and will be useful for all the football component to enrich their cultural heritage but especially to know how important is the work done by who acts in order to improve sport anyhow.

Sergio Campana
President AIC
Italian Footballers Association

As an F.C. Internazionale executive, and through dealing with the topic in UEFA and FIFA Commision meetings, I have been able to closely follow Piero Volpi's work in these past ten years. I have come to appreciate the preparation and ethics of a person who was "born" on the pitch and has since been through many experiences, first as footballer and later as a sports doctor. This interesting book is the result of an analysis of these two lives that run parallel, but which are not always necessarily linked in the professional world: theory is verified by practice, research addresses the demands and problems faced at the highest competitive levels.

The knowledge and possibilities offered by sports medicine today are far superior to those in my generation of footballers. Piero Volpi's book is the most recent and vivid example that today's athlete has the moral and professional duty to consider his own health as one of the most important and decisive aspects of his career – at the very level and on each occasion, starting from the basis, from the decisive transition between playing for fun and playing competitively.

Giacinto Facchetti
President, F.C. Internazionale Milano

Preface

Every child has a dream: to play football and become a famous footballer.

When I was 10 years old, my father was used to bring me to San Siro stadium on sunday afternoons to watch the Inter team playing. The great Inter was growing up. Sarti, Burnich, Facchetti… it was an absolute and sudden love.

Twenty years later I was climbing that prestigious stadium through the stairs from the locker rooms wearing the shirt of Como team to play a football match of the first league Italian championship against Inter.

Fifteen years later, I was sitting as a chief physician on the bench of the Moratti's Inter team, Ronaldo, Baggio, etc.

Sometimes dreams come true.

With the present book I accomplish today another wish. To enrich the wonderful world of football with scientific and specialistic contributions on football traumatology, a branch that has undergone important hits and progresses.

Football has constant need of medical, diagnostic, clinical, therapeutic knowledge to counteract the increased number of traumas occurring during sport performance in order to defend the integrity of its players, beeing them professionals, amateurs, young or women.

Thanks to my father, to him my dearest memory, thanks for bringing me to the stadium when I was a child.

Thanks to everybody that I met in the football world: players, team companions and competitors, trainers, managers, physicians, physiotherapists, press reporters, fans; I learned from everyone as in a real "gym of life".

Thanks to the Authors of this book, for their participation and for the high scientific level reached in their chapters.

Thanks to Springer who has been recipient of this idea and was able with great skill to make it real.

Piero Volpi

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Section I
General concepts

Epidemiology and Risk Factor

Piero Volpi

Introduction

Football is the world's most popular sport. According to the International Football Federation (FIFA), there are more than 200 club members in its 203 affiliated nations. The Union of European Football Associations (UEFA) has 23 members in 51 countries while the Italian Football Association (FIGC) estimates that there are 1 million players in Italy, including more than 3,000 professionals. Many players are obviously young, and the number of women footballers is constantly growing.

The greater frequency of accidents in recent years in professional and amateur football [1–3], youth football [4–6], and women's football [7, 8] has been the subject of several researches and publications. Many footballers have had to stop playing and undergo medical treatment and surgery followed by rehabilitation periods before being able to resume playing. Those who have been able and lucky enough to take part in professional soccer know that a second, or even a tenth of a second, is time enough for the occurrence of an accident that may require days, weeks, or months to put right and which results in considerable physical and mental distress. While it is true that markedly improved medical knowledge now ensures faster, more accurate, and more precise diagnosis, it is equally true that little is known about how to prevent injuries and little, indeed, is done to prevent them. Today's professionals must be on their guard against two great threats to their health and their career, namely, doping and accidents. In both cases, an understanding of preventive measures and the timely acquisition of clear, scientifically validated information constitute the first line of defence.

Many accidents are unforeseeable, as they are the natural consequences of a sport in which speed, strength, and explosive action are accompanied by physical contact, tackling, and collisions with an opponent. Many others, however, could be avoided because they stem from mistakes, underestimation of risk factors, or insufficient consideration of the ways in which they can be prevented. The current opinion is that while improvements are continually

sought in a player's physical, technical, and tactical performance, little is being done to prevent and contain the number of accidents.

Epidemiology

Epidemiological investigation of sport injuries assesses their incidence during the course of individual sports. Football gives rise to both sport-associated injuries, namely, those that stem from the specific ways in which it is played, and sport-occasioned injuries, that is, those that occur incidentally during training or the course of a match. The incidence of injuries can be expressed in several ways, including number per season, per match and/or training, per hours of activity, per exposure to a type of injury.

Epidemiological data are used to formulate appropriate preventive measures. In one of our recent studies [2], we collected the 5-year data for a top-level Italian professional team comprising 22–26 players per year. Account was taken of all match or training accidents that obliged a player to remain inactive for 3 or more days. An average of 245 training sessions were held per season (Table 1), and an average of 66 official matches were played (Italian League, Italy Cup, European Cups, same-level friendlies) (Table 2), giving a training-to-match ratio of 3.6:1. This ratio itself shows how little time is devoted to preparation as opposed to playing, especially since sessions prior to a match are devoted to the tactics to be adopted rather than to training while those conducted after it are mainly concerned with *unwinding*. It is also defective insofar as it takes into no account the matches in which many players, both Italian and foreign, take part as members of their national teams. We were the first to introduce this indicator, and it has since been adopted by other workers [9]. This index also helps trainers and coaches to prevent injuries: The greater the ratio of training to matches, the lower the risk of injury.

Table 1. Training sessions per season

Season	Number of sessions
1995/1996	235
1996/1997	248
1997/1998	231
1998/1999	256
1999/2000	243

Table 2. Matches and competitions per season. Numbers in brackets under Italian League indicate additional matches played to reach final league positions

Season	Italian League	Italy Cup	European Cup	Friendly matches	Total
1995/1996	34	6	2	22	64
1996/1997	34	7	12	14	67
1997/1998	34	6	11	18	69
1998/1999	34 (2)	8	10	16	70
1999/2000	34 (1)	8	0	18	61

As can be seen in Table 3, we recorded 335 injuries requiring inactivity for 3 or more days in 5 seasons, with an average of 67 per season. There was no significant difference between the number of accidents in matches and those during training. Compared to the past, this reflects an increase in the number of accidents that occur during training due to the greater intensity during the sessions. With respect to previous studies, there was no difference between the roles of players in accordance with their present characteristics, namely, defence, construction, and attack. There was no difference between the first and the second half of a match. There was a higher incidence of injuries when a team was attacking as opposed to defending. Elite footballers seemed more prone to injury. A study published in 2004 by the FIFAs Medical Assessment

Table 3. Types of injuries: 5 seasons – 1995/2000 (inactive for 3 or more days). Total number of injuries: 335

Kind of injuries	Number of cases	Percentage
Muscle injuries	103	30.7
Bruises	97	28.9
Sprains	58	17.3
Tendinopathies	33	9.8
Backaches	19	5.6
Cuts	11	3.2
Chondropathies	8	2.3
Fractures	6	1.7

and Research Centre (F-MARC) reported the incidence of injuries during the 2002 Korea-Japan World Cup: 171 injuries in 64 matches, corresponding to 2.7 injuries per match and 81 injuries per 1,000 h of playing time. The latter were calculated as 11 players x 90 min x number of returned injury report forms [10]. The incidence of injury in men's international matches ranged from 51 to 144 per 1,000 match hours, that is, 2 to 3 injuries per match [10].

We made a similar survey of the final stages of the 1990 World Cup in Italy in conjunction with the Coverciano Technical Sector [11]. There were 106 injuries, an average of 3 per match.

Risk Factors

Football is usually regarded as a sport in which injuries are the result of contact though in some respects it is similar to collision sports. Its risks have been the subject of several studies [12–18] that cannot be readily compared owing to the differences in their methods, parameters, and aims. A distinction must naturally be drawn between injuries occurring in young players, women, amateurs, and professionals. Consideration of the incidence due to multiple factors is also of importance [19–21].

Professionals, especially those at a high level, are exposed to the risks posed by many official matches, engagements within the course of the week, and overintense and overhasty inaugural seasons that shorten the period of physiological preparation (Table 4). Today, the *business* of a match in professional football is certainly higher than it was in the past. The cost of the *football product* and the corresponding interest of media, as demonstrated by the

Table 4. Risk factors

Direct	Collateral
- Play/technical and tactical aspects	- Age
(Rapid actions/faster play/intensity)	- Previous traumatic events
- Physical/athletic aspects (Increased muscle strength)	- Physical and biological characteristics
	- Life style Smoking/alcohol/diet
- Psycho-physical stress	- Severity of referees
(Number of matches/fatigue/ recovery/travels/business)	- Fair play
- Pitches and shoes	

relevant presence of television at every match, increase the interest and relevance of sports events. Such a type of involvement causes increased pressure and agonistic stress in players, trainers, and managers in order to reach the best possible outcome, even going as far as using unfair play.

Faster play, quicker movements and more intensive action, training methods differing both in quality and the amount of exercise and number of sessions per week, and changes in technique and tactics are other factors. Pressing in several parts of the pitch and offside and double marking are tactics practised with great intensity both in training and during a match and constitute other risk factors. Creation of a very compact or *short* team results in more contact between the players. It is common to see several players engaged in intense and rapid action on small portions of the pitch and hence increasing the likelihood of impacts and collisions.

Both in football and other sports, the essentially physical nature of the actions required and the great strength acquired by the muscles combine to form one of the main causes of both acute injuries (damaged knee ligaments, especially the anterior cruciate, and torn tendons) and chronic injuries (tendinopathies, enthesitis, cartilage disorders) since the joints and their surroundings, the tendons and the nervous control systems, are not always able to resist such an exaggerated concentration of muscular force. Stress and fatigue, too, may upset the workings of the proprioceptors. One of the most complicated questions to which an answer must be found, in fact, is how far stress and fatigue can be responsible for causing an injury.

Football pitches are also a potential source of injuries. Those used for both training and matches should be grassy with level surfaces and free from dangerous structures beside their edges. Matches should be played on similar surfaces. Both the FIFA and UEFA have recently looked into the possibility of using synthetic grass. Its characteristics, in fact, are technologically advanced and very similar to those of natural grass.

A footballer's footwear has always consisted of specially designed boots fitted with studs, bars, and the like to get a firmer grip on the ground. The constraints to the pitch imposed by this type of footwear during running alter both the correct mobility of the hindfoot when the heel strikes the ground and the function of the subtalar joint. The physiological movements of the forefoot during take off are also modified. Soles that assign priority to the maximum grip, as required by today's faster play, promote greater blocking of the ankle and foot coupled with greater vulnerability of the knee. New models fitted with such soles ensure higher grip coefficients with respect to slippage coefficients. Uneven pitches combined with ever more powerful leg muscles and rapid movements increase the risk of injury to muscles, tendons, and joints.

Many injuries are followed by reestablishment of function and hence full resumption of sport but not by total anatomical *restitutio in integrum*, and other disorders may ensue. Tendinopathy is a case in point. This can be cured, but the healed tissue, while functionally effective, has lost part of its elasticity. In the same way, surgical repair of an anterior cruciate lesion reestablishes the functional efficiency of the knee but not its original anatomy.

Many different constitutional factors may play a part even though several footballers have reached very high levels of performance without serious injury despite muscle and bone abnormalities that more readily lead, however, to overuse injuries. The most frequent pictures are lower extremity dissymetries, foot dysmorphisms, and femoral-tibial (varus-valgus) and femoral-patellar misalignments. Age is another factor because bones, muscles, and tendons lose their best features (strength, elasticity, resistance, absorption of knocks and traumas) over the course of time while a long career usually results in knee- and ankle-joint degeneration typical of sport-induced attrition.

Mention can also be made of the importance of fair play on the part of footballers, trainers, and managers, as well as the strictness of referees in defending and safeguarding the physical integrity of the players.

Injurious Mechanisms

Injuries are classed as acute due to overuse or chronic according to their characteristics, the moment when they occur, their type and location, and the mechanisms responsible. Acute lesions are the outcome of a single, usually macrotraumatic, low- or high-energy episode occurring either without contact with an opponent or during a tackle. They are typically fractures, sprains, and muscle or tendon injuries. Pain, generally acute and immediate, is the initial sign, followed by oedema, which may appear in the subsequent hours. Further training or play is often impossible. Immediate examination prior to the onset of oedema and reflex contractures is the best way of securing an already indicative diagnosis.

Overuse injuries, as their name suggests, are the result of repeated microtraumas and often attributable to the stresses associated with a particular sport. They primarily manifest as tendon and cartilage disorders, low back pain, fasciitis, and the like. All tissues may be involved when repeatedly subjected to maximum loads that eventually lead to inflammation, pain, and functional impotence and compel the suspension of sport. Their overture is insidious. After a warm up, indeed, they may be less intense or disappear, only to return as the training session or the match continues and thus become chronic. In about 2 out of 3, they appear without contact, during a swerve, for

example, or slowing down or landing after a jump, and take the form of a knee or ankle sprain or muscle injury. In contact injuries, tackles with the ball at the feet or sliding tackles and elbowing or butting during mid-air collisions predominate. The lower extremities are primarily involved. The knee and ankle are most frequently injured because the stresses and direct and indirect insults imposed by fast, explosive movements expose them to greater risks. The most recurrent knee lesions are sprains, especially of the tibial collateral and anterior cruciate, together with meniscus and cartilage lesions. Anterior cruciate injuries are nearly always indirect, without contact, whereas tibial collateral injuries are usually the result of collision with an opponent. The former are serious and often require surgical reconstruction followed in most cases by 5–6 months of inactivity [22–24].

More than 90% of ankle injuries are sprains, often underestimated in both diagnosis and treatment. All professional footballers have probably had an ankle sprain during their careers [25, 26]. Direct and indirect muscle lesions are numerically substantial [27–29], especially in the thigh, often underestimated, and the cause of troublesome recurrences. Overuse injuries are increasingly common, and bones, cartilages, ligaments, muscles, and tendons may be involved. Typical forms, such as pubalgia, patellar tendinitis, and chronic footballer's ankle, are determined by the subject's biological and physical characteristics, the use and choice of materials, and the stresses inherent to training.

Upper limb injuries, too, are increasing. Here, the goalkeeper is obviously involved because of the use of hands and arms. Clavicle, forearm, and finger fractures, along with dislocations of the shoulder, are suffered also by other players as the result of collisions or heavy falls. Craniofacial lesions are also much more frequent, particularly as the outcome of butting and elbowing in aerial tackles.

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Aspects of Biomechanics

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Introduction

Biomechanical analysis has brought certain benefits to the world of sport, not only because it has improved the knowledge of kinetics and kinematics of each sport, but also because of its practical repercussions on athletic performance, prevention, aimed therapy, sport-specific rehabilitation, and criteria for return to sport after injury. All this is even more important in soccer, both because of its media impact and the high number of players – 200 million registered with the International Football Federation (FIFA) in 186 countries [1, 2].

After the biomechanical studies on soccer in the 1960s and 1970s [3–5] during the 1980s, the biomechanical analysis applied to sport, and to soccer in particular, entered a new dimension and viewpoint thanks to ever-better instrumentation and dedicated personnel [6–12]. In that period in Perugia, we were among the first in Italy to undertake biomechanical studies on basic sport movements with the belief that they were useful not only for improving our scientific knowledge but also for their practical applications. Over the years, this science has become more and more important for athletes, coaches, sports rehabilitators, masso-physiotherapists, sports societies and others since a better knowledge of sport-specific movements, a morpho-functional analysis of the athlete under dynamic conditions, and research on environmental factors (field, equipment, type of training) allow a more specific and individual technical and athletic training, choice of appropriate equipment, and, in case of injury, a science-based rehabilitation in which the biomechanical evaluation of functional parameters play a fundamental role.

Biomechanical studies applied to soccer have allowed improvement in materials and equipment, and therefore, even industry can be an important

ally for scientific research. Obviously, biomechanical analysis needs adequate instrumentation and qualified personnel with the ability to interpret data and correlate them with the subject – the kinesiology and specific movements such as kicking, jumping, running, cutting, stopping, headers, and the like.

Biomechanical Analysis of Soccer

Biomechanical analysis applied to soccer allows the implementation of studies from basic science to practical application with clear repercussions on performance, prevention, and aetiologic treatment. Thanks to biomechanical analysis, it is possible to obtain objective and reproducible data on various aspects of the game:

- Soccer-specific gestures in terms of kinetics and kinematics;
- The involvement of the locomotor apparatus in qualitative and quantitative terms in the various movements;
- The player, both as a morphotype and functional status;
- Equipment, in particular shoes, a subject of great debate concerning the relationship between different field surfaces and the various shoe-cleat configurations;
- Materials, in order to guarantee a good performance and full protection (clothing, ball, goalie gloves, shin guards);
- Training methods, for adequate, individual, sport-specific, technical, athletic, physical, and mental training;
- Environmental factors, meaning not only ideal temperature but especially a regular and homogeneous pitch.

Of the six fundamental movement categories described by Nicholas [13] (stance, walking, running, jumping, kicking, throwing), soccer-specific categories are running, jumping, and kicking. Biomechanical evaluation of these movements can be performed not only in the laboratory but also on the field so as to reproduce "normal situations". Some movements occur during involvement with the ball, such as kicking, dribbling, and headers, while others occur without, such as running, cutting, and quick stops. In actions without involvement with the ball, the player puts himself or herself in position to receive the ball and perform the necessary movement (ball control, volley, first-touch passing, header), trying to obtain the best possible view of the play area, action, and the opponents' positions. Therefore, the running movement in most cases implies a different body position compared with track-and-field athletes.

Kicking is obviously the most important movement in soccer, both as shot on goal and passing shot. The latter should be judged from a biomechanical point of view as a translocation movement performed through the interaction between the foot and the ball. In 1971, Plagenhoef was one of the first to give a quantitative biomechanical description of kicking, having produced a kinetogram to illustrate the skill [4]. When preparing the ballistic action of kicking, the athlete must place himself or herself in order to favour the maximum acceleration of the body segments involved in running and in kicking and thus guarantee a powerful shot and great body balance. This implies adequate body inclination in comparison with the weight-bearing limb in which the foot must be in line with the ball and at a suitable distance. There are discordant opinions in the literature on this distance. In fact, in 1993, McLean and Tumilty reported foot placements in elite junior soccer players of 38 cm behind the ball centre whereas others reported a distance between the foot and the ball from 5 cm to 10 cm [14], and Hay, in 1985, suggested a distance from 5 cm to 28 cm [15]. Impact with the ball occurs during the swing phase of the kicking leg through a precise series of rotational movements. In this movement, the aim is to produce through the kinematic chain of body segments, high angular velocity to the foot [16, 17]. During the kicking movement, the foot rotates both on the transverse and vertical axis of the body [18].

In order to study kinematics of the locomotor apparatus in various movements, more sophisticated motion analysis systems have been used over the years, ranging from traditional optical recording systems (standard cameras provided with slow-motion and still images), cinematographic recordings with high-speed cameras (around 400 photograms per second), to the modern three-dimensional (3D) systems such as SIMI Motion, which we currently use in Perugia. These systems provide the application of external markers on the body surface in well-identifiable bone landmarks such as the iliac crest, greater trochanter, lateral femoral epicondyle, fibula head, fibula malleolus, heel, and head of the 5th metatarsal. These studies are able to provide precise numerical data on the global duration of motion and its phases, on angle variations of the lower limb, on angular and linear velocity, and on velocity variations of the body segments.

In 1991, during the International Congress "Biomechanics and Sport" that we organised in Terni, we presented the results of our biomechanical study on kicking performed on 6 professional soccer players (mid-fielders and/or play-makers) with a mean age of 25 (range 20–28) years and with an excellent technical basis [19]. These players were asked to perform different

types of kicks: inside of foot, inside of the instep, full instep, outside of the instep, and heel. Execution of these movements was recorded with a high-speed camera (400 photograms per second), which allowed a quantitative and qualitative evaluation of the movement, its global duration, and its phases and their duration. The evaluation of muscle electrical activity in the kicking leg was performed with Myosystem, a 16-channel telemetric electromyograph (EMG) that uses surface electrodes, provided with computerised data acquisition, analysis, and processing system. The muscles of the lower limb considered were the anterior rectus, vastus lateralis and medialis, hip adductor, ischio-cruralis, anterior tibialis, sural gemellus medialis, and lateralis. The EMG values obtained were then normalised relative to the maximum isometric activity of each muscle. Finally, we used a piezoelectric accelerometer and a vibration scanner to evaluate all speed variations and accelerations of the lower limb and the leg in relation to the thigh. For this reason, we placed the accelerometer externally over the anterior tibial tuberosity and fixed it to the skin with tape.

Our study gave the following results, which represent the mean value of each kick performed 5 times by every examined player. Evaluation with the high-speed camera allowed the division of the following phases in each kind of kick: run-up/approach, back-swing, wind-up, forward swing, follow-through, and recovery. Mean kick duration was:

- Inside of the foot: 1,540 m/s
- Inside of the instep: 1,720 m/s
- Full instep: 1,135 m/s
- Outside of the instep: 1,740 m/s
- Heel: 1,090 m/s

Furthermore, we calculated the duration of the single phases of each kick, focusing especially on wind-up and follow-through, which turned out to be the shortest but with an abrupt speed variation, as shown by the vibration

Table 1. Mean kick duration

Foot position	Wind-up (m/s)	Follow-through (m/s)
Inside of the foot	155	140
Inside of the instep	315	375
Full instep	245	240
Outside of the instep	265	350
Heel	85	

test. Mean duration results of these phases are shown in Table 1. Therefore, these two phases are potentially the most damaging since muscle, tendon, and joint structures involved must rapidly adapt themselves to these abrupt speed variations, otherwise biological damage, such as tendinopathies, muscle injuries, and chondral lesions, become inevitable.

EMG examination showed a different electrical activity in the examined muscles depending on the type of kick:

- Inside of the foot: maximum activity of sural muscles, hip adductors, and anterior tibialis;
- Inside of the instep: maximum activity of hip adductors, rectus anterior, and vastus medialis;
- Full instep: maximum activity of hip adductors, vastus lateralis, and sural muscles;
- Outside of the instep: maximum activity of rectus anterior, vastus lateralis, sural muscles, and hip adductors;
- Heel: maximum activity of sural muscles, hip adductors, anterior tibialis, and ischio-cruralis.

Therefore, the hip adductors are the most involved muscles in the various movements, and this is an important element to be considered in the aetiopathogenesis of groin pain in soccer players.

In this congress in Terni in 1991, other Authors [17, 20] also presented the results of their kinematic studies on the kicking movement, describing in particular the angular variations and velocities of the 3 most involved lower limb joints, i.e., hip, knee, and ankle. It has thus been observed that in the kicking limb during the back-swing, the hip hyper-extends, the knee flexes, and the ankle is plantar flexed. During the wind-up phase, with an approach speed of up to 100 km/h, the hip flexes while the knee reduces its flexion. Thus, at the moment of impact with the ball, the mean values calculated were a 9° hip flexion, a 41° knee flexion, and a 28° ankle plantar flexion. Furthermore, it has been observed that the best performance is achieved with slightly higher angular values than those reported above and therefore with a more flexed lower limb. In this phase, the ankle is in a fixed position so that the foot and leg form a whole to allow the most effective foot-ball contact and guarantee a different release velocity of the ball, depending on whether the aim is a passing shot, a lob, or a drive, up to a maximum speed of 100–120 km/h. Moreover, it has been observed that accuracy in kicking is the highest when the velocity of the ball is 80% of maximal velocity [21]. It has also been reported that effective performance of movement depends on the connection between the instant mechanical status of the player and the geometric condition of the ball–foot system, especially when the correlation

coefficient is calculated only on the basis of data concerning the best performances. In the following phase, the hip is still flexed, and the knee completes its flexion. Finally comes the recovery phase, in which hip flexion and knee extension are reduced. The weight-bearing limb must allow the other limb's movement and help maintain balance.

Other studies have been carried out on headers and the catch. In the header, the angular velocity of the head depends on the angular movement of the proximal body parts. The catch has been defined as an anelastic contact between the ball and the body, and it is a difficult movement for tall players with long lower limbs because of their reduced ability to amortise the impact between the foot and the ball and of the suboptimal relationship between linear momentum and the extent of bounce. This is due to the bigger body mass of players with long limbs [16]. Juggling is one of the young players' favourite movements and it is to be considered a technical training movement. The goalkeeper is a different issue since the specific movements required are different compared with those of other players, mainly because of the contact between hands and ball (fisting, two-handed catches, dive, punting, and release), which is optimised by footwork and body position.

Biomechanical studies on kicking are extremely important because precise qualitative and quantitative knowledge of the characteristics of every single movement and its performance (correct or incorrect) and identification of the most stressed locomotor apparatus structures provide important information to the trainer when organising schemes and training programs, which, obviously, should take into account morphological and functional individual characteristics, functional status of the player, and level of commitment to the sport. Thus, an optimal mechanical output of the most stressed locomotor apparatus structures and a reduction of damage due to mechanical stress is obtained.

Our laboratory examinations confirmed that hip adductors are the most stressed structures during kicking, cutting, and quick stop. Therefore, an optimal balance between hip adductors and abductors is necessary and can be achieved through an aimed training. General data on movements must, of course, be related to morpho-functional conditions of each player. This is why biomechanical evaluations are necessary to identify altered intrinsic factors (being anatomical and/or functional) and to correct them, thus reducing the risk of overuse pathologies in joints, myotendons, and bones.

The status of the proprioceptive system is one of the most interesting functional parameters examined. In the beginning of the 1990s, we verified the efficacy of proprioceptive exercises on unstable boards for anterior cruciate ligament (ACL) injury prevention in soccer players. If we add a precise

proprioceptive training program of increasing difficulty on unstable boards (rectangular, round, or multi-plane) to the traditional technical athletic training before and during the competitive season, it is possible to reduce non-contact ACL injuries in a statistically significant manner. In fact, we have followed 600 players in 20 teams for 3 consecutive seasons. Three hundred followed the traditional training program while the other 300 added proprioceptive and proprioceptive neuro-muscular facilitation (PNF) exercises. In the second group, non-contact ACL injuries per team and per season were 0.15 while in the first group they were 1.15 [22]. These data show application possibilities and practical repercussions of laboratory studies on the game. Moreover, the evolution of prevention exercises and dedicated rehabilitation make further prospective studies and objective data examination necessary both in the laboratory and on the field. We use functional biomechanical evaluations for objective qualitative and quantitative control of the various functional parameters implied [23].

The relationship between soccer player and clinical application of biomechanics is rather clear in our habitual management of ACL rupture. In these cases, along with the extent of knee instability, we focus on morpho-functional conditions of the athlete and general status of the locomotor apparatus. For this reason, we perform at the beginning and during the rehabilitation period various functional evaluations (arthrometric, stabilometric, and isokinetic examinations and gait analysis) that are useful in preoperative preparation to decide the kind of treatment or surgery and to organise the rehabilitation program and, if necessary, change it. The same examinations are performed in the following phase of sports preparation until return to the field. In fact, by controlling during and at the end of the program the various muscle (with isokinetic and EMG examinations), proprioceptive (with unstable boards, KAT 2000 and Bertec force platforms), joint kinematics (with 3D motion evaluations), and weight-bearing (with ground reaction forces measurements) functional parameters, as well as the performance of specific movements, it is possible to modulate the rehabilitation program following the aims of each phase.

Biomechanical evaluations certainly play a fundamental role in the prevention and aetiologic treatment of overuse pathologies in soccer players, such as the adductor or rectus-adductor syndrome and groin pain. In these cases, in fact, it is important to identify predisposing factors whether being individual, morphological (lower-limb dysmetria, axial deviations or torsion defects of the lower limbs, dysmorphisms or paramorphisms of the spine), or functional (muscle imbalance between hip adductors and abductors, proprioceptive disorders, joint defects). It is also important to identify the

extrinsic predisposing factors (inadequate field, inappropriate footwear) and the determining factors (bad movement performance and/or inadequate training).

As for the player's equipment, the shoe certainly plays a primary role. In fact, the stability of the foot-shoe-surface system allows a better sport-specific performance and prevents musculo-skeletal injury risks. Stress fractures, overload injuries of the locomotor apparatus, as well as ankle and knee sprains are in fact frequent both in amateur and professional players. In order to analyse the influence of the foot-shoe-surface interaction, we must consider various aspects, such as environment and equipment. An inadequate surface or an inappropriate shoe may represent the basis for injury in professional and, especially, amateur players who often play on different surfaces (grass, clay, synthetic), sometimes irregular, muddy, or icy, which represent an important risk factor. Thus, the importance of choosing the appropriate shoe in relation to the field, especially concerning the number of cleats, which influences grip, traction and stability, and may be fundamental in the prevention of overuse injuries. For this reason, we carried out a biomechanical study on professional players aged between 17 and 20 using soccer shoes with 6, 13, and lamellar cleats while they performed running followed by quick stop and kicking, cutting, and jumping on both synthetic and grass surface reproduced in our Laboratory of Clinical Biomechanics "Let People Move". Various parameters were analysed: 3D joint kinematics with the SIMI Motion movement analysis system, plantar load force distribution with Tek-Scan using FScan flexible soles, and anterior tibialis and lateral and medial gastrocnemius muscle electrical activity with TeleMyo telemetric EMG with surface electrodes.

The results we achieved show the importance of specific shoe/insole designs in preventing overuse injuries. In fact, only two areas showed different plantar distributions: the heel and the 2nd metatarsal. On natural grass, the 6 cleats produced a lower pressure distribution in both movements and on both areas (rearfoot/heel and 2nd metatarsal), reducing injury risk. On synthetic grass, the 6-cleat findings were the opposite as on the natural surface, leading to the conclusion that they cannot be used on synthetic surfaces. On synthetics surfaces, the selection between lamellar cleats or 13 cleats is not clear. It seems that 13 cleats produced lower pressure under the 2nd metatarsal during jumping whereas the lamellar cleats produced lower pressure under the 2nd metatarsal during cutting.

In conclusion, the results of the present study showed that current soccer footwear produce an excessive loading on the 2nd metatarsal, which increases the incidence of stress fractures, and an excessive loading on the heel, which increases pathologies of the rear foot, Achilles tendon, and patellar

tendon. Synthetic grass fields certainly represent a more homogeneous foot-shoe-surface contact, with indisputable advantages in the moments of contact and tackle with the opponent. This is because a better grip avoids any variation of angular forces on the lower-limb kinetic chain to add up to the high kinetic energy. On the other hand, during cutting and quick stop, this better grip might increase the risk of ankle and knee sprains, a situation that we are currently controlling.

The next step obviously is to optimise the shoe and cleats in relation to the surface, especially synthetic surfaces, focusing on the player's comfort, ability to perform sport-specific movements correctly, and mechanical effects on the locomotor apparatus through biomechanical evaluations.

Conclusion

Biomechanics applied to soccer, apart from enriching our scientific knowledge, brings important advantages to the whole world of sports: from societies, to supporters, and to players. The latter can take advantage of the practical application of our studies' results, not only in terms of competitive performance but also in terms of health status since he or she can reach optimal locomotor apparatus functional conditions. Furthermore, eliminating the intrinsic (and when possible also the extrinsic) predisposing factors, it is possible to reduce the incidence of pathologies that are typical in soccer. Biomechanical analysis gives objective and reproducible data on the main characteristics of specific movements (various kinds of kicking, cutting, jumping, etc.), on their performance by every single athlete, and on the functional status of the locomotor apparatus during the different phases of the competitive season so as to improve the results and reduce the risk of acute and/or overuse lesions. In this regard, the importance of muscle balance between agonist and antagonist muscles of the lower limb (for instance, surals, knee extensors and flexors, hip abductors and adductors) to reduce the risk of muscle injuries and/or typical tendinopathies of the game (Achilles, patellar, and adductor) is fundamental. Correction of postural disorders and weight-bearing asymmetries are equally important. The former are well shown by stabilometric analysis with unstable boards (KAT 2000) and force platforms and the latter through gait analysis. Gait analysis – performed with 3D motion analysis systems, ground reaction force systems, scanning system (S-Scan), and surface telemetric EMG to show muscle electrical activity during walking – is becoming an increasingly important examination for dynamic analysis, not only of soccer players but of humankind. Thus, biomechanical analysis of soccer players may positively

influence performance (improving movement and optimising function of the myotendon unit) and reduce the incidence of muscle injuries and tendon and joint pathologies typical of the game.

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Training Methods

Claudio Bordon

Introduction

Football is played by at least 200 million persons of both genders and all ages with wide ranges of experience and capability. A footballer's performance is influenced by technical and tactical factors as well as biomechanical, physiological, and psychological conditions, and the player must be sufficiently capable in every respect. In physiological terms, football is a highly intense, intermittent exercise. During a match, top-level players run about 10–13 km at an average intensity close to the anaerobic threshold [85–90% of the maximum heart rate (HR)]. This general picture of endurance is interspersed with numerous actions of a more impetuous kind: leaping, kicking, speed changing, sudden stopping, and sprinting. Neuromuscular qualities, such as explosive force, muscle elasticity, speedy recruitment of motor units, repetition of quick effort, and untiring execution of sudden spurts and accelerations are both characteristic and essential [1]. Repeated sprint ability (RSA) with short recovery intervals is an important component of performance [2], and its physiological features are not clear. Recovery seems partly dependent on the re-synthesis of phosphocreatine (PC) [3] whereas the role of a player's VO_2 max (maximum oxygen consumption) in this re-synthesis is disputed. Some workers maintain that the blood's buffer capacity is a significant feature of RSA [4]. Bangsbo et al. [5] are of the opinion that the intensity of football reflects a player's HR response and may be high enough to require substantial glycolysis.

Match Analysis

A player's efficiency may begin to wane after prolonged intense exercise or towards the end of a match. Fatigue of this kind is the outcome of concomitant metabolic processes [6]. There is thus a need to establish the performance patterns of élite footballers so that training parameter values can be approximated to those characteristic of a match.

Training is usually based on a programmed series of means and methods designed to stimulate the body to respond through specific functional adaptations. Understanding of the performance pattern from which the training pattern is to be derived is often geared to the experience and intuition of the trainer or physical coach. In the case of more complex activities, however, it is advisable to resort to more sophisticated methods to form appropriate opinions concerning the parameter values and the physical, technical, and tactical features of a player's performance. Expenditure of energy, ergonomic requirements, and the mechanical work needed in all kinds of sport form the essential background for the formulation of specific, purpose-oriented, training programmes.

In the literature, football is classed as an activity with alternate aerobic/anaerobic involvement, like all other sports in which there is a more or less regular, codified, causal or deliberate switch from one type of commitment to another. The distance covered in a match by élite footballers is about 10–13 km for full-field players and about 4 km for the goalkeeper (Table 1).

Many studies show that mid-fielders cover a greater distance and that professionals move about more than non-professionals [11, 12]. In the second half of the match, the distance covered decreases by 5–10% [1]. Wingers and sweepers sprint about 70 times (once every 90 s) whereas mid-fielders and stoppers sprint for an average of 2–4 s 40–50 times (once every 120 s) [5, 13]. Sprints account for 1–11% of the total distance [1, 11, 13] and 0.5–3.0% of the

Table 1. Distance covered per role

Level/nation	Defenders	Mid-fielders	Strikers	Method	Reference
1st division/England	11,472	13,827	-	Hand notation	Whitehead [7]
2nd division/England	10,826	11,184	-		
1st division/England	7,759	9,805	8,397	Tape recorder	Reilly and Thomas [8]
1st–4th division/Sweden	9,600	10,600	10,100	Hand notation	Ekblom [9]
University team/Belgium	9,902 (2)	10,710	9,820	Cine film	Van Gool et al. [10]
1st 2nd division/Denmark	10,100	11,400	10,500	Video	Bangsbo et al. [5]
1st division/Italy	10,800	11,500	10,500	Video analysis	Colli et al. [13]

actual playing time (i.e. when the ball is in play). Intense running (at 16–20 km/h) occurs about 100 times per match (roughly once a minute) while threshold running (at 12–16 km/h) ranges from about 220 times for mid-fielders (once every 25 s) to 180 for wingers and strikers (once every 30 s). Catch-up running at 8–12 km/h takes place every 10–15 s (Table 2) [13]. Every player changes speed about 1,000–1,200 times [1, 13], with a variation of intensity every 4–6 s (Table 3). Strikers and wingers sprint almost twice the distance of mid-fielders and defenders [11–13], and mid-fielders run further at anaerobic threshold speeds (Table 4).

Table 2. Quantity actions in speed categories per role

Role	Striker	Mid-fielder	Stopper	Winger	Sweeper
Sprint (over 20 km/h)	69	46	49	73	65
Intense running (16–20 km/h)	97	106	91	113	141
Threshold running (12–16 km/h)	172	218	185	196	250
Catch-up running (8–12 km/h)	247	336	296	303	352
Easy running (0–8 km/h)	325	378	360	370	404

Table 3. Speed of footballers in different positions during official matches

Role	Total metres	Speed variations
Strikers	10,566	910
Mid-fielders	11,507	1,084
Stoppers	10,808	981
Wingers	11,316	1,055
Sweepers	12,260	1,213

Table 4. Distance covered by footballers in different positions during official matches

Role	Striker	Mid-fielder	Stopper	Winger	Sweeper
Sprint (over 20 km/h)	1,163	688	753	1,196	859
Intense running (1620 km/h)	991	1,273	934	1,246	1,668
Threshold running (12–16 km/h)	1,486	2,518	1,793	1,857	2,751
Catch-up running (8–12 km/h)	1,591	2,338	2,146	2,011	2,381
Easy running (0–8 km/h)	5,334	4,690	5,182	5,006	4,600

Work Intensity

The energy consumed during a match is mainly derived from aerobic metabolism. The mean work intensity, expressed as the percentage of the maximum HR (HRmax), is close to the anaerobic threshold (i.e. the highest intensity at which the production and removal of lactate are equal, normally at 65–90% HRmax in footballers). A higher mean intensity could not be maintained because it would result in severe elevation of blood lactate (lactic acidosis).

Definition of work intensity as a mean for a 90-min match or for each of its halves, however, fails to provide some essential information. There are, in fact, short periods of very intense activity during which lactate is accumulated, and these must be followed by low-activity periods to allow its removal from the muscles. Oxygen consumption during a match has not been precisely determined but can be indirectly measured from the ratio between the percent of HRmax and VO_2 static contractions; however, exercises with small-muscle masses and psychological and thermal stresses can elevate the HR at a given oxygen consumption and alter the HR–VO_2 ratio [14]. Reilly et al. [2] have shown that this ratio is also valid during intermittent exercise by comparing this with continuous exercise in a laboratory test, and its validity in high-intensity intermittent exercise has been demonstrated [6]. Employment of this ratio to estimate oxygen consumption shows that a mean intensity of 85% of HRmax corresponds to about 75% of the VO_2max [14]. Stroyer et al. [15] have found that HRmax percentages during a match are higher in young élite players as opposed to non-élite players of the same age (Table 5).

Table 5. Heart rate as a percentage of the maximum (HRmax) during a football match

Level/nation	Type of play	HRmax (%)	Reference
Unknown/Czechoslovakia	10–min match	80	Seliger [16]
1st Division/Sweden	Official match	93	Agnevik [17]
Elite juniors/Norway	Official match	82.2	Helgerud et al [18]
Elite colts/Denmark	Official match	86.8	Stroyer et al [15]
Elite/Sweden	Official match	89–91	Ekblom [9]

Physiological Profile

Enhancement of maximum oxygen consumption in football and other sports has been evident in the last 20 years. This does not necessarily mean that match performance is determined by such consumption because changes in maximum aerobic power may simply be a side-effect of more intense and more frequent training. Aerobic metabolism supplies most of the energy used

in football. Even so, football's main actions (leaping, sprinting, sudden stops and changes of direction) are all fuelled by the release of anaerobic energy.

Players with a high VO_2max may have lower blood lactate concentrations due to their better recovery after high-intensity intermittent exercise via an enhanced aerobic response that promotes lactate removal and steps up PC resynthesis [19]. Exercise intensity equal to about 70% of HRmax ensures more efficient removal [14, 20]. The percentage of lactate removed, in fact, depends on its concentration, the work done during the recovery period, and the aerobic power: the higher the concentration, the higher the percentage [2]. The literature shows that the percent utilisation of aerobic power does not depend on a team's division whereas total calory expenditure is higher in top-level teams [21]. Owing to the type of exercise and the duration of football matches, the level of muscle and liver glycogen reserves is expected to influence a player's performance in the second half and extra time. In sedentary subjects, these reserves are usually reconstituted in 2 days whereas trained subjects require less time because of their increased glycogen synthase.

Histological and functional assessments of a footballer's lower limb muscles are of interest on account of the specific activity. A player can, in fact, be classed as a "fast" subject because of the approximately 60% of fast twitch (FT) fibres in the quadriceps [21] while the diameter of the quadriceps is greater than in controls. It is for this reason that the player's muscles are patently hypertrophic since hypertrophy is always the outcome of an increase in FT fibre cross-section due to training designed to improve force and explosive power.

Training of the Aerobic Metabolism

Intermittent Work

Intermittent training methods are widely employed to improve the supply of aerobic and anaerobic energy and enhance muscle strength, two qualities indispensable for resistance to fast force in many sports, including football [22]. The prime advantage of intermittent work is that it allows reiteration of the same expression of the force used in a match since it is based on the alternation of very elevated efforts with short recovery times.

During violent muscle exertion, there is a massive recruitment of FT fibres, and a great deal of biochemical energy is used, part of which is regenerated aerobically during the recovery stage, and stress is thus imposed on the respiratory and cardio-circulatory system [23]. Intermittent work enables large load volumes to be developed after a preparatory stage and during the special work stage. The lactate produced during intermittent work must not exceed 8–10 mmol/l.

Competitive football demands rapid moving around and facing opponents and quick recovery. The main biological feature of team games, therefore, is intermittent work in the form of rapid action that is nearly always followed by a sufficient recovery time that is neither fixed nor foreseeable since it depends on the current tactical and technical situation. Intermittent exercise is a pattern common to other sports in which periods of intense effort alternate with both active and passive recovery periods. Training methods and biology suggest that a physiological adaptation to training stimuli is more readily achieved, stabilised, and maintained with a substantial volume of work and intense effort [24]. Starting, braking, acceleration, lateral deceleration, running back and forth, and straight running can be used in specific exercises with the ball and in match situations.

HR and the amount of lactic acid produced at the end of the exercise are the parameters that need to be monitored in the assessment of intermittent work. One can thus establish identification of HR zones involving the minimum and maximum use of aerobic metabolism with either Mader's test (threshold at 2 and 4 mmol/l) or HRmax.

The actions set for each player and for different types of shifting position can take two forms: (1) continuous intense action lasting 7–8 s, and (2) intense action for 3 s repeated after a pause of less than 5 s. This situation can be repeated several times prior to a 20–30 s pause. This method can be used for an infinite number of motor actions associated with football, all of which involve acceleration and deceleration, offensive and defensive anteroposterior shifts, and short periods.

As already stated, the intermittent method in sport requires the maximum intensity (90–100%) for short periods (5–20 s). It does not teach the economy of running [22] but coordination and specific movements. In sports, in fact:

- The FT fibres always intervene;
- They are employed both in straight running and in speeding up and slowing down;
- The aerobic system specialises in fast PC recovery by remaining at 70–80% of the VO_2 max;
- It increases the aerobic power level;
- It can be used in specific ball exercises and in match situations;
- After exercise, it allows an elevated use of body fat by means of enhanced basal metabolism.

The quantity of intermittent work is comprised of periods amounting to more than 200 min a week and others requiring only 120–150 min a week [22]. As can be seen, when a player's HR is recorded during conditioning technical and tactical exercises, the volume and intensity illustrate progressive loading (increase in the total training time and gradually increasing work intensity) (Fig. 1). In intermittent work, therefore, the following points must

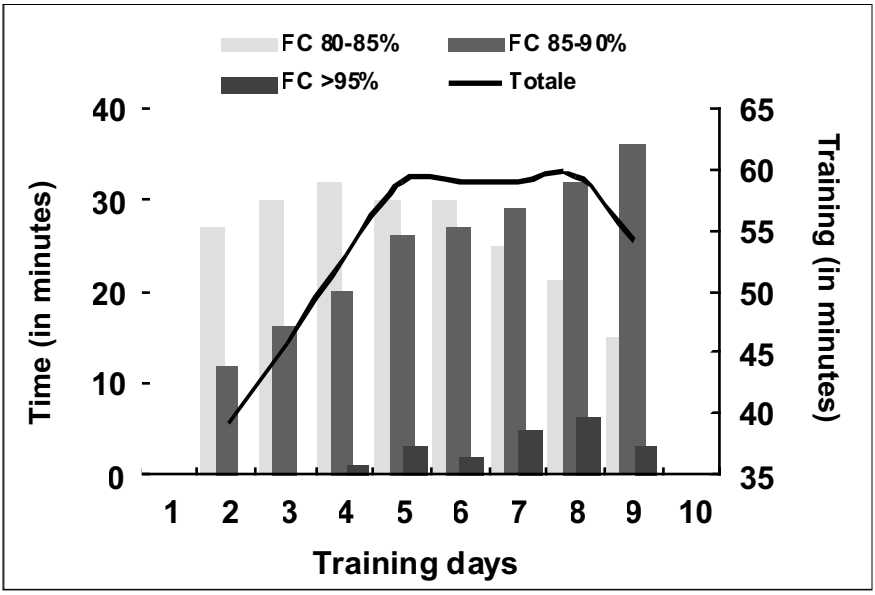

Fig. 1. Load intensity assessment

always be considered:

- the duration of the active stage and any minor actions prior to the pause, which can only be increased after a certain number of sessions have increased the volume of the intense actions;
- the amount of intense actions per series, which must be gradually augmented and can be regarded as the true volume of assessable work;
- the working HR values, which must be gradually increased, initially by shortening the pauses to bring the exercises closer together, then by making the latter a little longer (as in the first point). Some intense actions can be followed by an active recovery period at low aerobic intensity (minimum limit: 30 beats less than the anaerobic threshold HR at 4 mmol/l). This pause can be as long as 2–3 min. It must not be confused with the total recovery period between one series and the next.

Examples of intermittent training include Bosco's method for football [23] and the specific exercises proposed by D'Ottavio et al. [25] and Colli and Bordon [9].

Force Training

An élite footballer must possess the explosive lower-limb force needed for leaping, bounding, and shifting forwards, backwards, and sideways [23]. Anaerobic lactate metabolism is particularly important in this case because the high consumption of energy per unit of time means that the energy

required must come from ready-for-use sources [23, 24]. Bosco [23] suggests that maximum force, explosive force, and resistance to fast force should be improved simultaneously. The underlying concept of Bosco's new method is simultaneous stimulation of several biological properties to improve functional capabilities. In practical terms, a training session alternates maximum force loads with explosive force exercises that facilitate the maximum force work. Force training is wholly based on the management of two parameters: (1) the number of repetitions per series; this number must be such as to develop a power of not less than 90% during the exercise (usually 4–6 repetitions with loads between 70% and 80% of 1 RM); (2) short (60–90 s) recovery times that none the less allow repetition of the force exercises at the set power limit and the completion of 50–60 maximum force repetitions in 20 min.

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Evaluation of Whole Physical Condition

Fabio Pigozzi, Arrigo Glombini, Federica Fagnani, Valter Di Salvo

Assessment of Clinical and Functional Aspects in Soccer Players

The fundamental principles for medical screening should be no different in sport than in any other clinical setting. In professional sports such as soccer, the financial implications of players being unavailable for selection in competition are important. It is hence imperative that the medical team attempts to maintain players' optimal physical integrity. Player fitness is maintained and monitored in co-operation with the coaching staff with the aim of returning to play as quickly as possible in case of injury. The beneficiaries of the screening process are both the club and the individual player. Once a player is taken on by the club, the medical staff attempts to prevent injury and illness from interfering with participation at the highest level [1]. Thus, in professional football, investigations are undertaken to give advice to the club on the medical risks of signing an individual player (pre-signing medical) and to help the maintenance of the peak performance of all team members through a series of appropriate functional evaluation tests [2]. Comprehensive care is usually provided from a purpose-built training facility and includes a full medical team with a full-time doctor and full-time physiotherapists. All must be appropriately qualified, and other professionals can be accessed as required.

We would like to divide the evaluation of physical condition in soccer players into clinical and functional aspects. It is worth noting that "the status of professional athletes" has been recognised by Article 2 of the Italian Law 91 dated 23 March 1981 as pertaining to all athletes, coaches, sports managers, and athletic trainers who practice sports activity on a regular basis and get economic rewards in one of the disciplines regulated by the Italian National Olympic Committee and national sports federations regarding the difference between amateur and professional sports practice. The more recent ministerial decree on professional athletes' health-care laws of 13 March 1995 redefined the aim of health safeguards, which are guaranteed for all professional athletes who practice soccer as well as cycling, boxing, motorcycling, golf, and

basketball. All athletes who practice one of these professional sport activities must own a health card, as defined by Article 7, Law 91 (23 March, 1981) that, in accordance with the 1995 decree, should be maintained for the entire athletic life period and must be regularly updated (at least every 6 months) unless different provisions are dictated by specific sports activities. The health card certifies that all necessary medical tests have been undertaken and briefly outlines contraindications, even if temporary, to professional competitive sports practice. Sports associations are obliged to issue health cards, and it is the physician of the sport association who must fill in and regularly update the cards as the athletes start playing for the association. In case of an athlete's transfer to another sport association, the physician must update the health card at least 8 days before the transfer and must then send the card to the physician of the new association. The clinical evaluation includes the following tests: a general medical examination, including family history with particular regard to pathological and traumatological events; a general physical examination that necessarily includes anthropometric measures (weight and height); general and specific demographic data, including general and segmental assessment of organs and apparatus; and special attention to the cardiovascular, respiratory, and locomotor systems. Additional investigations can be performed in case of clinical suspicion. During periodic medical examinations, athletes undergo other instrumental and functional examinations, as well as blood tests. The medical examination must include a 12-lead resting electrocardiography (ECG) while a chest x-ray and a Doppler 2D echocardiography are required at the time of the first medical examination. Athletes must also undergo a resting and exercise ECG every year, and the Doppler 2D echocardiography must be repeated every 2 years in any case.

During the first medical examination, the following tests are necessary: blood count, glucose, uricaemia, transaminase, iron-complete urine test, serodiagnosis for syphilis (lues), polymerase chain reaction (PCR), total and fractionated bilirubinemia, determination of blood group Rh, blood urea nitrogen (BUN), sedimentation velocity, creatinemia, ferritinemia, hepatitis B surface antigen (HBsAg), hepatitis C virus (HCV), hepatitis markers (only if positive to HBsAg), glucose-loaded glycemic curve (only in case of well-founded clinical suspicion), lipidic profile, and electrophoretic protidemia. During the following medical examinations (every 6 months), all previous tests (except for serodiagnosis for syphilis, blood group and Rh, lipidic profile, and electrophoretic protidemia) must be repeated, and an alkaline phosphatase test must be added; lipidic profile and electrophoretic protidemia must be repeated every year. Athletes must undergo any other test "considered necessary in case of well-founded clinical suspicion" at any time.

With regards to the functional aspects, we would like to consider that in

competitive soccer, exercise is intermittent, and performance is related to the athlete's ability to repeatedly perform intense exercise. In fact, during a 90-min game, élite-level players run about 11.4 km at different intensities, and between 20% of this running time is spent close to the anaerobic threshold (AT). However, clear statistical differences exist for the averages of distances and intensities, depending on the positional roles of the players. For example, external and central mid-fielders run at a higher percentage intensity (26.3% and 24.8 %, respectively) while central defenders run at only 18.3%.

Throughout any game, numerous explosive bursts of activity are required, including jumping, kicking, tackling, turning, sprinting, changing pace, and sustaining forceful contractions to maintain balance and control of the ball against defensive pressure. Previous studies have demonstrated that players with a higher maximum aerobic power cover greater distances during a soccer game [3] and also complete more sprints. The average work intensity during a soccer match has been reported to be ~75–80% of maximum oxygen uptake (VO_2 max), resembling typical values of AT [3, 4]. It is therefore likely that despite their non-sport-specific design, both VO_2 max and AT are important measurements for soccer players. It would appear, therefore, that a high anaerobic power is required for success in top-class soccer players. The maximum oxygen intake for elite male players has been determined in several studies, with mean values between 56 ml and 69 ml/kg^{-1}·per min^{-1} having been reported [5]. Players in a top Norwegian team (Rosenborg FC) had higher VO_2 max values than players from a lower-ranked team playing in the same league (67.6 ml vs 59.9 ml/kg^{-1}·per min^{-1}) according to Wisloff et al. [6]. We think that VO_2 max is not a truly sensitive measure of performance capability in soccer and, in our opinion, cannot be used in isolation to evaluate fitness or to identify talent. Nevertheless, the consistent observations of mean VO_2 max values above 60 ml/kg^{-1}·per min^{-1} in élite teams implies a threshold below which an individual player is unlikely to possess the physiological attributes for success in top-class contemporary soccer.

Physiological measurements of VO_2 max and AT have commonly been used to monitor the fitness and training status of soccer players [7, 8]. Maximal oxygen uptake tests are usually performed continuously, with no rest between work increments, using a treadmill running protocol (Fig. 1). The ramp test is one of the most commonly used protocols. With this procedure, the speed is increased each minute for a total of 10–12 min, depending on the athlete's fitness. This relatively quick procedure produces a linear increase in oxygen uptake to the maximum level tolerated by the athlete. AT tests can be used to characterise training effects and evaluate physical fitness. In terms of soccer performance, this could mean that during a game, players with higher ATs are able to cover more distance at a higher intensity without accumulation of lactate compared to less-aerobically trained athletes. For the detection of

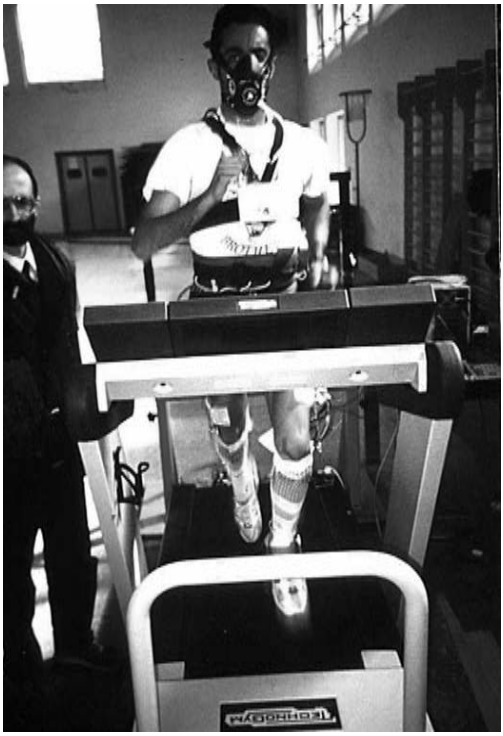

Fig. 1. Evaluation of maximum oxygen uptake (VO_2 max) with treadmill-running protocol

AT, several techniques and criteria are used on lactate concentration and ventilatory parameters during exercise. Measurement of AT using blood lactate concentration is directed at identifying either the initial rise in lactate above the resting baseline (LT) or the application of a fixed point at 4 mmol/l[1]. Although several methods are used to evaluate anaerobic performance (such as a supra-maximal test on a cycle ergometer or motor-driven running treadmill), as yet, no properly validated physiological measurement exists for determining anaerobic capacity. None of these factors by themselves accurately express the ability to perform prolonged, intermittent exercise with alternating intensities, as occurs in soccer [9]. For this reason, various field tests of performance endurance, which are highly valid for soccer, are used.

Recently, two field tests of relevance to soccer have been developed: the Yo-Yo intermittent endurance test and the Yo-Yo intermittent recovery test [10]. In the intermittent tests, players perform repeated 20-m shuttle runs interspersed with a short recovery period during which they jog. The time allowed for a shuttle, which is progressively decreased, is dictated by audio bleeps from an audio tape. The objective is to complete as many shuttles as possible [3]. The endurance test evaluates a player's ability to perform intense exercise repeatedly after prolonged intermittent exercise; players have a 5-s

rest between each shuttle. The aim is to determine a player's ability to recover from intense exercise; running speeds are higher than during the endurance test, and there are 10 s of jogging between shuttles.

Many activities in soccer are forceful and explosive. Power output during such activities is related to the strength of the muscles involved in the movements. Thus, it might be beneficial to have a high muscular strength, which also reduces the risk of injury [11]. Consistent with this suggestion is the finding that soccer players have higher muscular strength than untrained individuals [6]. The muscular strength of soccer players appears to be related to their position in the team. In a study of top-class Danish players, muscular strength evaluated with an isokinetic machine was lowest for mid-field players at all angular velocities measured. In contrast, full-backs generated lower forces than goalkeepers, forwards, and central defenders at high velocities [12]. Differences in muscular strength are probably due to selection of a specific type of player for a position rather than to a more pronounced development of strength as a result of playing in that position.

From observation of top-class soccer players in various studies, it is clear that the strength of the knee extensors alone does not determine the final impact on the ball when kicking [13]. Technical skill is also a major factor in kicking a football since the kick incorporates a complex series of synergistic muscle actions involving antagonistic as well as agonistic muscles. Quite apart from its relevance to game skills, muscle strength is relevant to more global aspects of competitive performance. Fowler and Reilly [14] reported that asymmetry in muscle strength, both between the left and right legs and between the leg flexors and leg extensors, together with the flexibility of the hamstrings and hip adductors, are factors in pre-disposing towards injury among professional players. Upper-body strength is also relevant for coping with the physical aspects of the game and for throwing in [15].

Different tests are used to evaluate strength parameters for élite soccer players. Most studies use isokinetic equipment to evaluate lower-limb strength; however, the use of different speeds and joint angles makes direct comparisons difficult [16]. Isotonic equipment, such as the Muscle Lab Bosco System, allows the evaluation of the stretch-shortening cycle during physiological movement. This guarantees much more specificity and reliability [17]. Muscular power has traditionally been measured by means of sprints and vertical jumps [18, 19]. Jumping ability can be evaluated with a Bosco jumping mat (ergojump), an optojump, or a force platform (Fig. 2). Players usually perform two different jumps – a squat jump (SJ) and a counter-movement jump (CM) with the arms kept in the akimbo position to minimize their contribution [6]. Three tests are carried out for each type of jump, and the best result is used. A 2-min rest is allowed between jumps to minimise the effect of fatigue. As regards sprints, players perform a series of 27-m sprints (Fig. 3).

Fig. 2. Jumping ability tested with a force platform

Fig. 3. A 27-m shuttle test to evaluate sprints

Speeds are measured with infrared photo-electric cells positioned at 9–18 m and 27 m from the start line. Players start on a visual signal from a standing position and run 27 m as fast as possible with two changes of direction (at 9 m and 18 m). Recovery periods are 20 s between each series.

It is fundamental to plan evaluation tests, carried out by the coach and the team physician, at the beginning of the competition season. In addition, besides the proposed tests carried out by the coach with the aim of checking physical condition and for the definition of training programmes, it is advisable to submit the athlete to evaluation tests aimed at physical integrity. Prevention in sport is more important nowadays because of the work load to which many soccer teams are subjected. It is possible that players compete twice a week without a suitable training programme during the pre-competitive season.

In recent years, the most important soccer teams have been engaged in stressful summer competition mainly planned for economic and merchandising purposes. The clear result of recent changes in the soccer world can be summed up as follows:

- More competitions;
- Lack of athletic training during the precompetition period because of economic pressures on the club;
- Media commitments;
- Additional work load for soccer players of different national teams who often do not have summer holidays.

To achieve a valid evaluation of physical integrity, every soccer player needs to keep a personal health card outlining their medical history, in addition to undergoing the clinical tests required by law. Also, it is advisable to carry out specific evaluation tests, such as RMN of hips, knees, and ankles at the time of recruitment. Other evaluation tests suggested at the beginning of the competitive season are:

- Tests for cardio-vascular and respiratory systems along with stress test;
- Haemato-chemical analyses that have to be carried out every 6 months and which should be repeated every 2–3 months to prevent possible disease and overtraining;
- Control of body composition for possible correction of diet;
- Isokinetic and isometric tests, such as the Muscle Lab, that are able to evaluate curve F/V and muscular strength. These tests are important for highlighting the clinical relevance of possible muscular imbalance (often a cause of muscular lesions). Such imbalances in athletes with lesions are also present several months after the injury date. Therefore, this evaluation needs to be carried out at least every 3 months.
- Additional exercises (scheduled outside of ordinary training) to prevent injuries. These types of exercises duly managed by medical staff, physio-

therapists, and coaches with individualized training programmes should improve proprioception and joint flexibility.

Conclusion

There are many factors that predispose towards a successful career in professional soccer. Foremost among these is excellence in games skills and the cognitive abilities to make correct decisions within the game. The player's health is the most important consideration and is accepted as being in both the player's and the club's best long-term interests. It is our opinion that elite players must possess moderate to high aerobic power, have good agility, good joint flexibility and muscular development, and be capable of generating high torques during fast movements. In addition, a higher level of anaerobic parameters is preferable and reduces the risk of injuries [6, 11]. Although non-genetic factors can be identified as markers of potential in soccer, the ability to tolerate systematic training is a clearly important factor. It is likely that even a multi-variate formula for predicting future success will remain elusive. However, from a practical standpoint, we believe that the use of information obtained by functional tests before and during the competitive season is important for coaches and trainers in order to adjust training regimes and concentrate on the variables that are specific for improving performance in soccer players and preventing injuries.

A final fundamental principle is the importance of establishing different training programmes appropriate to the player's positional role in the team [20].

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Biochemical and Haematological Parameters in Football Players

Giuseppe Banfi

Reference Values

The reference values usually listed in laboratory reports have been calculated on sedentary people and may not be useful for sports people. Athletes are, by definition, healthy and "normal" subjects, but they often show – owing to physical exercise, training, psychophysical stress, and peculiar environmental conditions – some biochemical, hormonal, and haematological values that are out of range. This particular behaviour of laboratory values must be properly interpreted to avoid incorrect treatment, expensive examinations, and possible cessation of training and competition.

For example, in four blood drawings performed in a year on 36 athletes (102 drawings) of an Italian professional team, we found 9 cases with total bilirubin >1.2 mg/dl due to haemolysis and related catabolism of haemoglobin, which is typical of sports people: the increase was of indirect bilirubin unaccompanied by increases of hepatic enzymes or other potentially pathological laboratory or clinical signs.

Creatinine and urea (although less frequently because of the lower sensitivity of urea), could show some values higher than 1.2 mg/dl in athletes with haemoconcentration and/or incorrectly rehydrated. The concentration is particularly important in football players when blood and urine are taken at the end of the match for anti-doping purposes. Recording and evaluation of specific gravity of urine is mandatory for judging the accuracy and validity of urine testing for biochemical and toxicological substances.

There is no system of reference values for athletes or for specific sports and, generally, there is a lack of data in this field. There are few studies on haematological values in football players [1, 2], and modern reference values for biochemical and haematological parameters are available [3, 4]. Nevertheless, reference values should be evaluated in comparison with biological variability (Vb). The ratio between intra-individual and inter-individual variability (CV_i/CV_g) is the "individuality index", and it provides information about the biological individuality of a specific analyte and, conse-

quently, about the need for using a reference range obtained from a general population. For instance, an index >1.4 identifies the utility of a reference range whilst an index <1.0 means that the reference interval is not useful for evaluating the value of an individual: only the evaluation of consecutive values of the same subject with the same analytical method is useful, obviously in association with the clinical and physiological findings. Table 1 presents some examples of analytes and their individuality index.

Table 1. Selected analytes and their individuality index. CV_i, intra-individual variability; CV_g, inter-individual variability; *ALT,* alanine aminotransferase; *CK,* creatine kinase

Analyte	CV_i (%)	CV_g (%)	Individuality index
ALT	24.3	41.6	0.58
Bilirubin	25.6	30.5	0.84
Calcium	1.9	2.8	0.68
CK	22.8	40	0.57
Creatinine	4.3	12.9	0.33
Haemoglobin	2.8	3.6	0.40
Platelets	9.1	21.9	0.42

Vb includes the cyclic variability of biological rhythms and the casual oscillation beyond homeostasis. The range of oscillation beyond homeostasis corresponds to the intra-individual Vb (CV_i) whereas the inter-individual Vb (CV_g) is calculated on a group or a population: the expression is a coefficient of variation (CV%). The Vb is important in sports medicine because it allows calculation of the critical difference, that is, the difference between two consecutive laboratory values, which is clinically significant. The critical difference is calculated on the basis of a constant (2.77 at 95% probability level) and total variability (Vt). Vt includes Vb and analytical variability. The critical difference should be the value used for judging the modifications of laboratory parameters.

Analytical Requirements

The modern clinical laboratory is highly automated. Clinical and haematological analysers allow analysis directly on serum, plasma, and blood under the supervision of laboratory personnel. The analysers are calibrated; that is, analytical methods are performed on materials having a known quantity of a

specific analyte, and the concentrations of that analyte's biological material are calculated on the basis of these measurements. It is mandatory to control daily, and sometimes more frequently, the accuracy of the measurements (and of the calibration). Quality control schemes have this aim: there are internal schemes that use materials with known concentration in a specific laboratory and external schemes that use materials with unknown concentration in various laboratories. Quality control must always be maintained even when analysers are used. The laboratory must supply, when requested, the analytical variability of each test for calculating total variability. Many laboratories are certified by external accredited agencies for quality control.

In recent years, some simple, portable, and small analysers have been developed for testing and measuring parameters at the bedside of the patient and also out of hospital. Point-of-care testing (POCT) is now part of a decentralised laboratory, allowing fast analyses with technology, informatics, and quality control similar to those of a central laboratory. POCT is useful for sports medicine, and its use for measuring lactate is the most widely known example; however, it could be used for many clinical chemistry analyses, including haematology and coagulation. Measures of creatine kinase (CK), especially during recovery or after intensive training or competition, could be taken by using POCT analysers. Trainers and sport physicians can judge from CK levels the complete recovery of the athlete after 48 h from competition and also detect possible muscular injuries if the values are higher than base levels.

Parameters to Be Analysed and Frequency of Analyses

The selection of analyses to be performed should be done by the team physician, taking into consideration the whole theam and an individual athlete's needs as well as unspecific and specific training loads. Some patterns could be identified even in the absence of specific symptoms and pathologies.

Evaluation of Apparatus

The liver should be evaluated by using enzymes as transaminases [aspartate aminotransferase (AST) and alanine aminotransferase (ALT)]: increases are linked to hepatocyte damage, gamma glutamyltransferase and lactate dehydrogenase (γGT and LAD increase is a sign of biliary obstruction, drug intoxication, or toxic hepatitis); surface antigen (HBsAg) and surface antibodies (HBsAb) should be analysed for evaluating the immune system against hepatitis B. Kidney function should be evaluated by creatinine, electrolytes [sodium (Na^+) and potassium (K^+)], and complete urine test. The immune system and presence of inflammation should be examined by C-reactive protein

(CRP) whilst erythrocyte sedimentation (ESR) rate is too unspecific. Metabolism should be evaluated by glucose, urate, calcium (Ca^{++}) and magnesium (Mg^{++}), total and high-density lipoprotein (HDL)-cholesterol, triglycerides, and, if necessary, thyroid hormones [free T4 (fT4) and thyroid stimulating hormone (TSH)]. Haematological testing is based on complete blood count (including leukocytes, erythrocytes, platelets, haemoglobin, packed cell volume, mean cell volume and related indexes, red cell distribution width, leukocyte differential) and reticulocytes (including count, volume, fraction of immature particles) whereas coagulation is evaluated by prothrombin time (PT) and activated partial thromboplastin time (APTT). Iron metabolism should be studied by ferritin, iron, transferrin, soluble receptor of transferrin (specific and sensitive for functional iron depletion), and transferrin saturation (or total iron binding capacity). Muscular damage should be studied by CK and myoglobin. Hormonal status should be evaluated by testosterone (index of anabolism), cortisol (index of catabolism), growth hormone, and insulin growth factor 1 only for anti-doping purposes.

Iron Metabolism

We underestimate the importance of constant and periodic evaluation of iron metabolism in soccer players. Iron metabolism must be followed in all athletes because of accelerated turnover of erythrocytes caused by haemolysis, gastrointestinal losses, and inadequate dietary intake. It should not be considered that iron depletion and the consequent so-called sports anemia are problems limited to female athletes or endurance athletes. In professional soccer players, some deficiencies in absorption, storage, and turnover of iron can be present, with evident consequences on the maturation of erythrocytes, oxygen transport, and physical performance [2, 5]. Football players generally show constant haematological values within the reference range and similar to athletes participating in other sports [6–8]. For example, mean values obtained in 923 professional football players of 39 teams with a total of 2,506 determinations of haemoglobin (Hb), ferritin, and soluble transferrin receptors were 14.82 g/dl, 84.2 ng/ml, and 1.25 mg/l, but ranges were 12.1–17.8, 14.9–249, and 0.50–3.20, respectively [1]. There were few athletes with values out of range, but it is crucial that they be screened and, if necessary, their diet, training, and/or food integration be modified. In Table 2, the iron metabolism of players on a professional team is illustrated: blood drawing was performed at the end of a period of heavy training and competition, and it is evident, especially from soluble transferrin receptor (sTfR) concentrations, that some athletes had an initial functional iron depletion.

Table 2. Iron metabolism of players on a professional team. Blood was drawn at the end of a period of heavy training and competition. *TFRh* transferrin; *sTfRh* soluble transferrin receptor, Bold: values out of range. Italic: values near to the lowest limit of the range

Ferritin (µg/dl)	Iron (µg/dl)	Transferrin (g/h)	TRF saturation (%)	sTfR (mg/l)
127	97	2.43	32	1.33
139	100	2.22	36	1.18
66	76	2.16	28	1.65
116	114	1.96	47	1.20
34	53	2.37	**18**	**1.92**
97	107	2.46	35	1.45
205	102	2.44	33	1.00
70	82	2.38	28	1.33
59	81	*3.15*	21	1.97
114	99	2.41	33	1.68
157	152	2.06	59	1.41
104	113	2.27	40	**2.28**
157	106	2.21	38	**1.78**
224	68	2.23	24	1.45
53	96	1.93	40	1.79
72	107	2.39	36	1.77
128	76	2.50	24	**1.90**
96	140	2.31	48	**1.96**
53	111	2.25	39	**1.90**
158	115	2.25	54	**1.91**
122	102	2.15	35	1.22

Specific Tests

In our 12-year experience, the evaluation of free testosterone/cortisol ratio (FTCR) is a useful index of incomplete recovery. Despite some criticisms from physiologists regarding the use of hormonal parameters for judging overload and incomplete recovery, and from some experts as regards the absence of over-reaching in football, we confirm that hormonal parameters are useful for

evaluating the entire team and for each athlete, if considered with other signs and with the collaboration of the sport physician and trainer. Football is a sport generally characterised by a risk of over-training lower than endurance sports, but we must consider that during the last 10 years, a noteworthy modification of training workload and of competition frequency in professional soccer athletes have been registered. The number of matches and of minutes played, the higher turnover of players, the frequent continental and intercontinental flights, and the higher frequency of matches, induce very high levels of stress, facilitating over-reaching. We are using a new model for interpretation of FTCR values based on a stratification of FTCR values in six categories for footballers, possibly useful also for other sports, which could be used in association with well-known interpretation schemes (modification of almost 30% of the previous value or absolute threshold value of $0.35x10^{-3}$). In Table 3, values of FTCR of athletes of a professional team are shown. We listed only the athletes who were present at all the blood drawings performed during the competitive season. The values depicted show the typical behaviour of the ratio in a football season: a decrease after high-level training, a small increase but a lower than basal one during the competition season, and stabilisation across the season with a slight increase in the final part of the season.

Table 3. Behaviour of free testosterone/cortisol ratio (FTCR) in a professional team

FTCR	Jul 02	Sep 02	Dec 02	Jan 03	Apr 03
	1.81	0.41	0.51	0.71	0.60
	1.00	0.37	0.40	0.59	0.72
	1.09	1.05	0.68	0.85	1.03
	1.69	1.26	2.21	1.13	0.85
	1.53	0.78	1.16	0.81	0.64
	1.19	1.23	0.73	0.78	1.17
	1.29	0.68	0.67	0.79	0.61
	1.07	0.96	0.99	0.90	1.07
	0.92	0.80	0.63	0.59	3.17
	1.11	0.47	0.65	0.47	0.67
	0.88	0.77	0.46	1.26	0.58
Mean	1.15	0.80	0.92	0.81	1.01
SD	0.47	0.30	0.59	0.23	0.74

We also performed in various football teams a study of specific free radicals. Exercise is a known source of production of oxygen free radicals, which can contribute to tissue damage and possibly trauma. The evaluation of the total antioxidant status and specific free radicals can be useful for adjusting diet and preventing performance problems. In Table 4, the values of members of a professional team are shown during a competitive season. Normalisation is generally reached by supplementation of antioxidants and by reducing workload, but some ethnic differences should be considered because Afro-Caribbean subjects have higher concentrations of free radicals than Europeans.

Table 4. Values of total antioxidant and free radicals in a professional team. *ARB*, arbitrary

Free radical ARB U/ml (250–300)	**Free radicals**	**Total antioxidant status mmol/l (>1.30)**	**Total antioxidant status**
Dec 02	Apr 03	Dec 02	Apr 03
299	221	1.36	1.35
396	226	1.43	1.58
216	242	1.38	1.34
364	248	1.19	1.20
235	214	1.38	1.33
271	250	1.40	1.35
283	240	1.39	1.39
222	208	1.43	1.42
377	371	1.31	1.31
239	231	1.60	1.48
222	228	1.40	1.31
245	229	1.36	1.34
344	239	1.58	1.47
245	258	1.48	1.42
354	310	1.09	1.03
383	363	1.36	1.48
259	270	1.45	1.36
284	264	1.43	1.34
325	344	1.26	1.29
255	227	1.56	1.57
249	228	1.43	1.34

Frequency of Testing

The ideal time period between two blood drawings in athletes has not yet been established. We can only treat this argument on the basis of pragmatism and experience. Too frequent analyses are no more informative than analyses performed with higher intervals but before and/or after significant differences in terms of training, competition, and health/disease status. Evaluation for hepatitis or vaccination could be done once a year at the start of the competitive season if specific clinical requirements do not suggest the need to verify immunological status at a different time. The semestral evaluation of general clinical chemistry and haematology, suggested by the rules of the Italian Football Federation, seems to be sufficient for professional players. The evaluation of some analytes strictly linked to training, recovery, and performance could be performed with an interval of 8–12 weeks. For instance, we recommend (for central and southern European countries) performing the general evaluation in July, at the begining of training. A second evaluation, using a simplified schedule of tests, should be performed in August/September, at the end of heavy training and before official competitions. A third evaluation should be performed at the end of the first part of the championship. A fourth examination should be performed in April before the end of competition. It is obvious that some analyses should be performed when clinical findings suggest the need for a check-up.

Pre-Analytical Conditions Could Affect Laboratory Data

It is mandatory to observe some pre-analytical conditions for obtaining valid and useful laboratory data. Physicians and pathologists must pay attention to materials used for blood drawings and collection and storage of biological materials [9]. For instance, there are numerous peptides and proteins of endocrinological interest used for research and routine purposes in sports medicine. Some of these molecules are fragile and could be fragmented in vitro by the action of several enzymes. Ethylenediaminetetraacetic acid (EDTA) generally protects labile molecules and can be recommended for all the substances that spontaneously present fragmentation *in vitro*. The addition of anti-proteolytic substances to EDTA, such as aprotinin, optimises stabilisation and storage of plasma. Aprotinin can inhibit the activity of many proteases such as kinins, trypsin, chymotrypsin, enzymes of coagulation and fibrinolysis, and lysosomal enzymes.

The use of stabilisers and iced water for transportation and refrigerated centrifugation with early separation of plasma can improve stabilisation Cardiac markers can be used to monitor myocardial involvement, particular-

ly in endurance or extreme performances. Myoglobin and CK isoenzyme MB (CKMB) should be measured in serum; EDTA should be avoided. Haemochromocytometric analysis must be performed in anti-coagulated blood; the usual anti-coagulant is EDTA potassium salt. The evaluation of haemoglobin blood cells and particles is usual in athletes in order to study iron metabolism, optimal oxygen transportation, possible infections, and other diseases. A precise and accurate ratio between anti-coagulant and blood drawn is crucial to perform correct analyses. The use of evacuated tubes with incorporated EDTA is recommended to assure a correct ratio: it is obviously important that the tubes are not over- or under-filled. The use of syringes and open systems often present in sports fields should be avoided. The stability of haematological parameters in EDTA anti-coagulated blood is 48 h for haemoglobin and haematocrit and 24 h for erythrocyte indices. The stability of leukocytes is good (48 h), but the differential count should be made before 6 h from drawing and the blood smear before 3 h. Storage should be at 4°C; it is important during transfer to avoid exposure of tubes to sunlight and hot temperatures to avoid erythrocyte shape modifications. Lactate may be the most used chemical parameter in sport to identify anaerobic metabolism. It is usually measured directly in fresh capillary whole blood collected from the ear lobe or finger: if the measurement is performed immediately after collection on the field, pre-analytical variables do not influence the data. Lactate tends to increase in vitro in plasma and serum; in which case, the heparinised whole blood is the recommended material, as it is also in the clinical laboratory with immediate analysis. Urine is a biological material frequently used in sports medicine for routine evaluation of hydroelectrolytic homeostasis and renal functionality but also for anti-doping controls. Urine was often proposed for the measurement of proteins, hormones, and substrates because the collection is easier than for blood. We understimate, however, that the problem with urine is the difficult control of production, and consequently, density and renal filtration rate, controlled by different mechanisms, such as hormonal (renin-angiotensin system, antidiuretic hormones), neurological (catecholamines), and dietary (fluid intake, particularly important during exercise). The ratio between the analytes under evaluation and creatinine or cystatin C is useful to minimise this variable. Saliva is another fluid that can be used for some analytes with low molecular weight, such as drugs and steroidal hormones. The concentration of cortisol seems to be independent from flow rate, and the rate of transfer by diffusion from plasma to saliva is rapid (1 min). Saliva, as with urine, presents a variable flow rate, and the collection is not easy to standardise although a special system based on a cotton roll to be chewed and centrifuged can supply salivary fluid without contaminants, foam, and viscosity.

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Prevention of Football Injuries

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Introduction

The ever-increasing number of football injuries in both genders and all age groups and their consequences indicate the need for proper prevention. These consequences not only have a negative influence on performance but raise recognisable socioeconomic problems. In the USA, the primary medical cost associated with football injuries is over $36 billion per year not including costs associated with the loss of competition or working days [1].

Due to the diversity of injuries and the causative risk factors, the required preventive strategy must be complex. Obviously, such a programme must be applicable for everyday practice at all skill levels of football without any problems. Despite about 200 million players playing football throughout the world, there are only a few clinical trials proving the real effectiveness of commonly used preventive measures. Until now, three studies have discussed the general preventive effects of multi-factorial injury prevention programmes [2–4], and seven others have evaluated the prevention of specific types of injury, namely, ankle sprains, severe injuries of the knee, and hamstring strains [5–11]. With the exception of one, all these publications conclude that both the general injury rate and specific types of injury can be reduced significantly by means of prevention (Table 1).

Despite scientific evidence of their success, these methods are not yet incorporated in the general training routine. Obviously, there are big differences in opportunities and circumstances of prevention between the big professionals and amateur football clubs, and there are also major differences from country to country. However, the final goal should be that general concepts become nearly the same in different clubs and different countries and become part of the training routine.

In this chapter, we give an overview for physicians and how they can contribute to and enrich scientific data on injury prevention, which can be especially applicable in the studied settings (e.g. in the club with which the physician works). Furthermore, we also present a general strategic concept that should help to reduce the overall injury rate.

Table 1. Controlled studies on the prevention of football injuries. *ACL*, anterior cruciate ligament

Study Authors	Population: gender, age (in years)	Sample size	Country	Type of injury	Type of intervention	Result
Ekstrand et al. [2]	Males, 17-37	12 teams	Sweden	All injuries	Multi-modal intervention programme	Significantly less injuries vs. control group by 75%
Heidt et al. [3]	Females, 14-18	300 players	USA	All injuries	Frappier acceleration training programme	Significantly less injuries vs. control group by 59%
Junge et al. [4]	Males, 14-19	194 players	Switzerland	All injuries	Multi-modal intervention programme	Significantly less injuries vs. control group by 21%
Tropp et al. [5]	Males, seniors	296 players	Sweden	Ankle sprains	Use of orthosis or ankle-disk training	Less injuries in players with previous history of ankle sprains using either technique
Surve et al. [6]	Males, seniors	504 players	South Africa	Ankle sprains	Instruction to wear a semi-rigid orthosis	Significant reduction of recurrent ankle sprains in players with previous history of ankle sprains
Caraffa et al. [7]	-	60 teams	Italy	ACL injuries	Proprioceptive training	Significant reduction of ACL injuries

Study Authors	Population: gender, age (in years)	Sample size	Country	Type of injury	Type of intervention	Result
Hewett et al. [8]	Females, 14–19	200 players	USA	Serious knee injuries	Pre-season neuromuscular training programme	A trend of reduced injuries in the trained v.s. control group
Söderman et al. [9]	Females, 21	140 players	Sweden	Severe knee injuries and ankle sprains	Balance-board training	No preventive effect
Askling et al. [10]	Males, 25	30 players	Sweden	Hamstring strains	Training with eccentric overload	Significantly less injuries vs. control group
Mandelbaum et al. [11]	Females, 14–18	5,703 players	USA	Non-contact ACL injuries	Multi-modal intervention programme	Significant reduction of ACL injuries

How to Assess the Possibilities of Injury Prevention?

Preventive measures in football – just like in any other sport – should be based on epidemiological research. Van Mechelen et al. suggested in 1992 that measures to prevent sports injuries should follow what they called a "sequence of prevention" [12]:

1. Identify the extent of the problem of injuries in football (hazard identification)
2. Identify the potential influential factors and mechanisms (risk assessment)
3. Introduce preventive measures that are likely to reduce the future risk and/or severity of injuries (risk minimisation)
4. Re-evaluate the maintenance of injury records being crucial, allowing the effectiveness of preventive measures and implemented management plans to be assessed (re-assessment).

Step One: Extent of the Injury Problem – Hazard Identification

Firstly, the extent of the injury problem should be identified and described. This first step of the sequence features two cornerstones of the whole prevention concept. One of these cornerstones is the use of standardised definitions for injury and especially for the description of the extent of the problem – injury incidence. The injury incidence should be expressed as the number of injuries per exposure time, usually per 10,000 h of participation. The lack of use of standard definitions and the shortage of reliable detailed epidemiological data hinders us gaining real evidence-based results, and it is furthermore the main obstruction to summarising the outcome of different studies by means of valuable meta-analyses.

On the other hand, any study of any size provides most benefits for the studied population – e.g. for a club – for any results hat study are most applicable to similar circumstances. This is one reason that there are not only great differences between subgroups of different ages, skill levels, and gender, but other important conditions varying from country to country must be considered. Thus, a fundamentally sound study helps the work of all professionals involved in the game but most of all the work of the experts carrying out the particular study.

Step Two: Potential Risk Factors and Mechanisms – Risk Assessment

The second step is to identify factors and mechanisms that play a part in injury occurrence. As there is always a complete interaction between these factors, the cause of injury is often multi-factorial. At the same time, most risk factors cannot be associated with only one or two types of injuries. In accor-

dance with the diversity of injuries in football, there are numerous risk factors, with changing levels of evidence proving their role in the development of a certain injury. (Read more about Risk Factor in Chapter 1).

However, there are already many factors that are identified by means of evidence-based medicine as important risk factors for numerous injuries, such as joint flexibility including pathological ligamentous laxity and muscle tightness [13], mechanical and functional joint instability [14–16], and some previous injuries and inadequate rehabilitation methods [15]. Causative extrinsic risk factors include training load [17], inadequate equipment (shin guards, taping, shoes), [2, 6, 14, 18, 19], playing field conditions [14–17], and foul play [20].

The present knowledge regarding risk factors seems to be biased by selection according to age, gender, and nature and level of play. In order to apply more effective preventive measures, the complex interaction of intrinsic (person-related) and extrinsic (environment-related) risk factors should be clarified, and their gender, age, and level specificity should be identified [21].

Step Three: Measures of Injury Prevention – Risk Minimisation

The third step is to introduce preventive measures. These measures should be based on aetiological factors and mechanisms, as identified in the second step. We should note here that addressing intrinsic risk factors reduces the incidence and severity of non-contact injuries (both acute and overuse) while consequences of direct trauma are generally reduced by the elimination of extrinsic risk factors.

As there are numerous risk factors, there are even more ways to address them. Below we give only a short overview of measurements according to the competence of different persons and bodies concerned with the game (Table 2).

Coach's Responsibilities

The coach has the main responsibility for the athletes' safety. They must teach them safety principles and appropriate football skills and ensure they are always in adequate condition. The coach should also master basic skills in the management of acute sports injuries. Besides the late season, injuries peak during *pre-season practices* [3, 10, 22]. All activities should be well controlled at this time, and coaching should apply injury-safe techniques and put a special focus on methods, which help injury prevention not just for the pre-season but the whole season. Not allowing the athlete's body to recover properly from training will eventually result in injury. The footballer's body needs time to rebuild itself to a stronger level before the next training session. Remember: athletes are not training when they are training, they are training when they are recovering! Sleep is also an important part of training. If they are not getting enough, get it sorted out!

Table 2. Preventive measures from different perspectives and competences

Trainers' perspective	Medical perspective	Players' perspective	Others (officials, governing bodies)
Risk-conscious education of players	Supervision of injury prevention; implementation and control of preventive measures	Co-operation with coaches, medical personnel, and management in realising the concept of injury prevention	Injury awareness of clubs and governing bodies
Structured, complex pre- and in-season training regimen (including proper complex muscle management and stamina training)	Risk-conscious attitude towards players; education of all staff members	Injury-conscious playing attitude: hard, but fair	Ensure safe pitch conditions
Appropriate warm up and cool down	Load-control: assistance to trainings	Good equipment, especially boots	Observance of the existing rules
Reduction of physical overload; appropriate game/training relationship	Pre-season and pre-signing examinations. Sufficient regard for complaints, ensuring sufficient recovery time and adequate rehabilitation	Using protective gear (shin guards, ankle taping, and bracing)	Improvement of rules of the game Reduction of foul play

Football players should participate in a well-supervised *pre-season physical conditioning* programme as well as focusing on cardio-respiratory stamina and good muscular condition (strength, dynamic power, endurance, flexibility, and proprioception). This should be started at least 6 weeks before the start of daily football practice so players will be in proper physical condition before the first day of practice. The conditioning programme should also include sports-specific exercises to prevent injury. Players should especially emphasise well-balanced strength and flexibility exercises for the lower back, abdominal muscles, and muscles of the lower extremity. Intensity, duration, and frequency of conditioning and skill practicing sessions should be increased gradually in order to prevent overuse injuries.

A similarly complex *in-season conditioning* programme should be followed during the course of the football season as well [3]. However, resistance-training programmes, which should help to prevent injuries by improv-

ing muscle balance, may also lead to injuries when they are not properly applied or supervised. Thus, in order to avoid injury, an athletic trainer with a thorough understanding of resistance training should supervise conditioning sessions personally. Before exercises, athletes should warm up (by means of stretching and low-intensity aerobic exercises) for more than 5–10 min. Particular muscle groups need to be conditioned only 2–3 times weekly to allow enough time for recovery and work-load adaptation between training sessions.

Stretching [2, 13, 23, 24] and *proprioceptive training* [5–7, 23, 25–27] sessions must be held on a regular basis and are best supervised by the athletic trainer. All training regimens should be defined for each player individually; competition among players regarding conditioning results should be discouraged.

Warming up is often overlooked but should be part of any injury-prevention routine [17, 24]. A good warm up will increase the temperature of muscles, blood flow and oxygen supply to the muscles, the speed of nerve impulses, and the range of motion at joints. It not only helps to avoid injury but also improves performance. The warm up should last between 15 and 30 min but must not be carried out too early; the benefits are lost after about 30 min of inactivity. Athletes should also implement a short warm-up session in case of any prolonged breaks (half time, etc.).

Similarly to the warm up, there must be a *cool down* after every practice session or game played [2, 17, 27–29]. Immediately stopping vigorous activities will cause a fast reduction of blood flow in the muscles, hindering the necessary transport of potentially harmful metabolites (hyperoxides, lactic acids, etc.) from the muscles. The cool down should consist of a gentle jog followed by light stretching.

Medical Perspectives – The Physician's Responsibilities

First of all, the most important role of a physician is to *supervise and coordinate injury prevention*, to implement preventive measures both for the team and for each individual, and to asses the effectiveness of the employed measures.

Wherever possible, teams should be encouraged to employ the services of athletic trainers or physical therapists who can assist the team physician in instituting and monitoring pre-participation fitness programmes [30] Furthermore, better *education* of coaches and players regarding injury prevention strategies is the responsibility of the physician in order to help them include such interventions as part of their regular training programmes and to increase general injury awareness among team members [29, 31].The team physician should help players and coaches/athletic trainers by means of *load control*, especially in the off-season. This includes detailed testing of cardio-

respiratory functions (using treadmill tests) and isokinetic dynamometric tests on the muscles of the lower extremities before starting and at the end of the pre-season conditioning period. Where appropriate, a previous injury should be addressed with an exercise programme.

The regular *pre-participation examination* is a basic component of football players' care and thus provides the best opportunity for individual injury prevention. The main goals of this examination are to assess (evaluate) overall health, detect conditions that may cause injury or that may disqualify the player from participating in the game, assess fitness, and make recommendations for the exercise programme. When a player signs with a new club, a similar screening should be performed, not only to avoid legal conflicts but also with the same goals as the pre-season examination [22, 30]. These examinations may include on demand (e.g. a recurrent overuse injury) a biomechanical analysis, which can help identify possible injury risks and the need for orthotic devices as necessary.

To minimise frequency of *re-injury*, new injuries must be addressed and managed properly, with *rehabilitation* of the appropriate intensity, quality, and length. Players must not be allowed to return to football until injuries are healed, range of motion is restored, and strength has been recovered. Proper rehabilitation may break the injury/re-injury cycle but only when the programme emphasises and attains a return to full function, not just symptom relief. The team physician must make sure injured players follow the rehabilitation prescription [14–16, 32].

Another important medical perspective is *nutrition and hydration*. Proper nutrition is important while good hydration is a fundamental factor in performance. A balanced diet is what should be aimed for: carbohydrates are important for re-fuelling muscles, protein re-builds muscles, and vitamins and minerals are required for a number of reasons related to recovery. If players become dehydrated, then less blood will flow through muscles, and the muscles will be more prone to injury. This should be avoided by using isotonic liquids before, during, and after sports activities for water intake.

Not only physicians but everyone on the club's staff should strictly follow anti-doping and anti-drug policies. Players must not be allowed to use steroids or any other performance-enhancing *drugs*! Be aware of the use of alcohol and recreational drugs.

Players' Responsibilities

Players must *co-operate* with conditioning programmes and coaches to improve the correct execution of sport-specific drills and techniques, wear protective equipment in all contact situations, and follow the rules in order reduce the risk of injury. They are obliged to report all, even minor, injuries to the coaching and/or medical staff and should show full compliance with

any injury treatment or rehabilitation programme. They should gain a professional, injury-conscious *attitude* without losing competitiveness – thus they should play hard but fair!

Each player should have and use *equipment* of good quality and condition. *Shoes* are the most important of any sports equipment in the prevention of overuse injuries to the lower leg. The ideal football boot should have a rigid heel counter, good depth in the upper, a flexible forefoot, a wide sole, and be slightly curved in shape; it should absorb shock well, be light and flexible, and have enough but not too much grip on the actual surface (different boots with different studding for different surface and weather conditions). If there is not enough support to the arches, insoles are recommended [22]. However, football boots should be used for sport-specific training sessions only; they are not suitable for other purposes, such as, for example, endurance training (running).

The use of *shin guards* is one of the first preventive measurements applied in football. They were introduced to reduce acute contact injuries to the shin. They cannot prevent all types of shin injuries (e.g. fractures) but can reduce their severity [19].

Ankle protection (braces and taping) is another evidence-based preventive measure, hindering the development of acute sprains and thus the development of chronic instability as well [2, 14, 15, 18, 25]. Knee braces are not applied as often for prevention but mostly to reduce the adverse effect of a previous ligament injury.

Clubs' Responsibilities

The management of a club must *provide all fundamentals* (human resources and financial background) to enable the ideal injury prevention concept to become practice. Clubs must realise the importance of their players (the player is the club's asset) and follow the total loss approach, avoiding injuries. Clubs should be totally committed to optimising the medical welfare of players of all ages and abilities. To ensure the health and safety of players, every effort must be made to further advance the practices involved with the prevention of injuries.

Each club should build a professional staff responsible for the management and prevention of sports injuries, capable of effectively assisting the coaching and conditioning staff in controlling the workload, and who can individualise the conditioning programme. At least one member of this staff should be present at all practices and all games. It is the club's responsibility to provide enough financial support to allow the medical team to work properly and effectively [22].

Good *playing-field conditions* represent another of the club's responsibility. Pitches should be well maintained and be free of holes or other hazards.

Clubs should prefer playing fields covered with natural grass over artificial turf, for there is a higher incidence of injuries reported on artificial playing surfaces [14–16, 25].

Officials' Responsibilities

All officials concerned with the game must keep in mind the health and safety of the players as the overall first priority. The sport's governing bodies and their delegated officials must have an *injury-awareness approach* and should continuously promote player safety at all levels. Football rules should be continuously evaluated to see whether they could be altered to help to reduce the frequency of injuries. Changes, which could be applied specifically to different genders or age groups, should be strictly enforced and then re-assessed, whether they have led to the desired decrease in the number of injuries or not.

Reviews by the International Football Federation (FIFA) *referees* have already required them to use stricter judgement on player-to-player challenges relating to the use of elbows and tackling from behind. It is expected that these changes will reduce the overall number of injuries [33].

Step Four: Controlling the Effects of the Employed Measures – Re-Assessment

Evaluate the effects of preventive measures and implemented management plans by re-assessing the incidence of injury by repeating step one. This step is often overlooked, but in fact, this proves if any measure had a real preventive effect. Furthermore, without re-assessing the incidence of a certain injury, methods of prevention cannot be improved.

Most Important Preventive Measures

After reviewing current available literature, we can state that the implementation of a *complex injury-prevention training programme* is an effective strategy for addressing multiple risk factors. In fact, these programmes seem to decrease or eliminate risk factors associated with the largest number of frequent football injuries (Table 3). However, incorporating elements of a complex prevention strategy is not all that can and should be done. Considering special problems of male and female soccer and youth, adult, and senior levels, physicians have to assess the injury problem further by means of the 4-step *sequence of prevention*. By so doing, we not only provide individualised help to our athletes but also contribute to the establishment of a broader, more evidence-based knowledge on the nature and prevention of injuries in football.

Table 3. Features of a multi-modal strategy for the prevention of football injuries. *ACL*, anterior cruciate ligament

Preventive measures	Addressed risk factor/effect
Cardiovascular stamina training	Better oxygen supply Less fatigue – improved stamina
Proprioceptive training including: - Sport-specific agility drills - Core proprioception - Lower-extremity proprioception	Better neuromuscular co-ordination: less injuries associated with landing and pivoting drills More muscular support to joints Fewer ACL and ankle injuries
Stretching	Increased muscle flexibility Muscles become more resistant to passive forces Less muscle injuries
Muscle strengthening – thigh muscles	Better hamstring-to-quadriceps power ratio (at least 2:3), better muscular balance Fewer injuries to thigh muscles
Muscle strengthening – lumbar spine and back muscles	Better muscular balance; stronger deep back muscles Less low-back pain Less groin injuries

Conclusions

Although it is considered to be the cornerstone of the prevention cycle, epidemiological information on football injuries is inconsistent and far from complete. Documenting incidence of and exposure to football injuries is vital in identifying the most serious problems and detecting their risk factors in order to design proper preventive measures [12].

In the future, well-designed, randomised studies are needed on preventive actions and devices that are in common use, such as pre-season medical screenings, warming up, proprioceptive training, stretching, muscle strengthening, taping, protective equipment, rehabilitation programmes, and education interventions (such as increasing general injury awareness among team members). The effect of a planned rule change on the injury risk in football could be tested via randomised, controlled trials before execution of the change [34].

Because the aetiology of football injuries may differ between different subgroups of the football population, different subgroups of football players (especially females and juniors) may need different prophylactic programmes to achieve a major reduction in the incidence and severity of injuries. Establishing a good surveillance system is inevitable from this point of view as well. Through this type of analysis, risks can be identified systematically and objectives and performance standards can be established and prioritised.

The practical implications of preventive strategies need to be thought through so as to avoid conflict with other demands in football. However, in this very challenging and vital quest to reduce injuries, every participant around the game – from players to coaches, from physicians to physiotherapists, and from governing bodies to clubs – must follow their well-defined roles.

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Organisation of a Professional Team's Medical Staff and the Physician's Role

Piero Volpi

Introduction

As in many other top-level sports, in recent years, professional football has been building up a relationship between sport and medicine which has grown considerably in terms of organisation, regulations, and general care. During the 1980s and 1990s, Italian professional clubs began, both on their own account and to meet their legal obligations, to secure the occasional and eventually continuous presence of a physician specialised in sports medicine to handle the medical side of their activities. Previously, it had in many cases been the club's masseur who set out to advance from simply treating player's muscles to caring for their health in general. Today's staffs have gradually grown to include a psychologist, a physiotherapist, a chiropractor, and other practitioners, together with a clutch of consultants that can themselves be said to constitute a team in the wings rather than on the pitch.

A club's chief physician is faced with many exacting tasks: organisation and supervision of the whole of its medical sector, assessment of the psychological and physical fitness of its players when they join the club and throughout the season, introduction of measures whereby illnesses and lesions can be prevented, and diagnosis and treatment of injured players, including their first-aid management. Furthermore, in conjunction with the trainer and the coach, the physician must monitor each player's performance and elaborate the training programmes, supply diet charts and give advice on eating habits, oversee the use of drugs and integrators not prescribed and authorised by the physician, undertake health investigations required by sports regulations, and be present during anti-doping tests.

Organisation

The way in which a club's medical sector is organised will be determined by its budget and the approach adopted by its directors. The model we have elab-

orated and applied for a club with a high Italian and European standing (Fig. 1) provides for the establishment of a scientific reference and supervising committee for the sector as a whole.

This is comprised of a president and four members, all renowned academics skilled in sports medicine: an internist, a haematologist, a cardiologist, an expert in legal medicine, and a traumatologist. The physician in charge (1) is an expert in orthopaedics, traumatology, and sports medicine; (2) runs the sector; (3) oversees its operations; (4) intervenes whenever summoned to deal with particularly serious and/or urgent questions. He and another sports medicine specialist are the official doctors of the club's first team and take care of the health of a roster of 24–28 players per year.

A medical secretary handles the bookings and due dates for the specialists' examinations and liaises with the auxiliary medical facilities and consultants. A psychologist assists the staff for specific purposes. Two masseurs, three physiotherapists, and a chiropractor work every day under direct medical supervision. One of the two coaches on the staff is responsible for the recovery of players injured in the gymnasium and on the pitch. The training

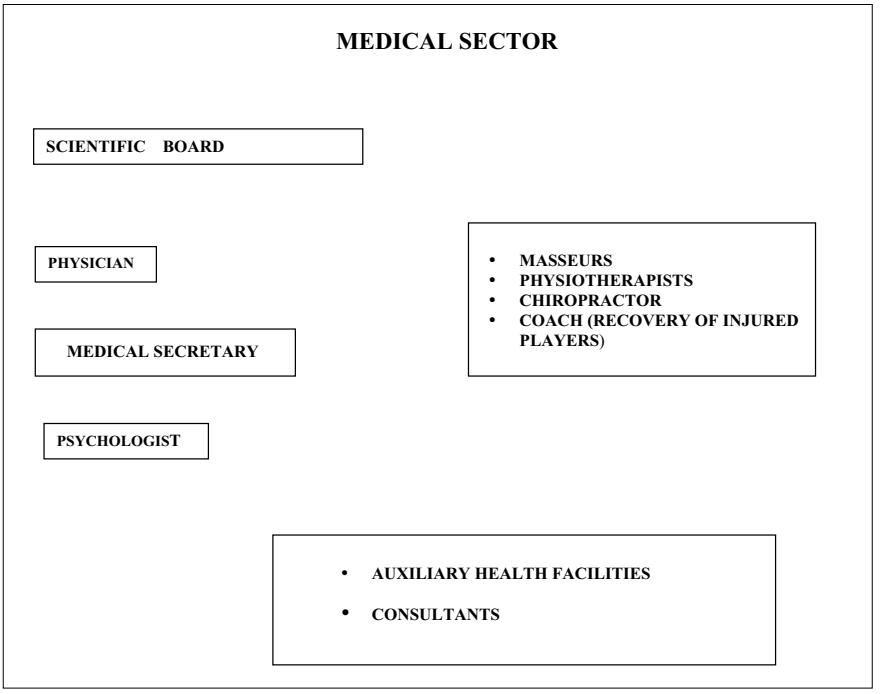

Fig. 1. Organisation of the medical sector

grounds comprise playing fields, changing rooms, accommodation for players and staff, restaurant, bar, assembly rooms, and more, together with a practitioner's surgery, a physical therapy room, and a gymnasium with the usual equipment.

The auxiliary facilities and consultants constitute a carefully chosen external service to which the club's medical staff and its players can turn to at any time. The facilities comprise:

- A sports medicine institute concerned with the legally obligatory yearly match-fitness examinations;
- An orthopaedics institute that provides first-aid services, traumatological evaluation and care, a radiology and instrumental diagnostics service, and carries out laboratory examinations;
- A pluri-specialist hospital for internal medicine and neurosurgical consultations;
- A legal medicine institute handling insurance and doping issues;
- A rehabilitation and applied biomechanics centre for the evaluation of newly acquired players and the elaboration of re-education programmes;
- A dentist for evaluation and care as required. The second-tier consultants include a dietologist, an ear nose and throat (ENT) specialist, an oculist, a dermatologist and the like.

The Italian sports regulations require a first-team physician to be responsible for the health, organisation, and care of junior teams.

Evaluation

Compulsory Evaluation

As already mentioned, Italian law requires all professional footballers to undergo a yearly match-fitness examination at an authorised sports medicine centre, with updates at least every 6 months (Law No. 91 of 23 March 1981; Ministerial Order 13 March 1995). Compliance with this obligation is seen to by the team doctor's duty. The examination consists of a general medical assessment, a cardiological evaluation including a resting and maximum-effort electrocardiogram (ECG), an echocardiogram (every 2 years), a chest x-ray (first visit), a spirogram, vision and hearing evaluation, and blood and urine tests (every 6 months). Other specialist examinations are conducted if a clinical suspicion is aroused. All the documentation is collected by the physician in charge and stored in the club's files. It is also summarised on the player's health record, which shows that the examinations have been carried out and that the player is fit. It accompanies the player throughout his or her career.

Recommended Evaluations

Further evaluations are undertaken to provide a fuller picture of a player's physical condition and traumatological history. These include (especially when a player first joins a club):

- Orthopaedic assessment and traumatological examination;
- Instrumental examinations consisting of radiography, magnetic resonance imaging (MRI), ultrasound (US), and other instrumental examinations of areas considered at risk or indicated by the individual player;
- Postural and dental analysis is recommended for an overall evaluation;
- Biomechanical assessment of the lower limbs (isokinetic, explosive force, and coordination tests);
- Field tests arranged with the coach.

One of the hardest tasks for a team doctor is to assure the club's directors and technical staff that a new player's bones, muscles, tendons, and joints are in perfect shape. An orthopaedic examination cannot always determine total fitness, especially now that accidents are becoming more frequent, particularly among professional footballers. Take, for example, a player with a reconstructed anterior cruciate ligament. Functional efficiency will certainly have been restored, but the anatomy of the knee is no longer the same. Our work with an elite Italian club showed that the incidence of spondylolisthesis with spondylolysis in 80 new players from various parts of the world over the course of 5 years was about twice as high as in the normal population. Furthermore, an echographic study of the patellar tendons of 12 of the club's new players at the start of the 1999–2000 season showed that 5/24 tendons displayed evident but asymptomatic abnormalities. As it turned out, none of these players reported patellar tendon pain during the season. It is very difficult, even for an expert traumatologist, to rule out the progression of such abnormalities on first inspection.

Prevention

Injury prevention is a sports doctor's principal objective. A distinction can be drawn between general and specific prevention. General prevention involves keeping an eye on a player's overall health through periodic, routine examinations; establishing work loads and training plans in conjunction with the trainer and the coach; elaborating individual prevention plans for players at risk for muscle, tendon, joint, and other types of injury; advising the use of protective devices such as taping, shin pads, and plantar arch supports; monitoring lifestyles (sleep, use of tobacco and alcohol, eating habits) and elabo-

rating diet and supplemental regimens; checking hygiene and accident-prevention measures in changing rooms, gymnasia, with equipment, and the playing fields; and planning vaccinations (especially anti-influenza, anti-tetanus, anti-typhoid, and anti-hepatitis A and B) since athletes are exposed to a high risk of infection.

Specific prevention requires the prompt perception of any departure from a player's optimum state of health so that appropriate treatment can be started. In this respect, the doctor can be viewed as an integral part of the team and hence his or her assiduous presence is necessary during training sessions, matches, pre-match retreats, and pre-seasonal gatherings.

Care

If a player has an accident or becomes ill, the team doctor must take care of that player and set about the process of diagnosis and treatment, if necessary with the assistance of the auxiliary facilities and the consultants. The physician must also follow the course of the injury or disease and make sure that effective therapeutic measures are being applied. This is achieved through:

- Medical and instrumental diagnosis;
- Immediate and subsequent care;
- Non-operative, surgical, rehabilitative management;
- Active repose;
- Recovery;
- Resumption of sport.

In the case of bone and muscle injuries, the clinical cure achieved by surgery or rehabilitation procedures is not enough to ensure certain resumption of sport, and a functional cure must be sought. In other words, the healing processes must be adapted to the specific stresses each sport requires. A clinical cure and medical approval of resumption are thus the prelude to three periods: differentiated field work, field work with the rest of the team, return to competition.

Three aims are pursued in differentiated field work: complete athletic recovery and an increase in stamina, muscle power, coordination, and other capacities; adaptation to specific exercises with the ball; and psychological recovery. The amount of time required will obviously depend on both the subject and the type of injury. Field work with the rest of the team sets the seal on full recovery of function. Guided by the trainer, the player will take part in the team's tactical exercises and become ready to play in minor matches and friendlies. Return to the field for a footballer corresponds to fully effi-

cient participation in an official match. It is the moment when the player feels truly recovered and is once again on the football scene. The team doctor is also required to handle the medical and insurance paperwork until the player is completely cured.

Doping

In addition to being an ethical issue and a question of loyalty among sports persons, the fight against doping must be pursued to safeguard the health of footballers, as in any other sports sector. Soccer has unfortunately been involved in doping episodes in recent years despite the fact that it has always been regarded as a sport in which a player's technical skills, dexterity, and a flair for tactics counted for more than physical constitution.

Now, however, winning is the be-all and end-all of every match and event, whether junior, amateur, or professional. In professional football, too, a calendar crowded with matches with a *high economic value*; the brief recovery times between one match and the next; and the excessive physical development required, as in all modern sports, are obvious risk factors. The constant drive for an ever better performance must be rationalised and perhaps channelled into better planning of match calendars, the elaboration of new training methods, and the devising and application of innovative solutions in nutrition – not in the provision of pharmacological support.

The Italian Footballers Association (AIC) has long been engaged in protecting the rights of footballers. It has discussed programmes, regulations, and questions of organisation in all the institutional forums but has concentrated its efforts on instructing and informing players. It has striven for clear, correct, and direct information and advised greater vigilance and consciousness-raising with regards to doping by promoting educational programmes through dedicated meetings, articles, teaching material, assessment questionnaires, and the like. After a congruous training period, the AIC has been pleased to find a greater awareness and understanding of health and doping among footballers. After much discussion of the scientific, legislative, and organisational sides of the question, blood and urine tests after Premier League and first-division matches came into force in Italy in 2003. The AIC approved this project, and its members readily and knowledgeably gave their consent. The constant increasing awareness of these topics on the part of players is probably the best foundation for future strategies.

We realise that future projects will seek closer co-ordination of anti-doping measures, at least in Europe, among the nations that take part in Continental soccer events. In addition to the European Cup series, therefore, there will be a better equilibrium in the number and means of testing in

national matches. The other priority is agreement with the dictates of the World Anti-Doping Agency (WADA) through the national institutions – the Italian National Olympic Committee (CONI) and the Italian Football Federation (FIGC) – namely, that preference should be given to unannounced (surprise) testing in a non-competitive setting as opposed to the numerous expected checks carried out after matches.

Injuries in Women's Football

Lars Engebretsen, Karin Steffen

Background

Whereas men's football has had a long and distinguished history, the same cannot be said for women's football. The sport was even banned for women in Europe by the European Football Association (EUFA) in 1921; the ban was finally lifted in 1971. The first European championship for women was held in 1982 in Italy and the first World Cup in China in 1991. An important development took place in the United States in 1972 when Title IX was enacted within the National Collegiate Athletic Association (NCAA) in an attempt to equalise the number of men's and women's athletic teams in the United States' collegiate environment [1]. This had a dramatic effect on the creation of women's football programmes. In 1896, there were only 230 such programmes in the NCAA; by 1997, there were 696. By 2005 in the United States, 43% of active football players were women, and it is estimated that 12.5–18.5 million people play soccer, with an estimated annual growth of approximately 22%. Women's football has now established itself as a sport at the highest level. Worldwide, women players account for approximately 22% of active players although this number is increasing. In the Scandinavian countries, football is the most popular women's team sport and the overall second-largest sport after men's soccer. In 1997, 204,000 Swedish football players were licensed, of which 20% were women, and this represents an increase of 7% since 1995 for female football players [2]. Currently, over 100,000 female football players are registered in the Norwegian Football Association (NFF), which accounts for about 25% of all players. From 2003 to 2004, the NFF saw a 21.9% increase in the number of registered female football players [3] (Fig. 1).

Physical Demands on Football Players

Football demands a combination of endurance running and discontinuous sprinting. There is reliance on lower extremity and trunk strength but also on

Fig. 1. A 21.9% increase in female youth football in Norway in 2004 [3]

balance and muscle coordination. A female athlete runs approximately 8–10 km during a game. The average sprint is 15 m long with an average of 100 sprints a game. The average percent of body fat is between 19–26% as opposed to approximately 15% in men. Until puberty, the physical and psychological differences between male and female football players are small. Puberty starts approximately 2 years earlier in girls (10–11 years of age) than boys (12–13 years of age) [4]. This means that girls develop earlier, which may indicate that they can sustain higher training intensity at an early age. During adolescence, girls develop better stamina and co-ordination, while boys develop better strength. The average maximum oxygen consumption is lower in women than in men (women have 70% of men's values) due to differences in body size and composition. The difference in capacity in relation to body weight is approximately 15%, but the difference is only 5–10% when comparing fat-free body weight. Even though men have higher overall work capacity, they have shorter time to exhaustion compared with women, at an intensity of 70–80% of maximum oxygen capacity. This is probably caused by an increased capacity in women to utilise fat consumption during work and thereby reduce the reliance on carbohydrates. Women have approximately 70% of men's muscle strength in the lower extremities and 50% in the upper extremities. However, much of this is dependent on the fact that women are smaller in size than men. If one compares strength per square muscle area, no difference can be detected. In addition, there seems to be no difference between genders when it comes to relative development of tiredness or the individual's experience of tiredness.

There are a few areas where women are different from men that are important to know from a sports medicine view on football. Women have lower haemoglobin concentration in general than men, and it has been estimated that approximately 10% of female athletes have iron values below the normal standard, leading to lower-than-wanted haemoglobin. As in regular athletic women who do high-intensity training, female football players also have menstrual disturbances. It has been shown that a combination of menstrual disturbances, low calorie intake, and low body fat may lead to an increase in stress fractures [5].

Incidence of Football Injuries in Women

Football is a contact sport, associated with a large number of injuries for both genders [6]. Of all injuries treated in emergency wards in Norway, 17% are sports injuries. Of all these, about one third is due to football: 42% of all injuries in men and 15% among women [7]. The many injuries in football are not necessarily due to the distinctive character of the sport but also to the fact that football is by far one of the most popular sports, with the largest number of active players.

Injuries in male football have been the subject of many studies, reporting a high incidence of injuries compared with other sports, but few studies exist concerning injuries in adult women's football and youth football.

It cannot be deduced whether the overall incidence of injury differs between male and female players because of limitations in studies and methodological problems, such as differences in injury definitions and study design. However, an interesting general development seems to have occurred from the early 1980s to the early 2000s. Studies from the world's largest youth tournament, the Norway Cup in 1984, revealed twice the incidence of injuries needing medical treatment in girls compared with boys [8]. A similar study in 1993 and 1997 (with identical study design) showed a 35% overall decrease in injury incidence, the majority of which occurred in girls [9]. Girls had more than 50% reduction in injury incidence in 10 years. The Authors speculated that this could be caused by the fact that young female football players in the 1990s were exposed to more football up through the years and therefore technically were better players than they were in the 1980s. The fact is that recent studies on injury incidence have shown little or no difference in the total injury rate among genders.

Whereas two studies of Scandinavian men's élite football reported injury incidence data to be 29 per 1,000 match hours [10] and 25.9 versus 5.2 per 1,000 match and training hours, respectively [11], two Swedish studies [2, 6] found the injury incidence in élite- and non-élite women's football to be 24

and 14.3 injuries per 1,000 match hours and 7 and 3.7 per 1,000 training hours, respectively. Soderman et al. [12] found the overall injury incidence rate (traumatic and overuse) among adolescent female players to be 6.8 per 1,000 hours of football (training and match). Injury incidence data from U.S. women's professional football was registered as 1.9 per 1,000 player hours (12.6 and 1.7 during match and training, respectively) [Women's United Soccer Association (WUSA)] [13]. Finally, as with international male players, data from top level athletes suggest a higher injury rate. Data from the World Cup in the United States in 1999 and the Olympic Games in Sydney, Australia, in 2000 shows an injury rate of 38 and 64 per 1,000 playing hours, respectively [14] (Table 1).

Although the general injury rate is about the same, however, the injury pattern seems to differ between genders. Serious knee injuries, such as anterior cruciate ligament (ACL) injuries, are of particular concern in female sports. The highest incidence is seen in adolescents playing pivoting sports, such as basketball, team handball, and football, and the incidence of an ACL injury is 3–7 times higher in women than in men [6, 15–17]. Women also injure their knees at an earlier age than men [15, 18, 19]. The risk for an ACL injury in junior football (15–19 years) has been estimated to be 5.4 times higher for girls compared with their male counterparts [17, 19].

The most common injuries leading to absence from match or training are knee injuries (26%), followed by foot (12%), ankle (11%), and thigh [2]. In a study from US high school sports, Powell and Barber-Foss [17] observed a slightly different injury panorama among female youth football players, with 33.5% ankle/foot, 25.8% hip/thigh/leg, and 19.4% knee injuries. In female youth players, contusions (45%), sprains (25%), and lacerations (14%) are the main causes for medical attention in a large tournament [8]. Soderman et al. [20] found a similar injury incidence in Sweden in adolescent female players, and 42% of their injuries occurred in the knee and ankle. Ankle sprains were the most frequent injury (22%), and more than half of those were re-injuries. Fourteen percent of their 79 reported injuries were classified as serious, leading to more than 30 days away from the sport. In Swedish élite players, the injury pattern was similar although the overall rate was three times higher: 49% of injuries were located in the knee and ankle, and 15% of their 78 injuries were classified as major [6]. The NCAA data base shows that lower-extremity injuries are the most common, with the knee and ankle as the main regions. Concussions were the third-most frequent region and four times more common in women than men. Stress fractures were more common in women, with a ratio of 3.5:1. The main finding from this study was the fact that non-contact injuries to the ACL were almost 6 times higher in women compared with men during games [15]. In the US professional league, the knee (32%) and head (11%) were the most common regions, with again

injuries to the ACL being a major problem. International Football Federation (FIFA) data from major tournaments (World Cup 1999 and Olympic Games 2000) are limited to only 60 injuries altogether and suggest that head and lower leg are major regions of concern [14]. All these studies underline the high exposure of the lower extremities to injuries in football.

The high incidence of injuries among female football players constitutes a significant problem, both for the player, for her club, and in a socioeconomic perspective. A major knee injury will in many cases causes severe consequences, such as an interrupted career, but the risk for early knee osteoarthritis with permanent disability for the player must also be considered [6, 21, 22]. Consequently, there is every reason to emphasise the prevention aspect of injuries in football and to develop and implement prevention programmes for young players as early as possible.

Multi-factorial Aetiology for Soccer Injury

As a first step towards injury prevention, the causes must be established. This includes information on why a particular athlete may be at risk in a given situation (i.e. risk factors) or how injuries happen (i.e. injury mechanisms). Murphy et al. [23] recently reviewed the literature on risk factors for lower-extremity injuries, demonstrating that our understanding of injury causation is limited. Many risk factors have been implicated; however, there is little agreement with respect to the findings. Partly, this can be attributed to limitations in study design and the statistical methods used to assess the results. Murphy et al. [23] conclude that more prospective studies are needed, emphasising the need for proper design and sufficient sample sizes. Meeuwisse's dynamic, multi-factorial model divides causes in intrinsic and extrinsic risk factors and describes the injury mechanism for the inciting event [24] (Fig. 2).

A review of the literature shows that there are hardly any studies that have made use of a multi-factorial approach. Risk factor analyses of football injuries show that higher age and the number of years one has played increases the risk of injury among men and women [2, 25, 26]. A high percentage of re-injuries are reported in both male [27–29] and female players [12]. Hormonal influences have been linked to injuries in female athletes although with conflicting results. Studies have shown an increasing risk of ACL injuries in the late luteal phase [30] and the ovulation phase [31]. The female athlete triad, with menstrual disturbances, disordered eating, and premature osteoporosis, has been hypothesised to increase injury risk (stress fractures) [1, 5]. Other risk factors discussed in women are the following: lack of physical fitness, joint laxity, lower-extremity strength, and muscular imbalances [20].

Table 1. Epidemiological studies on incidence for all injuries in females

Reference Design, country, and period	Population	Injury recording	Injury definition	Injury incidence per 1,000 h	
				Game	Practice
Giza et al. [13] Retrospective cohort USA, 24 months	Elite *n*=202	Database of league insurance company	?	12.6 Overall traumatic: 6.2	1.2
Soderman et al. [20] Prospective cohort Sweden, 7 months	Youth *n*=153 15.9±1.2 years	Physical therapist by personal visits and phone contact in co-operation with the coaches, injury protocol	Time loss = absence from at least one practice session Traumatic: sudden onset Overuse: gradual onset without any known trauma Player-defined injured until she was able to participate fully in games and/or practice sessions	9.1 Overall traumatic: 4.4 Overall traumatic and overuse: 6.8	1.5
Ostenberg and Roos [2] Prospective cohort Sweden, 7 months	Elite and amateur *n*=123 20.7±4.6 years	Physical therapist connected to the team Phone contact by the author	Time loss = absence from at least one practice session Traumatic: sudden onset Overuse: gradual onset without any known trauma Player-defined injured until she was able to participate fully in games and/or practice sessions	14.3	3.7

Reference Design, country, and period	Population	Injury recording	Injury definition	Injury incidence per 1,000 h	
				Game	Practice
Engstrom et al. [6] Prospective cohort Sweden, 12 months	Senior elite *n*=41 21 years	Four medical students	Time loss = absence from at least one practice session	24	7
Maehlum et al. [9] Prospective Norway, 1 week	Youth	Tournament medical staff	?	Overall traumatic: 8	
Maehlum et al. [8] Prospective Norway, 1 week	Youth *n*=c. 3,900	Tournament medical staff	?	Overall traumatic: 17.6 (per 1,000 player hours)	

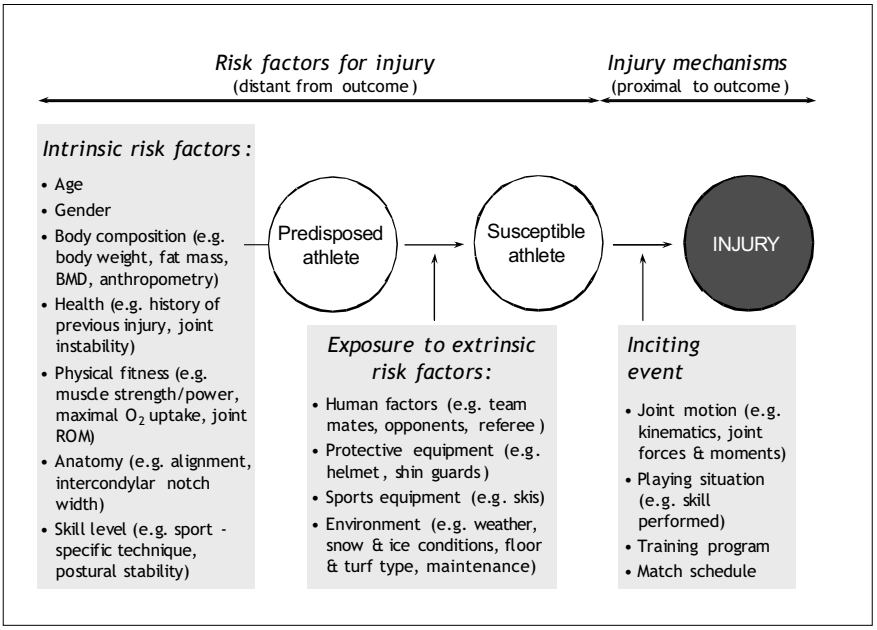

Fig. 2. Potential causes of athletic injuries (Meeuwisse 1994, modified by Bahr and Holme 2003 [24]). *BMD*, bone mineral density; *ROM*, range of motion

Some studies indicate that general joint laxity, mechanical instability, and functional instability may be factors pre-disposing for injury [2, 27, 32, 33].

Prevention of Football Injuries

Although football is one of the world's most popular sports and the risk of injury is high, surprisingly little has been done to prevent football injuries in general, and injuries in female players in particular. So far, only eight studies have been carried out on a world basis – only two of them in women's football – even though the first study was done as long ago as 20 years and with very promising results [34]. In this study, the intervention group sustained 75% fewer injuries than the control group. In the following years, a small number of intervention studies in men's football were published. Research groups have used different prevention approaches, like orthoses, balance training, and eccentric hamstring training, and all reported a reduction of injuries after the intervention period [32, 35–39].

We do not know if this knowledge obtained in studies of men's football

can be transferred to women. In women's football, even less scientific work has been done, and until now, there are only two studies published on female football players alone [12, 40] and one study with team sports athletes including female football players [41]. These studies focused on the prevention of injuries in general [40] or on specific types of injuries, such as ankle and knee injuries [12, 41] (Table 2). Heidt et al. [40] examined the effect of a 7-week pre-season training programme among 300 female football players at the high school level (aged 14–19 years) in a non-randomised study. Of the group of 300 players, 42 went through a training programme consisting of warm-up exercises, plyometrics (jump training), and strength and flexibility training. The training group had significantly fewer injuries than the control group. However, the results must be evaluated with caution due to the low study power. Soderman et al. [12] examined the effect of balance-board training among senior female football players on various levels. Players were randomised to training on a balance board daily for 30 days then 3 times a week during the season, or to the control group, which trained as normal. In contrast to the results of Caraffa et al. [36] who performed a similar study on men, Soderman found more ACL injuries in the intervention group than in the control group. A conclusion of these results could be that balance-board training alone, at least based on a home-training programme, is not sufficient to prevent ACL injuries in women. Again, the power of the study was low, which suggest careful interpretation of the results. Hewett et al. [41] evaluated the effect of neuromuscular training on the incidence of knee injuries in female team-sport players where 290 were football players. They found a trend towards a higher incidence of ACL and medial contralateral ligament (MCL) injuries in untrained female players compared with trained female players. A large, unpublished study on the prevention of overall injuries focusing on proper landing techniques and strength suggests a major reduction of ACL injuries in female football players among adolescents and later among NCAA players in the United States [42]. This study is important since a high number of study participants strengthens the power and overall design such that results may be viewed with a certain amount of optimism. Three recent studies with the goal of reducing injuries in team handball showed remarkable results [21, 43, 44]. These studies showed that training programs highlighting co-ordination and strength excercises reduced the knee injuries by approximately 50%. However, the transfer value from handball to other team sports may be limited since injury mechanisms vary between sports. There are less ACL injuries in football players compared with handball players and less non-contact ACL injuries in football compared with handball [13, 21] and basketball [45] even though females football players sustain more ACL injuries than male players [41, 45].

Table 2. Injury prevention studies designed to reduce the risk of injuries in the lower extremities

Reference Design Type of prevention	Level Country	Study group	Injury definition and registration	Comparison of intervention	Follow-up	Results
Heidt et al. [40] Prospective cohort Pre-season conditioning with strength, running, and plyometrics, primary	High school USA	*n*=300 *n*=42 players in IG *n*=258 players in CG Age: 14–18 years	Acute and overuse: time loss Athletic trainer: injury form (reported at the end of the year)	IG: 7-week training program: sport-specific conditioning, plyometric training, sport-cord drills, strength and flexibility exercises 1–2 times weekly during preseason CG: Train and play as usual	1 year	Significantly lower injury incidence in IG
Soderman et al. [12] RCT (of teams) Balance board, primary	Div. II–III Sweden	*n*=221 7 teams (62 of 121 players) in IG 6 teams (78 of 100 players) in CG Mean age: 20 years	Acute: time loss Coach: injury form (regular personal visits and telephone calls)	IG: 10–15 min balance board initially each day at home for 30 days, then 3 times per week during the rest of the season CG: Train and play as usual	7 months (April – October)	Balance-board training did not reduce the number, incidence or type of acute injuries of the lower limbs. A significantly higher incidence of major injuries was seen in IG

Reference Design Type of prevention	Level Country	Study group	Injury definition and registration	Comparison of intervention	Follow-up	Results
Hewett et al. [41] Prospective cohort Plyometrics and weight training, stretching, primary	High School: volleyball soccer basketball USA	*n*=290 *n*=97 IG *n*=193 CG Age: 14–18 years	ACL and MCL Athletic trainer: injury form (reported weekly)	IG: 60-90 min neuromuscular training (flexibility, plyometrics, weight training) 3 times weekly during 6 months in pre-season CG: Train and play as usual	1 year	A trend towards a higher incidence of ACL and MCL injuries in CG compared with IG

IG, intervention group; *CG*, control group; *RCT*, randomized controlled trial; *ACL*, anterior cruciate ligament; *MCL*, medial contralateral ligament

Conclusion

In conclusion, there is still a lack of studies in injury prevention among female footballers, and one reason could be that there is limited knowledge of the causes and mechanisms of injuries, which again makes it difficult to develop targeted preventive measures. There is a need for increased focus on the prevention of injuries in football in general and in women's football in particular.

A specific training programme called F-MARC 11 has recently been designed by an expert group within the FIFA Medical Assessment Research Centre (F-MARC) in order to prevent the four most common injury types in football, i.e. injuries to the ankles, knees, groin, and hamstrings, respectively. The programme is developed on the basis of established principles for rehabilitation of such injuries. It is a time-efficient programme (15–20 min) and is designed to be part of a warm-up period at each training session. F-MARC 11 should replace similar exercises, which are often done during warm-up. The programme includes the ten exercises within core training, balance training, dynamic stabilisation, and eccentric hamstring training.

There are now ongoing projects in Switzerland (senior football, men's and women's) and in Norway (female youth football) using F-MARC 11 as a preventive strategy to reduce the high number of injuries in football.

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Referee Lesions

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Introduction

Referees are a fundamental group in the professional sports world. However, no articles about referee injuries have been published over the past few years. This article is an attempt to review the most frequent lesions in this group. In a telephone survey of Portuguese referees, we found that the most frequent lesions among this group were muscle lesions and ankle sprains. Those lesions occur especially in reverse running or sprinting and in most cases, at the beginning or end of the game.

Muscle Injuries

Muscle injuries are among the most frequent sport lesions in football – referees included. They can be caused by a direct blow (extrinsic) or, more frequently, an indirect mechanism (intrinsic). The bi-articular muscles are more prone to indirect injuries, as they can change from asynergy to violent contraction with simultaneous stretch due to hip flexion and knee extension. The muscles most frequently affected are the quadriceps (specially the anterior rectus) and the hamstrings.

The quadriceps is one of the most common site of running injuries. Different anatomical sites within this structure can be involved. A thorough examination of the extensor mechanism includes an assessment of quadriceps strength, contracture, and the position of the vastus medialis obliquus, as well as position, tracking, stability, and mobility of the patella.

Hamstring injuries are a frequent source of acute injury and chronic pain in referees. Hamstring muscle injuries primarily occur proximally and laterally, and they usually involve the biceps femoris. Changing direction is the most common cause of biceps rupture in two different ways: the biceps acts as an engine in valgus, flexion, external rotation with 5 antagonists (quadriceps, semi-membranosus, semi-tendinosus, sartorius, and gracilis). On the

other hand, the 5 antagonists are the engine in varus, flexion, internal rotation, and braking. Injuries often occur at the proximal myotendinous junction. In the biceps femoris, this junction extends over most of its entire length. Injury usually does not occur within the tendon itself unless there is a pre-existing pathology. Careful evaluation and correct diagnosis is crucial. Bony avulsion at the ischial origin may occur as well but usually is associated with sudden, large-force, hip-flexion injuries. Avulsions are commonly seen in people when the knee is extended and the hip is suddenly flexed.

The great majority of muscle lesions do not need surgical treatment and can be repaired with conservative measures. Nevertheless, surgery is indicated in specific cases. Reasons for surgical treatment are:
- Failure of conservative measures (orthopaedic, physiotherapy or others)
- Risk of long-term sport inactivity
- An acute lesion that became chronic.

Anatomo-physiology

The skeletal muscle consists of a contractile tissue with a tendino-aponeurotic skeleton. In the long muscles of the upper leg, the short disposition of the fibres creates a great isometric force but a poor length variation and shortening velocity. Myoaponeurotic and myotendinous transitions, where stiffness of the connective tissue opposes muscle tissue, are the weakest part of the muscle, being the region more frequently affected in muscle injuries.

Risk Factors

Several factors increase the risk of muscle injuries, among them, age, previous lesion, fatigue, inadequate warming up, bi-articular muscles, and a large quantity of type II fibres. A sports-related injury is most likely to occur when the referee experiences any change in use of the involved structure. This is a rate-dependent process. If training is within the physiological range, there is cellular homeostasis. Insufficient training is associated with disuse, which can cause a catabolic response that ultimately leads to injury. It is common that referees do not have enough time to train adequately. Transitional risks include: 1) improper training; 2) changes in equipment; 3) environmental changes, such as new surfaces or different altitudes; 4) alteration in frequency, intensity, or duration of training; 5) attempts to master new techniques; 6) a return to sports activity too soon after an injury with poor muscle recovery; 7) an unbalanced diet; 8) technical errors; 9) drug ingestion; 10) poor game-field conditions. Commonly affected muscles beyond quadriceps and hamstring are calf and adductors.

Diagnosis

Diagnosis of muscular lesions is clinical with image confirmation. Clinical observations are:
- Sudden pain;
- Dilaceration sensation;
- Functional incapacity;
- Inspection: swelling, bruises, and tension;
- Palpation: painful spot, depression, or muscular contraction;
- Pain on passive stretching or resistive contracture.

Imaging

An x-ray is mandatory in diagnosing a muscle injury because it can show a bony avulsion or a calcified myositis. Ultrasound is the test choice for studying the traumatised muscle. It detects haematoma, discontinuity of muscular fibres, muscle aponeurosis, or myotendinous rupture. Magnetic resonance imaging (MRI) is also highly sensitive and specific for muscle-lesion diagnosis. In MRI, muscle appears with intermediate density wrapped in low signal intensity (aponeurosis). Fat suppression enhances muscular signal. T2-weighted MRI is best for ruptures and T1-weighted MRI for haematomas. Indications for MRI are:
- Study of profound muscles
- Disagreement between clinical examination and ultrasound
- Professional level of the referee.

Acute Muscle Injuries

Direct trauma (extrinsic mechanism):
- Contusion

Indirect trauma (intrinsic mechanism):
- Contracture
- Distension
- Rupture.

Lesions near the insertion point cause large haematomas with large clots difficult to evacuate by aspiration. The only formal indication for surgical treatment of the hamstring is a complete rupture at or near the origin of the ischial tuberosity or distally at its insertion point (either a soft tissue avulsion with a large defect, a bony avulsion from the isquion with displacement superior to 2 cm or a bony distal avulsion). These must be operated and re-insert-

Table 1. Rodineau and Durey classification

Type 0	Type I	Type II	Type III	Type IV
Reversible	Irreversible in some muscle fibres with full integrity of the supporting connectin tissue	Irreversible with moderate involvement of connective tissue with no disorganisation	Muscular lesion with significant involvement of connective tissue	Total or near-total rupture
Moderate pain	Recovery in a few days	Selective pain with no immediate stopping during sport	Marked muscular disorganisation with localised muscular haematoma	Functional impairment
Muscle contraction with impaired strength		Healing in 10–15 days	Acute, severe pain with immediate functional incapacity	Surgery must be considered depending on location
Recovery in hours		Return to sporting activity after recovering muscle strength; stretching and resistive contraction with no pain	Recovery in 6–12 weeks	12–18 weeks of recovery
			Special care when recovering force and elasticity	

ed to avoid pain and residual loss of power and function. Late repair is more difficult because of scar tissue and adhesions. The use of anchors is helpful to obtain proper reinsertion. In rare cases, symptoms of sciatic dysfunction can appear, and nerve dissection is necessary.

The length of immobilisation is, however, dependent on the grade of injury and should be optimised so that the scar bears the pulling forces that

operate on it without re-rupture. Early mobilisation is required to regain original muscle strength and to achieve good final results in resorption of the connective tissue scar and vascularisation of the damaged area. Another important aim of early mobilisation, especially in sports activities, is to avoid atrophy and loss of strength and extensibility.

Surgical indications are very rare and include:

- Muscle hernia if painful and stretched between the aponeurosis;
- Complete and large muscle belly ruptures with large haematoma in high-level athletes;
- Traumatic, complete muscle dilaceration;
- Open injuries.

Surgical technique

Surgical treatment of muscle injuries is not comparable with tendon repair, specially the end-to-end rule. It is not necessary to suture the muscle end to end. It is important to remove clots, haematoma, and necrotic tissue and clean the cavity. Necrotic fibres have the ability to form an anarchic tissue, which can progress to calcification. The purpose of surgery is three-fold: decrease pain related to tension, avoid formation of an anarchic painful scar tissue, and achieve the best local tension to maintain muscle strength.

Chronic Lesions

Chronic muscle lesions are related to an event with a more than 3-month evolution period. Patients, either high-level or leisure-sport athletes, present after unsuccessful conservative treatment and often following several unsuccessful attempts to re-initiate physical activity and after a long period of inactivity. Another typical case is the patient with recovery, early return to sports, and re-injury. Re-injury rates from 5% to 77% are reported in the literature and are related with areas of calcification, inflammation, and scar tissue. Chronic lesions are: painful scar tissue, cysts, and myositis ossificans. Myositis ossificans can occur as a muscle ossification of a deep haematoma after trauma and must be distinguished from osteogenic sarcoma. It is often an asymptomatic radiographic discovery. It can be the result of repeated ruptures (in our series, from 1 to 12) or direct trauma and appears 3–6 weeks after the first accident. Usually, the diagnosis was not made, and the athlete had a premature return to sports activities. Rehabilitative treatment includes early and deep massage, active mobilisation, early return to sports, and infiltrations. The best treatment is prevention, and the first treatment measures are conservative without massage. Surgical treatment must only be proposed after failure of all the other procedures and always after "cooling" of the bone scan.

Treatment

Objective

Treatment objectives are to:
- Obtain global healing, it being important to achieve good vascularity, tissue innervation, and traction (strain) for the fibres;
- Avoid chronicity;
- Avoid recurrence.

Principles

Principles to be attained are to:
- Limit haematoma volume;
- Obtain early tension of muscular fibres;
- Utilise anti-fibrosis measures (ultrasound, decorin, corticosteroids, and platelet growth factors);
- Maintain treatment protocol for a longer period if lesion severity demands it.

Therapeutic protocol

The treatment protocol consists of:
- Rest, ice, compression, elevation (RICE) and muscle relaxants;
- Athermic physiotherapy, non-vibrating; draining massage at 48 h;
- Between 3rd and 10th day (according to lesion type), passive stretching and physiotherapy (heat and low-frequency current);
- Postural stretching, active isotonic contraction, and resistive contraction;
- Proprioceptive exercises;
- No-load exercise (bike, swimming);
- Return to normal activity with progressive intensity if no pain. Before maximum load, perform maximum stretching with no pain;

Treating muscular lesions and early mobilisation are beneficial because they:
- Increase initial haematoma;
- Increase the number of inflammatory cells;
- Increase absorption;
- Facilitate regeneration, achieving better fibre orientation.

Frequency

In our series, the frequency of muscle lesions represented more than 50% of referee injuries. Hamstring injuries represented 30% of lower-extremity injuries in sports medicine consultation over the last 3 years in persons aged

15–25 years and more than 50% of all muscle lesions. These injuries most often occur in sports in which the hamstrings can be stretched eccentrically at high speed with sprinting or running contact sports, such as football. Recreational sports, such as water skiing, in which the knee is fully extended during injury, also are common causes of hamstring injuries. In 256 individuals with hamstring injuries 74% of injuries were in football, 16% in track, 8% in rugby, and less than 2% in tennis, squash, ballet, and gymnastics.

Rehabilitation Program

Treatment varies according to injury severity and surgical difficulties. Up to 1 week, the focus of therapy is to limit pain, inflammation, and swelling. Rest, ice, and elevation are recommended. Icing for 20 min, 4 times per day provides pain relief. Following surgery, some individuals need immobilisation with the in knee extension for 1–5 days to prevent contracture formation and damage to the suture/re-insertion. Crutches enable ambulation while resting the injured leg. After several days, most patients may begin pain-free isometric exercises, pool therapies (after skin healing), and upper-body exercises. Isometric exercises are performed at various knee angles in increments of 20°. Generally, treatment 1–6 weeks postoperatively focuses on strengthening, improving range of motion (ROM), and flexibility. Passive static stretching may begin at this stage. Electrical stimulation may be used in conjunction with ice for added pain relief. The patient may exercise, preferably with a therapist, to strengthen the muscle within the available pain-free ROM. Then the patient begins isotonic exercises with resistance increasing gradually as tolerated. As healing continues, high-speed, low-resistance isokinetic exercises can begin. Resistance is increased gradually while exercise speed is decreased. Over time, the patient progresses from concentric to eccentric strengthening exercises. Prior to returning to play, sports-specific training maximises recovery and minimises chances for additional injury.

Return to Activity

A common threshold for return to the field is when the strength of the injured muscle has at least 90% of the strength of the unaffected side and when the patient has full ROM. In a hamstring lesion, at least a 50–60% hamstring-to-quadriceps ratio is desired prior to return to play. Strength testing is performed using isokinetic exercise equipment. In addition, it is also important to ensure return of normal flexibility and endurance prior to return to play; re-injury most often is due to lack of both. Therapy that incorporates sports-specific activities can help minimise the risk of re-injury.

Complications

Complications following treatment include:
- Re-injury;
- Failure of healing related to early active mobilisation;
- Relapse of painful scar tissue;
- Relapse of calcification;
- Atrophy and decrease of strength.

Surgery

Surgical treatment of muscular lesions is rare and with a very few and precise indications. In acute lesions, indications are as follows:
- Compressive haematoma (with risk of a compartment syndrome) secondary to near-total ruptures;
- Complete proximal or distal disinsertions;
- Total rupture with important retraction;
- Sciatic nerve dysfunction;

For chronic lesions, indications are as follows:
- Painful scar;
- Painful fibrous muscular scar;
- Chronicle seroma or cyst;
- Painful muscular hernia;
- Painful ossification of the periosseous.

Prevention

Prevention is the key to avoiding muscular lesions and could be divided into three main parts as follows:
- Primary prevention
  - Medical examinations
  - Dietary control
  - Training flexibility and stretching
  - Avoiding drugs
  - Warming up and cooling down
  - Progressive training
  - Personal training
  - Hygiene
- Secondary prevention
  - Information
  - Education
- Prevention
  - Legislation
  - Budget
  - Substructures.

Ankle Instability

Acute ligament lesions of the ankle are a common problem in sports trauma and in daily life. The incidence of ankle sprains in the community is 1/10,000 a day (Davis and Trevino). Even with appropriate treatment, some patients develop chronic instability with difficulties in sports activities and even in daily life. Ankle sprain, due to its high incidence among referees, deserves mention in this article. Ankle ligament lesions may be classified as:

- Type I – minor lesion;
- Type II – partial ligament rupture;
- Type III – total ligament rupture.

The deltoid ligament, responsible for medial stabilisation of the ankle, is damaged during an eversion/abduction movement. The anterior and posterior talofibular ligament and the calcaneofibular ligament are responsible for ankle stabilisation during inversion movement.

Diagnosis

After ankle sprain, it is important to understand the lesion mechanism in order to identify damaged structures. Local inflammatory signs, stress tests (inversion, eversion, and anterior posterior), and inspection can provide valuable information for classification and diagnostic. They are sometimes of low sensitivity in the acute phase; however, they are useful for late evaluation of a sprain sequela. MRI may be of interest for diagnosis, showing which ligaments are damaged and to what extent.

Treatment

The objective of the treatment is to obtain a good functional result and avoid chronicle ankle instability. In the acute phase, all ankle sprains should be submitted to rest, ice (48 h), compression, and elevation. Types I and II lesions should be protected with a brace during the initial face, being accepted that early mobilisation is of advantage. In respect to type III lesions, most studies have shown that early mobilisation followed by a period of physiotherapy with neuromuscular training achieves good results, even superior to surgical treatment. On the other hand, ligament reconstruction can always be performed secondarily, with results being similar to those achieved by primary surgical treatment. Surgical indications are as follow:
- Large osseous avulsion fragments;
- Severe ligament lesions;
- Recurrent type III lesions;
- Conjoint medial and lateral collateral ligament (LCL) lesions.

Chronic Instability

Until recently, surgical treatment of chronic ankle instability was not effective in returning athletes to sports activities. In 1998, we conducted a study to evaluate the dimensions of the internal malleolus by tri-dimensional computed tomography (CT) scan in 30 cases. This study showed a significant relation between a short medial malleolus, number and severity of sprains, chronic ankle instability, and osteoarthritis ($p<0.05$). Arthroscopic treatment with radiofrequency is nowadays a solution available for use by orthopaedic surgeons. This technique uses thermal shrinkage of the capsule and ligaments with molecular remodelling. We have been using this surgical approach in chronic ankle instability since 1999.

Materials and Methods

In our study, we used arthroscopy equipment with a 2.7-mm, RF generator Mitek and VAPR-T probe. From May 1999 to April 2003, we operated 98 ankles with chronic instability, including 3 referees. We selected those whose surgery was performed more than 12 months earlier and reviewed 88. There were 36 males and 52 females, with a mean age of 22 (14–45) years. The stress x-ray showed a mean external opening of 9.2º (5.4–21.9º). The mean number of sprains per year was 5 (4–12). The mean lesion duration was 5 (3–7) years. We selected patients with no improvement after 6 months of rehabilitation (with or without local corticoid injection), more than 4 sprains a year, pain on the LCL and external tilt difference from side to side >5º (TELOS). In this series, 51 patients performed sports activity and 37 patients were sedentary. Postoperatively, ankles were protected with a brace for 6 weeks and a brace was used for more than 3 weeks. At 6 weeks, all the patients were submitted to physiotherapy with proprioceptive work and fibular muscles reinforcement.

Results

All patients were revised with clinical examination, the American Orthopaedic Foot and Ankle Society (AOFAS) score, and varus and anterior drawer stress x-ray. Mean follow-up was 42 (12–64) months). The external opening decreased more than 50%, and the mean number of sprains decreased from 5 to 0.2. All referees returned to their previous level of competition and would have the same surgery again. Overall results were:

- Preoperative AOFAS mean score: 58.2;
- Postoperative AOFAS mean score: 89.5;
- Average AOFAS increased 31.3 points;
- 75 patients would have the same surgery again.

Relevance

Our results were good and promising. Ankle stability improved in all patients. Even in those with an augmented external opening, the number of sprains and pain decreased. We found good and excellent results in 67% of patients in this series, and those results remained with time (mean follow-up 42 months). We believed that this technique has a place in the treatment of chronic ankle instability.

Conclusion

There are no references in the literature regarding referees lesions. It is important to encourage work in that area and to stimulate prospective studies among those professionals. There is no doubt that referees deserve attention and careful care.

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Future Treatments for Football Injuries

Mario Ferretti, Boris A. Zelle, Freddie H. Fu

Introduction

Football, or soccer as it is called in North America, is the most popular sport in the world, with about 200 million participants [1]. It is characterized by a complex collection of movements including running, short sprints, rapid deceleration, turning, kicking, and tackling. These characteristics predispose participants to the relatively high injury rate of 12–35 injuries per 1,000 player game hours in adult men's football [2]. The lower extremities are more often injured. Muscular strains are the most common traumatic injuries in football players, followed by contusions and ligamentous sprains [2–4]. All these injuries have an impact on the players' lives and hinder them from participating in training and matches. Furthermore, injuries can lead to a 22% re-injury rate [3]. Moreover, injuries such as anterior cruciate ligament (ACL) tears may force a player into retiring early. ACL tears can have a rate as high as 0.41 tears per 1,000 game hours at the male competitive level [5]. In addition to the injury and its consequences for the players, associated costs of treatment are also a relevant issue [2]. Therefore, it is important to know about preventive methods to avoid injuries, as well as future types of treatment using modern biological approaches.

Injury Prevention and Preventive Methods

In order to decrease the incidence of injuries in the near future, the International Football Federation (FIFA) Medical Assessment and Research Centre (F-MARC) has promoted injury prevention [1, 6]. Van Mechelen [7] recommended four steps to be followed for injury prevention in sports: (1) identify the frequency of common and serious injuries; (2) identify risk factors; (3) introduce preventive methods; and (4) monitor ongoing surveillance of preventive methods.

Improving the structure and content of the training by education and supervision of coaches and players has shown a 21% decrease in the number of injuries [6]. This preventive method is based on general interventions such as improvements in warm-ups, regular cool-downs, taping of unstable ankles, adequate rehabilitation, and promotion of fair play, as well as applying a specially designed set of 10 exercises (F-MARC Bricks) to improve stability of the ankle and knee joints; flexibility and strength of the trunk, hip, and leg muscles; and player coordination, reaction time, and endurance.

Prevention of muscle injuries has been described by using strengthening and stretching [8, 9]. The eccentric strengthening preventive method has been promoted to reduce hamstring strains rates (Fig. 1) [8]. The recurrence of ankle sprains can be minimized by using orthoses or by training proprioception on an ankle disk [10, 11] (Fig. 2); 54% of ankle sprains occur during tackles, and fair play education may decrease this rate [12]. Preventive treatments also decrease the incidence of ACL injury. Proprioceptive training with wobble boards, flexibility, plyometrics, and muscular strength training are described as effective methods to prevent ACL injury [13, 14].

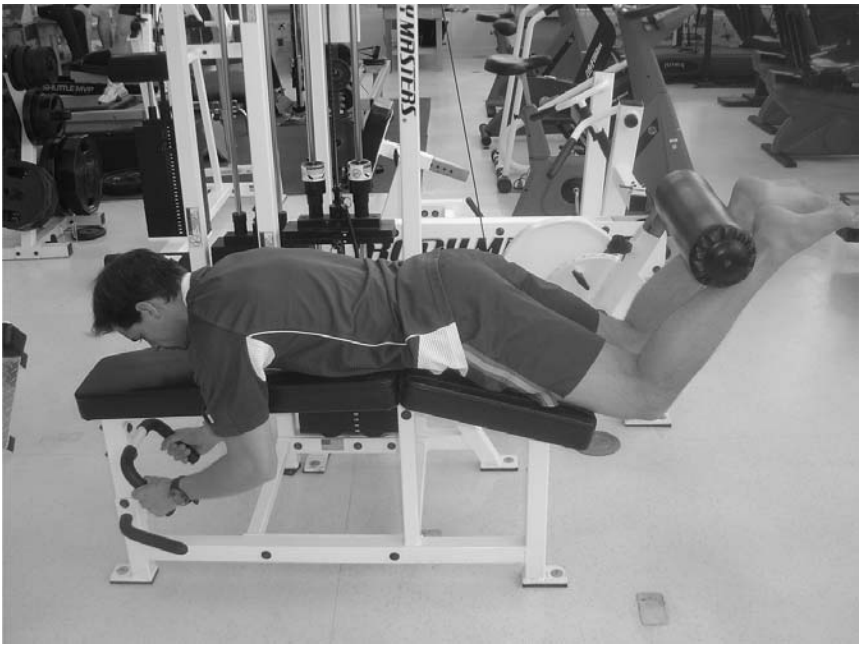

Fig. 1. The athlete exerts strength against the weight while extending the knee, performing the eccentric strengthening of the hamstrings

Fig. 2. Athlete performing the proprioceptive balance-board training. This training is used as prevention for sprains in the lower extremities

Biological Approaches

For injuries that could not be avoided by prevention, biological approaches may be an important tool for future treatments. Biological approaches have been used to avoid fibrosis formation in muscle, as well as to better heal tendon, ligaments, cartilage, and menisci. These approaches are based on the application of growth factors or cytokines. Growth factors are capable of stimulating cell proliferation, migration, and differentiation, as well as matrix synthesis and providing better tissue healing [15]. Direct application of growth factors is realized with very high dosages and repeated injections of these proteins because of their relatively short biological half-lives. In addition, growth factor effects are hindered by the difficulty of delivering to only the specific injured site. Different methods have been developed for administration and delivery of growth factors. Gene therapy techniques have shown the most promise. Using viral or non-viral delivery vehicles (vectors), the genetic information, usually a complementary deoxyribonucleic acid (cDNA), encoding the protein of interest is inserted into a living cell. The genetically modified cell has the potential to express the protein in a sus-

tained manner, making the long-term delivery of a protein or growth factor possible [16]. Tissue engineering is a technology based on the development of biological substitutes used for tissue repair using biomaterials capable of integrating molecules (e.g. growth factors). The biomaterial can also be used with stem cells from different sources (bone marrow, muscle, fat, etc.) [15]. Stem cells differentiate in specific cells according to the tissue environment and produce growth factors and proteins to heal the tissue. Tissue engineering and gene therapy are not yet a realistic therapeutic technique for the treatment of orthopaedic diseases. However, we believe that it has great potential for clinical applications in the near future and might help football players mainly for injuries in tissues with low capacity of healing, such as ligaments, menisci, and articular cartilage.

Muscle

Muscle strain is the most common injury in football [2–4]. The muscle healing process after an injury consists of three distinct phases. The first phase, of degeneration and inflammation, occurs in the first few days post-injury and is characterized by necrosis mediated by intrinsic proteases, local swelling, haematoma formation, and degeneration. Subsequently, the necrosis area is invaded by inflammatory cells. The lymphocytes secrete several cytokines and growth factors. Cytokines, such as interleukins and tumour necrosis factor-α (TNF-α), play a wide range of functions in the inflammatory process. At the injured site, growth factors, such as insulin-like growth factor-1 (IGF-1), hepatocyte growth factor (HGF), transforming growth factor (TGF-β and TGF-α), and platelet-derived growth factors (PDGF-AA and PDGF-BB) regulate myoblast proliferation and differentiation to promote the second phase of muscle healing, the phase of regeneration and repair. In this phase, not only do the growth factors have an important role but also the satellite cells located between basal lamina and plasma membrane. These cells are responsible for the formation of new myofibres and muscle regeneration. This phase occurs 7–10 days after injury and peaks around 2 weeks and then decreases at 3–4 weeks post-injury. The TGF-β1 from the regeneration phase also triggers the formation of fibrosis, the third phase of muscle healing. The fibrosis-forming phase begins between the second and third weeks post-injury [17]. Despite the many treatments available, the formation of scar tissue seems to be the end product of muscle repair [17–20]. Therefore, complete regeneration of muscle tissue cannot occur. Based on the biological aspects of muscle healing, research has been conducted to better heal the muscle by enhancing muscle regeneration and by preventing muscle fibrosis [19, 20].

The literature has shown that either high-dosage serial injections of

growth factors, such as IGF-1 and basic fibroblast growth factor (b-FGF), applied directly at the injured site, or IGF-1 secretion mediated by gene therapy improve muscle healing [17–20]. However, neither can prevent fibrosis formation. This necessitates the use of anti-fibrotic agents in order to block scar tissue formation. Because of the critical role of TGF-β1 in the development of fibrosis formation, it is essential to create approaches to antagonize this molecule. Decorin, gamma-interferon, and suramin have been described as promissory agents to avoid fibrosis formation in lacerated muscle by blocking TGF-β1 [15, 17–20]. Suramin promotes significantly less fibrous scar formation in muscle strain and provides better strength recovery [19]. It is also important to note that treatment with IGF-1 and decorin is not better than treatment with decorin alone in terms of reducing fibrosis [20]. This fact suggests that the blocking of fibrous scar tissue is the key for complete muscle healing (Fig. 3).

All the experiments performed in animal models encourage the clinical trial of anti-fibrotic agents. Moreover, some of these agents are approved by the Food and Drug Administration (FDA) and have already been used in humans for different pathologies [17]. Further research is needed before beginning to use growth factors and/or anti-fibrotic agents in athletes. However, we believe that these biological approaches are the future for muscle strain in football players.

Ligaments

The ACL tear is a major lesion that hinders play in athletes for approximately 6 months. Only 60% of professional players affected return at the same level

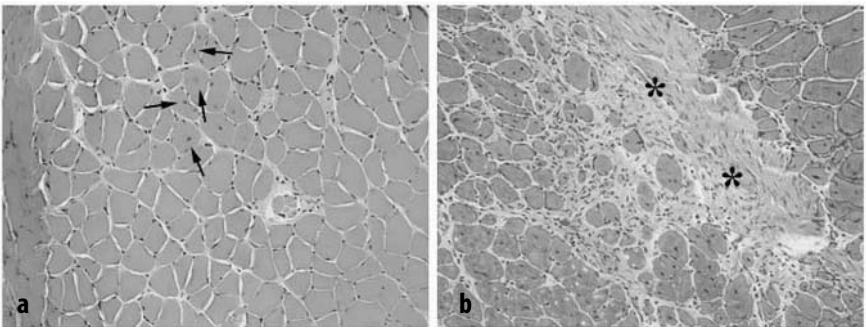

Fig. 3. Histologic evaluation (hematoxylin and eosin stained: x200) of injured skeletal muscle. Injured muscle treated with suramin showing fewer fibrotic areas (**a**) than untreated control sample (**b**). *Arrows* indicate regenerated myofibres in each sample. *Asteriks* show fibrotic area (Courtesy: Dr. Johny Huard and Dr. Jong Li)

as before the injury [5]. We believe that this reduced level of skill may be associated to the lack of rotation stability after ACL reconstruction [21]. Turning, a factor of rotational stability, is essential for football practice and associated movements. The ACL consists of two distinct bundles: the anteromedial and the posterolateral. In order to increase rotational stability and improve outcomes of ACL reconstruction, anatomic ACL double-bundle reconstructions have been performed on patients [22] (Fig. 4). Biomechanical study of anatomic ACL double-bundle reconstruction has been shown to be a superior technique when compared with single-bundle reconstruction by better restoring the rotation of the knee joint [23]. Further clinical comparative long-term studies are needed to definitively demonstrate that anatomic double-bundle ACL reconstruction is the best option for ACL tear treatment. However, based on the anatomy, biomechanics, and *in vivo* kinematics, we believe that this double-bundle concept may improve the quality of ACL reconstruction in athletes and provide a better recovery to the pre-injury level of skillfulness.

Biological approaches have been tested to improve the healing of the tendon graft in the bone tunnel in animal models [24]. Semi-tendinosus tendon

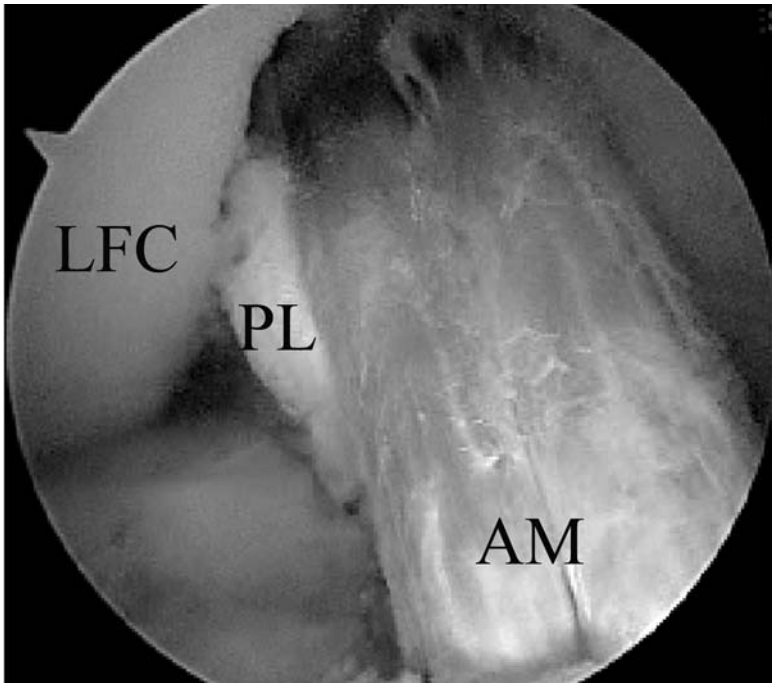

Fig. 4. Arthroscopic view of an anatomic anterior cruciate ligament (ACL) double-bundle reconstruction. *AM*, anteromedial bundle; *PL*, posterolateral bundle; *LFC*, lateral femoral condyle

grafts were transduced in vitro with adenovirus BMP-2 and implanted as ACL grafts in rabbits. This experiment showed a tendon-bone interface histology close to a normal ACL, and the stiffness and ultimate load at failure were significantly enhanced when compared with the control group (without AdBMP-2) [24]. This study suggests that gene therapy can improve the integration of soft tissue graft into bone and may be an important tool in the treatment of ACL tear, as well as in different ligaments such as in the ankle.

The direct administration of PDGF and IGF to injured ligaments and tendons has been shown to promote healing of ligaments and tendons [15]. Therefore, the possibility of using gene therapy to enhance ligament and tendon healing is a treatment to be used in the near future.

Cartilage and Meniscus

The treatment of chondral lesions is a challenge for physicians, with several techniques described. However, none of the techniques used for treatment is recognized as being outstanding. Cartilage has a poor regenerative capacity, with lack of stem-cell availability, poor vascularization, and low cellular turnover [15]. Tissue engineering and gene therapy have been used in animal models to deliver growth factors and proteins and better heal the cartilage injury and degeneration [15, 16]. The use of BMP-2, BMP-7, epidermal growth factor (EGF), IGF-1, and TGF-β1 has been shown to positively affect cartilage healing [15]. This is a potential research field to be explored for clinical applications. The same principle of gene therapy is also applied to the meniscus. Potential growth factors are bFGF, PDGF AB, TGF-α and β, BMP-2, and EGF [15].

Bone

Fractures are less common in football players-around 3% [3]. However, a fracture can lead to a long term without playing. The concept of tissue engineering and gene therapy is also functional to the bone. The BMPs (BMP-2, 4, 7) are already described as good proteins for bone healing and can improve and accelerate the bone-healing process [15, 16]. We believe that players experiencing fracture treatment will benefit from these therapies and may heal faster and better.

Overview

The use of preventive programs to avoid injuries, better surgical technique and rehabilitation, and biological approaches with high technology to better heal tissue will become the treatment of injuries for football players in the

near future. These treatments aim to restore histology, anatomy, and mechanical properties of tissue, providing athletes with optimal performance.

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Section II
Specific Injuries

Maxillo-Facial Traumatology

Roberto Brusati, Federico Biglioli

According to the literature, the incidence of maxillo-facial trauma with a sports aetiology ranges from 4% to 15% [1]. This depends on the country in which the study was conducted and on the most common sporting activities there. For example, in Italy, Scandinavia, and Latin American countries, soccer is undoubtedly the most commonly played sport [2]. In soccer, most facial fractures are caused by collisions between opponents, and only rare cases are attributable to falls. Generally, because the force involved is low, most trauma involves single fractures, and multiple and comminuted fractures are rare [3].

Every sport has its specific mechanical aetiopathologies, which frequently involve a characteristic type of fracture. In soccer, fractures of the zygoma, or nasal bones, are most frequent because of either blows with the elbow or *headers* during the aerial phase of the game [4, 5]. Such accidents, the intensity with which they occur, and the percentage of maxillo-facial fractures increase progressively going from recreational soccer to training sessions to competitive matches [6].

The case histories in this chapter were collected at the Maxillo-Facial Unit of San Paolo University Hospital of Milan (Italy) in the period 1994–2004. Of 796 maxillo-facial fracture cases treated, 173 (21.7%) resulted from sports activities, and 57 (7.6%) were soccer injuries [7]. Dento-alveolar fractures were excluded from the statistical study because they are more important to dental practice. Of the patients examined, 85% were resident in Milan or its environs, 9% lived in Lombardy, and 6% were from other areas. The types of fractures seen and their incidence in the athletes examined are summarised in Tables 1 and 2. Considering soccer aetiology only, following the general rules of maxillo-facial traumatology, the bones most often struck (23–28%) were the more exposed facial bones, i.e. the mandible, zygoma, and nose. Of the more protected skeletal areas, the orbital floor was most frequently involved (8%) owing to fractures produced by the blow-out mechanism. Multiple fractures involving more than one facial bone simultaneously represent only 7% of cases. There were fewer lacerations associated with the fractures (18%) compared

Table 1. Prevalence of the sporting disciplines in maxillo-facial traumatology (173 patients). Case histories from the Maxillo-Facial Surgical Unit, San Paolo University Hospital, Milan, Italy. Observation period: 1994–2004

Discipline	%
Football	57
Skiing	12
Cycling	9
Volleyball	7
Martial arts	4
Others (golf, gymnastics, basket, trekking, etc.)	11

Table 2. Typology of maxillo-facial fractures that occurred during soccer activity. Case histories from the Maxillo-Facial Surgical Unit, San Paolo University Hospital, Milan, Italy. Observation period: 1994–2004

Type	Number
Mandible	15 (27%)
Nose	13 (23%)
Zygoma +/- orbit	16 (28%)
Orbit	5 (8%)
Maxilla	4 (7%)
Multiple fractures	4 (7%)
Total	57

with the trauma that results from a car accident or fight. This is probably because of the modest forces involved in the trauma and because the collision is most often with an opponent's body rather than with rigid material.

As found in other studies, this type of trauma was much more common in males than in females, with a 10:1 ratio. The peak incidence in the study group was for ages between 16 and 30 years (54%); 33% of the subjects were older, and only 13% were younger. In addition, the temporal distribution of accidents was skewed: 48% occurred in winter, 42% in spring, 2% in summer, and 8% in autumn.

The relatively high percentage of facial trauma with a sports aetiology (21.7%) seen by us compared with that in the literature [7] might result from the low number of motor vehicle accident patients seen at the San Paolo

Hospital. The latter accidents involve a high degree of force and often result in loss of consciousness, requiring the services of a neurosurgeon, which is not available at San Paolo Hospital.

Introduction to Maxillo-Facial Traumatology

The facial skeleton is fundamental for several functions of the head: mastication, deglutition, respiration, hearing, sight, smell, facial mobility, and facial sensitivity. Furthermore, the facial skeleton has an important aesthetic value because it acts as support for the soft tissues. Therefore, a fracture can disrupt more than one function simultaneously and can have a negative effect on facial appearance [8].

The structures most frequently affected by trauma are the more exposed structures: the nasal pyramid, zygoma, and mental symphysis. As most of the facial skeleton is immobile, except the mandible, generally such trauma does not produce much pain. When injured, the mandible tends to be immobilised by muscle contraction, with a consequent reduction in pain, which is aggravated by opening the mouth and deglutition. For the sake of clarity, we describe the fractures of each facial bone separately although several bones can be affected simultaneously by the trauma.

Frontal Bone

The frontal bone constitutes the superior border of the orbit and is partially pneumatised from the frontal sinus. The front wall is thin and tends to cave in readily with trauma, which has a negative aesthetic effect. If the frontal duct is obstructed, the pressure inside the sinus can build up to the point where the thin floor of the sinus *explodes* downwards. The sinus floor constitutes the roof of the orbit, and, from an anatomical perspective, such a fracture is called a *blow-in* fracture (it bursts towards the inside). Bone fragments in the orbit can cause exophthalmos and diplopia if they interfere with the superior rectus or oblique muscles. The orbital frame is very strong, but, between the middle and lateral third, there is a weak area where the supraorbital foramen allows the passage of the homonymous nerve. A fracture at this point can alter the superior profile of the orbit and produce anaesthesia/hypoaesthesia of the omolateral frontal area. Fractures of the posterior wall of the frontal sinus are not considered here because of their neurosurgical importance.

Nose

The nose can be divided into two principal units: the septum and the pyramid. The cephalic third consists of the nasal bones, which are extremely thin

and can break easily. The lower two thirds consist of the nasal cartilage, which better absorbs trauma. The cartilage seldom breaks although it may be separated from the nasal bones. Frontal impacts tend to displace the pyramid backwards, changing the profile of the nose. If there is much hollowing, the septum is compressed by the displaced bone segments. The resulting folds of the septum can obstruct nasal respiration. Lateral trauma shifts the nasal bones medially or displaces the entire pyramid causing consequent respiratory obstruction and obvious aesthetic problems. Epistaxis is generally present.

Orbit

The orbit is the bony structure containing the eye. It is pyramidal in form with its axis directed anteroposteriorly and slightly inclined latero-medially. It can be subdivided into an external portion formed by the orbital margins; a middle portion consisting of the lateral and medial walls, floor, and roof; and the apex through which the optic nerve, ophthalmic artery, oculomotor and abducens nerves, and the first branch of the trigeminal nerve pass. The orbit is formed by the union of many bones. Consequently, the fracture of one bone, such as the zygoma or frontal bone, necessarily involves an orbital fracture. Fractures of the orbital margins generally create aesthetic problems and can impair the function of the supra-orbital and infra-orbital nerves [6]. Palpation of the margin with a finger readily identifies a *bony step*.

Fractures of the intermediate portion of the orbit result from two different mechanisms: blow-out and blow-in mechanisms. The latter has been described in fractures of the frontal sinus. The blow-out mechanism (explosion towards the outside) gives rise to a pure orbital fracture. A blow to the eyeball, such as an impact with a tennis ball, increases the intra-orbital pressure, which is transmitted to the orbit walls. As a rule, the mechanical resistance of the eyeball is greater than that of the thin orbital floor and medial wall formed from the orbital plate of the ethmoid bone. Consequently, the orbital floor breaks most often. The intra-orbital tissues cross the fracture line, which is sometimes very wide, and are displaced into the maxillary or ethmoid sinus, giving rise to enophthalmos. This is obvious immediately after the trauma but is masked by oedema of the tissues after a few hours and can be reduced spontaneously after 5–6 days when the oedema has regressed. The altered position of the eyeball causes incorrect co-ordination between the eyes with consequent double vision or diplopia. Diplopia can have another pathogenesis: the extrinsic muscles of the eye, particularly the inferior rectus and medial rectus, can become trapped in the fracture, which limits their function and consequently ocular mobility, creating diplopia in a few quadrants. For example, with a fracture of the floor of the orbit, the trapping of the inferior rectus muscle can lead to diplopia in the superior quadrant because it is impossible to raise the eyeball. Clinically, such injuries can be detected by

moving an object slowly through the entire visual field and asking when the patient has double vision. The observer simultaneously watches the struck eye for the inability to make a required movement. Specific exams, such as the Hess–Lancaster test, are subsequently conducted. A trapped extrinsic muscle in the fracture line is confirmed by the forced duction test. Fractures of the orbital floor often imply reduced function of the infra-orbital nerve. By contrast, fractures of the apex of the orbit give rise to ophthalmoplegia and mydriasis resulting from compression of the cranial nerves passing through the superior orbital foramen. Compression of the optic nerve or stretching caused by retro-bulbar angioma may lead to the loss of vision.

The leakage of air into the orbital and lid tissues from the maxillary sinus, generally following a sneeze or blowing the nose, leads to emphysema. Therefore, the patient is advised to avoid these maneuvres. Emphysema can be detected by palpation and is visible on computed tomography (CT).

Zygoma

The zygoma has extensions towards the frontal, temporal, and maxillary bones. Its body is quite resistant, but the projections are frequently fractured with frontal or lateral trauma. The displacement of the zygoma produces asymmetry of the middle third of the face. With the patient supine and exerting modest pressure on the soft tissues, the physician can assess the position of the zygomatic knob. The infra-orbital nerve and zygomatic-facial branch of the trigeminal nerve are often hypo-functional, with consequent hypo-aesthesia of the skin of the lower eyelid, cheek, hemi-nose, and superior hemi-lip. Posterior displacement of the body of the zygoma can interfere with the coronoid processes of the mandible, limiting opening of the mouth. Displacement of the zygoma in toto necessarily involves a fracture of the side wall and floor of the orbit, with the clinical signs described above.

An isolated fracture of the zygomatic arch, which consists of a thin extension of the zygoma fused with an analogous extension of the temporal bone, results from a lateral blow. Clinically, it causes a depression because the tissue covering the arch is especially thin in this area. Radiographically, the displaced bones assume the form of *seagull's wings* (Figs. 1, 2). Coronoid-zygomatic interference, with consequent limited opening of the mouth, is often present.

Maxilla

The maxilla is the base for insertion of the teeth of the superior arcade and the musculature of the soft palate. It also supports the soft tissues of the central face and nasal septum. Maxillary fractures can be classified as Le Fort I, II, and III based on the more cranial position of the fracture line. By gently gripping the superior arcade between index and thumb and applying traction

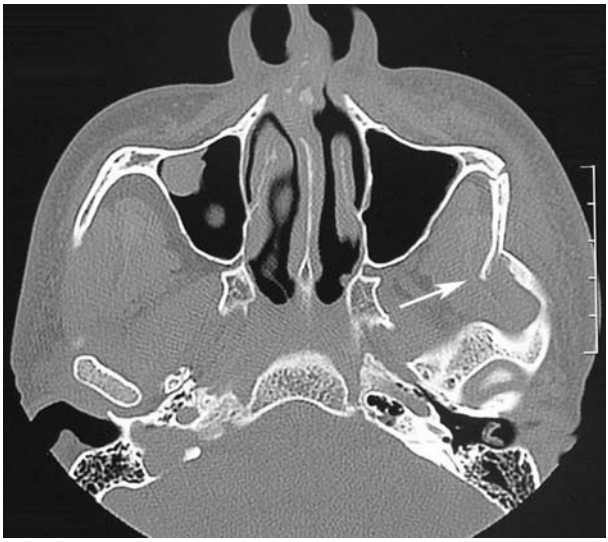

Fig. 1. Zygomatic arch fracture with the typical form of *seagull's wings* (*white arrow*)

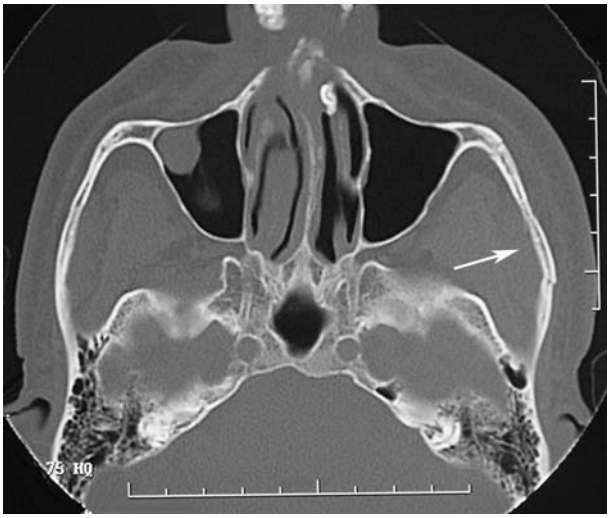

Fig. 2. Good reduction of the fractured bone segments (*white arrow*)

inferiorly, it is possible to observe the movement of the fracture segment. With a Le Fort I fracture, only the dental arcade is mobile; in Le Fort II, both the arcade and nasal pyramid are mobile; in Le Fort III, the arcade, nasal pyramid, and zygoma all move.

In patients who are not completely edentulous, even slight displacement of the maxilla disrupts occlusion, which is also typical of mandibular fractures, and the patient is immediately aware of this. Moreover, respiration can be limited by oedema of the mucosa of the nasal fossae or rearward displacement of the soft palate. With Le Fort II and III fractures, the infra-orbital nerve can be damaged in its tunnel, formed by the fusion of the zygoma and maxilla. Marked displacement of the fracture generally causes an obvious facial deformity. Such patients tend to avoid eating to prevent pain.

Mandible

Mandible fractures are classified according to anatomic location. As the chin is more exposed to trauma, these fractures are the most frequent. Condylar fractures are second most common and involve the transmission of a force applied in the canine area to the contra-lateral condyle, which is typically thin and offers little resistance to such force. Clinically, ecchymosis of the mucosa and diastases of the dental elements are often observed along with the bony lesions. Furthermore, as with maxillary fractures, a loss of occlusion is observed also with minimal dislocations. Oedema and haematoma of the oral floor can give rise to dangerous respiratory obstructions. In addition, respiratory distress can result from a para-symphyseal double fracture with consequent rear ptosis of the base of the tongue, which loses its front anchorage. The patient will voluntarily avoid mastication, phonation, and deglutition to maintain an antalgic position immobilising the mandible. Lost or broken teeth are further factors limiting mastication.

Radiographic Findings

The classic radiographic exam to detect frontal, zygomatic, and maxillary fractures is a Waters' projection of the skull. This view also shows the medial or lateral displacement of nasal fractures well. To examine the displacement of nasal bones on the sagittal axis, the lateral projection is more precise. Today, these classic radiographic projections are often replaced with CT exams, possibly accompanied by three-dimensional reconstruction of the images. In particular, orbital fractures must be investigated using axial and frontal CT. This last projection is mandatory for studying the floor and roof of the orbit. In addition, the axial projection adds details on the upper two thirds of the face.

The Panorex is useful for studying maxillary fractures, especially comminuted fractures, and it is the best radiographic exam for the mandible. When there is concern over condyle fractures, axial and frontal CTs are necessary to locate the condylar head in the articular fossa and the degree of dislocation of the bone segment.

Principles of Therapy

The classic approach to the patient with maxillo-facial trauma first involves maintaining the airway and controlling bleeding. Such problems are rare in the sporting traumas we analysed because of the low forces involved. In such cases, the only situation that requires the immediate management of homeostasis is epistaxis owing to a nasal fracture, which is generally managed with anterior nasal packing.

Facial fractures are treated with open or closed reduction. The latter, which is less aggressive, is reserved for compound maxillary and mandibular fractures, a few condylar fractures, nasal fractures, and zygomatic arch fractures. The remaining displaced maxillo-mandibular fractures, as well as orbital, zygomatic, and frontal fractures, are managed with open reduction.

In order to preserve facial aesthetics, incisions are positioned so as to be completely hidden, such as above the hairline, inside the mouth, or in the lid mucosa. When a cutaneous scar is unavoidable, it should be placed along a wrinkle or natural fold in the skin. Finally, when the trauma produces cutaneous wounds, these may sometimes be used to access the fractured segment although in most cases they are not located in a useful position.

Frontal Bone (Zygoma)

When the bony segments are displaced, these fractures must be treated using the classic principles of traumatology: reduce and stabilise the fractures. Obviously, given the modest degree of muscular traction involved, any plates and screws used are small. The plates are usually 0.5–1 mm thick, and the screws are sized accordingly.

Zygomatic Arch

The fracture is normally reduced percutaneously under local anaesthesia. A suitable lever is positioned deep to the sunken bone fragments, and the reduction is obtained using outwards traction. The surgeon can usually feel when the bone fragments are positioned correctly, but this must obviously be confirmed radiographically. The bony fragments are generally self-retaining and do not need stabilisation. When good reduction is not obtained or the fragments become displaced again, the fracture site can be explored surgically and stabilised with a titanium micro-plate.

Nose

Nasal fractures must be treated within the first few days following trauma. The nasal bones are so thin that ossification takes place rapidly. Moreover, the initial scar tissue leads to an inaccurate reduction of the fracture with obvi-

ous aesthetic damage. The fracture is treated under local anaesthesia with Walsham forceps, which allow manipulation of the fractured bones to their original position. The fracture is stabilised from below the nasal vault using thick nasal packing to provide support during the fracture consolidation phase. In addition, a cast is applied to the fronto-nasal area and taped to the skin. The packing material is generally removed after 1 week.

Orbit

In a pure orbital fracture, the bone fragments are extremely thin and are often comminuted to involve a wide area. Consequently, any gaps secondary to the trauma must be filled with autologous (bone or cartilage) or alloplastic (titanium mesh, plastic, or silicon sheets) material and be extremely smooth. Such a reconstruction will support the intra-orbital tissues and allow correct ocular muscle function.

Maxillae and Mandible

With compound fractures, treatment is conservative, consisting of wiring metal arches to both dental arcades under local anaesthesia. This will stabilise the fractures for the 15–20 days required for maxillo-mandibular fixation. Regarding condylar fractures, the intra-articular fractures, extra-articular fractures with inclination of the bony stump less than 60°, and all paediatric fractures are treated conservatively. In such cases, the time of maxillo-mandibular fixation is reduced to 5–7 days. This is followed by 1–2 months of functional therapy aimed at restoring articular function, with short periods of maxillo-mandibular fixation (a few hours during the day or night). Alternating maxillo-mandibular fixation with functional therapy has the double purpose of restoring dental occlusion and mandibular movement. For displaced condylar fractures in adults with inclination of the bony stump exceeding 60°, and all remaining maxillo-mandibular fractures, treatment involves reduction of the bony segments and their stabilisation with mini-plates. The reduction must be very precise at the dental level, as a displacement of only 1 mm can result in the loss of dental occlusion, causing much discomfort for the patient.

Multiple Fractures

The approach to such trauma requires careful planning before and during surgery and remarkable technical skills. Therefore, they should only be treated by an expert surgeon. The general plan during surgery involves a centripetal progression, reducing and stabilising central fractures before peripheral fractures (forehead, zygomatic arches, mandible, etc.), going on to the zygoma and maxillae, and finishing with the nose.

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Upper Extremity Injuries

Piero Volpi, Roberto Pozzoni, Marco Galli, Corrado Bait

Introduction

Upper limb injuries in association with football have been increasing in recent years [1, 2]. Goalkeepers, of course, are afflicted by typical chronic disorders. Among the other players, however, there is a disturbing incidence of acute traumas, including forearm and metacarpal fractures and acromioclavicular and gleno-humeral dislocations. The upper extremities, in fact, are exposed to significant risks from falls occasioned by the speed at which tackling takes place and certain fouls resulting in the knocking down of an opponent heading for the goal. These impacts are very similar to those associated with American football, which is a contact sport par excellence, so much so that its players wear specific protective devices. Falling after a header tackle, too, is a relatively frequent cause of upper limb injury [3].

A goalkeeper's actions are primarily founded on the use of the upper limbs. Goalkeepers must be in possession of special gifts brought to perfection by training in the art of falling without injury, especially since they are likely to fall on their shoulders as many as 200 times a week. The sliding mechanism employed to protect joints on these occasions ensures that the stresses are distributed over several points of the shoulder. A fully trained goalkeeper, in fact, does not land directly on the shoulder but curves it to spread the impact over a round surface. When catching the ball during a save, too, flexed hands are set in such a way that the forces exerted on the palms are transmitted to the forearms and elbows.

Although the goalkeeping role mainly exposes the player to direct injuries as the result of contact with opponents, the ground, and the goal posts, injuries also occur without contact. Modern tactics, in fact, sometimes place the goalkeeper in the position of the last open-field defender. In this respect, the incidence of injuries is similar to that of other players in addition to those peculiar to the particular actions and movements associated with this position.

Generic Upper Limb Injuries (Field Players and Goalkeepers)

Field players are essentially prone to acute lesions (bruises, lacerations, and sprains) whereas fractures and dislocations are less common. Gleno-humeral dislocation is certainly not rare on a football pitch though it is more frequent in other sports (skiing, judo, rugby). It is usually anterior, with displacement of the humeral head resulting in damage to the labrum, ligaments, capsule, and bone. Common causes are a fall on an arm in external rotation, a direct fall, or a tackle with abnormal traction of the arm by the opponent. In goalkeepers, it may be the outcome of a dive save against an opponent, with the arm abducted and laterally rotated and often with posterior impact on the ground. The altered profile of the shoulder, loss of movement, and acute pain indicate the correct diagnosis and call for immediate treatment. Radiographic confirmation of the diagnosis is followed by reduction by expert hands, usually those of an orthopaedic specialist at a first-aid station. Immobilisation with a brace for 20–25 days followed by rehabilitation to recover full efficiency is normally sufficient though recurrences cannot be entirely ruled out. In very carefully selected cases, surgery (usually arthroscopic) is undertaken after the first episode to suture and ensure better reestablishment of the labrum and anterior ligaments and hence reduce the risk of recurrences. Reeducation begins a month of relative safeguarding of the shoulder, and sport is resumed after 3 months. Recurrent dislocations demand arthroscopic or open-field management.

Acromio-clavicular dislocations are due either directly to a fall on the shoulder with the arm adducted or indirectly to a fall on an extended arm. Six degrees of injuries (sprains, subluxation, and dislocation) are distinguished in accordance with the anatomical damage they cause. Pain at the anterior surface of the shoulder, sharp pain provoked by pressure on the joint, and alteration of the profile of the lateral end of the clavicle (typical of dislocations) indicate the correct diagnosis prior to radiographic confirmation. Surgery followed by resumption of sport after 2 months is recommended. Goalkeepers, especially after many seasons, may display chronic joint pain due to repeated traumas or microscopic lesions.

Elbow dislocations, generally posterior, are not common. They are provoked by contact with an opponent when running or by a fall after a header. The elbow may be injured directly though dislocation is often caused indirectly by a fall on the palm with the arm extended and retroposed. Deformity, functional impotence, and pain indicate the correct diagnosis and call for radiographic confirmation and immediate reduction.

Metacarpal fractures are caused when a player's hand is trodden on while it is on the ground. The studs of the other player's boots usually cause a shaft fracture, sometimes with concomitant bruising and laceration. Pain and

swelling are immediately evident. Radiography is always advisable. Four weeks in plaster are usually sufficient. Surgical reduction and union is preferred in the event of displacement, shortening in excess of 2 mm, and angulation greater than 15°. Field players need not give up playing completely. They can continue their general physical training until the resumption of sport, which will be promptly achieved when the hand is protected with a brace. Goalkeepers are more prone to fractures of the shaft or neck of the third metacarpal since this is the longest bone and is exposed to direct injury when the ball is driven back into play during a save.

Specific Goalkeeper Injuries

As already mentioned, in modern soccer, goalkeepers use their hands to catch or punch back the ball in their penalty area and are thus exposed to injuries similar to those of the other players in addition to specific acute and chronic lesions, particularly of the upper limbs.

Reception of the ball brings the hands with the fingers open to block it and the fingers closed to repel it. The palms and second, third, and fourth metacarpo-phalangeals may thus be injured. The name "goalkeeper's thumb" [4] is used to describe subluxation of the metacarpo-phalangeal joint of the thumb with rupture of the ulnar collateral ligament (UCL) and possible fracture of the base of the first metacarpal. The force of the ball when caught is concentrated on the proximal phalanx of the thumb and subjects the ligament to heavy stress.

An acute injury results in pain accompanied by functional limitation and swelling. Rupture of the ligament renders the joint unstable. The diagnosis is clinical and radiographic. Treatment is non-operative (4 weeks in plaster) in the case of partial lesions whereas surgery is mandatory in the event of complete rupture of the ligament and bone detachment with displacement of the fragment.

Distal phalanx fractures are unusual whereas fractures of the base due to avulsion of the flexor [5] or extensor [6] tendon are more common. This type of injury, especially detachment of the deep flexor tendon, often necessitates surgical reduction and re-instatement of the bone fragment. Sprains of the (usually proximal) inter-phalangeal joints are relatively frequent while involvement of the collateral ligaments may result in instability. The joint is swollen and locally painful, with occasional loss of congruity.

The wrist and its many bones are the site of sprains, lunate dislocations, and scaphoid fractures usually due to falling with the hand in a defensive attitude. Scaphoid fractures are the most frequent, though uncommon, because goalkeepers mainly stress their metacarpals. Chronic fractures and pseudo-

arthroses sometimes escape notice. During a save, in fact, the scaphoid is between two convergent forces, one due to the impact of the ball on the palm with the fingers extended and the other due to the forward movement of the radius in the direction of the ball [7]. All types of scaphoid fractures require long abstention from sport. Clinical and radiographic healing enables competitive play to be resumed after 3–6 months.

A goalkeeper's elbow is mainly subject to sprains. Serious injury is unlikely as the result of contact with the ground even though the elbow pads used in other sports are not worn. There are, however, rare instances of capitulum humeri lesions due to abnormal falls with the hand in a defensive attitude, as well as isolated olecranon fractures. Overuse disorders include olecranic bursitis, often accompanied by oedema owing to continuous direct micro-traumata, together with insertion disorders of the triceps tendon caused both by direct repetitive traumata in flying saves and by the forces of reception of the ball with the elbows flexed. This disorder may eventually give rise to a typical olecranic spur varying in shape and size, which becomes evident at the end of a player's career.

Chronic shoulder disorders that disable goalkeepers from playing and specific daily training include the various forms of rotator cuff tendinopathies, traumatic derangement of the tendon of the long head of the biceps, and micro-traumatic instability with the various features of a "slap lesion".

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Shoulder Dislocation

Raul Zini

Introduction

The game of football presents a multiple variety of traumas that, due to the characteristics of the game itself, commonly concern the lower limbs and to a lesser extent the upper limbs. Volpi [1] reported various case studies that published very high percentages of traumas to the lower limbs with respect to the upper limbs in professional football players [2–5]. More recently, Morgan and Oberlander [6] reported 77%, and an epidemiological study by the Committee on Sports Medicine and Fitness of the American Academy of Paediatrics [7] confirmed percentage values of lower-limb injuries between 61% and 80% in comparison to 2.3–7.7% for upper limbs. Within this percentage, those involving the shoulder were between 1.8% and 2.6%. Another epidemiological study carried out on 50 Asian football matches by Yoon et al. [8] reported 5.4% of shoulder injuries. Junge et al. [9] examined the main football injury cases in the 4-year period from 1998 to 2001 reporting shoulder injuries between 2% and 13%.

Therefore, the shoulder is not frequently injured in football even though recently published documentation shows an increase in the trend, above all for the most traumatic injuries, such as fractures and dislocations. In fact, the evolution of the game of football in recent years with a notable increase in speed, choice of tactical solutions such as pressing and marking, and the ever-increasing recourse to tactical fouls, augmented the number of legal and illegal physical contacts, with a subsequent increase in trauma caused by falling on the ground.

Shoulder dislocation is one of the most frequent possibilities in the field of pathological trauma in the upper limbs of football players and, certainly, in light of the most recent suggestions in the literature, offers inspiration for a plethora of debates on prognosis and treatment.

Pathogenesis and Clinical Aspects

Gleno-humeral dislocation occurs when the upper part of the humerus is forced past its normally permitted limits; following traumas of varying extents and characteristics, the humeral head can be pushed against the acromion edge, which works as a fulcrum, and if the bone does not break, causes the dislocation of the humeral epiphysis from the joint cavity. There are five varieties of scapular-humeral dislocations: subglenoid, subcoracoid, subclavicular, subspinatus, or posterior, erected. The most common are the anterior subcoracoid dislocations, which will be the focus of attention here.

Trauma mechanisms that most frequently determine a gleno-humeral dislocation in footballers are due to a double strain of abduction and external rotation, which act violently on the humerus. On occasion, it can be a mechanism of extreme retropositioning with the arm abducted or simply, in more violent traumas, only extreme abduction; in other cases, not uncommon, the trauma is purely indirect due to a fall on the palm of the hand or the elbow of an abducted arm with the resulting force along the diaphysis of the humerus. This type of injury almost always involves traumas from falling following either a legal or illegal contact, which can concern all cases without any particular predisposition.

Epidemiological research conducted by the Committee on Sports Medicine and Fitness of the American Academy of Paediatrics [7] showed how young athletes are statistically more susceptible to upper-limb traumas and to gleno-humeral dislocations; the same study showed more generically how there are more risks during an official match than during a training session, that the majority of detrimental cases (44–74%) were caused by physical contact between two players, and that during an indoor match, the risk of this type of trauma is approximately six times greater than during an outdoor match. According to Morgan and Oberlander [6], neither the player's role nor age affects the seriousness of the reported accidents. The goalkeeper deserves a separate mention, however, who – considering the frequency of falls following either dives or exits from the goal area – is more susceptible to damaging the gleno-humeral "track". Volpi [1] noted how the goalkeeper suffers on average approximately 200 shoulder-ground contacts per week, which are generally kept under control without damage by exercising a sliding motion that protects the joint by distributing the trauma to many different parts of the shoulder. When in exceptional circumstances this type of protection is not possible and the trauma is abnormal or more violent, joint dislocation can occur.

An acute gleno-humeral dislocation is characterised by complete loss of connection between the humeral head and the glenoid cavity of the scapula. The upper limb seems almost lengthened with respect to the limb on the

opposite side and is positioned slightly abducted and rotated inwards; the elbow is flexed and the forearm lifted against the trunk and held by the other hand; the neck is inclined towards the side of the injury to give an analgesic effect. There is exaggerated swelling of the acromion process due to the cavity at the height of the shoulder, as the humeral head is found at the height of the armpit. There is complete functional powerlessness, and passive movement is very painful. Even though the clinical aspect is very significant and permits an effortless diagnosis even on the football field, it is always advisable to take x-rays, which will ascertain whether or not there is a fracture.

Anatomo-Pathology of the First Dislocation

A precise definition of the anatomo-pathology of an acute shoulder dislocation is possible, above all thanks to a series of arthroscopic studies made during the 1990s by different Authors [10–13] who wanted to classify the frequency and entity of joint injuries. Specifically, following a traumatic gleno-humeral dislocation, the following anatomo-pathology case histories are present:

- *Bankart lesion* consists of the disconnection of the glenoid labrum together with the medial gleno-humeral ligament and the superior aspect of the inferior gleno-humeral (Fig. 1); it is present in almost all cases, with a documented percentage between 87% [11] and 100% [10, 13].

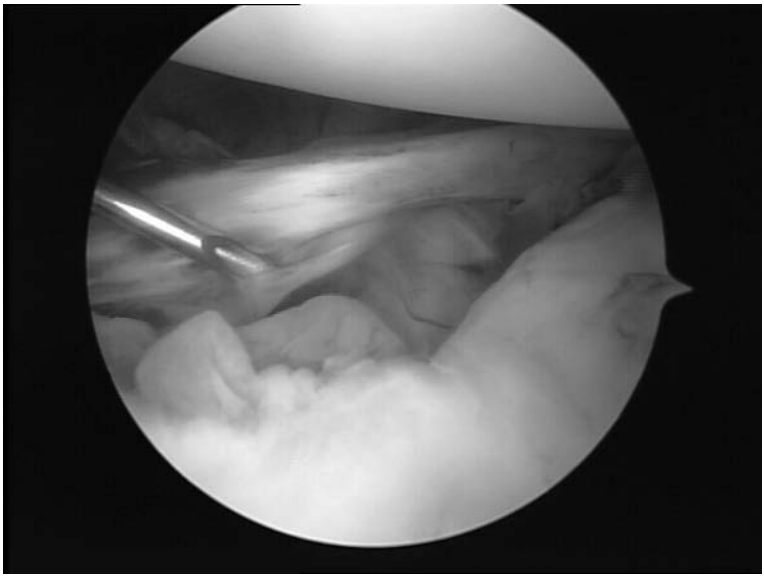

Fig. 1. Bankart lesion

- *Hill-Sachs lesion* is an injury from impact of the posterior aspect of the humeral head, with damage to the cartilage and exposure of the subchondral bone (Fig. 2); it is described as showing high percentages – between 90% [8] and 100% [10].
- *Glenoid bone fracture (bony-Bankart) or humeral head* are not considered as rare injuries, bearing in mind the percentages between 9% [7] and 13% [10].
- *Superior labrum tear – anterior to posterior (SLAP) lesions and disconnection of the bicipital tendon* are not rare, with a documented percentage of approximately 10%.
- *Rotator cuff tears* are the rarest injuries, ranging between 0% [8] and 11% [11]. In all these cases, enarthrosis and haemorrhagic synovitis is furthermore present.

Injury Evolution

Habermeyer [14] compared the present anatomo-pathological injuries following a first traumatic dislocation of the shoulder (group 1) with respect to a recurring dislocation (groups 2, 3, 4), finding the following injuries in group 1: Bankart lesion, 90%; capsular injury, 30% (10% damage, 20% elongation); SLAP injury, 20%; bicipital tendon subluxation, 10%; rotator cuff

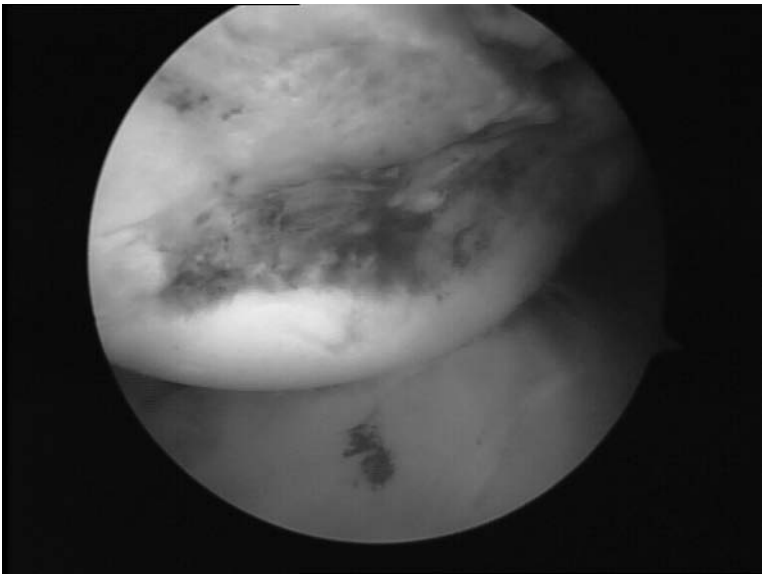

Fig. 2. Hill-Sachs lesion

injury, 50% (40% partial, 10% total), Hill-Sachs injury, 80%; glenoid cartilage injury, 20%. With the increase in cases of dislocation, a characteristic series of joint modifications was found, more serious in proportion to the number of dislocations. A constant increase in joint volume was found, mainly noticeable after the second episode. There was also an extension of the front ligament complex that gradually tears or presents significant degenerative signs; if the labrum is still not disconnected, it tends to reattach in an abnormal position with the aspect defined as an anterior labrum periosteal sleeve avulsion (ALPSA). There was a notable increase in the SLAP injury; Hill-Sachs injury increased in volume in a distinct manner after the fifth dislocation. With the increase of dislocations and age, there was a notable rise in the number of injuries to the rotator cuff. The Authors concluded that it is necessary to prevent recurring dislocations to avoid progressive and serious damage to the joint structures.

Unfortunately, however, the clinical progress of a traumatic dislocation of the shoulder shows a high percentage of a recurring injury. According to documented evidence, the percentage shown is vague, varying between limited values such as 17% and as high as 96%, with an overall average of approximately 67% [15]. Subsequent research closely examined the progress of the dislocation of a gleno-humeral joint, comparing it to the clinical characteristics of the patients, and a better determination of the risk of a recurring dislocation was found. Hovelius [16] established the relationship of recidivism with the age of the injured person, concluding that risk decreases as age increases: in patients younger than 22 years old, recurrence occurs in 55% of cases; between 23 and 29 years in 37% of cases; and between 30 and 40 years only in 12% of cases. Other Authors have associated the risk of dislocation to the age of the injured person and sport, concluding that athletes younger than 25 years have a potential risk of recurrence higher than 80% despite adequate conservative treatment. Henry [17] reported a percentage of 95% in this age range, Simonet et al. [18] 82%, Wheeler et al. [13] 92%, and Arciero et al. [19] 80%. Patients identified as having such a high risk of the injury becoming chronic have been scheduled for immediate surgery for injuries resulting from the first dislocation event.

Non-Operative Treatment

Non-operative treatment consists of immobilisation of the joint after reduction of the fracture and subsequent rehabilitative treatment. In the past, the most common treatment was immobilisation by plaster or starched cast with the shoulder in a functional adduction and intra-rotation position. In time, the plaster was replaced by a soft bandage or shoulder brace, which is better

tolerated and offers the same guarantees. Itoi et al. [20] published a clear improvement in the results of conservative treatment with immobilisation of the shoulder in the neutral position, potentially extra-rotated rather than intra-rotated; the results were confirmed by a study on a cadaver by Miller et al. [21], which compared how the extra-rotated position increases the contact force between the glenoid labrum and favours the curing of Bankart lesions; there are currently larger indications for immobilisation, with the extra-rotated limb in a bandage or shoulder brace, when a bloodless treatment is chosen.

The duration of immobilisation varies between 3 and 6 weeks, but there are no prospective studies that show a connection between duration of immobilisation and incidence of possible relapse. Amongst the post-immobilisation rehabilitative methods, one of the most followed is that of Kirkley et al. [22], which divides rehabilitation into different subsequent stages: during the first 3 weeks, the patient can remove the shoulder brace to wash and do exercises of active mobilisation of the wrist and elbow; from the fourth week, after having removed the shoulder brace, active movements are allowed, with extra-rotation limited to 20; from the seventh week, isometric exercises are introduced, and extra-rotation is allowed up to 45; from the ninth week, isotonic, stretching, and building-up exercises of the scapular muscles are started; from the third month, it is possible to return to work and non-contact sports; from the fourth month it is possible to take up contact sports.

The advantages of conservative treatments are mainly low social costs, avoidance of general surgery risks, and above all, the high percentage of curing adult patients with sedentary activities (no sportsmen). The disadvantages are definitely greater, above all in young patients and sports people, due to the many cases of recurrence and the imperfect repair of joint injuries, which in time leads to joint degeneration.

Surgical Treatment

Surgery has the advantage of immediately identifying and curing existing injuries and therefore allows for an immediate repair of the same: the obvious consequence is a distinct decrease in re-occurring cases, which is above all ideal for young athletes. The disadvantages are connected to the generic risks concerning surgery, and costs that, obviously, are higher compared to bloodless treatments. Surgery on first dislocation can be carried out in acute stages or in the weeks that follow, always, however, before a possible second dislocation. Uribe et al. [23] and Valentin et al. [24] found no difference between surgery carried out during an acute stage or one delayed by 3 weeks from dislocation. It has now been shown in the literature that during the acute stage, arthroscopic surgery is recommended rather than traditional open-wound surgery.

Initially during the 1980s and 1990s, arthroscopy allowed for a close examination of the anatomo-pathology of injuries and treatment in a mini-invasive, selective, and more precise manner. In time, different arthroscopic techniques were suggested to deal with a first dislocation of the shoulder in young sports people. Wintzell et al. [25] suggested simple washes of the joint, with a 20% recurrence. Wheeler et al. [13] published their results following abrasion to the glenoid and detached labrum. At the same time, various authors started to carry out stabilisation of labrum injuries using different techniques. Arciero et al. [19] published results after treatment of capsular re-insertion with thermally assisted capsular shrinkage (TACS) with 4% recurrence. Caspari's technique was used by Molé [26] and Hehl et al. [27], with recurrences of 9.5% and 7% respectively. Re-insertion with anchors was first described by Jakobsen et al. [28], with recurrences in 3% of cases; stabilisation of labrum injuries using anchors was subsequently the acclaimed arthroscopic technique, associated with treatment of joint injuries possibly present.

Acute surgery on first shoulder dislocations in young sports people was an ever more common practice in 2000, with publications of different prospective research and retrospective revision works, amongst them Zini et al. [29]. From all this research emerged a fundamental improved outcome in surgery compared with the traditional conservative treatment. Boszotta and Helperstofer [30] published a study in which 93% of cases were cured using arthroscopic surgery compared with 10% using the bloodless treatment. Larrain [31] cured 96% of cases arthroscopically compared with 4% of cases using bloodless treatment. De Berardino et al. [32] reported 88% of cases were cured following acute surgery on high-risk patients such as West Point Academy cadets. Bottoni et al. [33] reported that following arthroscopic treatment, 89% of cases were cured compared with 25% of non-operative cases. Yanmis et al. [34] confirmed that acute arthroscopic treatment is the surgery chosen following first dislocations in young athletes, noting a significant increase in the Constant score in patients operated on compared with the group treated with bloodless therapy.

Arthroscopic Technique and Personal Case Histories

Arthroscopic surgery for the stabilisation of an unstable shoulder due to a first traumatic dislocation is carried out in our experience with the patient in lateral position. It can also be carried out, at the surgeon's discretion, in the so-called "beach-chair" position, that is to say, with the patient sitting on the operating table. The operation is carried out with a local-regional anaesthetic with interscalenic brachial plexus, therefore with the patient awake and

easier to check on during the operation. The portals usually used are the posterior, the inferior anterior, and the superoanterior (Fig. 3).

Arthroscopy begins with creation of the posterior portal and exploration of the entire joint. Therefore, a work cannula is introduced through the inferoanterior portal, through which the possible associated injuries can be treated. Another superoanterior portal is created through which the third cannula is introduced. This cannula is used as a work cannula for introducing arthroscopic instruments and for recovering threads or for introducing the television camera and checking the injury from the front and repairing it. After having assessed the Bankart lesion, the labrum is completely mobilised where a delicate *débridement* is carried out. The anterior bone edge of the glenoid is thus superficially incised where the labrum and the anterior capsular will be reinserted (Fig. 4). Using a perforator, small holes are made on the anterior edge of the glenoid for inserting the anchors (Fig. 5). Generally, three to four anchors are needed, spaced 4–5 mm from one another, in order to adequately stabilise the lesion. It is very important that the most distal anchor is preferably lower so as to be able to re-insert the lowest part of the capsular using a correct amount of pressure and therefore avoid the risk of a recurring dislocation.

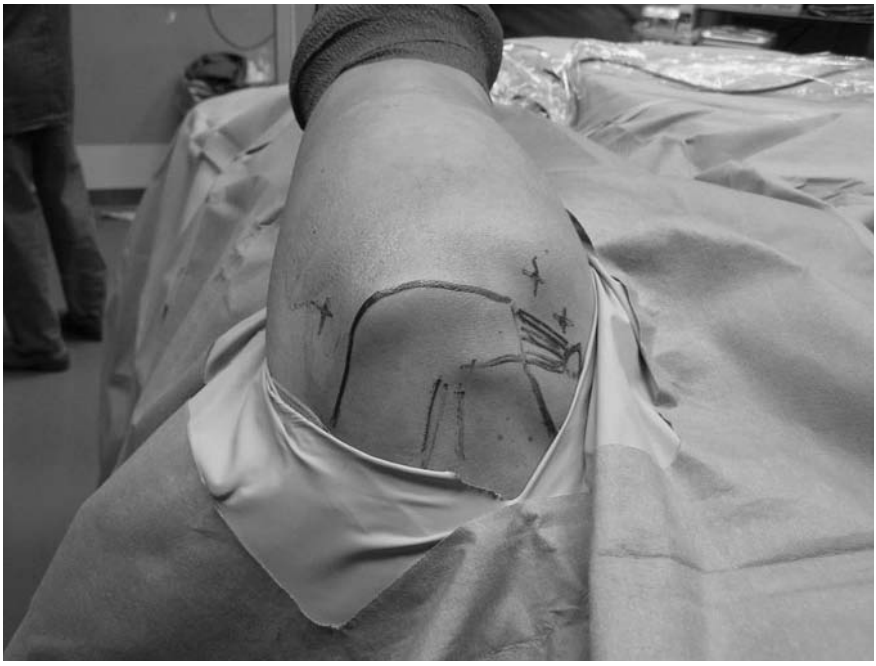

Fig. 3. Arthroscopic portals

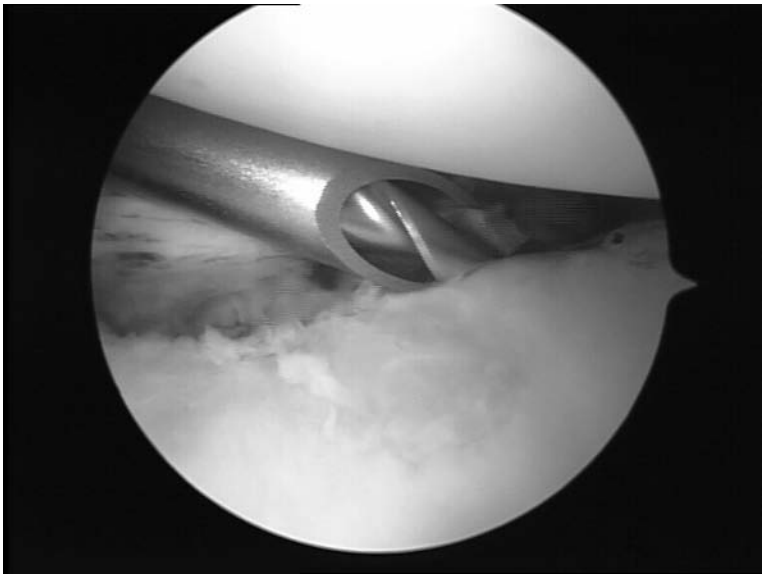

Fig. 4. Bleeding of the glenoid edge

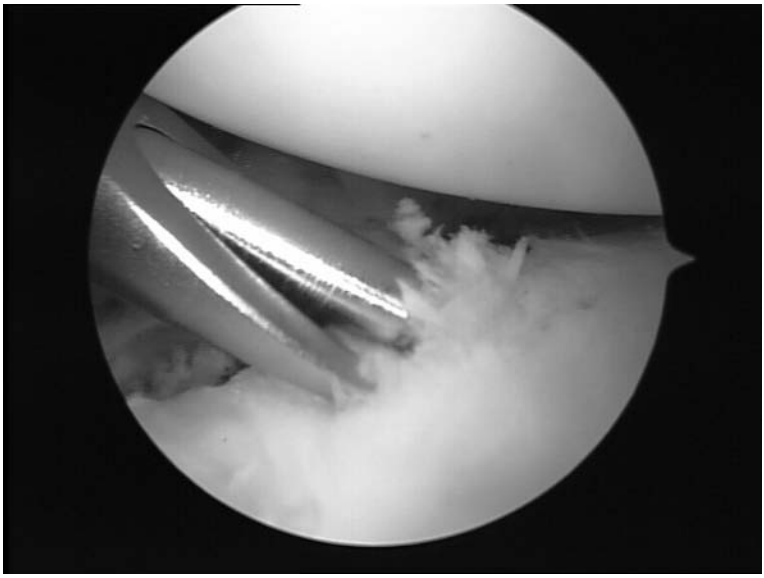

Fig. 5. Glenoid perforation

By means of the anterior cannula, the most distal anchor is inserted (Fig. 6). One end of the threads inserted on the anchor is threaded using special hooks and a transporting thread through the most distal point of the capsular injury in such a way that when the thread is knotted, the free edge is strongly attached using a correct amount of pressure on the glenoid.

The knots are made outside the articulation and pushed through the cannula using an appropriate arthroscopic knot pusher (Fig. 7), which allows the knots to close properly and firmly. This is how it begins: starting from the bottom and stretching the capsular to repair the injury. Using the same procedure, the other, closer anchors are inserted (Fig. 8) until the fracture is completely sealed. The Bankart lesion is therefore completely repaired (Fig. 9).

Exact articular haemostasis is carried out using a radio-frequency electrode, and joint stability is assessed with rotating movements. Using single stitches, the three arthroscopic portals are closed, and an orthopaedic shoulder brace is put on. The patient is admitted into day surgery and therefore released the morning after the operation. The shoulder brace is kept on for 3 weeks; at the end of this period, rehabilitation begins. Active sports using physical contact can be resumed 3–4 months after the operation, according to each individual case.

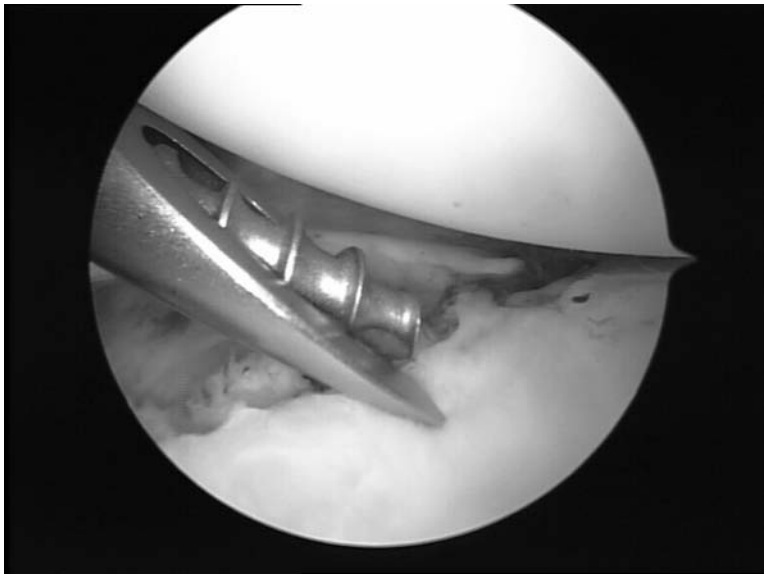

Fig. 6. Insertion of the anchor-screw

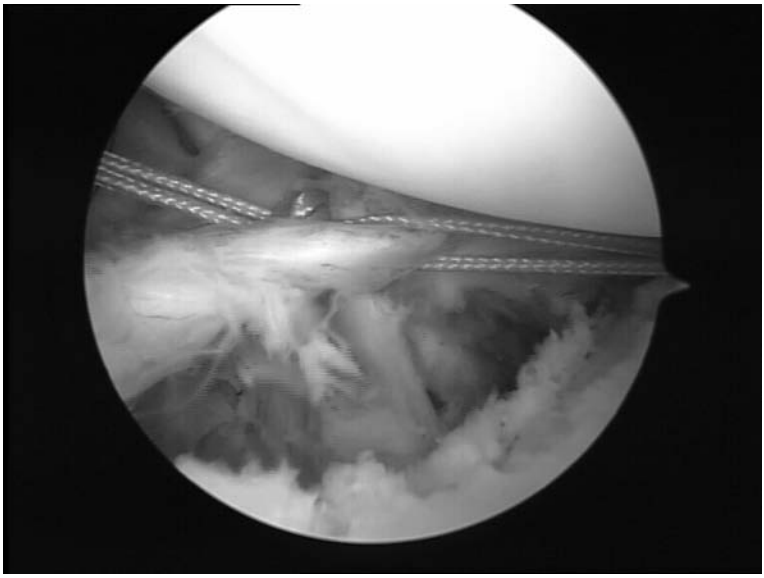

Fig. 7. Knotting of the thread

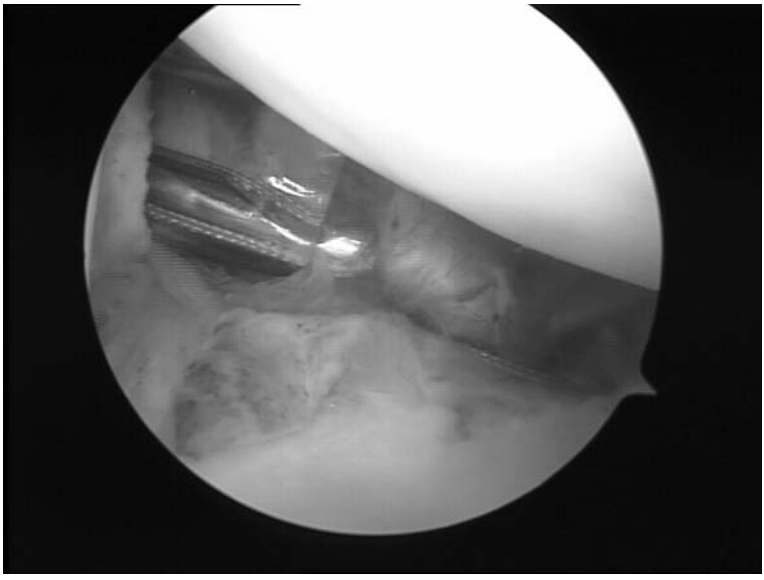

Fig. 8. Knotting of the second thread

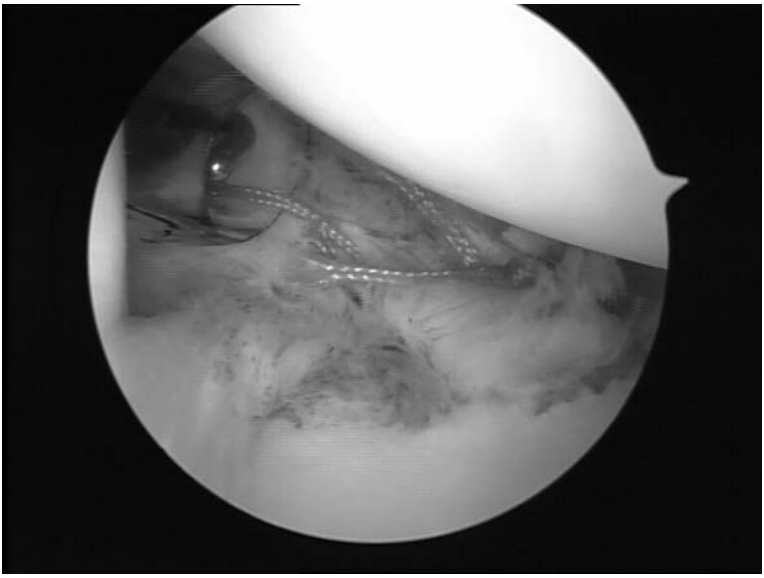

Fig. 9. Completed repair work

In 35 patients between 1997 and 2004 (27 men, 8 women), ages ranging from 15 to 25 (average 21) years, arthroscopic surgery was carried out within 10 days of dislocation. Thirteen patients were competitive football players; the remaining injuries occurred in basketball (14 cases), rugby (5 cases), and volleyball (3 cases). During a statistical check carried out in 2004, all patients operated on were followed up; only one recurrent case was found (in a rugby player). Therefore, the percentage of non-success was 2.85%. The average preoperative Constant score was 48 (range 35–60), whilst the postoperative Constant score improved significantly, with an average of 92 (range 70–100).

Conclusion

Data in the printed documentation shows that there is a high incidence of recurrence following conservative treatment for acute anterior dislocation of the shoulder in young patients participating in a high level of sport. The footballer who, following an injury during a match or training, suffers a dislocation trauma of the shoulder is a perfect candidate for undergoing acute arthroscopic surgery. According to our records of patients to be operated on, arthroscopic surgery is highly recommended for footballers younger than 25 years whilst it is debatable and advisable for players between 25 and 30 years. For players older than this, conservative treatment is recommended, with

immobilisation according to Itoi et al. [20] and subsequent rehabilitation according to Kirkley et al. [22].

In all cases operated on, an unmistakable anatomo-pathology history with disconnection of the labrum (Bankart lesion), Hill-Sachs lesion, and enarthrosis were found; associated injuries such as SLAP injury, cuff tears, osteochondral lesions of the humeral head, or glenoid injuries were found in limited percentages. In the acute stage, lesions from tears of the capsulolabral complex are the best conditions to be repaired; in fact, there is no degeneration of the labrum nor capsular retraction, as is almost always seen in chronic injuries. Therefore, excellent results may be expected with selective treatment and particularly with arthroscopy. Also, during the arthroscopic surgery, any possible associated joint injuries can be diagnosed in advance and properly treated, thus obtaining the best possible prognosis with respect to any other treatment, be it conservative or traditional surgery.

Obviously, only purely traumatic injuries are treated, and patients who have not suffered real traumatic dislocations but only a single subluxation not in need of a real reduction are excluded from the acute treatment. Pre-existing, multi-directional instabilities together with previous pathologies of any other nature suffered by the traumatised shoulder are the only real contraindications to acute surgery. There are no published papers that take shoulder dislocation into consideration in football players, even if numerous articles clearly highlight the excellent outcome of arthroscopic stabilisation in young sports people.

Our statistics overlap with those in the literature as distant results and therefore contribute to advising sports traumatologists on what to recommend – specifically in football and other sports where contact with other players is involved – arthroscopic surgery for a first gleno-humeral dislocation.

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Back Problems

Éric Rolland, Gérard Saillant

Introduction

The causes and origins of a footballer's back problems may be discal, articular, muscular, or ligamentous. They are often intricate, sometimes associated with neurological signs, and reflect overuse of the spine due to repeated and powerful execution of the movements required during training sessions and matches. A diagnostic, therapeutic, and prognostic distinction must be drawn between acute disorders with severe, crippling pain and less painful but persistent and recurrent chronic forms.

General Notions: Physiopathology

Spinal overuse injuries are the outcome of an age-related interaction between microtraumatic mechanical factors inherent in football itself (extrinsic factors) and a player's own morphological and degenerative factors (intrinsic factors).

Extrinsic Factors

The changes of position, physical contacts, interceptions, and marking of opponents required in modern football are the cause of overuse and overloading of the spine, together with contusions due to direct blows. Shooting at the goal, dribbling, heading, and sliding tackles are the main sources of injury:

- A classic shot at the goal is made with the supporting leg half flexed and internal rotation of the hip. The ball is struck by the other foot with the leg in adduction, rotation and flexion of the hip, and extension of the knee. Sudden rotation of the pelvis is offset by counter-rotation of the shoulder girdle and the upper dorsal spine. The maximum stresses are thus imposed on the lumbar spine and its dorso-lumbar and lumbo-sacral hinges. Shooting demands a strong abdominal musculature to allow locking of the pelvis and the dorso-lumbar spine.

- Players run with or without the ball, sprint, counter-attack, dribble, swerve, and cut.
- Heading demands hyper-extension of the spine and rotation of its cervical and thoracic regions. Uncushioned landing after a jump often results in a fall and hence an additional risk of injury.
- A sliding tackle is a defensive manœuvre that stresses the pelvis and lumbar spine. It is carried out with the knee and ankle extended, external rotation of the hip, and hyper-extension of the lumbar spine.

In practical terms, shooting at the goal, heading, and sliding tackles are more likely to give rise to disc lesions whereas dribbling, counter-attacking, and sudden cutting and dashing result in posterior joint disorders, falls and contacts, and bruises.

Intrinsic Factors

These may be anatomical or functional. Anatomical malformations, whether congenital (transitional vertebrae, mega-transverse process) or acquired during adolescence (scoliosis, spondylolisthesis, or Scheuermann's disease), underlie spinal static defects that may be incompatible with the stresses imposed by intensive sport. Functional factors stem from the fact that an adult footballer is often bradymorphic and hypermyotrophic with a particular lumbo-pelvi-femoral equilibrium. Hyperlordosis, in effect, may be associated with excessive anteversion of the pelvis that promotes a conflict of the hip and hypertrophy of the thigh muscles, with weakness of the abdominal wall: the adductors, hamstrings, and anterior straight muscles are usually powerful, short, and retracted whereas the musculi abdominis are scanty, deficient (especially in black people), and thus unable to provide protection against spinal stresses. Lastly, repeated sport-induced stressing of this kind of vulnerable somatotype may give rise to precocious, microtraumatic degeneracy of the discs and ligaments, resulting in discopathies and posterior arthrosis that carry the risk of painful chronic disorders.

Acute Disorders

Pain may be the immediate consequence of an action or injury. More frequently, it increases progressively after the activity and is often accompanied by muscle locking and contractures with pain-relieving attitudes. Assessment of the mechanism responsible for the lesion, clinical examination, and the progress of the functional signs over a period of 48 h will guide the diagnosis of discopathy (herniation), joint disorder (minor vertebral derangement), or muscle disorder (bruises, haematomas) and the prescription of further examinations for the elaboration of appropriate treatments.

Traumatic Discopathy

Typically, unilateral spinal-nerve-root pain (L5 or S1 sciatica) associated with a strong rachidian pain syndrome is indicative of a traumatic disc disorder. Clinical examination is required to corroborate disc-root impingement from the dura mater signs (impulsivity on coughing, Lasègue's manœuvre, Achilles reflex analysis), clarify the extent of the spinal rigidity, and look for neurological signs of sensory and/or motor-root deficiencies (assessment of the strength of the extensor hallucis longus, the common extensor, and the lateral peroneals for root L5, and the triceps and the gluteus maximus for root S1). Treatment always starts with complete rest coupled with medical management of the symptoms (analgesics, anti-inflammatory agents, decontractants) to sedate the pain. After 2–3 days of medical treatment, three clinical situations must be looked for:

- The most frequent picture is gradual improvement of rigidity and lessening of lumbar and root pain. Systematic radiography is used to detect anatomical abnormalities and evaluate arthrotic degeneration. A computed tomography (CT) scan will confirm a diagnosis of disc herniation. Medical management is then continued without resumption of sport until the clinical tests (rigidity, Lasègue's manœuvre) return to normal, followed by progressive training with re-education of spinal equilibrium to reduce the risk of recurrences or chronicity.
- On some occasions, despite the initial medical management, including the parenteral administration of major analgesics, substantial pain persists without an antalgic position in the form of acute hyperalgia. Imaging [CT or magnetic resonance imaging (MRI)] will both clinch the diagnosis and illustrate the topography and size of the hernia so as to determine the appropriate treatment. A voluminous and refractory hernia raises the question of prompt surgical correction whereas smaller forms with less severe impingement may respond to cortisone filtration under radiographic or scanographic control.
- Lastly, one must look for a falsely reassuring clinical form that progresses in three stages: initially, acute lumbago with no root signs, then hyperalgic sciatica with disappearance of the spinal syndrome, and finally, disappearance of sciatica and the establishment of a severe motor impairment.

This picture reflects progressive migration of the hernia to the point of exclusion within the canal and compression of the nerve root. Muscle strength must thus always be checked after the disappearance of pain by analysis of tip-toe and heel walking and testing of muscles (particularly the extensors and peroneals). The presence of a painless neurological impairment syndrome, *a fortiori*, associated with perineal signs (saddle-block anaesthesia), demands urgent MRI to confirm the diagnosis of excluded her-

nia, determine its topography, and guide urgent surgical ablation of the fragment of the disc.

All in all, if a traumatic discopathy is diagnosed clinically, its anatomical features (topography, size, and exclusion) demand imaging (CT or MRI after systematic radiography) to guide the choice of (occasionally surgical) management.

Minor Inter-Vertebral Derangement (MID)

The posterior joints are vulnerable to exaggerated stresses at the origin of a projection beyond the physiological alignment of their facets, especially during a single-foot stance and sudden rotation to regain balance. MID refers to unilateral distress of a posterior joint. It takes the form of elective mechanical root pain irradiating to the half girdle or towards the iliac crest, with enhancement during lateral or rotatory movements, with localised para-vertebral contractures and pain during palpation and rolling of the skin in the dermatomic area concerned. T11–T12 MID (Maigne's syndrome) is particularly frequent in footballers. Manipulations are usually effective, combined with decontractants and analgesics. Even so, one must be on one's guard against the secondary sedative effects of decontractants, which are particularly dangerous in this type of sport. The ever-increasing inclusion of osteopathic kinesitherapists as members of a team's staff illustrates both the importance of this type of treatment the frequency of MID.

Muscle Traumas

The traumatic context is evident following a direct blow from an opponent's foot or knee. Pain is immediate, aggravated by stopping of the activity, with substantial muscle contractures leading to a pain-relieving attitude. Root signs are absent, however, and the neurological picture is normal. Radiographic assessment may disclose a transverse apophyseal fracture. More important, ultrasonography allows examination of the contracture and differentiation, for therapeutic and prognostic purposes, of a simple bruise (requiring massage and physiotherapy) from an intra-muscular haematoma, which requires a follow-up and sometimes a puncture in accordance with the protocols for extrinsically induced muscle lesions. Renal assessment (haematuria, ultrasonography) must be systematic to look for uncommon but possible complications.

Chronic Disorders

These are revealed by the onset of spinal pain, sometimes associated with atypical (sacroiliac, lower-limb) pains whose functional disturbance may be

permanent or cyclic in function of the intensity of the sport. They may reflect anatomical abnormalities (spondylolisthesis, sequelae of Scheuermann's disease, scoliosis, mega-transverse process, or transitional vertebra), overuse due to intensive training and too frequent matches, or precocious arthrotic degeneration (discopathy, posterior arthrosis) provoked by repeated microtraumata. Diagnosis is usually radiographic. Medical and/or functional management will be determined by both the cause of the disorder and its type of disturbance and stage.

Spondylolisthesis

The lumbo-sacral spine is heavily stressed in football. The onset and progression of lysis of the pars interarticularis of one of its vertebrae with forward displacement of the body on the vertebra below it, or upon the sacrum, may be secondary to an acute fracture of the pars due to a severe injury in lumbar hyper-extension but is usually the expression of a fatigue fracture provoked by repeated "cigar-cutter" stresses with compression of the pars pinched between the articular facets of the vertebrae above and below it. A poorly or non-immobilised fatigue fracture progresses to pseudoarthrosis and lysis of the pars to spondylolisthesis in the event of deterioration of the underlying disc, with forward displacement of the body of the vertebra on the vertebra below it, or upon the sacrum, which is no longer restrained by the anterior discs and ligaments and the posterior bones.

This disorder is not a contra-indication to normal sport. Even so, both the stage of the displacement and the degree of dysplasia must be determined to assess the risks of aggravated progression, monitor the disorder, and decide how to treat it in the light of the clinical and radiographic findings. Meyerding has described four stages of displacement vis-à-vis the underlying vertebra. Roy-Camille has stressed the relationship between the extent of the local and regional dysplasia and the risk of progression. His three grades of dysplasia involving the vertebra, the sacrum, and the inter-vertebral disc are:

- Grade I: minimum dysplasia confined to the body of the vertebra, which is slightly trapezoidal, and sometimes accompanied by occult spina bifida.
- Grade II: more marked dysplasia. The body is trapezoidal, the sacral cupula is readily S-shaped, the sacrum is hooked, and the dysplasia of the components of the posterior arch is more marked than in grade I.
- Grade III: major dysplasia, with a re-worked, truly trapezium body, elongated pars interarticularis, and dome-like sacral plateau. This grade carries the greatest risk of displacement to the point of spondyloptosis.

The risk of progression is dependent on the following intrinsic factors peculiar to the patient and extrinsic factors associated with sport:

- Intrinsic factors: age, the extent of the dysplasia and the height of the disc.
- Extrinsic factors: frequency, repetition, and intensity of sport responsible

for stresses by microtraumata and mechanical overuse.

Most lyses of the pars observed in footballers are grade I, with good functional tolerance and a low risk of displacement, both compatible with engagement in even high-level sport. A grade III spondylolisthesis is obviously a contra-indication to football and *a fortiori* to competitive football on account of its inevitable progression. Grade II forms in young adults pose the most therapeutic uncertainties. Annual radiological and clinical surveillance is needed to determine whether sport should be abandoned on the appearance of displacement and to consider the resort to arthrodesis in the event of excessively aggravated displacement.

Muscle Overuse Disorders

These take the form of dorso-lumbar para-vertebral myalgia due to muscle fatigue induced by repeated tension and often reflect qualitative or quantitative over-employment of the spinal muscles in relation to their current physiological capacities. They are more the outcome of spasmodic contraction during a movement rather than the movement itself. Clinical examination discloses widespread contractures with no sign of disco-radicular impingement or specific evidence of posterior joint attack. The radiographical picture is normal, with no indication of advanced degeneration. Treatment is functional (massage and physiotherapy). It must be primarily preventive since these myalgias reflect a problem of physical preparation and/or recovery after sport.

Degenerative Disorders

The spine starts to be stressed at a very early age, substantially during football, and thus the onset and progression of precocious degeneration associated with discopathies owing to giving way of a disc and posterior arthrosis. Often asymptomatic, such degeneration may result in chronic spinal pain associated at times with atypical and poorly systematised nerve-root pains. Radiological diagnosis shows the number of levels attacked, and CT is used to confirm arthrosis and rule out disc herniation. Treatment is primarily functional, with reestablishment of lumbo-pelvi-femoral equilibrium (by stretching and checking the position of the pelvis), reinforcement of the musculi abdominis, and development of lumbar locking in the specific actions. Combination of physiotherapy and massage with manual techniques and sometimes with infiltrations may be useful at the start of a re-education programme.

Therapeutic Indications and Practical Advice

Treatment of footballer's back problems is always medical at the start, then, in function of their aetiology (clarify from the progression in 48 h and the complementary examinations), manipulation, corseting, radio-guided infiltration, or occasionally surgery may be necessary. Functional preventive management, however, will always be mandatory to avoid recurrences and chronicity.

Medical Treatment

Rest is scaled to the intensity of pain and must be absolute in decubitus if needed to obtain painlessness, coupled with systematic prescription of analgesics, anti-inflammatory agents, and muscle relaxants. Massage and physiotherapy coupled with heat and low-frequency antalgic currents also help to secure speedy amelioration of pain and allow clinical examination and complementary aetiological examinations to detect disc herniation, MID, or simple overuse with reflex-muscle contractures.

Specific Complementary Treatments

Traumatic discopathy in footballers can usually be non-operative, apart from the case of a paralysing excluded hernia. Persistent nerve-root pain when resting, however, can be treated by radio- or CT-guided infiltration at the disco-radicular impingement site and maintenance of an analgesic attitude by tight corseting. In the absence of a hernia and after systematic radiographic evaluation, osteopathic assessment may authorise the use of manual manipulations to treat an MID. Lastly, mesotherapy is being increasingly employed for athletes on account of its beneficial effects on reflex-muscle contractures.

Criteria for the Resumption of Sport

Sport may be resumed in function of pain improvement and control of spinal freedom of movement. It must be regular and progressive but by degrees in terms of intensity and duration of exercise and in function of clinical tolerance. In all cases, correction of factors promoting recurrences, especially intrinsic anatomical and, above all, muscular factors, should allow veritable prevention.

Prevention

A footballer's training alternates physical preparation for running and "technical-tactical" sessions. To be able to assimilate this training, the basic work must comprise specific muscle work and well-codified stretchings. One can, in the best of cases, rely upon an inter-seasonal isokinetic evaluation of the spine. The advantage of this muscle assessment is that muscle performance is studied functionally. After a 10–15 min warm-up, it is carried out at a slow 60/s and a fast 150/s to reveal muscle insufficiency and check the effectiveness of preventive measures. The aim is to strengthen the muscular stays of the spinal column. Special emphasis is placed on the work of the abdominals and deep spinals to oppose hyper-extension of the lumbar spine. The great rectus and the obliqui and transversi must be strengthened. For the great rectus, use is made of both concentric work and eccentric strengthening. The latter is of great importance because it is eccentric contraction of this muscle that controls extension of the lumbar spine during a shot at the goal. The deep spinals are worked by active axial stretching on the part of the subject. Stretching is done to ease sub-pelvic rigidity and avoid anterior tilting of the pelvis. It is personalised in function of an essential morpho-static evaluation. Particular attention will be devoted to the rectus anterior, which is often retracted, but also to the pelvi-trochanterians and the ischio-crurals. These stretchings must constitute an integral part of the general physical preparation programme. Each training session must start and end with a series of stretchings. Slow stretchings are preceded by static contraction of the muscle to be stretched to secure better muscle relaxation. Maximum stretching must be maintained for 30 s. A lumbar strap may be adopted as a temporary measure to improve proprioceptive control of the spine during sport. Lastly, prevention depends on both a good physical condition and training in the actions required, together with footwear appropriate to the quality of the terrain and strict personal hygiene.

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Muscle Strains

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Introduction

The muscle's physical characteristics enable it to absorb energy. An injury occurs when the energy generated exceeds this intrinsic capacity.

Muscle injuries can be classified into four main groups:

1. Delayed-onset muscle soreness (DOMS)
2. Strains:
- First degree: local fibril and filament damage with no solution of continuity
- Second degree: interruption of a certain number of muscles fibres without involvement of a grossly recognisable portion of the belly
- Third degree: rupture of a large portion of the belly with clinically evident solution of continuity
3. Contusions:
- Intermuscular haematoma
- Intramuscular haematoma
4. Avulsions:
- Osseous
- Apophyseal
- Muscular

In the case of strains, the muscle-tendon junction is particularly at risk due to its lesser extensibility and the sudden reduction of local circulation in the tendon compared with the muscle and the bi-articular muscles, especially the hamstring with their greater number of type 2, fast-contraction fibres [1–3]. The clinical and functional picture of strains is worse the greater the tissue damage (Table 1).

Biomechanical Principles

Examination of the epidemiological and clinical features of muscle injuries in

Table 1. Clinical and functional picture of muscle strains

Degree	Pain and contracture	Swelling and effusion	Loss of range of motion	Loss of function	Recovery time (days)
First	+	Minimal	Minimal	Minimal	7–20
Second	++	Moderate	Significant	Significant	21–50
Third	+++	Extensive	Severe	Complete	60–180

football must be preceded by an account of the biomechanical principles involved in kicking a ball since this action is likely to damage specific groups of muscles. A kick comprises six biomechanical components [4]:

1. Approach to the ball: frontal or at an angle
2. Ground reaction forces on the supporting limb side
3. Loading of the swinging limb
4. Flexion of the hip and extension of the knee
5. Foot contact with the ball
6. Follow-through.

The main difference between skilled and unskilled players is the share of movement they devote to each component. In unskilled players, kicking is poorly co-ordinated and dominated by the approach phase whereas in skilled players, it is determined by a combination of the approach and both backward and forward swinging of the leg. During loading of the swinging limb, the hip flexors and knee extensors develop negative power due to their eccentric activation by the backward swing of the limb. When the limb begins to swing forward, their activity becomes concentric, and the force vector changes from negative to positive. Before contact with the ball, hip flexors and knee extensors develop a peak force of 280 Nm and 230 Nm, respectively [5]. Intense contraction of the flexors swings the thigh forward and downward. The leg and foot move as a single unit and passively follow the swing. When the thigh's angular velocity decreases, both leg and foot start to accelerate due to a shift of the moment of force along the kinetic chain and to support against the elastic and contractile components in the knee extensors [6]. Better co-ordination and definition of movements enable skilled players to kick the ball further than unskilled players, with less and prevalently eccentric antagonistic muscle activity.

On contact with the ball, the hip flexors and knee extensors act concentrically and synergically whereas at other points during a kick, they act eccentrically as antagonists in keeping with the *the soccer paradox* whereby flexor activity is dominant during extension and extensor activity during flexion. DeProft et al. showed that the activity of the quadriceps and the hamstring is greatest

when they oppose the movement of the limb: the quadriceps during the loading phase and the hamstring during the forward swing [7]. Robertson and Mosher, too, observed no extensor activity immediately prior to ball contact. This phase, in fact, is dominated by the eccentric activity of the hamstring, which reduces the angular velocity of the knee and prevents its hyperextension [5].

The foot/ball contact lasts 6–16 ms, depending on the ball pressure, and takes place within the last few degrees of knee extension. The angular velocity at the knee 15 ms before contact is between 1,500° and 2,000°/s while the estimated impact force is 1–1.1 kN [8–11]. The hip is flexed when the impact occurs. This means that the angular velocity of the thigh is minimal and has little effect on the outcome of the kick. Follow-through is a protective mechanism for the swinging limb since it dissipates the muscle and elastic forces generated during loading [6]. It consists of concentric hip flexion followed by eccentric hamstring activity and then by hip extension [5].

Epidemiology

The literature shows that the quadriceps, hamstring, and adductors are the muscles most involved although to varying degree. Morgan and Oberlander's study of 10 US major league teams found that strains constituted 21% of all injuries that required at least 1 week's rest and that the hip adductors were primarily involved [12]. Ekstrand and Gillquist found that strains accounted for 25% of all injuries and that of these, the hamstring accounted for 47% [13]. The hamstring was also the most involved in the prospective study by Witvrouw et al.: of 146 Belgian professionals during a single season [14]. Our survey of strains in an Italian major league team during a 5-year period showed that they accounted for 30% of the total injuries. The quadriceps was most frequently involved (32%), followed by the hamstring (28%), adductors (19%), and gastrocnemius (13%) [15].

Risk Factors

Many factors predispose to strains [13, 16–24]:

- Flexibility deficiencies;
- Concentric-eccentric muscle weakness;
- Agonist-antagonist strength imbalance;
- Lack of training;
- Insufficient warming up;
- Excessive fatigue;
- Previous muscle injury;
- Insufficient rehabilitation.

There are conflicting opinions about the role of flexibility in preventing strains. Orchard et al. found no correlation between deficient hamstring flexibility and the risk of injury in Australian footballers [25] whereas the prospective study by Witvrouw et al. revealed that players with a hamstring or quadriceps injury had a significantly lower flexibility at the start of the season [14]. Because previous muscle injury is a well-established intrinsic risk factor [26], the authors excluded all players with a history of lower-limb muscle strain in the previous 2 years. Flexibility of the quadriceps, hamstring, adductor, and gastrocnemius was measured by means of specific tests. The quadriceps was evaluated goniometrically with the subject prone, and the knee was passively and maximally flexed with the contra-lateral foot on the floor and the hip flexed at 90°. Hamstring flexibility was measured with the subject supine and by passively flexing the hip with the knee extended to the point where knee flexion showed that the hamstring flexibility limit had been reached. There was a significant correlation between hamstring flexibility of less than 90° and the risk of an injury. Adductor flexibility was evaluated with the subject supine and legs together. The leg being tested was passively moved away from the mid-line until femoral rotation indicated the end of adductor flexibility. Flexibility of the gastrocnemius was tested with the subject standing with the tested leg behind its fellow and the sole of the foot resting on the ground along the sagittal axis. The tested ankle was dorsally flexed, and the maximum angle reached was measured. There were too few adductor and gastrocnemius injuries to establish any correlation with flexibility. Ekstrand and Gillquist found a correlation between adductor tightness and injury [27], whereas Tyler et al. did not [28]. Ekstrand and Gillquist also showed that insufficient warming up is another strain predisposing factor. They noted that hamstring accidents were more common in teams that did not employ an appropriate muscle stretching programme and that quadriceps strains were more likely to be caused by shooting at the goal before warming up [29]. Gabbe et al. conducted a prospective study of 222 Australian rules footballers to identify intrinsic risk factors for hamstring injuries. During the season, there were 31 hamstring strains. A hamstring injury in the previous 12 months was recorded in 29 players. Since these subjects displayed marked flexibility, the Authors suggest that over-emphasis on recovery of flexibility during rehabilitation may not help to prevent recurrences [30].

Since strains frequently occur during eccentric activation, it has been suggested that the microscopic damage provoked by eccentric exercise may sometimes result in a gross lesion. This depends on the optimum muscle length for active tension, in other words, the optimum angle for peak activity torque. Brockett et al. found that in a previously injured hamstring, torque peaked at significantly shorter lengths compared with both the contra-lateral muscle and those of uninjured subjects, whose optimum angles lay between

16° and 34° of knee flexion. If a typical value for an uninjured hamstring is about 20°, subjects with significantly higher values are potentially at risk. The case series in this study, however, was too small to endow this figure with any predictive value [31].

Following biomechanical principles, we have determined the extent to which eccentric contraction of the hamstring during forward swinging of the leg dissipates kinetic energy. Eccentric contractions produce microscopic damages and may eventually result in the formation of areas of structural weakness, from which a major tear may arise [32]. Dissimilar lengthening of the sarcomeres, presumably beyond the optimum muscle length for active tension where sarcomere length is unstable, has been posited as the pathogenetic mechanism [33, 34]. When a muscle is stretched beyond its optimum length, in fact, the longer sarcomeres will adapt more quickly to the stretching imposed by an eccentric contraction. A muscle with a short optimum length for active tension is more likely to be injured because more of its working range will lie in the region where microscopic damage occurs. Muscles are thought to adapt to the microscopic damage inflicted by eccentric exercise by increasing the number of sarcomeres in series in their fibres to augment the length of the optimum working angle and reduce the risk of damage [32] whereas concentric exercise decreases their number, and a shorter length is needed to produce peak torque [35]. Eccentric training of the hamstring is justified because eccentric activation is an integral part of their functional repertoire [36, 37]. The fact that hamstrings are often damaged during eccentric exercise is not the only consideration that must be borne in mind when drafting a rehabilitation programme. Angular velocity and joint angle are equally important. Angular velocity of the knee during a sprint may be as high as 600–700°/s. Hamstring is injured during its maximum eccentric activation to slow the forward movement of the foot and the leg in the late forward swing phase until the leg is stopped at about 30° of knee extension. Electromyography has shown hamstring activity is intense in the late swing phase during a sprint [38]. Muscle weakness and agonist–antagonist strength imbalance are controversial strain risk factors. In Australian rules players, Orchard et al. found that hamstring injuries were significantly associated with a low peak torque and flexor/extensor ratio evaluated with an isokinetic dynamometer [25] whereas Bennel et al. found in Australian rules players [39] and Grace et al. in American football players [40] no correlation between pre-season isokinetic peak torques and subsequent risk of hamstring injury. Strengthening has in any event been advocated to prevent hamstring injuries [41] in the light of animal studies showing that a strong muscle absorbs more energy prior to failure than a weak one [42]. Askling et al. reported that specific pre-season eccentric strengthening of the hamstring reduced the incidence of strains in 30 Swedish soccer players. In addition, 2 of the 6 players in

the study group with a prior history of hamstring strain and all 4 subjects in the control group were re-injured [43]. The marked tendency of muscle injuries to recur [4] means that great attention must be devoted to injured players. Prevention of recurrences, not quick recovery, is the primary goal of rehabilitation. It should always be borne in mind that whereas spontaneous resolution of tissue damage is the rule, previous or residual biomechanical loss of function can only be made good by re-education.

Lack of training is also regarded as a cause of muscle strains. In a personal 5-year survey of an Italian professional team, the training/match ratio was 3.6:1. Since sessions the day before and the day after a match concentrate on tactics and winding down, respectively, this ratio drops to less than 2:1 [15]. The role of training in preventing muscle injuries has been assessed by comparing 8 Danish with 14 Swedish professional teams. In Denmark, the soccer season runs from the autumn to the spring, as in the rest of Western Europe, whereas in Sweden it runs from spring to autumn. In Denmark, the pre-season lasts about 4 weeks, starting from the second half of July, and there is a mid-season break focused on training from the end of December to February. In Sweden, the pre-season lasts 12–16 weeks, usually from December to March. The authors maintain that this longer pre-season allows more time to be devoted to muscle strengthening and general conditioning. During the season, in fact, the incidence of muscle strains in the Danish teams was almost twice that in the Swedish teams [44].

Principles of Rehabilitation

Slight injuries are repaired by mononuclear satellite cells that differentiate into myoblasts. More serious injuries are mainly repaired by the formation of scar tissue. The way in which recovery of function progresses is of fundamental importance because it guides the correct repair of newly formed tissue.

As we have seen, hamstring lesions are particularly frequent in soccer. An account will now be given of the principles applicable during the rehabilitation of a subject with a stage II injury [45]. Three time phases are distinguished: the acute phase, remodelling, and recovery of function (Table 2) [46–48]. The first step requires application of a compressive elastic bandage and local cryotherapy [20 min/h). This phase is marked by local haemorrhage, myofibrillar retraction, and oedema due to enhanced capillary permeability. The goal of the RICE (rest, ice, compression, and elevation) procedure is to limit the initial damage. There are conflicting views concerning the real effectiveness of compressive bandaging. In a prospective study by Thorsson et al., compression within 5 min of thigh and calf injuries in 19 subjects was

Table 2. Repair phases of a stage II injury

Acute phase		Time
1	Lesion	0–6 h
2	Inflammatory reaction	6–24 h
3	Phagocytosis	24–48 h
Remodelling		
4	Initial repair	3–6 days
5	Advanced repair	7–14 days
Functional recovery		
6	Recovery of function	15–60 days

not more effective in reducing haematoma and recovery times compared with 20 treated with ice and elevation only and in some cases with compression after 10 30 min [49]. Non-steroidal anti-inflammatory drugs (NSAIDs), muscle relaxants, and other drugs are not used. Pain, in fact, is not usually such as to require analgesic cover. The usefulness of NSAIDs to limit inflammation in cases of DOMS and strains is uncertain [50]. Biochemical and histochemical studies have shown that indomethacin reduces local muscle damage [51]. Reynolds et al., however, found that NSAIDs are not useful in the acute phase of hamstring injuries [52]. Administration of corticosteroids at this stage hinders healing [50].

After 24–48 h, marked oedema is associated with mechanical muscle weakness due to massive macrophage invasion. An over-aggressive approach at this stage may cause further tissue damage, prolong the inflammation, and delay repair. The outstanding neuro-reflex action of transcutaneous electric nerve stimulation (TENS) can be safely and effectively used to relieve pain [53, 54]. Weight-bearing walking with two crutches can be started, if tolerated. One crutch is usually abandoned on the third day, and the bandage is removed for echography and magnetic resonance imaging (MRI), if required. The crutches are abandoned when a normal gait is resumed with no local pain. Recovery of normal walking can also be attained in water at decreasing depths since its hydrodynamic, hydrostatic, proprioceptive [55], and thermal effects allow the commencement of active mobilisation and re-instatement of the range of motion (ROM).

Initial remodelling starts on the third to sixth day when fibroblasts deposit collagen. Healing is encouraged by the capillary neovascularization thus induced since it brings the oxygen and nutrients needed for regenerative and reparative metabolism [56]. Biostimulation can be enhanced by physical

therapies, such as the neodymium-YAG laser and the capacitive and resistive transfer biostimulation therapy (TECAR therapy) [57], according to specific protocols and in function of the echographic picture, though sufficient scientific evidence of their effectiveness is still lacking. Recovery of a normal, painless gait is followed by cautious passive stretching in water to promote relaxation of the muscle fibres around the lesion. Massage is not employed at this stage. Isometric muscle contraction is begun in the form of submaximal exercise below the pain threshold.

During the advanced repair stage (7th–14th day), fibre regeneration is accompanied by approximately 50% loss of muscle strength compared with the situation prior to the accident. This is thought to be more correctly attributable to the inflammatory nature of the repair process, as shown by oedema and pain, rather than a real reduction of contractility. There is a high risk of recurrence in this stage; pain and function improve, but the patient is still vulnerable structurally. When satisfactory hamstring elasticity is reached, submaximal concentric and then eccentric muscle strengthening is begun against manual resistance.

Recovery and maintenance of tissue elasticity are achieved by means of specific passive stretching exercises. Bandy is of the opinion that daily 30-s passive stretching sessions are optimal and that 60-s stretching is not accompanied by a parallel increase in flexibility [58–60]. Aerobic exercises on a bicycle ergometer and step machine are introduced at this stage whereas running is postponed until the recovery of function phase. Proprioceptive reeducation exercises are conducted at three progressive levels [61]: joint positioning/repositioning (cortical level), eyes open/shut one/two foot-balance training (subcortical level), and reflex dynamic stabilisation exercises and sport-specific activities (spinal level). During functional recovery (15th–60th day), maturation of collagen and full recovery of voluntary muscle control mean rehabilitation must be directed to the total recovery of strength and functional efficiency. Normal gait, complete muscle elasticity, and painless maximum isometric contraction are usually achieved by the third or fourth week. The progress of fibre remodelling in the scar site is checked echographically at this time.

Heiser et al. advise commencement of running when the peak moment of force at 60°/s is not less than 70% of the other limb [62]. This, however, requires the execution of a maximum isokinetic test that could itself result in a recurrence. In addition, the isokinetic dynamometer evaluates muscle strength in open kinetic chain and without loading. Joint kinematics in a closed kinetic chain system and with loading therefore cannot be reproduced, and this instrument cannot be used to evaluate the musculature's role as a dynamic joint stabiliser. Aagaard et al. have suggested interpolation of the eccentric force peak of the hamstring with the concentric force peak of the

quadriceps (Ham ecc/Quad con) to obtain the knee extensor functional ratio [63] in which the hamstring force peak is directly proportional to angular velocity at which the exercise is carried out and inversely proportional to the degree of knee flexion. A study by Dauty et al. of professional players found that a knee extensor functional ratio at 60°/s lower than 0.6 identified those who had resumed sport after a hamstring strain but could not predict a recurrence or a new hamstring muscle injury [64]. Initially, submaximal eccentric isokinetic work at increasing speeds (starting from 60°/s) is carried out no more than 3 times a week. Overloads are avoided to prevent muscle fatigue. Because hamstring injuries classically occur during the eccentric phase of contraction, a specific eccentric strengthening programme is required. Work at high angular velocities is the hub around which the rehabilitation programme revolves. As Jonhagen et al. have shown, recurrences in athletes whose sport demands higher angular velocities, such as sprinters, are often attributable to a loss of eccentric hamstring strength [65].

Electrostimulation, in our opinion, is not appropriate and there is no evidence of its utility in the literature. Unlimited training is commenced when the strength values are not less than 80% of those of the other limb and there is no muscle fatigue after protracted running.

Athletes who have suffered a hamstring injury must never give up eccentric work. The marked tendency of muscle injuries, especially those of the hamstring, to recur poses an additional difficulty for the physician in an attempt to strike an even balance between the primary need to prevent relapses and the need for quick recovery. It is hoped that resumption of competitive sport is possible when there has been a sufficient regaining of muscle force, strength and flexibility and neuromuscular control [2].

The site and extent of a lesion, the motivation and state of mental and physical health of the subject, the adequacy of the re-educative techniques employed, and perfect integration of the work of all components of the medical staff are all aspects that influence complete recovery of function. Recurrence within 2 months from the resumption of sport is a clear indication of the inappropriateness of the rehabilitation programme employed.

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Tendon Injuries

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Introduction

Injuries to the muscle-tendon complex are among the commonest injuries sustained in football players [1]. Tendon injuries were ranked fifth after muscle injuries, joint injuries, contusions, and low back pain in an analysis of 254 injuries of a top Italian football team. In a similar study of the Australian Football League (AFL), patellar tendon injuries were ranked fifth, and Achilles tendon injuries were ranked seventh in terms of incidence of number of injuries per team season [2].

The management of tendon injuries can be frustrating and prolonged. The recurrence of Achilles and patellar tendon injuries in AFL players was 23% and 20%, respectively [2]. This high rate of recurrence results from early return to play in a competitive professional environment. It is therefore important to understand the exact aetiology and pathogenesis of these injuries.

Achilles Tendinopathy

Aetiology

Excessive loading of the tendon during vigorous physical training is considered to be the main pathological stimulus for Achilles tendinopathy [3]. The tendon may respond to this repetitive excessive overload beyond physiological limits by either inflammation of its sheath or damage with failed healing response of its body, or by a combination of these two mechanisms [4]. Tendon damage can also occur if the tendon is stressed within its physiological limits, as frequent cumulative microtrauma may not leave enough time for recovery and repair. Microtrauma may result from non-uniform stress distribution within the Achilles tendon as a result of different forces from the gastrocnemius and the soleus, producing abnormal concentrations of load

within the tendon, frictional forces between the fibrils, and localised damage to fibres [5]. Tissue hypoxia and consequent reperfusion injury and exercise-induced hyperthermia have been implicated in the pathogenesis of Achilles tendinopathy.

Relatively poor vascularity of the tendon, dysfunction of the gastrocnemius-soleus, age, gender, body-weight and height, pes cavus deformity, and lateral instability of the ankle are common intrinsic factors that predispose to Achilles tendinopathy. Changes in training pattern, poor technique, previous injuries, footwear, and environmental factors such as training on hard, slippery, or slanting surfaces are extrinsic factors that predispose to Achilles tendinopathy.

Histologically, there is non-inflammatory intra-tendinous collagen disruption, disorientation and thinning of fibres, hypercellularity, scattered vascular in-growth, and increased inter-fibrillar glycosaminoglycans. The process is characterised by absence of inflammatory cells and a poor, haphazard, healing response [6].

In Achilles tendinopathy, pain and swelling are located in and around the tendon 2–6 cm proximal to the Achilles tendon insertion on the calcaneus. Initially, pain occurs after exercise. As the pathology progresses, pain may occur during exercise and, in severe cases, it may interfere with activities of daily living. The *painful arc* sign helps to distinguish between lesions of the tendon and paratendon. In paratendinopathy, the area of maximum thickening and tenderness remains fixed in relation to the malleoli from full dorsiflexion to plantar flexion. Lesions within the tendon will move with movement of the ankle [7].

Investigations

Ultrasound (US) scan and magnetic resonance imaging (MRI) will help identify the location and extent of the lesion. MR scanning helps to differentiate between paratendinopathy and tendinopathy of the main body of the tendon.

Management

Recovery is slow due to low oxygen consumption and slow synthesis of structural protein and can be further slowed by continuing excessive load. Conservative management aims to identify and correct the possible aetiological factors, at times using a symptom-related approach. It includes relative rest, analgesia, physiotherapy, and orthoses. Reducing training intensity and duration or a reduction in other aggravating activities may be beneficial, as

tendon loading stimulates repair and remodels the collagen fibres. Achilles tendinopathy is not inflammatory in origin: non-steroidal anti-inflammatory drugs (NSAIDs) should therefore not be used. Piroxicam did not benefit patients with Achilles tendinopathy in a double-blind, placebo-controlled study using NSAIDs [8]. Corticosteroid injections are contra-indicated [9]. Aprotinin (2–4 injections of 62,500 IU with local anaesthetic in the para-tendinous space) may offer lasting pain relief.

Physiotherapy techniques include cryotherapy, deep friction massage, augmented soft-tissue mobilisation, gentle stretching, and eccentric strengthening exercises. Eccentric strengthening exercises are performed by standing on tip-toes on a step and dropping the heel(s) down below the level of the step in a controlled manner, then raising the heels back to the starting position. The exercises are done with the knee straight (to eccentrically strengthen the gastrocnemius) or slightly flexed (soleus). Calf raises back to the starting position are controlled with the asymptomatic leg. Exercises are preceded by warm up and stretch, with ice massage to the tendon for 15 min after; 3x15 repetitions twice daily per week are recommended. Exercises are continued through mild or moderate pain and stopped if pain becomes unbearable. Progression to the subsequent step occurs when exercise can be completed with no pain or discomfort (Table 1). A prospective study demonstrated the benefit of eccentric strengthening exercises in non-insertional tendinopathy [10]. This regimen helped 89% of patients with non-insertional tendinopathy compared with 32% of patients with insertional tendinopathy of the Achilles tendon in a randomised, controlled trial [11].

Table 1. Sequence of progression of eccentric strengthening exercises regimen for Achilles tendinopathy

Step number	Exercise
1	Double heel drops: slow pace
2	Double heel drops: fast pace
3	Double heel drops with weights: slow pace
4	Double heel drops with weights: fast pace
5	Single heel drops: slow pace
6	Single heel drops: fast pace
7	Single heel drops with weights: slow pace
8	Single heel drops with weights: fast pace
9	Dynamic push-offs from step

Surgical Management

Surgery is recommended if non-operative management has proved ineffective for at least 3 months; 24–45.5% of the patients with Achilles tendon problem fail to respond to conservative treatment and eventually require surgical intervention [12–14]. Surgical technique is based on the principles of excision of fibrotic adhesions, removal of degenerated nodules, and making multiple longitudinal incisions in the tendon to identify and excise intra-tendinous lesions, restoring vascularity, and possibly stimulating the remaining viable cells to initiate cell matrix response and healing [15, 16]. Excellent or good results in up to 85% of patients are reported [17].

A longitudinal, medially placed, curved incision with the concave part toward the tendon is centred over the tendon lesion. This incision avoids injury to the sural nerve and the short saphenous vein and prevents direct exposure of the tendon in case of skin breakdown [18, 19] (Fig. 1). The paratenon and crural fascia are incised and dissected from the underlying tendon. The tendon is freed from adhesions on the posterior, medial, and lateral aspects. The paratenon should be excised obliquely to avoid post-operative formation of a constriction ring [20]. Areas of thickened, fibrotic, and inflamed tendon identified by the change in texture and color of the tendon

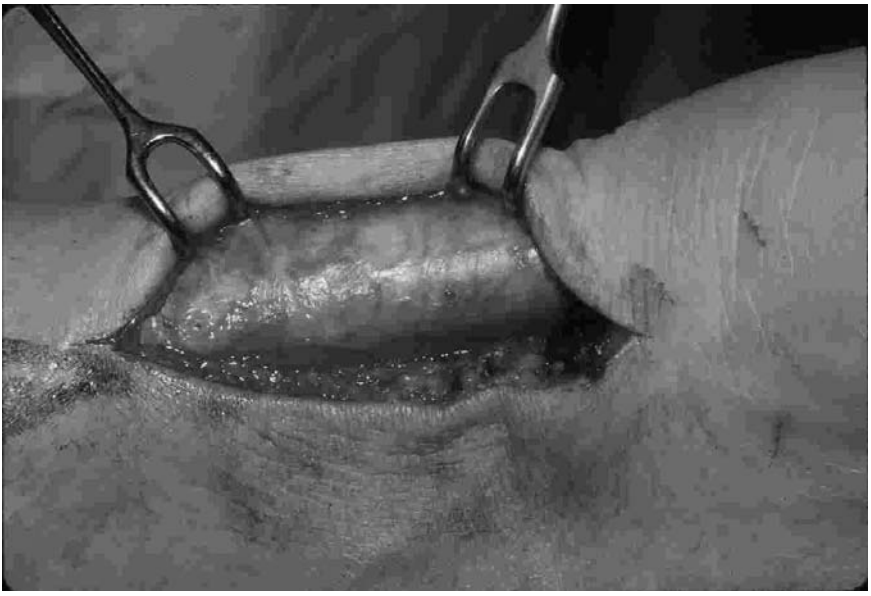

Fig. 1. Through a medial curved incision, inflamed paratenon and tendinopathic Achilles are exposed

are excised. The defect can either be sutured side-to-side or left open. Open procedures on the Achilles tendon can lead to difficulty with wound healing from the tenuous blood supply and increased chance of wound breakdown and infection. Good haemostasis speeds up recovery and diminishes wound infection and possible fibrotic inflammatory reaction. In patients with isolated Achilles tendinopathy with no paratendinous involvement and a well-defined nodular lesion less that 2.5 cm long, multiple percutaneous longitudinal tenotomies can be used when conservative management has failed. An US scan helps to locate the precise area of Achilles tendinopathy [21].

Long-standing tendinopathy may predispose to Achilles tendon rupture, as ruptured tendons show more advanced degenerative changes than tendinopathic tendons [22]. Acute Achilles tendon ruptures are more common in racquet-sport players than among footballers. Patients present with sudden onset of pain, snapping sensation in the calf, and absent calf squeeze test. We prefer percutaneous repair as it reduces the risk of infection, and functional results are comparable to open repair. We advocate early weight bearing and mobilisation as pain allows on the affected limb following surgical intervention of a ruptured Achilles tendon [23].

Insertional Achilles tendinopathy may present alone or as a triad in combination with retrocalcaneal bursitis and Haglund's deformity. It is seen in older, overweight athletes. Tight Achilles tendon, hyperpronation, pes cavus, and obesity can predispose to degeneration, attrition, mechanical abrasion, and chemical irritation that could lead to a chronic inflammatory response at the heel. Stress shielding and internal shear forces producing heat may also play a role in pathogenesis [24, 25]. Insertional Achilles tendinopathy presents with early-morning stiffness, pain localising to the insertion of the Achilles tendon that worsens after exercise, climbing stairs, running on hard surfaces, or heel running. Radiographs may reveal calcification at the insertion of the Achilles tendon or a spur (fishhook osteophyte) on the superior portion of the calcaneum (Fig. 2). Conservative management produces 85–95% success rate with rest, ice, modification of training, heel lift/orthoses [26, 27]. Surgery to excise the spur, the tendinopathic tendon, and bursa help alleviate symptoms if conservative management fails.

Patellar Tendinopathy

In patellar tendinopathy, anterior knee pain starts insidiously, and a specific activity may make the pain worse. Pain is well localised to a small area over the anterior aspect of the knee, and early in the pathological process, it may ease completely while exercising. With time and continued activity, the pain

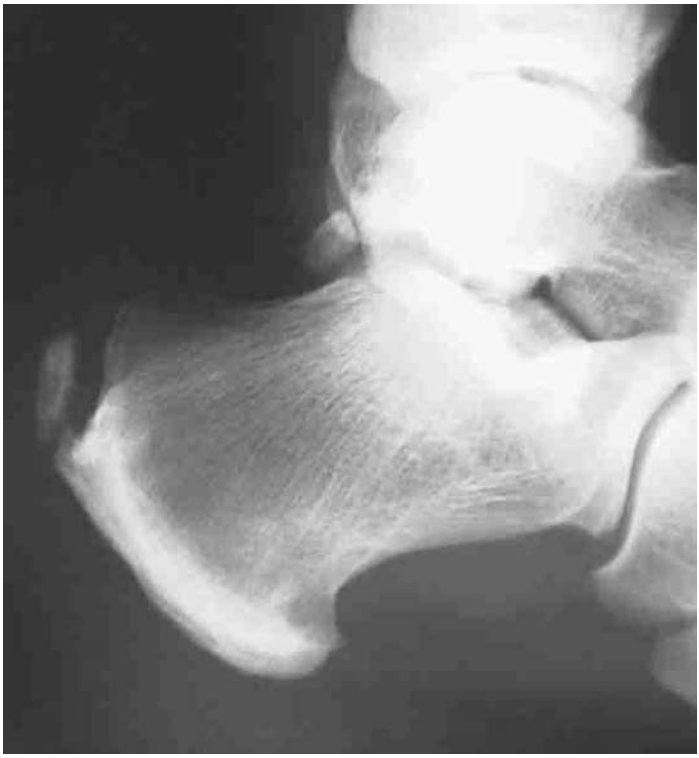

Fig. 2. Lateral radiograph demonstrating calcification of Achilles insertion and Haglund's deformity

worsens and limits sporting performance. Eventually, anterior knee pain can develop during activities of daily living and can even be present at rest.

Examination reveals tenderness at the junction of the patella and the patellar tendon. Palpation of the tendon attachment at the inferior pole of the patella is the classic physical examination technique for detecting patellar tendinopathy, but mild tenderness at this site is not unusual in a normal tendon in athletes. Only moderate and severe tenderness is significantly associated with tendon abnormality at ultrasonography. The criteria of Blazina et al. are used for clinical staging of patellar tendinopathy [28] (Table 2). Patello-femoral joint syndrome and patellar tendinopathy may co-exist.

Investigations

MRI will demonstrate an oval or round area of high signal intensity at the tendon attachment on T1-, T2-, and the proton-density-weighted images [29, 30]. It identifies the exact location and extent of tendon involvement and

Table 2. Clinical staging of patellar tendinopathy

Stage 1	Pain is present only after athletic participation with no undue functional impairment
Stage 2	Pain during and after activity, but still able to perform at a satisfactory level
Stage 3	Pain is present during and after activity, but is more prolonged, with progressive difficulty in performing at a satisfactory level
Stage 4	Patellar tendon is ruptured

helps to exclude other conditions such as bursitis and chondromalacia. Ultrasonography should include both knees, and it shows a focal hypoechoic area combined with swelling of the surrounding tendon (Fig. 3). Hyperechoic regions within the tendon correspond with dystrophic ossification. US scan helps surgeons to accurately locate the area of tendinopathy for correct placement of the scalpel blade while performing multiple percutaneous longitudinal tenotomies [31].

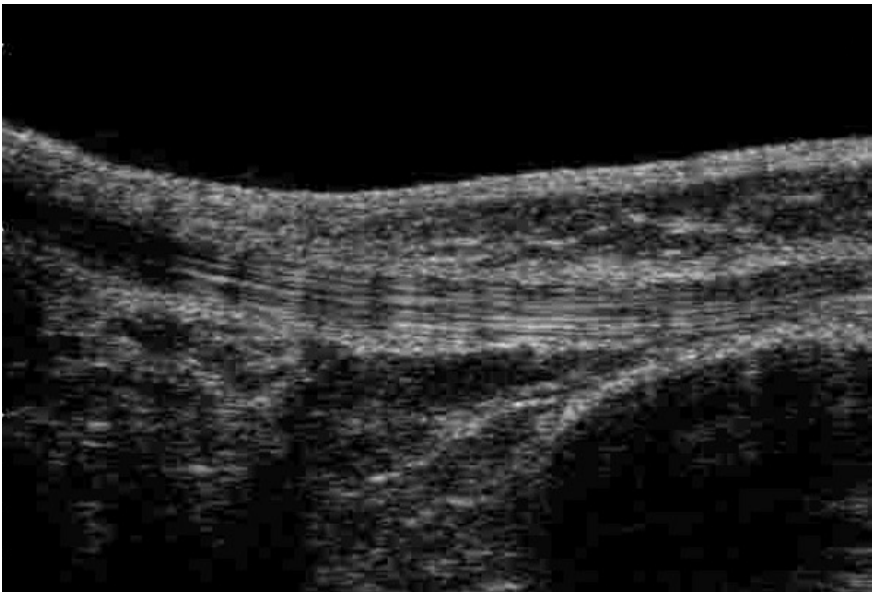

Fig. 3. Ultrasound of the patellar tendon demonstrating hypoechoic area distal to the inferior pole of the patella

Conservative Management of Patellar Tendinopathy

Conservative management includes measures to reduce the load on the tendon, eccentric strengthening exercises, remedial massage, cryotherapy, and drugs:

1. Decreasing load on the tendon can be achieved by reducing overall activity, correcting biomechanics to improve the energy-absorbing capacity of the limb (for example, flatfoot landing vs. forefoot landing [32]). Pes planus and hyperpronation should be addressed if present. Shoe orthoses are helpful. Low flexibility and weakness of quadriceps, hamstrings, iliotibial band, calf muscles, gluteal, and lower abdominals must be corrected.
2. Eccentric strengthening is the keystone to successful management of tendinopathies, including patellar tendinopathy [33, 34]. The key exercise is a drop squat from standing to about 100–120° of knee flexion. Patients perform 3 sets of 10 repetitions per day. Symptoms may resolve after 6–8 weeks of exercises. Progression to the next level of the programme should only be performed if the previous workload is easily managed, pain is controlled, and function is satisfactory. Rehabilitation and strength training must also continue through the return to sports. However, the simple addition of eccentric exercises continuing the normal training routine does not decrease the pain from patellar tendinopathy [35].
3. Cryotherapy and other physical modalities: cryotherapy decreases blood flow and metabolic rate, thereby limiting tissue damage. Other modalities include US, heat, interferential therapy, magnetic fields, pulsed magnetic and electromagnetic fields, transcutaneous electrical neural stimulation (TENS), and laser. The true effects of these modalities remain unknown.
4. Remedial massage decreases the loads on the tendons, improving muscle-stretching capabilities. Deep friction massage may activate mesenchymal stem cells to stimulate a healing response [36].
5. Drug management: The use of anti-inflammatory medication seems paradoxical in this condition though they are the most commonly used for controlling symptoms. Topical ketoprofen in patellar tendinopathy reached the target tissue, but the clinical outcome has not been assessed [37]. NSAIDs may act in ways other than their anti-inflammatory mode. Infiltration of corticosteroids by means such as iontophoresis has a dramatic effect on symptoms arising from inflamed synovial structures [38]. Corticosteroid injections are not recommended in management of tendinopathy (see above). Aprotinin was used in a randomised, control trail and found to be effective [39]. In the short term, it offers better chances of pain relief than do corticosteroids. As aprotinin is a polyvalent inhibitor of proteolytic enzymes, its administration is probably only warranted in athletes with relatively short symptom duration.

Surgical Management

Patellar tendon surgery is performed when the patient has not improved with at least 6 months of conservative management. The surgical methods for management of patellar tendinopathy include: drilling of the inferior pole of the patella, resection of the tibial attachment of the patellar tendon with realignment, excision of macroscopic degenerated areas, repair of macroscopic defects, scarification (i.e., multiple longitudinal tenotomy of the tendon), percutaneous needling, percutaneous longitudinal tenotomy, and arthroscopic débridement.

Arthroscopic procedure involves soft-tissue *débridement* over the retropatellar tendon surface to aid the visualisation of the patellar tendon insertion into the inferior pole. An 18-gauge needle is advanced through the area of maximum tenderness that is confirmed with imaging. The needle is visualised arthroscopically, and an inferior patellar portal is created perpendicular to the inferior pole of the patella; 8–10 ml of inferior pole of the patella are resected with a shaver.

Patellar tendon surgery has an unpredictable outcome, with a variable success rate of 46–100% [40]. The mean time for return to pre-injury level of sport varied from 4 months to more than 9 months. A long-term study of outcome reported that only 54% of open patellar tenotomy and 46% of arthroscopic tenotomy returned to previous levels of sport activity [41]. However, the median time to return to the pre-injury level of activity was 10 months for open and 6 months for arthroscopic tenotomy patients.

Quadriceps Tendinopathy

Quadriceps tendinopathy is much less frequent than patellar tendinopathy. This may be related to the superior strength, mechanical advantage, or vascularity of the quadriceps tendon. Patients with quadriceps tendinopathy complain of pain at the proximal pole of the patella. The pain is typically insidious and is often associated with a recent increase in jumping, climbing, kicking, or running. Physical examination reveals tenderness over the superior pole of the patella and discomfort with resistance to extension with the knee hyperflexed. Patients should be evaluated for malalignments such as femoral anteversion, increased Q angle, and tibial torsion. Quadriceps strength and hamstring flexibility should also be assessed.

Investigations

Degenerative changes such as calcification in the tendon or spur formation at the superior pole of the patella may be present on radiographs. MRI may demonstrate degenerative changes at the posterior insertion of the tendon.

Management

Conservative management consisting of activity modification, anti-inflammatory medications, and physical therapy is successful in the vast majority of patients. Once the pain improves, physiotherapy for quadriceps-strengthening exercises and increasing hamstring flexibility is initiated. Strengthening should focus on eccentric training of the muscle-tendon complex performed in 3 sets of 10 repetitions. Most cases resolve within 2–3 weeks. Surgery is rarely required. Indications include persistent tendinopathy in patients who have failed a 3- to 6-month trial of non-operative management. Surgical principles include *débridement* of degenerative, diseased tissue and promotion of healing by stimulating a vascular response either by longitudinal tenotomy and/or needling.

Partial Quadriceps Tendon Rupture

Partial ruptures of the quadriceps tendon are rare, and present with pain in the quadriceps tendon region and weakness of knee extension. Typically, a traumatic knee injury during sporting activity occurs in a pre-existing quadriceps injury. If the vastus intermedius tendon is detached, which is common, there may be no deformity. Examination reveals weakness of extension; strength from a flexed position needs to be compared with the contralateral side. Strength measurements help to document the deficit, but there is a risk of completing the tear with maximum resistance.

Investigations

Plain films are usually normal but may demonstrate degenerative calcific changes within the tendon. MRI helps in identifying the location and extent of the injury.

If tears involve more than 50% of the quadriceps tendon or are diagnosed late, surgical repair is preferred. When the diagnosis is made immediately in the acute situation, if the tear involves less than 50% of the tendon and there is no tendon retraction, conservative management with brace immobilisation for 6–8 weeks may be considered. As healing progresses, the amount of knee flexion allowed by the brace may be increased. Surgical repair of the chronic, partially torn tendon is recommended. With a longitudinal incision, the rectus femoris tendon is split to gain access to the vastus intermedius tendon. We use strong absorbable sutures, passing them through the vastus intermedius tendon and any detached tendons. The attachment site on the anterior half of the superior patella is abraded to bleeding bone using a curette and/or burr. The tendon is then sutured to the patella through drill holes or with bone anchors.

Semi-membranosus Tendinopathy

Semi-membranosus tendinopathy, a rare cause of medial knee pain [42], may be primary or secondary to primary knee abnormality such as patellofemoral disorders. Semi-membranosus tendinopathy causes aching pain at the posteromedial aspect of the knee, aggravated by prolonged jogging, climbing, or lifting. Clinical examination reveals tenderness at the posteromedial corner of the knee just inferior to the joint line. A thorough knee examination is mandatory to exclude intra-articular pathology.

Investigations

Bone scan demonstrates increased tracer uptake at the posteromedial aspect of the proximal tibia. MRI scanning will confirm the diagnosis and exclude a medial meniscal tear or pes anserinus bursitis.

Management

The initial management of rest, hamstring-stretching exercises, and analgesia is successful in the vast majority of patients with primary semi-membranosus tendinopathy. Surgical intervention is advised after failed conservative management of at least 3 months. It includes diagnostic arthroscopy to address intra-articular pathologies. An incision is made over the insertion of the direct head of the semi-membranosus tendon, and the sheath over the tendon is opened. Areas of degenerative tissue are excised, and several longitudinal tenotomies are performed. The insertion site is drilled with a small Kirschner wire to promote a healing vascular response. Nine out of 10 patients returned to sporting activities at an average of 12 months following surgical intervention [42]. Secondary semi-membranosus tendinopathy is managed by first addressing the primary knee disorder.

Popliteus Tendinopathy

Popliteus tendinopathy causes knee pain [43] along the posterolateral portion of the knee. It is insidious and occurs on weight bearing with the knee in flexion of 15–30° or during the early part of the swing phase. There may be a recent increase in activity levels, especially down-hill running or running on a banked surface. Pain is typically aggravated by running and relieved with rest. Examination reveals localised tenderness over the tendinous insertion of the popliteus over the lateral femoral condyle. This tendon is best palpated with the leg in the *figure-of-four* position. If there is no rest pain, the patient is run down hill prior to the physical examination to assist in localising the pathology. An injection of local anaesthetic into the tendon sheath may help in diagnosis.

Investigations

Plain radiographs are usually normal, but in patients with chronic involvement radiodensities in the area of the popliteus tendon may be seen. MRI helps exclude intra-articular pathology or a tendon avulsion.

Differential diagnosis: lateral meniscal pathology, iliotibial band syndrome, discoid lateral meniscus, degenerative joint disease, lateral collateral ligament injury, osteochondritis dissecans, intra-articular loose body, occult cyst of the lateral meniscus, and proximal tibiofibular instability.

Management

Most cases are acute and respond to a 2-week course of rest and analgesics. Upon return to activity, training modifications avoiding down-hill running, can help alleviate the stresses imposed on the popliteus tendon. Chronic cases usually require a longer period of restricted activities.

Popliteus Tendon Avulsion

Isolated rupture of the popliteus tendon is extremely uncommon [44–46]. The mechanism reported is external rotation of the lower leg with the knee in slight flexion [47]. Patients present with acute haemarthrosis and lateral knee pain. Pain and weakness may be present during active internal rotation of the tibia against resistance or during passive external rotation of the tibia with the knee flexed at 90°. A complete ligamentous examination should be performed to exclude injury to the lateral, posterior, or posterolateral structures.

Investigations

Plain radiographs may demonstrate a small, faintly visible, ossified fragment in the lateral gutter of the knee. MRI helps to confirm the diagnosis and exclude concomitant injury.

Management

Reduction and fixation of the tendon restores the motor, ligamentous, and proprioceptive functions of the popliteus complex.

Tibialis Posterior Tendinopathy

Tibialis posterior pathology and valgus flatfoot-pronation deformity are closely related. Repeated excessive pronation leads to tibialis posterior overuse. On the other hand, tibialis posterior tendinopathy causes eversion of the

hindfoot and fall of the medial longitudinal plantar arch, resulting in a pronated valgus flatfoot.

In tibialis posterior tendinopathy, there is a non-specific response to tissue injury with fibroblast hypercellularity, chondroid metaplasia, mucinous degeneration, and neovascularisation [48]. Collagen structure and orientation are disrupted, and the tendon is predisposed to rupture under physiologic loads. Factors predisposing to posterior tibialis tendinopathy:

- Mechanical: ligamentous laxity, articular hypermobility, a shallow retromalleolar groove, a tight flexor retinaculum, and navicular bone abnormalities (accessory scaphoid, prominent navicular tubercle) [49, 50].
- There is an area of hypovascularity immediately posterior to medial malleolus.

Examination will reveal whether there is a flatfoot, a valgus hindfoot (*too many toes* sign), and abduction of the forefoot. Subtle signs of tibialis posterior tendinopathy are detectable by the toe-raise test (inability to perform a single-foot tip-toe raise). The position of the calcaneus is checked. In a normal foot, the hindfoot is inverted. In tibialis posterior tendinopathy, the hindfoot remains neutral or valgus. Oedema and tenderness are present around the tibialis posterior tendon or at its insertion point. Flexion and inversion of the hindfoot against resistance are assessed and compared with the unaffected limb. Range of motion of the ankle, subtalar joint, hindfoot, and midfoot should be assessed. In advanced stages, a fixed valgus deformity, failure of the deltoid ligament, and degenerative joint disease of the lateral compartment of the ankle may be present. At this time, fixed supination of the forefoot can occur, keeping the foot plantigrade with fixed valgus hindfoot.

Investigations

US and MRI provide information about the extent of the condition and the degree of tendon pathology. US identifies paratendinopathy and intra-tendinous pathology. MRI is more sensitive for tears of the posterior tibialis tendon. Both are useful to monitor tendon pathology in athletes. Various clinical forms of tibialis posterior tendinopathy are described and can be considered different stages of the same pathology.

Acute and Chronic Paratendinopathy

The synovium surrounding the tendon can be hypertrophic and oedematous, but the main body of the tendon is not, or only minimally, involved. Symptoms onset is gradual and relatively aspecific. Pain worsens by prolonged weight bearing and walking. Pain is also elicited by retromalleolar palpation, passive eversion and abduction, and active inversion and adduction. Local swelling is often present, particularly in the posteroinferior portion of

the retromalleolar sulcus. Rarely, in chronic paratendinopathy, intra-tendinous calcifications can be found at US or MRI.

Partial and Complete Rupture

Ruptures of the posterior tibialis tendon may be missed, as they are relatively uncommon in footballers. Partial tears are more frequent in younger athletes, and total ruptures are more typical of middle-aged or former athletes. The patient has a pronated forefoot and an acquired flatfoot on the affected side. Pain and swelling are generalised on the medial aspect of the ankle, the tibialis posterior is inactive on manual testing, and the patient is usually unable to perform a single-leg heel raising. Clinical signs of tendon failure may be absent, but local pain and swelling, pain on resisted eversion and inversion, and an enlarged tibialis posterior tendon can point to the diagnosis.

In classifying dysfunction of the posterior tibial tendon, Johnson and Strom described three clinical stages [51] and Myerson later added a fourth stage [52].

- Stage 1: This stage is characterised by pain and swelling of the medial aspect of the foot and ankle. The length of the tendon is normal, and tendinopathy may be associated with mild degeneration. Mild weakness and minimum deformity are present.
- Stage 2: The tendon is torn, the limb is weak, and the patient is unable to stand on tiptoe on the affected side. Secondary deformity is present as the midfoot pronates and the forefoot abducts at the transverse tarsal joint. The subtalar joint, however, remains flexible.
- Stage 3: Degeneration of the tendon is present, the deformity is more severe, and the hindfoot is rigid.
- Stage 4: There is valgus angulation of the talus and early degeneration of the ankle joint.

Management includes rest, analgesia and orthotics with a longitudinal arch support, which prevents eversion. Excision of the thickened paratenon, longitudinal tenotomy of the tendon, and reconstruction of the retinaculum are useful if friction or stenosis is identified. For total rupture and avulsion of the tendon, surgery is mandatory. In general, this condition is rare in footballers, as it seems to be associated with increased age. We have only occasionally observed early stage I tendinopathy.

Flexor Hallucis Longus Tendinopathy

The tendon of flexor hallucis longus (FHL) inserts into the distal phalanx of the hallux and has three possible zones of stenosis along its course:

1. The bony-fibrous tunnel behind the medial malleolus where the tendon runs between the talus and the medial malleolus; its synovium may become irritated. The presence of an accessory tendon or muscle belly [53], an enlarged os trigonum, calcaneal fractures, and soft-tissue scars can entrap the tendon of FHL at this level [54].
2. At Henry's node on the plantar aspect of the first metatarsal.
3. Over the two sesamoid bones at the plantar side of the head of the first metatarsal [55].

Hyperpronation strains the FHL tendon at the retinaculum predisposing it to intra-tendinous pathology. Clinical features include pain and discomfort in the medial retromalleolar region elicited by flexion of the toes. Sometimes, hyperaesthesia or crepitation may be present. Fullness between the Achilles tendon and the tibia due to oedema and inflamed synovium within the tunnel are present. The hallux is stiff, with limited dorsiflexion at the metatarsophalangeal joint when the ankle is extended. MRI is the imaging modality of choice. Early recognition of tendinopathy of flexor hallucis longus is important. In the acute phase, rest, analgesia, and avoidance of extreme plantar flexion can be helpful. Surgical management is controversial. Conservative management is preferred, as the scar tissue produced by surgery may lead to persistent pain [56]. When conservative treatment has failed, surgery must be undertaken to remove the cause of stenosis, free the tendon, and remove degenerated intra-tendinous areas.

Conclusion

Tendinopathy of the lower limb in footballers is secondary to overuse, insufficient warm up, or various other mechanical factors. Reducing training intensity and duration or addressing other aggravating factors is beneficial in the early stages of tendinopathy. Eccentric strengthening exercises are effective in tendinopathies. We do not recommend non-steroidal anti-inflammatory medications or corticosteroids. Aprotinin injections in the peri-tendinous region are beneficial for short- to medium-term relief. If conservative measures fail, surgical management may relieve symptoms, but the success depends on the tendon affected and the severity and chronicity of the condition.

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Groin Pain

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Introduction

Groin pain refers to pain in the area where the abdomen ends and the legs begin. For males, the terms *groin* and *testicle* are sometimes used interchangeably. However, what causes pain in one will not necessarily do so in the other. Groin pain is also referred to as lower abdominal pain, genital pain, and perineal pain. In football players, groin pain is considered a pain in the pubic or lower abdominal or adductor region, which can be monolateral or bilateral. In this chapter, we deal with groin pain in football athletes considering anatomy, etiopathogenesis, clinical presentation, diagnosis, and treatment.

Anatomy and Biomechanics

Anatomically, the adductor (longus, magnus, brevis), the pectineus, and the gracilis muscles comprise the muscular component of the medial aspect of the thigh. These muscles have their origin at the symphysis pubis and the inferior pubic rami before traveling along the medial aspect of the thigh and inserting on the linea aspera of the femur. The lone exception is the gracilis, which inserts on the medial aspect of the proximal tibia. The tendinous and aponeurotic attachments of the rectus abdominis and the internal oblique muscles are also found at or near the adductor's origin. The close proximity of these various muscles often makes it difficult to distinguish the true source of the athlete's pain. Anatomically, the pelvis is between two major muscular systems, the back and the lower limbs, and is the junction point of tensile forces coming up from the rectus abdominis and the internal oblique muscles and down from adductors. Moreover, loads of up to 8 times body weight have been demonstrated in the hip joint during jogging, with potentially greater loads present during vigorous athletic competition. The structures about the hip are uniquely adapted to transfer such forces. The body's centre of gravity is located within the pelvis anterior to the second sacral vertebra; thus, the loads that are generated or transferred through this area are important in virtually every athletic endeavour.

Etiopathogenesis

The pathogenesis of groin pain can be extrinsic, such as overload or repetitive microtrauma, or intrinsic, such as anatomical disorder of the pelvis, hip, or groin. Many different theories about the cause of groin pain have been proposed based on the findings of the physical examination or the operation. One of the most frequent causes of groin pain is disruption of the musculotendinous elements of the groin. This injury affects the external oblique aponeurosis, the conjoined tendon, and the inguinal ligament, resulting in weakness of the lower abdominal wall and occult hernias. Disruption of the external oblique aponeurosis also pre-disposes to entrapment or irritation of branches of the ilioinguinal or iliohypogastric nerves. Enthesopathy at the site of insertion of the abdominal and adductor muscles to the pubic bone has been described as another cause of groin pain. Among various causes of groin pain that have been proposed, a symptomatic, non-palpable hernia has been described, with an incidence of 36% to 90%. In the literature, the terms *sportman's hernia* and *sports hernia* are used for "the syndrome of a weakness of the posterior inguinal wall without a clinically recognisable hernia", "bulge in the posterior inguinal wall consistent with a incipient direct (medial) inguinal hernia", or "an imminent, but not demonstrable, inguinal hernia". The term *athletic pubalgia* refers to chronic inguinal or pubic-area pain in athletes that is exertional only and not explainable preoperatively by a palpable hernia or other medical diagnosis and so may be a more appropriate term for these injuries. Possible causes of groin pain are summarised in Table 1.

Table 1. Common disorders of the hip and groin area (modified from [2])

History	Cause
Acute onset	Muscle
	Contusions (hip pointer)
	Avulsion and apophyseal injuries
	Hip sprains and subluxations
	Acetabular labral tears and loose bodies
	Proximal femur fractures
Insidious onset	Sports hernias and athletic pubalgia
	Tendinopathies
	Osteitis pubis
	Bursitis
	Snapping hip syndrome
	Osteoarthritis
Other disorders	Lumbar spine abnormalities
	Entrapment neuropathies

Incidence

Chronic groin pain is a well-recognised and problematic entity in athletes. Besides football, sports such as ice hockey, cross-country skiing, and others that require repetitive kicking, evasive or side-to-side motion, and physical contact are a common cause of groin pain, but the incidence of athletic pubalgia is lower. Groin pain among (professional) football players has an estimated incidence of 0.5–6.2% [1, 2] and is responsible for a large proportion of time lost from sport and work. Interestingly, this syndrome is rarely seen in female athletes.

Classification

Classification of groin pain can be based on either the site of pain or on symptoms. Classification according the site of pain is:

- Adductor localisation;
- Abdominal localisation;
- Mixed localisation.

  All these localisations can be uni- or bilateral.

  Classification according to severity of symptoms is [3]:
- Stage 0: no pain;
- Stage 1: pain only after intense sports activity; no undue functional impairment;
- Stage 2: pain at the beginning and after sports activity; still able to perform at a satisfactory level;
- Stage 3: pain during sports activity; increasing difficulty in performing at a satisfactory level;
- Stage 4: pain during sports activity; unable to participate in sport at a satisfactory level;
- Stage 5: pain during daily activity; unable to participate in sport at any level.

History

The history of the injury may vary from an insidious onset with progressive pain during intense pre-season or in-season training to a sudden, simple, painful event. Tendon damage at the adductor origin or at the lower abdominal insertion may commonly occur as an overuse injury. Pain localisation is also important to determine which structure may be causing the pain. However, sometimes the pain may be poorly localised and felt in a number of different areas simultaneously. It is important to establish the time course of

the pain, as pain worsening after exercises, especially the following day, and gradually decreasing during exercise is indicative of an inflammatory condition, such as tendonitis. It is important to establish which movements aggravate the pain, especially movements such as kicking, which may suggest an iliopsoas or rectus femoris strain; twisting, which may suggest an adductor muscle strain; or sit-ups, which may suggest a rectus abdominis injury or hernia.

Clinical Approach

In patients with groin pain it is important to localise the area of pathology. The pathology may be in the adductor muscles or in the lower abdominal musculature and may be unilateral or bilateral. Abdominal-wall-muscle injuries have been increasingly recognised as a source of chronic inguinal or pubic area pain in athletes and should be included in the differential diagnosis. The majority of these patients have lower abdominal pain with exertion; however, a small minority of patients have pure adductor-related pain. History and the physical examination can be difficult to distinguish from those seen with a sports hernia. These athletes commonly recall a distinct injury involving a combination of abdominal hyper-extension and thigh hyper-abduction and may complain of pain when attempting to perform a sit-up.

On physical examination, each region of the groin that has the potential to produce groin pain must be examined. This includes adductor muscles, pelvic bones, hip joint and its surrounds, hip flexors (including tensor fascia lata and sartorius) and lower abdominal muscles. The lumbar spine and sacroiliac joints are also examined. Pelvis alignment must be assessed, and any leg-length discrepancy noted. Pain can be reproduced with adduction of the hip against resistance (Fig. 1) if the adductor muscles are involved or with flexion of the spine against resistance in an abdominal localisation. Occasionally, there is a pubic or peri-pubic tenderness along the adductor tendons near the pubis (Fig. 2), making this quite difficult to distinguish from a pure adductor strain. The site of pain can usually be identified with palpation along the course of the musculotendinous unit against mild to moderate resistance in adduction and on passive abduction and external rotation.

The clinician must not overlook the less common but important causes of pain in this region, such as intra-abdominal pathology (e.g., appendicitis), urinary tract pathology, gynaecological pathology, and rheumatological disorders (e.g., ankylosing spondylitis). Infection such as osteomyelitis should also be considered. Patients with tumours, such as testicular tumours, occasionally present with groin pain. Moreover, abnormalities of the abdominal wall, including inguinal hernias and microscopic tears or avulsions of the

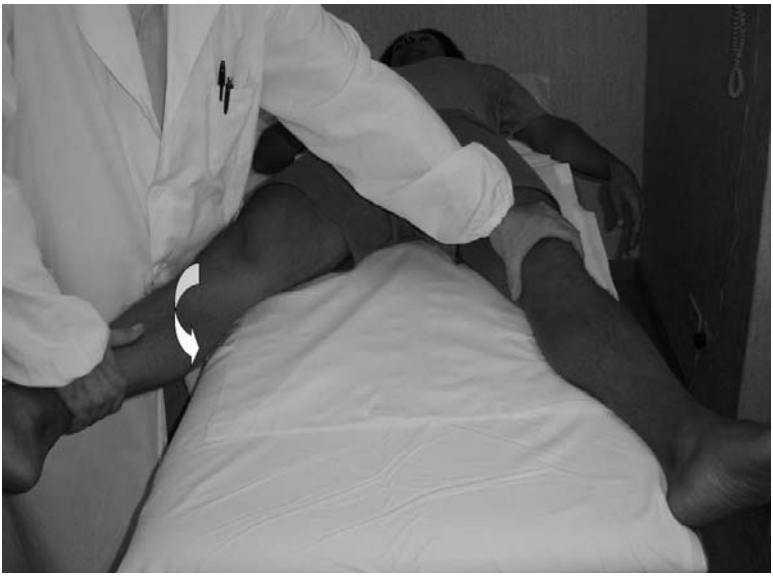

Fig. 1. Resisted movement: hip adduction. This should be performed with varying degrees of rotation to stress different adductor muscles. The neutral position stresses adductor longus preferentially, internal rotation stresses the pectineus, and external rotation the adductor magnus

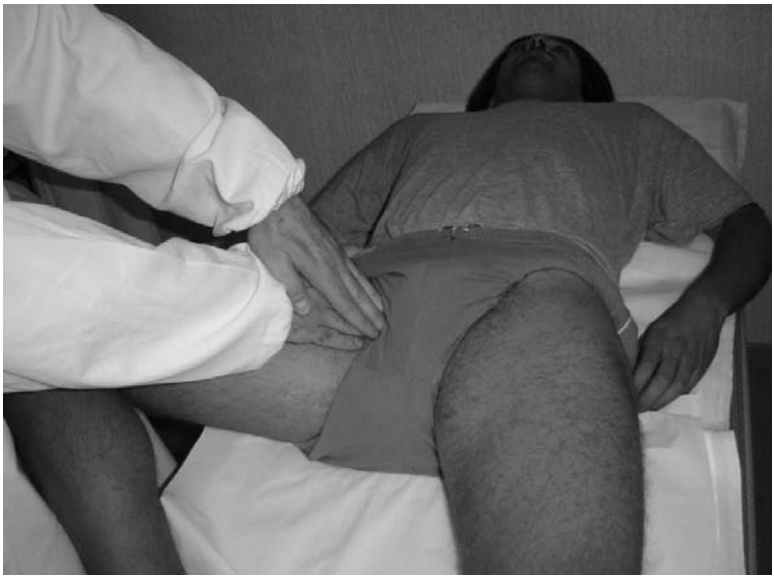

Fig. 2. Palpation: the groin region is palpated from the pubis symphysis laterally to the anterior superior iliac spine. The patient should be relaxed with the hip abducted and the knee flexed

internal oblique muscle, can be an overlooked source of groin pain in the athlete [4]. Differential diagnosis must be made from other rare causes of groin pain originating from urological and orthopaedic diseases of the spine, the pelvis, and the hip, such as tenoperiostitis, hip-joint arthritis, osteochondritis, and infective pubis osteitis.

Investigations

Investigations have a significant role to play in the management of groin pain. As with all investigations, they should be used to confirm a diagnosis suspected as a result of clinical examination. X-ray of the pelvis may reveal characteristic changes of the osteitis pubis, hip joint pathology (e.g., osteoarthritis), or stress fracture of the neck of the femur or pubic ramus. Sclerosis or osteophyte formation around the sacroiliac joint (SIJ) indicates that this joint may be a cause of referred pain although frequently, x-ray does not reveal SIJ abnormalities.

Radioisotopic bone scan shows a characteristic pattern of increased uptake in osteitis pubis and may confirm a suspected stress fracture in those cases where x-ray fails to demonstrate the fracture. Ultrasonography is useful in diagnosis of insertional tendinopathies of adductors muscles or lower abdominal muscles or in groin pain secondary to acute muscles strains, but its accuracy is debatable and it is operator dependent. Magnetic resonance imaging (MRI) is useful to study soft tissues and tendons and for detecting abnormalities within the muscles or pubic symphysis. MRI findings, such as bone marrow oedema of the symphysis pubis, have been reported in 50–70% of athletes with groin pain [5, 6]. Peritoneography may be an appropriate investigation to confirm the presence of a direct or indirect inguinal hernia or reveal a bulge of the posterior wall of the inguinal canal that may not be detectable clinically. However, in all cases of groin pain, it is important to investigate the cause of pain with a multidisciplinary approach (orthopaedic, surgical, urologic, and radiologic).

Treatment

The treatment of an athletic pubalgia is complex, especially because diagnosis could be difficult and symptoms underestimated. In the initial phase of the pain, reduction and/or modification of sport activity and training sessions is indicated to treat the pain. Ice and nonsteroidal anti-inflammatory drugs (NSAIDs) orally are useful in the acute inflammatory stage, such as a local injection of NSAIDs (mesotherapy) or electrotherapeutic modalities, such as laser and ultrasound.

The balance of the muscles of the upper thigh, particularly the adductor muscles, with those of the lower abdomen is important to prevent pain and recurrence of sintomatology, so a stretching and strengthening program of those muscles should be done, usually with postural exercises (Figs. 3–10). Strengthening exercises progressing from concentric to eccentric for the major muscles groups in the groin, the hips, and the back are recommended, especially for the abdominal muscles. The athlete can begin functional exercises in water. Moreover, the athlete must repeat stretching exercises of abductors muscles before and after training sections. Isokinetic exercises can also be done in the initial period of treatment.

Injections of corticosteroids and other medicaments, are occasionally required when conservative measures have failed or have been only partially successful. Injection should be regarded as only a part of the total management program. Repeated injections into the tendon should be avoided due to the risk of complete tendon rupture.

Surgical treatment is occasionally required for those few patients resistant to conservative treatment for more than 3 months and for high-level athletes. Correction of the anatomical imbalance – as adductor tenotomy in cases of adductor localisation or abdominoplasty (herniorrhaphy) in cases of weakness of abdominal wall – is the goal of each surgical technique for groin pain. *Pelvic floor repair* refers to a broad surgical re-attachment of the inferolateral edge of the rectus abdominis muscle with its fascial investment to the pubis

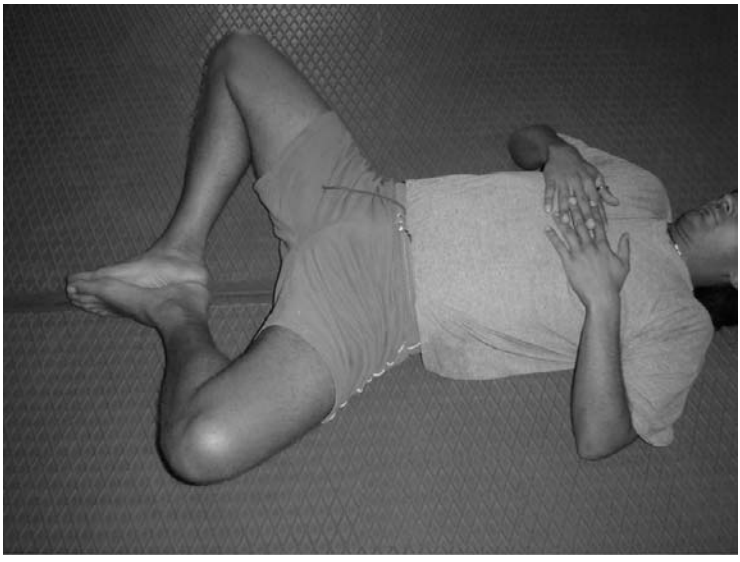

Fig. 3. The athlete relaxes with knees bent and soles of the feet together. This comfortable position stretches the groin. The stretch is held for 30 s

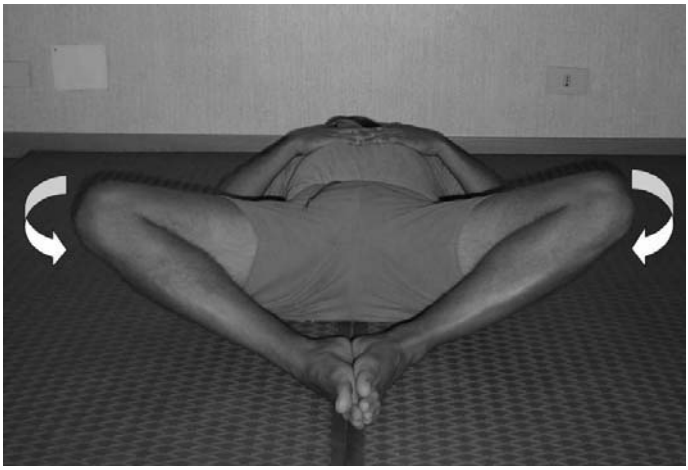

Fig. 4. From the lying groin stretch, the athlete gently rocks the legs as a unit back and forth about 10–12 times. These are easy movements of no more than 1 inch in either direction. Movements are initiated from the top of the hips. This stretch gently limbers up the groin and hips

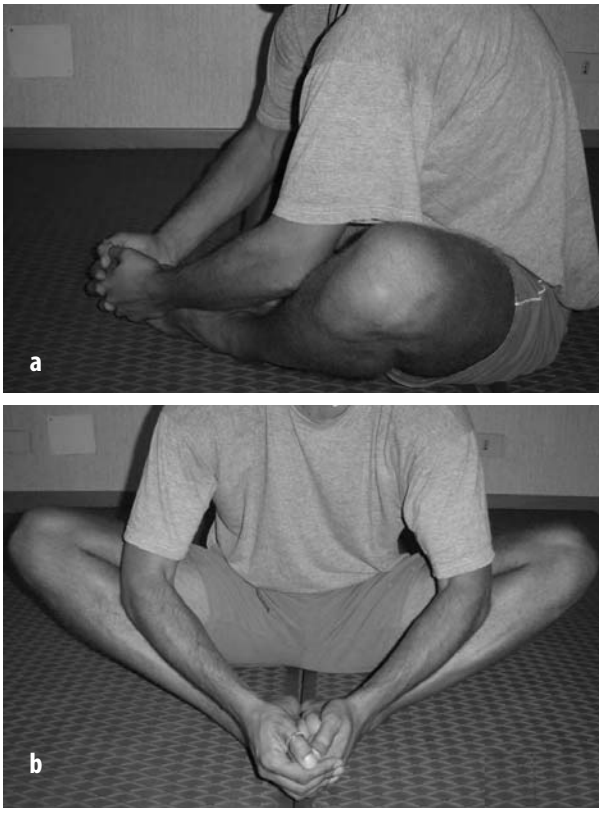

Fig. 5a, b. The athlete puts the soles of the feet together and holds on to the feet. The athlete contracts the abdominals while gently pulling forward, bending at the hips, until a mild stretch is felt in the groin. The stretch is held for 20–40 s

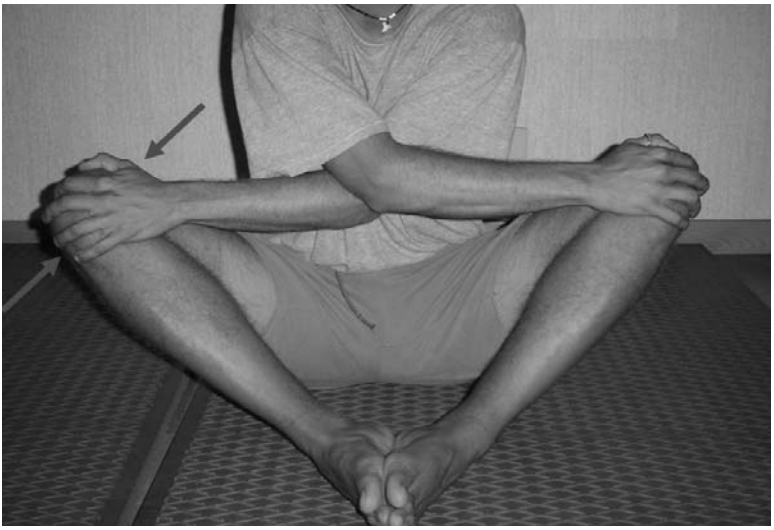

Fig. 6. From the lying groin stretch, the athlete gently rocks the legs as a unit back and forth about 10–12 times. These are easy movements of no more than 1 inch in either direction. Movements are initiated from the top of the hips. This stretch gently limbers up the groin and hips

Fig. 7. The athlete pulls the knee across the body towards the opposite shoulder until an easy stretch is felt on the side of the hip. The stretch is held for 30 s and is done on both sides

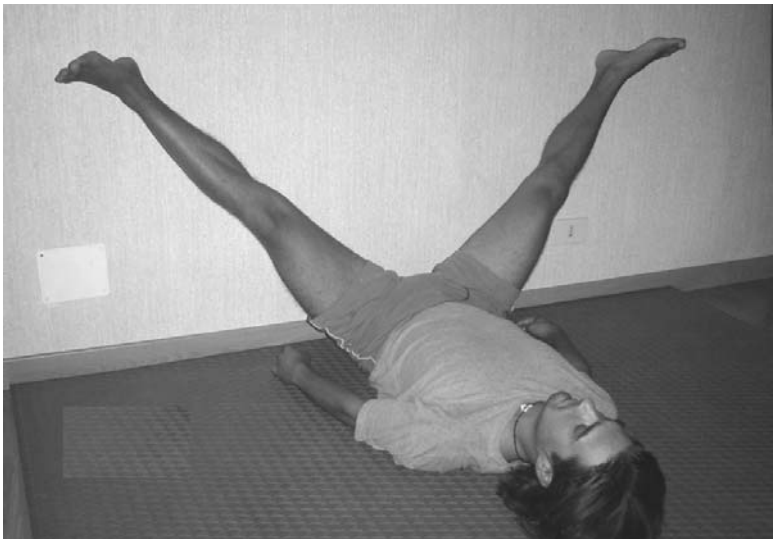

Fig. 8. It is possible to stretch the groin from this legs-elevated position by slowly separating the legs, with the heels resting on the wall, until an easy stretch is felt. The stretch is held for 30 s, then the athlete relaxes

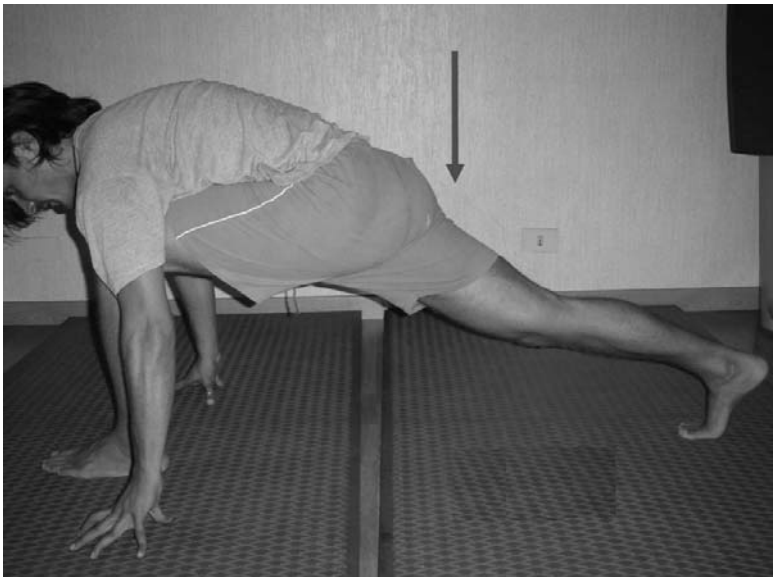

Fig. 9. The athlete places one leg forwards until the knee of the forward leg is directly over the ankle. The other knee should be resting on the floor. Without changing the position of the knee on the floor or the forward foot, the athlete lowers the front of the hip downwards to create an easy stretch. This stretch should be felt in front of the hip and possibly in the hamstrings and groin. The stretch is held for 30 s

Fig. 10. The athlete places the ball of the foot up on a secure support of some kind. The down leg is pointed straight ahead. The athlete bends the knee of the up leg while moving the hips forwards. This movement should stretch the groin, hamstrings, and front of the hip. This stretch is held for 30 s. Both legs are stretched

and adjacent anterior ligaments. This operation is similar but not identical to a Bassini hernia repair. Pelvic floor repair focuses on attachment of the rectus abdominis muscle fascia to the pubis rather than protection of the inguinal floor near the internal ring. Therefore, the principal difference between this surgery and a Bassini repair is in the orientation of the sutures. The internal ring is usually left intact. "Adductor release" refers to complete division of all the anterior epimysial fibres of the adductor longus muscle about 2–3 cm from the pubic insertion, leaving the muscle belly intact. Multiple longitudinal incisions into the tendinous insertion site on the pubis and bone drilling in the pubis are also made, similar to surgery for *tennis elbow* or *jumper's knee*. Some recent studies have shown a high incidence of occult hernia and especially sports hernia in athletes with undiagnosed groin pain, and the herniorrhaphy has had reported success with either a conventional or laparoscopic approach. Frequently, the abdominal wall is reinforced with mesh during these repairs. Moreover, the use of a diagnostic endoscopy could be justified in undiagnosed groin pain in athletes because with this

procedure, abnormalities can be diagnosed and treated. Postoperatively, athletes can return to sports within 6–12 weeks after specific rehabilitation targeted at abdominal strengthening, adductor muscle flexibility, and a graduated return to activity. Laparoscopic pre-peritoneal hernia repair should be considered as a treatment modality in athletes presenting with chronic groin pain and allows good results at long-term follow-up [7–9].

The Authors' orientation in surgical treatment of groin pain in based on the site of pain and clinical evaluation. In cases with abdominal and adductors pain, we prefer an association of abdominoplasty of Bassini and an adductor tenotomy with scarification of tendons at insertion site and bone drilling. In cases with only adductors pain, adductor tenotomy should be enough. In this case, the return to sport activities is quicker – within 2–3 months. However, surgical treatment ensures a resolution or significative improvement in pain in no more than 70% of athletes and after a prolonged period of post-operative rehabilitation. Therefore, surgery should be performed cautiously in top-level athletes [3].

Conclusion

In conclusion, athletic pubalgia remains a vague diagnosis, with a long duration of symptoms of various origins and pathogenesis and is a therapeutic challenge. Further studies are required for a better assessment of incidence, natural course, optimal clinical and imaging evaluation, and selection of the most effective forms of treatment [10, 11].

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Meniscal Lesions

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Introduction

The menisci are of fundamental importance in knee biomechanics. They are crescent-shaped, fibrocartilage discs formed almost entirely (90%) of type 1 collagen. They are located between the tibia and the femur and serve to increase the congruity of the joint between the tibial plateau and the femoral condyles. The medial meniscus ensures a contact surface of about 50% whereas the lateral meniscus ensures more than 75%. The menisci are thus engaged in the absorption and distribution of loads. They stabilise the knee and are also involved in the proprioception mechanisms.

Peripheral vascularisation of the meniscal surface is provided by arteries that penetrate from the capsule. This means that only 10–30% and 10–25% of the medial and lateral menisci, respectively, are vascularised. A meniscus is divided into three areas from its outer surface to its free edge: red–red (0–3 mm); red–white (3–5 mm); white–white (>5 mm) [1–3]. The lateral meniscus moves forwards and backwards, respectively, during extension and flexion of the knee [4]. It is more mobile than the medial meniscus.

Epidemiology and Risk Factors

The literature has little to offer with regards to the epidemiology of meniscus injuries in general and those occurring during soccer in particular. A Danish hospital study [5] has shown a frequency of 0.7 per 1,000 inhabitants per year, with a higher percentage among non-athletic subjects. The medial meniscus is mainly involved although the lateral is more frequently injured (as a percentage) in the event of a concomitant anterior cruciate ligament (ACL) lesion. A 41% incidence of meniscus lesions has been reported in subjects with an ACL injury [6]: medial 30%; lateral 21%; both menisci 7%.

There are obviously differences between one sport and another. A 13-year survey of the Italian alpine skiing team (357 skiers, 567 injuries, including 242

knee injuries) showed that meniscus lesions accounted for 41% – 15% isolated, 26% with a ligament injury, medial 6%, lateral 7%, 2% both menisci [7, 8]. Another study of skiers [9], however, found only a 23% incidence of meniscus injuries (13% lateral, 10% medial) associated with acute ACL lesions. The Author of that study also considered that the risk of such injuries was less in skiing than in other sports. A 5-season personal survey of an Italian first-division volleyball team (32 players, 24 injuries) showed that meniscus lesions accounted for 21% of the total (17% medial, 4% lateral), including 17% isolated [10], and an 8-season personal survey of an Italian first-division basketball team (76 players, 31 injuries) showed that meniscus lesions (all medial) represented 13% of the total (6.4% isolated) [10].

Turning to soccer, Volpi's whole-season study of all the Italian premiership teams (670 players, 235 injuries, including 49 knee injuries) revealed that meniscus lesions constituted 4.5% of the total and 61% of knee injuries [11]. In a 5-season study of a single premiership team (335 injuries, including 58 knee injuries), meniscus lesions represented 7% of the total, with a 5% prevalence of the medials [11].

Meniscus injuries are classified in relation to their associated lesions (they are frequently the outcome of anterior knee instability), the lapse of time since their occurrence (acute or chronic), their site (mostly the posteromedial third), the area involved (red–red, red–white, or white–white), their extent (a bucket-handle tear occupies nearly the whole of the edge), and their type (longitudinal, radial, horizontal, flap, or complex). Meniscus injuries have increased in number in recent years, especially in association with cruciate lesions that were not diagnosed in the past. The causes of this increase lie in the growth of the active sport population, the advent of new sports (including five-a-side football), and the fact that women have started to take up soccer. Another facet of this constant increase is that the earlier commencement of competitive sport is leading to the occurrence of meniscus injuries in younger age groups.

Risk factors, too, are represented by the greater number of professional soccer matches, a decrease in the training/match ratio, and the use of footwear that ensures a firmer ground grip but augments the risk of knee sprains since a decreased impact on the ankles discharges the forces to the knees. Soccer has also become more aggressive due to greater competitiveness, with training sessions that are more exacting in terms of quality and quantity coupled with an increase in physical and tactical training and of the muscle mass. It has been calculated that the quadriceps and hamstring concentric peak torque in professional footballers has increased by 45% [12]. This increase in the muscle mass, however, is not accompanied by a corresponding increase in the capacity of the menisci, ligaments, and tendons, which thus continue to constitute the weak joint chain.

Injury Mechanisms

Tackling is the origin of most meniscus injuries. Lesions of the capsuloligamentous structures capable of involving all the joint formations may occur during a knee sprain. The most frequent mechanisms are a trauma inflicted on the lateral compartment with the foot firm on the ground, or contraction of the quadriceps with valgus deviation of the knee accompanied by external rotation of the leg. They result in a violent outwards force that may damage the medial collateral ligament, then the ACL, and hence the medial meniscus.

Traumas inflicted on the medial compartment with varus stress or contraction of the quadriceps with varus deviation of the knee accompanied by internal rotation of the leg are rare owing to the protection afforded by the contralateral limb. A "leg against leg" trauma may result in a meniscus tear associated with a posterior cruciate injury.

Several mechanisms give rise to "non-collision" meniscus injuries: torsion pivot, landing after a leap (after a header), sudden changes of direction, and sudden spurts or stops. Hyperextension of the leg as the result of a "missed kick" and sudden contraction of the quadriceps with the knee either flexed or extended are other causes of knee joint injuries. A trauma with an extended knee typically produces a tear of the anterior horn of the lateral meniscus.

Diagnosis

A thorough assessment of the patient's prior history, age, occupation, body mass index, and type of sport is required as a prelude to determining how the injury was caused since this will give a more precise idea of the type of lesion involved. Attention must also be directed to the features and sources of pain as well as other symptoms, such as effusion, locking of the joint, and feelings of instability. Objective evaluation of the morphology of the knee and muscle tone and trophism is necessary, along with tests to assess the joint structures in general and those specific for the menisci: palpation of the articular hemirimae, Apley test, Steinmann test, and McMurray "click" test. The varus morphology typical of footballers will obviously be more prone to a medial meniscus injury and a valgus morphology to a lateral meniscus injury. Clinical evaluation must be accompanied by instrumental examinations: standard anteroposterior and lateral knee radiograms, total lower-limb radiograms under load, Rosenberg projection radiograms (for older subjects) and magnetic resonance imaging (MRI).

Treatment, however, must be determined in light of the clinical picture. MRI is an almost essential aid to diagnosis. In expert hands, its sensitivity, specificity, and accuracy exceed 90%. Our patients, however, differ in level

and come from different areas where examinations are sometimes performed prior to a super-specialist clinical evaluation. In a personal arthroscopic [13] surgery series, in fact, we found that only 53% of medial meniscus and 25% of the lateral meniscus lesions (all isolated) of 137 patients had been correctly diagnosed by MRI in 58 centres. This goes to show that MRI should be conducted in highly specialised areas and cannot be relied upon for the choice of treatment.

Types of Treatment

Arthroscopy is essential since it both ensures a correct diagnosis and allows immediate treatment in the form of either suture or meniscectomy. Current thinking favours the maximum possible conservation of a meniscus and hence the maximum residual function so as to avoid the deleterious degeneration of a joint deprived of two structures that are fundamental for its normal kinematics. Even so, excision should be envisaged when the white–white area is damaged or in the event of degeneration in other areas rendering reconstruction impracticable. Meniscectomy must naturally be as selective as possible. Unstable portions, however, must always be removed since they could be the cause of a recurrence.

Suture techniques (all inside, out-in and in-out) have been improved in recent years. Certain prior conditions must none the less be satisfied: good knee stability, an interval of preferably not more than 3 months since the injury, and a lesion in a well-vascularised area [14]. Patients should be allowed to opt for one form of treatment rather than the other after being duly informed about the risk of complications and the 20–30% suture failure rate, with the subsequent need for meniscectomy [15].

Saving a young player's meniscus is obviously desirable. In the case of an élite professional footballer who may be around 30, however, the situation is very different for the simple reason that 5–6 months are required for rehabilitation and the resumption of sport after a suture compared with a mere 2–3 weeks after a meniscectomy. It is thus very unlikely that a professional will choose a treatment that entails long rehabilitation and the loss of a season and also carries the risk that a second operation may be required. Account must equally be taken of the point during the season at which the injury occurred since this will determine the amount of time that can be devoted to rehabilitation.

Sutures are more widely used when a ligament reconstruction is also required because the rehabilitation times are the same and the success rate is improved by bleeding within the joint. When dealing with élite players, therefore, we prefer to excise isolated lesions. Repair of a meniscus is confined to

cases where this is chosen by the patient, when the patient can stop playing for a long time, or when a ligament reconstruction is also required since the rehabilitation protocol and hence the period before sport can be resumed are the same for both interventions.

Rehabilitation

Selective meniscectomy is immediately followed by the application of ice and the administration of anti-inflammatory drugs. Active movements of the knee and isometric exercises of the quadriceps and hamstring must also be started immediately to secure rapid recovery of range of motion (ROM) and joint function. Unaided walking at a normal pace with the leg fully extended should begin on the first morning after the operation. Open- and closed-chain exercises are introduced later. As already mentioned, resumption of activities is achieved after 2–3 weeks: running after about 10 days and sport after 2–3 weeks, depending on the patient's characteristics and degree of rehabilitation. Generally speaking, rehabilitation is much the same after a medial and a lateral lesion even though the latter is certainly more disabling in biomechanical terms.

The aim of management after suturing is to protect the tissues during their repair. This is done with progressive and protective joint arches. Full ROM is only reached after 3 weeks. Protection is also provided with a brace. Progressive partial and total weight bearing is achieved in accordance with the rehabilitation programme followed after an ACL reconstruction.

Prevention

Prevention of meniscus injuries in soccer must identify the specific risk factors, quality of warming up, training/match ratio, quality of pitches, and footwear. Account must also be taken of the ability of a professional as opposed to a non-professional player to "manage" the increasingly sophisticated footwear currently employed.

Prevention of meniscus injuries must certainly start with the prevention of sprains in general. The employment of braces for the purpose of prevention is allowed by the regulations but has not proved beneficial [16] whereas specific training programmes can significantly reduce injuries [17]. The proprioceptive programme [17] was found to reduce the number of injuries from 1.15 to 0.15 per team in a comparative study of 40 soccer teams. Soderman et al. [18], however, found that no benefit was derived from the use or non-use of a proprioceptive programme on the part of 215 female footballers. They also emphasised the fact that balance board training cannot prevent knee

sprains. Jung et al. [19] maintain that training of neuromuscular and proprioceptive performance, as well as improvement of jumping and landing techniques, seems to decrease the incidence of ACL injuries in female athletes. They also stress that more methodologically well-designed studies are required to evaluate the effects of specific preventive interventions.

Despite this absence of a clear scientific standpoint, however, we feel that no harm can be caused by the inclusion of a programme of concentric and eccentric strengthening exercises to re-establish the force of the flexors, along with a programme of "Nordic hamstring" progressive eccentric exercises. Consideration, in fact, should be given to the fact that the muscle protective reaction time is longer than that of the injury mechanism, which means that any attempt on the part of the athlete to obtain muscle recovery from a sprain injury would have little preventive effect [20].

Poor pitch surfaces (defective grass cover, frozen ground in winter) are a factor that must be handled to secure greater prevention, especially in the minor leagues. It is still uncertain whether the eventual use of synthetic pitches will alter the epidemiology of meniscus lesions. In the case of professional players, the risk of injury could be lowered by reducing the number of matches and altering some of the rules to place a tighter rein on tackling.

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Anterior Cruciate Ligament Injuries

Paolo Aglietti, Francesco Giron, Pierluigi Cuomo

Introduction

An anterior cruciate ligament (ACL) injury represents one of the most dramatic events in the career of a soccer player. This ligament acts as a restrain to the anterior tibial translation during twisting, jumping, and cutting manoeuvres. In case of a tear, it cannot heal, and surgical reconstruction is mandatory to recover the full function of the knee and allow the player to go back to the game. Unfortunately, we still do not know if restoration of adequate function of the knee changes the long-term destiny of this injury. Nevertheless, the increased number of people of both genders and all ages involved in soccer activities around the world, changes in rules with a progressive increase in the speed of the game, and the great diffusion of this sport activity, mainly in Europe, has turned the ACL in the most extensively studied ligament of the body. In this chapter, we try to concentrate on what we currently know about ACL injuries in terms of incidence, risk factors, natural history, clinical evaluation, surgical treatment, and outcome results.

Incidence

Up to one fifth of football injuries involve the knee joint [1]. In most cases, sprains or overuse injuries are encountered; however, major injuries such as ACL tears are not infrequent, and they seriously affect a player's career, with both short- and long-term consequences. Bjordal et al. [2] retrospectively reviewed ACL injuries that occurred in their local community in Norway over a period of 10 years to 176 players involved in organised football. An overall incidence of 0.063 injuries per 1,000 game hours was recorded. Women had a significantly higher incidence (0.1 injuries per 1,000 game hours) than men (0.05). Overall, ACL injury incidence during training was significantly lower (0.013) than during games (0.063). Giza et al. [3] in professional female foot-

ball players observed as well that the risk of ACL injury is doubled in games with respect to training. Gwinn et al. [4] calculated the incidence of ACL injury in all US Naval Academy midshipmen over 6 years both at a professional and amateur level. In intercollegiate football, the male injury rate was 0.08 per 1,000 athletic exposures and was 9 times inferior to women's risk. When intramural coed soccer was examined, male injury risk was higher (0.3 injuries per 1,000 exposures), and the female/male ratio inferior (6x), thus suggesting that a specific training at a higher level protects the knee joint. Arendt and Dick [1] examined a larger sample consisting of 739 National Collegiate Athletic Association (NCAA) football teams over a 5-year period (1989–1993). ACL injury incidence in men was 0.13 and was significantly lower than in women (0.31). The same group of researchers [5] more recently extended their investigation on the same sample teams to year 2002. A 13-year time interval was therefore examined. In the final year of the study period, ACL injury incidence in men was significantly lower (0.07 injuries per 1,000 h exposure) than in the first year (0.12). This trend was not observed in women, which in 2002 still showed an incidence of 0.28 injuries per 1,000 h of exposure.

Mechanism of Injury

Most ACL lesions in football are caused by non-contact injuries. Agel et al. [5] in a 13-year study period found the incidence of non-contact injuries to vary from 28% to 85% in men and from 42% to 70% in women, with an average of 58% and 50% non-contact injuries for women and men, respectively. This difference was statistically significant. Other investigators [2] in a smaller sample found non-contact injuries to be 54% and 42% in men and women, respectively. Non-contact injuries are usually the result of a deceleration such as the one that occurs when a player wants to change direction. In this situation, increased quadriceps contraction induces an anterior force, and if the tibia is externally rotated and a valgus moment is present, the ACL is particularly at risk. A concomitant meniscal or medial collateral ligament lesion is likely to occur in such conditions.

An exclusively non-contact soccer injury mechanism is knee hyper-extension resulting from missing the ball while kicking. Contact injuries, on the other hand, are mainly the result of tackling. Bjordal et al. [2] in their series found that 58% and 42% of ACL injuries were due to tackling in women and men, respectively. In men, most tackles (64%) were from the side. In women, ACL injuries were due both to side (44%) and front (32%) tackles.

Risk Factors

The clear identification of ACL injury risk factors in football players is attractive in the attempt to set up prevention strategies. At the same time, this task is quite challenging, as large series would be necessary to be prospectively examined, and the incidence of ACL injuries in football is quite limited [6]. Risk factors can be easily distinguished into modifiable and non-modifiable. The main non-modifiable ACL injury risk factor is gender. As previously discussed, female football players are at higher risk of experiencing an ACL injury during their career. A more narrow intercondylar notch, hormonal influences, and inadequate neuro-muscular control have been considered responsible of the higher propensity of female players to injuries [7]. The exact role and mechanism of hormones on the ACL has not been clarified, and controversial results have been presented. On the other hand, in recent years, great attention has been dedicated to understanding the neuro-muscular control differences between men and women with the attempt to set up interventional strategies to prevent non-contact injuries. Women athletes have been found to have different landing and cutting patterns from men. In particular, women have the tendency to land with the knee more extended than do men, and in cutting manoeuvres, the knee is in a more extended, externally rotated, and valgus position than in men [8]. The recognition of these patterns should help to prevent injuries by instructing athletes and correcting their movement patterns.

Among non-modifiable risk factors is the importance of previous injuries, which has been emphasised by some. Arnason et al. [9] analysed injury data of 306 football players over one season in Iceland and recorded 5 ACL injuries with an overall incidence of 0.15 per 1,000 h of exposure. Though with multivariate analysis no clear risk factor could be isolated due to the paucity of ACL lesions, players who sustained knee sprains had a significantly positive history of previous injuries. Chomiak et al. [10] followed 398 football players from the Czech Republic for 1 year and found that previous injuries or a pre-existing knee instability are predisposing factors to new knee injuries. In their series they also found that less-experienced soccer players had a higher incidence of ACL injuries.

Among the modifiable risk factors, the only significant correlation has been identified with shoe design. Lambson et al. [11] compared 4 types of football shoes and evaluated the incidence of ACL tears among 3,119 high school football players during the 1989–1991 competitive seasons. They found that longer, irregular peripheric cleats produced significantly higher torsional resistance than the other designs with flatter cleats and were associated with a significantly higher ACL injury rate.

Clinical Evaluation

A good diagnosis starts by obtaining a good history. ACL injury in most situations is a major traumatic event with a characteristic history. The athlete usually describes the knee as "coming apart", and in most cases, the injury type is non-contact. An audible "pop" is heard or felt in about 50% of cases. An effusion secondary to bleeding occurs within 6–12 h after the injury; full weight bearing and full extension of the knee are usually difficult [12]. In acute injuries, physical examination could be difficult due to knee pain. The examiner should avoid performing potentially painful and unnecessary components of full knee examination and should concentrate only on ligament examination. A comfortable and relaxed patient is essential. We suggest evaluating the uninjured knee first to gain patient confidence and obtain a baseline for comparison. The Lachman test is considered the most reliable and reproducible investigation method to confirm an ACL injury. In an acute setting and when performed correctly, this test has a sensitivity of 87–98% for detecting an ACL tear [13]. An increased anterior tibial translation and a soft or absent end point compared with the opposite healthy knee can confirm the ACL injury. Another important test is the pivot-shift test. However, creating this can be difficult in an acute setting secondary to patient guarding. This test can provide additional confirmation of ACL injury, however.

After the ACL has been evaluated, the collateral ligaments, the posterior cruciate ligament, and the menisci need to be assessed for injury. In some cases, the status of the ACL may remain in question at the conclusion of a detailed history and examination. In these cases, it is helpful to reduce pain and swelling and to re-evaluate the knee several days later. Imaging should include standard weight-bearing anteroposterior (AP) view, a lateral view, and a patellofemoral Merchant view. Most often, plain x-rays are normal; however, an avulsion or an osteochondral fracture may be seen. A Segond fracture may be seen on AP view and is considered highly predictive of an ACL injury. Magnetic resonance imaging (MRI) usually is not needed to evaluate an acute knee injury because of the accuracy of the history and physical examination. More important than confirming an ACL injury is the additional information that can be obtained with regard to the menisci, the subchondral bone, and the other ligamentous structures. In chronic ACL-deficient knees, an MRI can detect associated meniscal injuries or a new bone bruise from a recent giving way.

Natural History

Several studies have attempted to define the disability, associated pathologies, and degenerative changes of the knee after an isolated untreated lesion of the

ACL. However, this is a controversial issue because most studies do not reflect the true natural history of the ACL-deficient knee due to the fact that only symptomatic knees have been observed and followed up. Moreover, many studies have short or incomplete follow-ups, without objective documentation, or flawed by inconsistent and varying treatment between cases. Nevertheless, even in studies where the documentation is incomplete, important information can be obtained regarding functional impairment, incidence of associated injuries, and effectiveness of rehabilitation regimens.

A recent review of the literature [14] considering only studies with more than 5-years follow-up, revealled that radiographic degenerative arthritis significantly increases after all knee injuries compared with the uninjured joint of the same patient. Partial or total ruptures of the ACL without major concomitant injuries seem to increase the risk 10-fold (15–20% incidence of degenerative arthritis) compared with an age-matched, un-injured population. Thus, an ACL injury combined with a significant meniscal tear or other knee ligament injuries results in knee arthritis in most patients. Ten to 20 years after ACL injury, knee arthritis often presents a slight joint-space narrowing and usually is not associated with major clinical symptoms. The progression of the arthritis is slow, and in some cases, the condition seems to remain stable. Time is an important determinant for the degree of arthritis, and other significant symptoms requiring treatment may be delayed and only encountered more than 30 years from the initial trauma. However, it must be noted that in the case of ACL injury, most patients are not able to return to the un-restricted pre-injury level of function and complain of pain, swelling, and giving-way symptoms during sports activities [15–17].

In "cutting-sports performers", such as soccer players, the persistence of sports activity at the same intensity level is a high risk factor for additional ipsilateral knee lesions, most commonly meniscal or cartilage tears, with a dramatic increase in the incidence of early degenerative arthritis [18]. Von Porat el al. [19], evaluating 154 soccer athletes 14 years after an anterior cruciate injury, found that only 8% were still participating in organised soccer; 80% reported reduced activity level, and of these, the majority (69%) reported the knee injury as the cause. No difference in radiographic outcome was determined between those treated within or without surgery. Radiographic changes were found in 78% of the injured knees, and of these, radiographic osteoarthritis equivalent to Kellgren and Lawrence's grade 2 was seen in 41%.

Male soccer players have an increased risk of knee osteoarthritis [18– 21]. Injuries to the menisci and the cruciate ligaments are believed to be the main reason for this, but when injuries are excluded, a relation between elite soccer and osteoarthritis persists [21]. This means that a player who returns to soccer after an ACL injury has an even higher risk of osteoarthritis both because of soccer itself and because of the injury.

Treatment of the Injury

A unified treatment approach to ACL injuries is yet to be defined. Young and motivated athletes most often desire to recover pre-injury sport activity and are unable to do so unless surgical reconstruction allows them to achieve reasonable knee stability. Older and recreational athletes often prefer to decrease their activity level, thus avoiding giving-way episodes, and to postpone surgical reconstruction, which will keep them away from work for a protracted time.

Unfortunately, whether a conservative or a surgical treatment is used, the natural history of an ACL-deficient knee seems to show that occasional episodes of giving way are still present in all patients and that it is not possible to prevent early joint degeneration in about one half of those knees. Thus, at the present time, counselling patients as to the most appropriate treatment can only be based on factors that are known to place the knee at risk for further injury and relating those factors to the individual patient.

Many patients who tear the ACL will elect not to undergo reconstruction. For the skeletally immature athlete, this decision is only a temporary one. The adult, however, may have few limitations during sport or daily living activities and may feel no need to undergo surgical treatment. The main key for the appropriate treatment is the patient's desire and motivation to recover soccer activity at the same level as prior to the knee injury.

Regardless of the situation, the initial management of an acute ACL tear involves decreasing the patient's pain and swelling, followed by restoring full range of motion with rest, ice, isometric, and extension exercises. Strengthening exercises, with closed kinetic chain rehabilitation programs, begin once extension is restored and flexion exceeds 110°. In the last phase of the rehabilitation program, the patient is progressively introduced to activities that are believed to be safe based on the patient's confidence and knee stability. Returning to high-risk activities usually is not possible without sustaining additional giving-way episodes.

Surgical treatment is reserved for those players who want to continue playing soccer. The goals of ACL reconstruction are to re-establish a normal knee with full range of motion, good stability, and strength and to prevent additional meniscal and/or chondral damage. To achieve those goals, a great variety of surgical techniques have been developed throughout the last 40 years. The most popular is single-bundle intra-articular ACL reconstruction with tibial and femoral bony tunnels. This technique aims to recreate one of the two major fibre bundles of the ACL: the anteromedial (AM) bundle. This is the most isometric bundle of the native ACL and shows quite constant tension behaviour throughout the range of motion.

Several different type of grafts have also been employed for ACL recon-

struction, and actually, the most accepted ones are autografts – including the central third of the patellar tendon, the quadriceps tendon, and the hamstring tendons – and fresh-frozen allografts, represented by the central third of the patellar tendon and the tibialis anterior tendon. Based on the allograft employed, a wide variety of fixation methods have been introduced to achieve rigid fixation of the graft to the bone. However, apart from the technique employed, the main principles for a successful ACL reconstruction are a graft showing biomechanical properties similar to those of the native ACL, a precise placement of the bony tunnels, and a strong and rigid fixation of the graft in the correct tension.

Despite the high rate of satisfactory results achieved with the single-bundle technique, and though successful in reducing the anterior drawer, recently, several investigators [22–26] have complained about the inability of this type of reconstruction to completely restore the rotational laxity and avoid the pivot-shift phenomenon. Single-bundle reconstruction has been developed in an attempt to replicate the function of the more isometric AM bundle. In this reconstruction technique, the femoral tunnel is close to the roof of the femoral inter-condylar notch. Consequently, it is also close to the axis of axial rotation of the tibia; thus, the graft is insufficient to resist externally applied rotatory loads. Recent laboratory studies using different investigation devices [22–26] demonstrated that the single-bundle reconstruction is certainly effective in restoring tibiofemoral anterior laxity in response to a tibial anterior translation force, but it is not so effective in reducing the coupled anterior tibial translation and rotation resulting from a combined tibial valgus and internal rotation torque. Based on these findings, it has been suggested that both the AM and the posterolateral (PL) bundles should be reconstructed. The PL bundle resists the majority of the anterior draw force in the native ACL [27] and in ACL reconstructions [28] when the knee is almost fully extended.

Laboratory studies comparing single-bundle to double-bundle ACL reconstruction have reported a significant superiority of the double-bundle reconstruction in restoring normal anterior tibial translation and avoiding the pivot-shift phenomenon [28–30]. Unfortunately, to date in the literature, all clinical studies [31–33] have failed to demonstrate a clear improvement in terms of anterior knee stability, incidence of residual pivot-shift, and postoperative proprioceptive function. Double-bundle ACL reconstruction is certainly an appealing technique, but further studies are needed to support its routine clinical use.

Postoperative rehabilitation is as important as the surgical technique to achieve a stable knee. An adequate rehabilitation program should emphasise rapid recovery of the full range of motion, fighting with pain and swelling. Usually, the graft needs about 2–3 months to incorporate into the bone, and

the remodelling process allows the graft to sustain vigorous loads only after 6 months from surgery. Strengthening exercises should start when the full range of motion has been recovered and the swelling has disappeared. It is important to avoid exercises that could apply excessive tension on the graft. In the first postoperative month, it is safe to start with isometric exercises; in the second postoperative month, it is possible to introduce close kinetic chain exercises; at the beginning of the third postoperative month, open kinetic chain exercises can be gradually added. For soccer players, return to sport-specific training should not be allowed until up to the fourth or fifth month postoperatively while return to competition should be delayed to the sixth or eighth month postoperatively based on the grade of knee stability and muscular strength recovered.

Outcome Results

A vast number of studies reporting outcome results of ACL reconstruction have been published in the literature. While after an ACL injury the return rate to cutting sports is low with conservative treatment [16, 17], the mid- to long-term results of operative treatment with the central third of the patellar tendon and hamstring tendon autografts are good with regard to stability improvement and the ability to return to demanding knee activities [34–36]. Most reports on ACL reconstructions, however, deal with heterogeneous populations, with varying demands on knee function and different levels of sport performance. Soccer players represent a homogeneous group with similar and high demands on knee function, and only few papers [37, 38] report outcome results of ACL reconstruction in this sport category.

In a questionnaire survey of ACL injuries reported to Swedish insurance companies, Roos et al. [38] found that only 26% of ACL-reconstructed players were still active in soccer after 7 years, and none of the élite players were active at the same level. Interestingly, there was no difference in the return rate to soccer after either surgical or conservative treatment. The majority of players give up the sport for social reasons, and this is very likely 7 years after an injury. More recently, Bak et al. [37], evaluating 132 soccer players who underwent primary ACL reconstruction with an iliotibial band autograft, found that the majority of players (68%) were still active at a median of 4 years after reconstruction, and only 11% of those who were not active at the follow-up claimed that knee problems were the cause. Moreover, evaluating gender differences, an unacceptable rerupture rate was seen in female soccer players. Women participating in cutting sports are subjected to a significantly higher risk of sustaining an ACL injury than are men [5] and are therefore probably also at higher risk of graft failure.

Long-term radiographic analysis showed no differences between men and women in terms of osteoarthritis risk. More than 10 years from ACL injury, both genders showed an high prevalence of either radiographic or symptomatic knee osteoarthritis. Lohmander et al. [39], investigating 103 female soccer players 12 years after an ACL injury, reported that 82% had radiographic changes in their index knee, and 51% fulfilled the criteria for radiographic knee osteoarthritis. Seventy-five percent reported having knee-related symptoms that affected their quality of life, and 42% were considered to have symptomatic radiographic knee osteoarthritis. Slightly more than 60% had undergone reconstructive surgery of the ACL. Using multi-variate analyses, surgical reconstruction was found to have no significant influence on knee symptoms. Von Porat et al. [19], in a cohort of 154 male soccer players 14 years after the initial ACL injury, found radiographic changes in 78% of injured knees. No difference on radiographic outcome was determined between those treated with or without surgery. Only 12 (8%) of the 154 participants were still participating in organised soccer.

Conclusion

Soccer is the most popular sport worldwide with around 200 million licensed players according to the Federation of International Football Associations (FIFA). However, this is a high-risk sports activity for the integrity of the ACL. Many factors have been recognized that increase the risk of ACL injury, particularly in women. The only consistent solution to regain knee stability and to return to the sport at the pre-injury activity level is surgical reconstruction. However, reconstruction, though effective in restoring short-term knee function, is not able to prevent long-term joint degeneration. Efforts should be made to obtain a precise definition of the injury mechanism and how to prevent it. Today, prevention represents the only chance to guarantee a soccer player a long career.

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Anteromedial Knee Instability

Maurilio marcacci, Stefano Zaffagnini

Introduction

Anterior cruciate ligament (ACL) lesion is a frequent injury in sport activities, particularly in high-contact sports. ACL functional insufficiency causes an increased anterior tibial translation (straight or primary instability) and an increased internal tibial axial rotation. Single-bundle ACL reconstruction was indicated by the literature and by the largest number of orthopaedic surgeons as the gold standard for acute or chronic ACL lesions, which could be easily diagnosed through clinical examination and magnetic resonance imaging (MRI).

Rotational instability of the knee consequent to an ACL lesion is a stimulating argument both from a biomechanical point of view and from a surgical one. Clinically, rotational instability is referred to as a bi-axial combined instability during cutting movements, mainly during fast changes of directions such as a football player performs in dribbling. Hugston [1] classically distinguished anterior rotational instability in:

- Anteromedial rotational instability (AMRI) due to a lesion of the medial compartment, including posterior oblique ligament (POL) ruptures. AMRI is increased in associated ACL lesions;
- Anterolateral rotational instability (ALRI) caused by a tear of the central third and the lateral capsular ligament. Also, ALRI is empowered by associated ACL lesions.

ACL functional insufficiency has been indicated as the main cause of AMRI, most of all with the knee near to its complete extension; however, several biomechanical studies reported in the current literature have shown that the medial collateral ligament (MCL) plays an important role in anteromedial rotational stabilisation, mainly at 30° of flexion. An unrecognized MCL instability consequent to a grade 3 MCL lesion can be a predisposing factor for ACL reconstruction failure. In fact, chronic MCL deficiency has been noted to increase stress on an ACL graft, particularly during the critical interval of the neoligamentisation [2]. Moreover, other authors have demonstrat-

ed that associated ligamentous functional deficit either not discovered or underestimated in preoperative planning can represent the main causes of ACL reconstruction failure [3].

Anatomical and Functional Considerations

The anatomical pattern of the medial side of the knee has been well investigated by Hughston et al., Warren et al., Warren and Marshall [1, 4, 5]. To better understand the pathophysiology of traumatic mechanisms that lead to rotational instability, it is fundamental to have a good understanding of anatomical structures of the medial side of the knee. According to Warren and Marshall's studies [4, 5], structures of the medial side of the knee are divided into three layers. Each layer is separated from the other by a virtual space, the active muscular component (semi-membranosus, gracilis, sartorius, medial head of the gastrocnemius) contributes to an important and strong stabilisation of the medial side of the knee.

On the first superficial layer, we find the "deep fascia" or "crural fascia", the plane of which is defined by the fascia that covers the sartorius muscle's anterior surface where it is fused with the deeper layers while posteriorly it covers the two heads of the gastrocnemius muscle. Anteriorly, it contributes to form the medial patellar retinaculum. Inferiorly, the gracilis and semi-tendinosus tendons separate this layer from the second layer. The second layer is formed by the superficial portion of the MCL, which is formed by an anterior portion in which the fibres are straight and a more posterior portion in which the fibres are oblique and fused with the third layer. The semi-membranosus tendon and its sheath insert into:

- The distal femoral condyle (fused with the POL);
- The proximal tibia (fused to the superficial portion of the MCL);
- The popliteal fossa, enforcing the posteromedial capsule (PMC) (popliteus oblique ligament).

This complex and multi-directional structure contributes to an active stabilisation of the medial compartment of the knee, and this action is also important for moving the internal meniscus during flexion and extension. The most anterior portion of the second layer is the region with the largest range of individual variability, but it is possible to individuate a defined anatomical pattern. The fibrils anterior to the MCL travel superiorly to the vastus medialis and join the first layer, forming the parapatellar retinacular fibres. From the insertional area of the superficial portion of the MCL, transverse fibres run forwards in the second layer towards the patella, forming the patellofemoral ligament.

The third layer is the deepest one and is formed by the joint capsule. The anterior part is thin and plays no role in knee stabilisation and includes the

fat Hoffa pad and a thin covering on both anterior and intra-articular surfaces of the patella. On the mid portion of the capsule is the deep portion of the MCL. It extends from the femur to the mid portion of the peripheral margin of the meniscus and tibia. Posteriorly, the third layer merges with the second layer, and their combined fibres envelop the posteromedial corner of the joint, forming a composite structure.

Voshell [6] described two portions of the tibial collateral ligament: one anterior, with a parallel arrangement of fibres, and a more posterior, oblique portion. This more oblique group of fibres was described by Hughston et al. [1, 7] as the ligamentum popliteum obliquum, or POL (Figs. 1, 2). This anatomically distinguished structure originates from the adductor tubercle, and distally divides into three separate arms or expansions:

- The tibial arm, inserting closely the articular surface;
- The capsular arm, continuous with the posterior capsule;
- The superficial arm, associated to the semi-membranosus tendon.

The functional concept of the posteromedial corner has been well studied by Hughston et al. [1, 7]. Muller [8] echoed this concept, saying that "the pos-

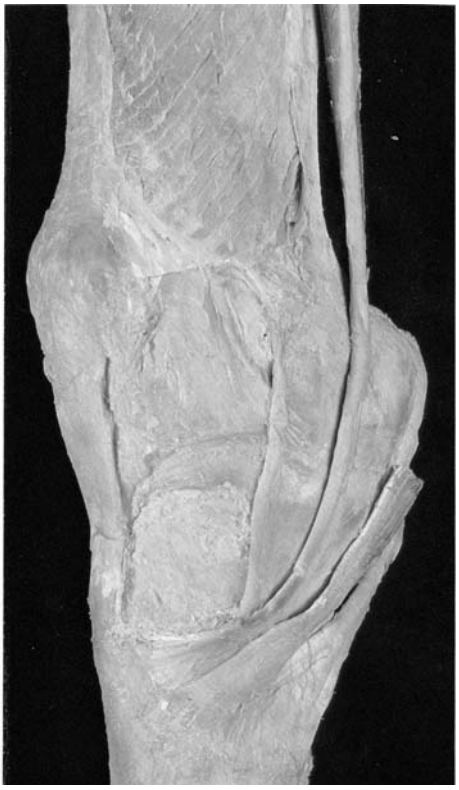

Fig. 1. Anatomical specimen of the medial side of the knee

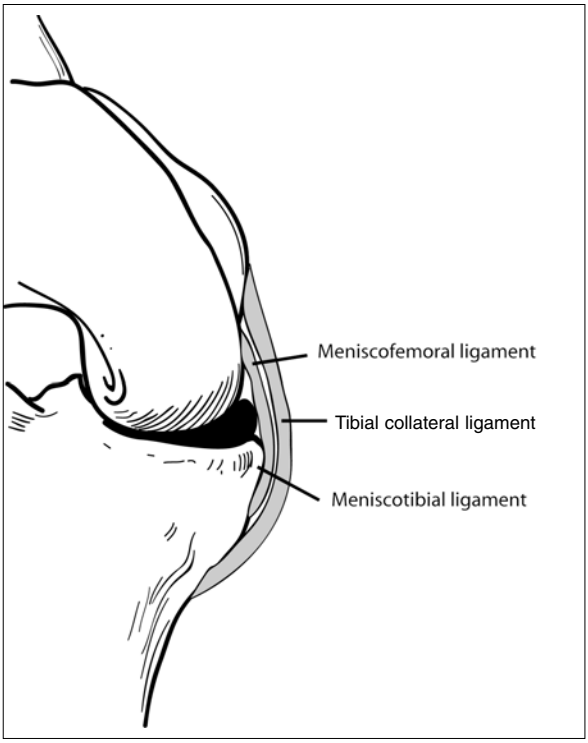

Fig. 2. Model of medial-side structures

teromedial corner is fundamentally different in nature and function from the medial collateral ligament itself". Anatomical structures that contribute to the function of the posteromedial corner are the posterior horn of the internal meniscus, the POL, the semi-membranosus expansion, the meniscotibial ligaments, and the oblique popliteal ligament. The active functional restraint on this region is the semi-membranosus tendon, well described by Muller [8], who called this area "the semi-membranosus corner". The tendon inserts distally by five expansions:

- The reflexed part directly inserting on the tibia;
- The direct posteromedial tibial insertion;
- The oblique popliteal ligament insertion;
- The expansion to the POL;
- The popliteal aponevrosis expansion.

This complex tendon insertion is fundamental for knee medial stabilisation and is also important for the internal meniscus motion pattern during flexion and extension. The meniscocapsular complex has a dynamic co-ordination, which contributes to anteromedial rotational stabilisation during the complete normal range of motion both in static and dynamic conditions.

Quantitative analysis of knee specimens shows that the MCL is structured in three functional units. The first is the superficial medial ligament, the second is the deep medial ligament, and the third is represented by the posterior oblique fibres. Experimental cutting studies, have shown the relative functional importance of the different structures located in the medial side of the knee [4, 5]. During valgus stresses, an important role is played by the superficial medial ligament while the deep medial ligament plays a secondary role. From a clinical point of view, valgus instability demonstrated with the knee flexed up to 45° indicates a lesion of the anterior long fibres. An important and evident instability probably indicates a rupture of the oblique fibres and of the deep portion of the MCL. It is also current opinion that valgus instability with the knee completely extended indicates a significant functional deficit of both cruciate ligaments.

Functional correlations between ACL and MCL have also been well investigated. The term "motion limit" refers to the equilibrium position reached by the tibia in respect to the femur when a force or a moment is externally acting on the lateral compartment of the knee joint. The role of the ACL and MCL in controlling motion limit has been studied by different investigators. These studies have established that the ACL is the primary restraint that determines the maximum limit of anterior tibial translation when an anterior force is applied. The ACL plays a secondary role in the stabilisation of internal-external rotation and of abduction-adduction rotational instability against intra- and extra rotational and abduction-adduction stresses.

It has been well demonstrated that the ACL is the primary stabiliser opposing anterior tibial translation while the MCL in this case plays a secondary role. Different degrees of instability were evaluated at 30° or 90° of flexion in an ACL-deficient knee, and it is possible to find great individual variability in terms of anterior instability in ACL lesions, mostly due to muscle stabilisation differences. At 30° of flexion, the MCL is the primary stabilising structure against abduction and external rotational stresses while the ACL plays a secondary role.

When POL and PMC are involved in a combined injury, we find increased anterior tibial translation, increased external rotational instability, and positive valgus stress test. These structures, the posterior horn of the internal meniscus, and the semi-membranosus arm play an important role in controlling AMRI because they represent the most important anatomical structure of the posteromedial joint portion [4, 5]. To detect a possible lesion of these structures and to choose the best kind of treatment, it is important to perform a clinical test at 30° or 90° of flexion to show pathological anteroposterior (AP) tibial translation and at 0° or 30° of flexion to show increased abduction and rotational instability. A rotational instability combined with abduction stress can justify some functional instabilities in patients clinical-

ly characterised by a minimal joint looseness. Straight fibres of the MCL are tighter when the knee is nearly fully extended (20–30° of flexion) while the PMC and oblique ligament are tighter at higher flexion angles.

Nielsen et al. [9] have shown the direct connection between concomitant MCL and PMC disruption and clinically important valgus and anteromedial rotational instability. PMC lesions alone are not true clinical evidence [10]. Reporting clinical examinations and intra-operative findings in 68 knees, Hughston [7] concluded that anteromedial instability is based on a tear of the medial compartment ligaments, including the POL. To oppose external torque, valgus moment, and anterior tibial translation, the MCL works in co-operation with the ACL if high forces are applied, and this effect is mostly evident in the flexed knee. After an ACL reconstruction, a patient with a residual valgus laxity due to a previous MCL injury can have an increased risk of a second lesion, caused by excessive stresses concentrated on the neo-ACL [2].

It is easy to understand that complex lesions combining passive restraints, such as the ligamentous structures involved, and dynamic restraints, such as muscular insertion of the semi-membranosus and its expansion on the medial capsule, are difficult to evaluate. Even more difficult, however, is to select the correct treatment options according to the degree of instability, which is different in every patient. In fact, without an intra-operative tool capable of accurately assessing the severity of the pathological laxity, it is extremely difficult to decide whether to address anteromedial knee instability conservatively or surgically.

Classification and Pathogenic Mechanism

Knee ligaments and capsular lesions are very common in sportspeople, but considering the high number of people involved in common sport activities, they are still a low percentage. The knee is a heavily beard structure that, in most over-stressing situations, is able to avoid possible ruptures. Conservation of its structural integrity against powerful stresses is guaranteed by neuromuscular correlation that enables it to change position and flexion angle through muscular co-ordinated contraction in response to stressing mechanisms. That is why, for example, a football player receiving a stress force on the lateral side of the knee reacts to the possibly traumatic stress with a contraction of the hamstring to subconsciously flex the knee and reduce the injuring forces on the medial side. This compensation mechanism is naturally not sufficient if the foot is stably planted on the field, in which case the knee cannot avoid the stressing mechanism. The lesion occurs when the intensity of the stressing forces overwhelms the ligament's capacity to resist the straining forces.

Certainly, in many patients, instability is a complex combination of the kinds of lesions previously described. There are different traumatic mechanisms that could determine capsular or ligamentous lesions in the knee. By referring to the study of Marchetti et al. [11], we can recognize 2 or 3 kinds of injuries that can determine an anteromedial instability (Figs. 3–5).

Valgus forces in extension or flexion (specially if associated to 2 or 3);
- External rotation forces;
- Tibial anterior over-stress.

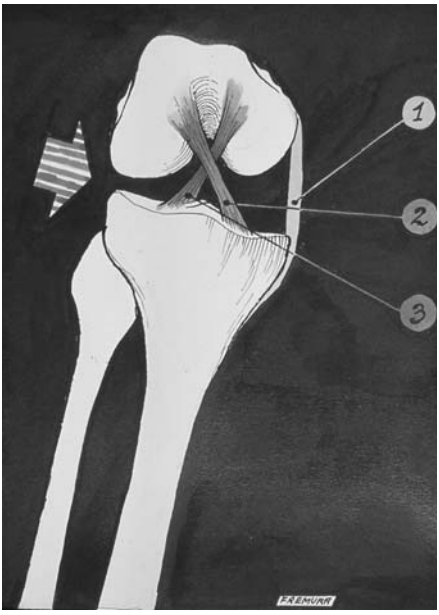

Fig. 3. Structures involved in valgus over-stress

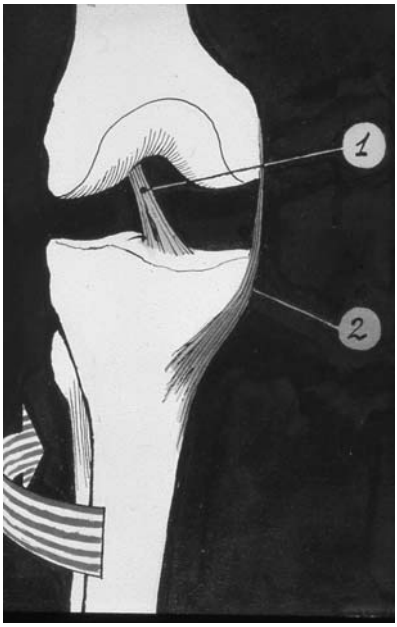

Fig. 4. Structures involved in external rotation over-stress

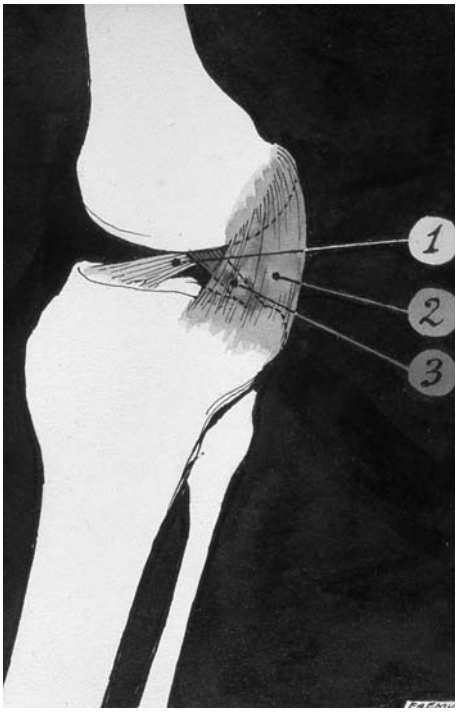

Fig. 5. Structures involved in medial recurvatum over-stress

Physical Examination

Before performing a physical examination, it is necessary to obtain the patient's history, with a description of the traumatic mechanism. During normal activities such as walking, knee range of motion is between 0° and 20° of flexion. In this condition, femoral condyles are "locked" on the tibial plate, and no rotational stresses are present. During running activities, the maximum flexion angle is directly related to rate. With flexion angles higher than 20°, the femoral condyle with intact ACL and PCL starts rotating on tibial plates. Rotation begins when the knee starts flexing; maximum rotation occurs between 60° and 90° of flexion. In sport activities that require pivoting, cutting, or rapid stopping, rotational instability, if present, will express itself at the maximum grade.

Functional tests are important to recognise this kind of lesion. Patients should perform fast jogging, hop test with both legs one by one, out-to-the-side test, and squatting producing a valgus stress on the knee and with the unaffected leg crossed over the affected leg producing a varus stress to the knee. Any difference between the two legs during tests should be recorded. Rotational instability can result as a combination of previously described

instabilities. AMRI is often found together with a posteromedial one, as well as anterolateral and posterolateral instabilities [12, 13].

Clinical Tests

The intimate relation between the posterior horn of the medial meniscus, the POL, and the semi-membranosus expansion is critical for dynamic stabilisation of the medial side of the knee. Any injury to the components of this complex structure can result in a loss of this co-ordinated balance [14]. Performing a complete pattern of clinical tests is important to determine and assess an anteromedial instability.

It is important to recognise whether the ACL is involved, with a lesion of its structure, or if there is just a lesion, simple or complex, to the medial side of the knee. Lachman test is important in evaluating any pathologically increased AP translation of the tibia with respect to the femur. It is positive in case of ACL deficiency. The test is performed with the knee at approximately 15–30° of flexion with the foot resting on the examining table. A firm endpoint should be obvious when the ACL is intact. If the ACL is deficient, the tibia will translate forwards excessively. The test can be performed with the patient awake or under anaesthesia. In case of ACL rupture, both variants have a high level of accuracy. The drawer test is performed in three positions. The knee is flexed at 60–90° with the foot resting on examining table. When the test is performed with foot in neutral rotation, the ACL is the main stabiliser against anterior tibial translation, providing the 87% of resistance to this kind of stress. Severity of instability is graded on a scale of 1–3 depending on the amount of displacement (grade 1 >5 mm; grade 2 >5 mm–1 cm; grade 3 >1 cm). The examiner's experience is a fundamental factor in the correct use of this kind of scale. The most important position in recognising AMRI is with the foot in external rotation (about 15°) when the medial structures of the knee are tightened. If the medial tibial plate moves excessively forwards compared with the one on the opposite side, it can be concluded that the medial tissues in the involved knee are stretching. Therefore, it is important to compare any laxity with the opposite knee in the same position. If the ACL is completely torn, the amount of anterior translation will be increased. In internal rotation, lateral structures can be tested with nearly the same consideration as for those of the medial side.

Finally, in conjunction with these positive tests, focal tenderness along the posteromedial aspect of the joint line in the area of the meniscotibial ligament and medial meniscus instability with abduction stress testing (medial subluxation of the meniscus) may be present in case of injury to the meniscotibial medial ligament.

Treatment

In the past, the most popular treatment was generally to use a cast for 8 weeks, but as a consequence of the poor results observed with non-operative treatment, there was a swing towards operative strategies. An ideal algorithm to better assess whether there is an indication for surgical reconstruction has to consider the individual kind of injury and its intrinsic potential to heal, the natural history of injury mechanism, and the effects of complex and combined injuries. As well as we know that ACL lesions do not heal while PCL tears do heal, we can observe that isolated MCL injuries heal while lateral-side combined injuries with multiple torn structures do not spontaneously heal.

MCL injuries combined with ACL lesions create different and complex problems, and the goal is to understand the basic principles of each ligament tear pattern and the relative treatment possibilities. After a complete initial evaluation, it is important to pay attention to the eventual swelling, investigating whether there is a large hemarthrosis and if it is contained within the joint capsule, in order to obtain a first indication about the severity of the injury. For combined medial-side and ACL injuries, we observe a high incidence of knee stiffness and arthrofibrosis in acute surgical procedures.

MRI observation can be helpful in clarifying the diagnosis and, even in severe grade 3 injuries, MRI has shown it is possible for the MCL to heal with continuity. MRI can help assess the position of the MCL lesion and determine if it is proximal or distal. Proximal injuries are usually more painful, show a large amount of swelling, cause the knee to stiffen quickly, and remain in flexed position. Distal injuries are usually less swollen, and the patient feels more comfortable with the knee in extension. A potential non-operative treatment of combined ACL and medial-side lesions must consider these differences. Proximal injuries cause the knee to stiffen quickly, reflecting a faster healing process, which requires a shorter casting – generally 4 weeks. Distal injuries, on the contrary, cause less swelling and a slower and less-intense stiffness of the joint, requiring a longer period of casting to obtain complete MCL healing.

Our general approach is to provide non-operative treatment to allow complete healing of the injured MCL and then to surgically reconstruct the torn ACL. The key to a successful result is to obtain complete healing of the MCL, which is why we prefer a brace for 3 weeks to allow weight bearing and to make the patient feel more comfortable during the process. The brace can be periodically removed to re-evaluate the healing process, and we allow mobilisation. If some residual laxity persists after 3 weeks, we put on a functional brace for 2 weeks and allow rehabilitation in order to gain range of motion (ROM) and muscle trophism. When severe residual laxity remains, it is possible to completely address and correct it during ACL surgery.

For every kind of surgical repair of the MCL, it is fundamental to observe some basic principles. Firstly, the critical point is restoration of isometry at the medial femoral epicondyle. Secondly, the surgeon has to pay attention to the redundancy of the PMC to avoid its significant anterior advancement or to avoid overtighting it because of the risk of postoperative flexor muscle contracture. The surgical approaches include a straight medial incision extending 6–8 cm from the medial epicondyle distally and incising the sartorial fascia at the posterior border of the superficial MCL and the anterior border of the POL, and a curved medial access extending from the medial epicondyle to 6 cm inferior to the medial joint line and 2 cm medial to the tibial tuberosity, paying attention to protect the sartorial branch of the saphenous nerve located between the sartorius and gracilis.

Surgical repair of acute MCL injuries, indicated in rare cases as complete disruptions, requires exposure of the tibial insertion of the MCL, retracting the sartorial fascia, the sartorius muscle, and other components of the pes anserinus. The sequence of repair should progress from deep to superficial and posterior to anterior. If repair of the superficial band of the MCL is necessary, it is important to preserve the broad and flat configuration of the ligament, choosing to use sutures, suture anchors, staples, or screw posts after the evaluation of lesion orientation. Tissues eventually avulsed from bone insertion have to be reattached with suture anchors or by performing osseous tunnels. Tears of the weaker meniscotibial portion of the medial capsular ligament are frequently combined with tears of the medial meniscus or its peripheral capsular attachment. It is important to repair all tears in the peripheral meniscal attachment and all tears within the outer one third of the meniscus structure, paying attention not to tie meniscal sutures until the medial-side reparation is complete. If medial-side tears are extensive and reparation looks fragile, we prefer to add other procedures to provide reinforcement and dynamic support. Some of these medial-side augmentation procedures include suturing the semi-membranosus tendon to the posteromedial corner to reinforce the POL, suturing the semi-membranosus into the posterior aspect of the MCL, advancing the gracilis and sartorius, performing a pes anserinus plasty, transposing anteriorly the vastus medialis, and performing a Bosworth procedure [15].

For surgical repair of chronic anteromedial knee instabilities, after ACL reconstruction, it is important to determine if residual laxity is present at 0° or 30° of flexion. Principles of medial-side reconstruction are:

- Repair/retention of medial meniscus if possible;
- Reconstruction of capsular structures, especially the posterior capsule;
- Restoration of the meniscotibial connection of the semi-membranosus complex;
- Reconstruction of the POL at the deep posterior corner;

- Reconstruction of the MCL.

Severe valgus instability usually requires tightening of the MCL in addition to the PMC and mid-medial capsular ligament. If the laxity is mainly meniscofemoral, we usually perform a tightening of the medial capsular ligament by proximal advancement if the meniscotibial portion is intact. If the laxity is mainly meniscotibial and the coronary ligament portion of the capsule is markedly lax with an intact peripheral attachment of the meniscus, a distal advancement of the posteromedial capsule may be more difficult. Rather than excising the meniscus, we prefer to detach the posteromedial attachments of the meniscus along its capsular attachment, then we advance the capsule distally and suture the meniscus to the tightened capsule [16, 17] (Fig. 6).

If a residual medial laxity is still present, the direct head of the semi-membranosus tendon can be advanced anteriorly. This takes advantage of the fact that the direct head inserts into the posterior tibial tubercle, consisting of the strongest soft-tissue anchor point at the posteromedial corner. A Bosworth reconstruction [15] has a good prognosis in residual laxities and consists in harvesting the proximal attachment of the semi-tendinosus tendon, leaving

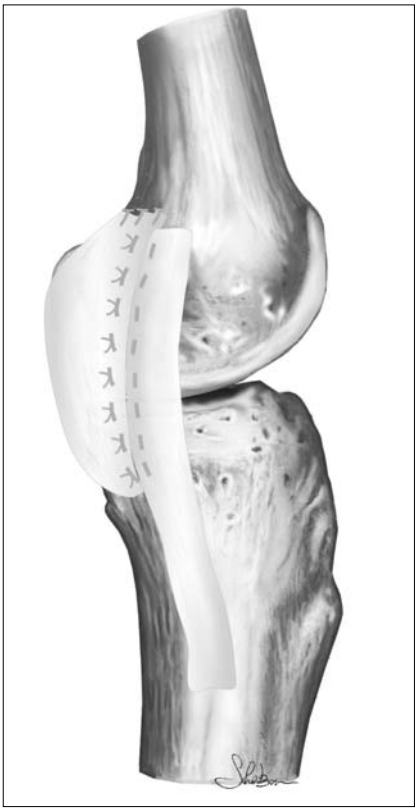

Fig. 6. Posteromedial capsule advancement

its distal insertion intact, passing the tendon over a suture post and a washer at the medial epicondyle before the proximal portion of the tendon is secured to the posteromedial corner of the tibia. To test and confirm the isometric fixation point, a Kirschner wire can be placed at the medial epicondyle, and then the knee can be mobilised in flexion and extension.

MCL reconstruction can now take advantage of the use of allograft tendons, especially when a chronic instability has been present for a long time and the intact residual medial-side structures may provide some stability. In this condition, to sacrifice and harvest the semi-tendinosus tendon or other medial-side structures may cause medial instability. The most frequently used allografts are semi-tendinosus tendon, anterior and posterior tibialis, patellar tendon, and most of all – in our experience – the Achilles tendon, the advantage of which is not to require its duplication and to guarantee adequate length for reconstruction. Allograft is usually fixed with a biodegradable interference screw or staples into the femoral medial epicondyle. On the tibial side, it is secured to roughened bone with a screw and spiked soft tissue washer or a staple at the distal end of the tibial MCL insertion site beneath the pes anserinus, similar to the Bosworth [15] procedure, or a tunnel can be created to reproduce both anterior and posterior borders of the native MCL (Fig. 7). The knee can be moved through its complete ROM to determine correct isometric placement of the graft on the femoral side before definitive fixation.

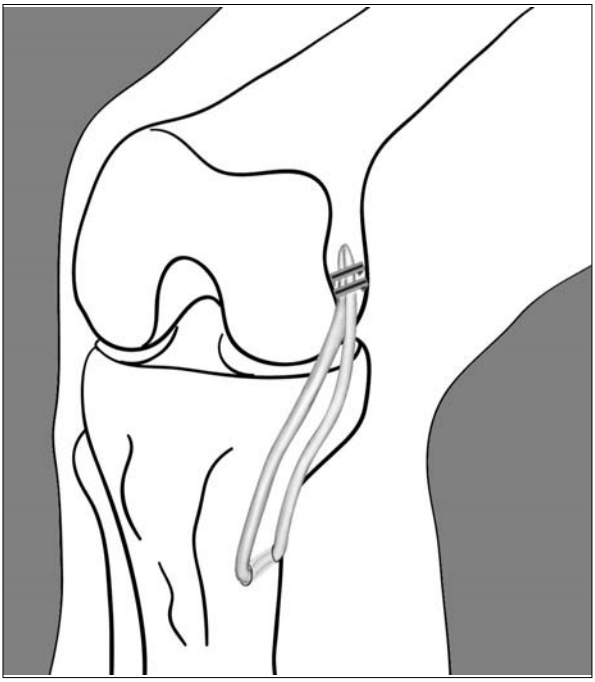

Fig. 7. Model of anatomical medial collateral ligament reconstruction using a hamstring allograft

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Section II
Specific Injuries

Medial-Side Injury of the Knee

Paolo Adravanti, Aldo Ampollini

Introduction

Whereas in lateral-side injuries of the knee, clear distinctions have been made between the lateral and posterolateral structures as separate entities, both of which are important in controlling varus laxity and external rotation, medial-sided knee injuries have often been identified with injuries to the superficial medial collateral ligament (MCL). In actual fact, the internal aspect of the knee is a far more complex structure, and the posteromedial corner, with its static and dynamic stabilisers, plays a fundamental role in medial knee stability, in controlling rotation, in the stabilisation of the medial meniscus, and in controlling anterior stability when the anterior cruciate ligament (ACL) is insufficient.

Anatomy

The anatomy of the medial aspect of the knee has been adequately described in the literature [1–3]. It extends from the medial edge of the patella to the medial edge of the posterior cruciate ligament (PCL). It can be divided into anatomical areas [2] or layers [1] whereas on a functional level, it is composed of static capsular and non-capsular ligaments and dynamic stabilisers, constituted by the semimembranosus tendon with its relative expansions. According to Sims and Jacobson [4], in the past, the contribution of the dynamic stabilisers and the effects of injury to the same have been underestimated, as most studies are performed on cadavers. Warren and Marshall divided the internal aspect of the knee into 3 layers according to cadaveric dissection [1]. The most superficial layer is constituted by the sartorius and its fascia. The pes anserinus tendons are located between the first and second layer. The second layer is constituted by the fibres of the anterior retinaculum, which blends with the first layer to the front, inserting the anteromedial tibial periosteum by the superficial MCL and to the posterior by the semimem-

branosus tendon. The superficial MCL extends from the medial femoral epicondyle and inserts the proximal tibia 8–10 cm below the articular margin and, deep down, the pes anserinus tendons. The thicker anterior fibres are arranged in a parallel manner whereas the posterior ones are oblique and blend with the posterior capsule of the posteromedial corner of the knee that forms the third layer. This third, deeper layer is constituted by the articular capsule, which is slender to the front and grows thicker centrally and to the rear. Centrally, the articular capsule is reinforced by the deep MCL composed of the meniscofemoral and meniscotibial ligaments. The posteromedial capsule blends with the posterior oblique fibres of the superficial MCL and is reinforced by the insertions of the semimembranosus tendon and the posterior oblique ligament (PCL). The PCL continues to the posterior and obliquely and inserts the adductor tubercle. It is constituted by the upper, middle, and lower bands [5]. The upper most proximal band is attached to the posterior capsule and connects below and behind in the expansion of the semimembranosus. From the adductor tubercle, the middle band inserts the meniscus posterior to the deep MCL and the proximal tibia, just above the direct arm of the semimembranosus. The lower band inserts distally on the semimembranosus and the tibia. It is intimately connected to the meniscus and the posterior capsular, and together with the expansions of the semimembranosus tendon, they constitute the posteromedial corner of the knee. This particular anatomical structure was described by Hughston et al. [2] and later by Müller et al. [3] as a static and dynamic stabiliser during coupled motion of the knee, restricting anteromedial rotation (Fig. 1). Five semi-

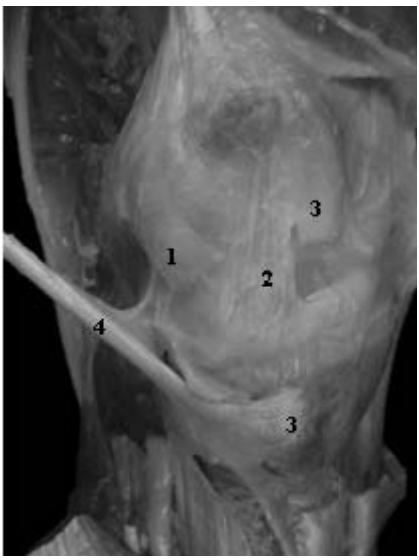

Fig. 1. Medial side's ligament of the knee and semimembranosus tendon. *1,* Posterior oblique ligament; *2,* deep fascicle of medial ligament; *3,* superficial fascicle of cut medial ligament; *4,* semimembranosus tendon

membranosus expansions have been described [1]. The broadest expansion is the direct arm that inserts on the posteromedial corner of the tibia. The pars reflexa passes in depth to the superficial MCL on the medial edge of the tibia. The third superomedial expansion inserts at the PCL, the posterior capsule, and the medial meniscus, reinforcing them. The fourth insertion is the popliteal oblique ligament and extends laterally to reinforce the posterior capsule, whilst the fifth expansion continues distally and to the rear, blending with the popliteal fascia (Fig. 2).

Functional Anatomy

Müller et al. [3] and Warren and Marshall [6] stated that the anterior fibres of the superficial MCL are tight during flexion and lax during extension whereas the posterior fibres exhibit an opposite behaviour. These concepts were confirmed by Gardiner et al. [7] in their studies with sensors positioned on the structures of the medial aspect. The deep MCL is tight during flexion and lax during extension. The posterior capsule and the PCL are tight during extension and lax during flexion; however, their function as dynamic stabilisers cannot be evaluated during usual investigations on cadavers [4]. It has been suggested that thanks to the insertions on the PCL, on the posterior horn of the inner meniscus and on the posterior capsule, the semimembranosus tightens structures that should be lax when the knee is flexed and contributes to

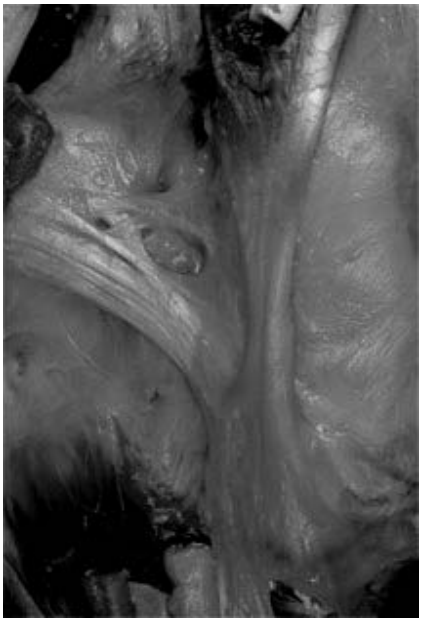

Fig. 2. Semimembranosus tendon and expansions

meniscus retraction during the flexion of the knee, thus preventing impingement between the femur and the tibia [3, 8, 9]. In their papers, Hughston et al. [2, 9, 10] emphasised the role of the PCL as the main medial stabiliser against valgus stress and introduced the concept of anteromedial rotational instability (AMRI), intended as an abnormal excess opening of the medial joint space at 30° of knee flexion, with an anterior dislocation of the medial tibial plateau in relation to the medial femoral condyle. The concept of the PCL as a dynamic stabiliser through the expansions of the semimembranosus was taken up by Müller et al. [3], who wrote: "despite its closed topographic relation to the medial collateral ligament, the posteromedial corner is fundamentally different in nature and function from the tibial collateral ligament". Warren and Marshall [1], in contrast with Hughston et al. and Müller et al., described the superficial MCL as the main medial stabiliser to valgus stress; however, in their study, they did not find the PCL as a separate anatomical entity.

In studies on cadavers, the section of the superficial MCL causes between 2° and 5° of medial laxity at 30° of knee flexion. The total amount of valgus laxity is limited. The cutting of the PCL and the posteromedial capsule increases medial laxity from 7° to 10° [6, 11–14]. There is agreement in stating that the rupture of the superficial MCL causes laxity during flexion and association with the PCL and the posteromedial capsule in extension. In addition, the cutting of the superficial MCL causes an increase in external rotation [6, 12, 14–17], which is 4–10° greater when the knee is flexed whereas it does not affect internal rotation.

The cutting of the posterior capsule and the PCL causes a 5–10° increase in extra-rotation. Internal rotation also increases considerably after additionally cutting the posteromedial complex [12, 14]. The fibres of the PCL are parallel at the PCL and resist posterior translation and internal rotation. Through their insertion on the posterior horn of the inner meniscus, the PCL and the posterior capsule act as secondary stabilisers to the anterior translation of the knee when the ACL is injured [3, 8, 18]. The deep MCL does not appear to play an important role in stability in valgus tests but, rather, acts as a medial meniscus stabiliser through the meniscotibial ligament. In the event of a tear of the meniscotibial ligament, there is an increase in the stress on the other knee structures, which resist anterior and anteromedial dislocation, with risks of condyle and inner meniscus damage [19]. In conclusion, one may state that:

- Rupture of the superficial MCL causes a valgus laxity of 2–5° when the knee is flexed and an increase in external rotation when the knee is flexed at 4–10°. The knee is stable in extension.
- Rupture of the deep MCL does not cause an increase in medial instability; however, rupture of the meniscotibial ligament increases mobility of the medial meniscus.

- Rupture of the PCL and the posteromedial capsule occurs in association with superficial MCL injury and causes a 7–8° increase in valgus laxity. Laxity is also present in extension. External rotation with the knee flexed increases by 10–20°, and internal rotation also increases. The meniscotibial fibres contribute to the stability of the medial meniscus.

Injury Mechanism

Medial-sided knee injuries are usually caused by an injury of the lower thigh or on the side of the upper leg. They can also be caused by distortion injuries in external rotation. These distortion injuries usually cause injury to the PCL and the posterior fibres of the MCL and may cause a partial or total tear of the ACL. When the valgus injury is associated with external rotation, simultaneous rotation of the MCL, PCL, and ACL is almost certain [20].

Diagnosis

Clinical inspection is important in identifying the site of tumefaction. The presence of medial tumefaction suggests pathology of the medial aspect and must be differentiated from a haemarthrosis, an indication of ACL tearing. The site of tumefaction and pain, especially when pressed with the fingertips, indicates the site of the injury. The valgus test at 30° of flexion remains the most important test for evaluating medial knee damage. As with all clinical tests performed on the knee, it must be compared with the healthy side. It must be performed with the patient relaxed and the muscles not contracted. Extension of the hip helps to keep the ischiocrural muscles relaxed. The thigh rests on the bed, and the knee is flexed by 30°, allowing the leg to hang from the edge of the bed, supporting the limb with a hand whilst the other hand grips the foot and the ankle. In order to better appreciate rotatory instability, which consists in the simultaneous valgus stress opening of the knee associated with the anterior dislocation of the medial tibial plateau in relation to the medial femoral condyle, Sims and Jacobson suggest supporting the sole of the foot rather than the ankle [4].

The positive outcome of the anterior drawer test in external rotation is another index of rotatory instability. This test is positive when the medial tibial plateau dislocates anterior to the medial condyle whereas the external aspect of the knee remains stable [10, 21]. According to certain Authors, it is important to evaluate the presence and absence of a rigid stop during the execution of valgus stress tests. However, opinions differ regarding the interpretation of this test, as according to Indelicato [20], the absence of a rigid stop indicates a simultaneous tear of the ACL whereas other Authors indicate the

rupture of the superficial MCL and the PCL. The valgus test must then be repeated with the leg extended. Positive valgus stress tests with the leg extended are an index of a rupture of the superficial MCL and the PCL. A combined tear of the ACL and the PCL must be suspected. As regards the classification of medial stability, as Ballmer and Jakob observed, there is no standardised method or evaluation uniformity [22].

Many Authors divide medial injuries into 3 grades: grade I (first-grade sprain with minimal disruption of fibres, tenderness, solid end point), 0 to 5-mm laxity; grade II (second-grade sprain with more fibre disruption, firm endpoint), 5 to 10-mm laxity in flexion; grade III (complete rupture, no endpoint), >10-mm opening of the joint in flexion and extension [23–25]. From this imprecise classification, it is not clear which ligaments are ruptured. The classification method put forward by Hughston et al. [10] also entails a distinction between 3 grades; however, it is more precise in quantifying laxity and the ligaments concerned:

- Grade I: rupture of few fibres, localised pain, and no instability;
- Grade II: rupture of more fibres, pain is more widespread; however, there is no valgus instability;
- Grade III: complete rupture of the ligament with valgus instability. This instability is in turn broken down according to the medial opening: 1+ (<5 mm, IIIA), 2+ (6–10 mm, IIIB), and 3+ (>10 mm, IIIC). A 1+ laxity indicates complete rupture of the superficial MCL, and the extension test is negative. A 2+ or 3+ laxity in flexion indicates rupture of the superficial MCL, the PCL, and the posteromedial capsule. Medial laxity is also present in extension.

Treatment

Treatment of isolated medial-sided knee injuries has changed over the past 20 years. Whereas conservative treatment in grades I, II, and IIIA and B injuries is universally accepted, there is disagreement on whether conservative or surgical treatment is best suited to severe grade IIIC injuries, especially in world-class athletes, although most Authors favour the conservative treatment option. Grade IIIA and B injuries of the MCL are treated using an orthosis in extension and the use of crutches to relieve pain. As soon as pain permits, the patient starts a rehabilitation programme for joint recovery and isotonic, isometric and, when possible, isokinetic exercises. Load is encouraged with the orthosis, and crutches are gradually eliminated. Various rehabilitation protocols can be followed for this type of lesion [26]. The resumption of sports activities depends on the type of sport and the gravity of the injury. On average, grades I and II injuries require 10 days and grade III injuries 3–6 weeks.

However, in the sports at greatest risk, such as football, the period of inactivity could be longer [26]. In those patients who experience persistent pain caused by lesions to the femoral insertion of the MCL, immobilisation must be maintained until the disappearance of symptoms [20]. If pain and tumefaction appear during rehabilitation, a fracture of the meniscus or cartilage damage must be suspected. The persistence of pain at the articular margin beyond 3 weeks also suggests meniscal injury.

According to many Authors, isolated injuries with valgus laxity of 2+ heal well with conservative therapy although some patients heal with slight residual laxity [27–34]. Isolated grade IIIC lesions will also heal with functional conservative treatment [22, 31, 32, 34–36]. Fetto et al. [27] and Sandberg et al. [37] found no improvement after operative treatment of isolated grade III lesions. Conservative treatment has also been used on world-class athletes with excellent results [32]. Reider et al. [36] reported outstanding results in 35 athletes monitored for approximately 5 years; 19 patients resumed sports practice within 8 weeks, of whom 16 resumed sports practice within 4 weeks. The treatment proposed for grade IIIC injuries of the medial aspect [20] consists of a knee brace in extension for 2 weeks, however, commencing articular recovery without limitations compatible to pain, load with crutches just bearable, and without crutches when the patient has achieved a noticeable limp. The knee brace is gradually removed after 3–4 weeks. Training can be resumed when the limb has regained 80% of its strength in isokinetic tests. In one series, Kannus [33, 38] observed a higher percentage of arthritic degeneration following conservative treatment of severe medial laxity in grade IIIC injuries.

The criticism of systematic conservative treatment of even isolated medial-side knee injuries lies in the fact that such series often do not specify the severity of medial instability [39]. Hughston holds that when talking of grade III lesions, the severity of grade IIIC injuries must be specified and claims that they must be treated surgically. Sims and Jacobson [4] emphasise the concept of AMRI, presented previously by Hughston, as an index of complex rupture of the medial and posteromedial structures of the knee, which requires surgical treatment even in the case of isolated injuries. PCL repair and, when necessary, repair of the semimembranosus tendon and meniscotibial ligament insertion is of fundamental importance to countering anteromedial rotatory instability. Furthermore, in the event of meniscotibial ligament rupture, the tibia slides forwards, and the stabilising action of the meniscus is therefore lacking and the articular cartilage and the meniscus risks further damage and at higher stress level, as do the other joint structures that resist anterior and anteromedial dislocation of the knee.

In conclusion, many authors believe that acute isolated grade IIIB and C injuries can be treated conservatively, thus saving surgery for acute-phase

bony ligament avulsions. Others believe that medial injury repair is more suitable, even in isolated injuries where medial laxity is higher than 10 mm with anteromedial rotatory instability. Chronic instability of the MCL/posterior MCL is a relatively rare operation indication [40]. Shahane et al. [41] reported favourable results after an "isometric" proximal advancement and recession of the MCL. Kim et al. [42] and Borden et al. [43] reported anatomic-like reconstructions with semitendinosus tenodesis for repair of the superficial MCL and PCL, but results were not available. A strip of semitendinosus tendon can also be used to support the PCL [3] (Fig. 3).

Surgical Technique

Surgical technique for medial-side knee injury repair is broken down into four stages:

1. Repairing the meniscus and the posteromedial capsule, fastening it to the tibia
2. Repairing the superficial MCL (tight in flexion)
3. Repairing the PCL and the posteromedial capsule (tight in extension)
4. Repairing the semimembranosus and the capsular extensions thereof.

Firstly, an arthroscopy is performed to exclude intra-articular injury. This examination makes it possible to assess two indirect signs of posteromedial corner injury: meniscal rise during adduction stress at 30° of flexion (Fig. 4)

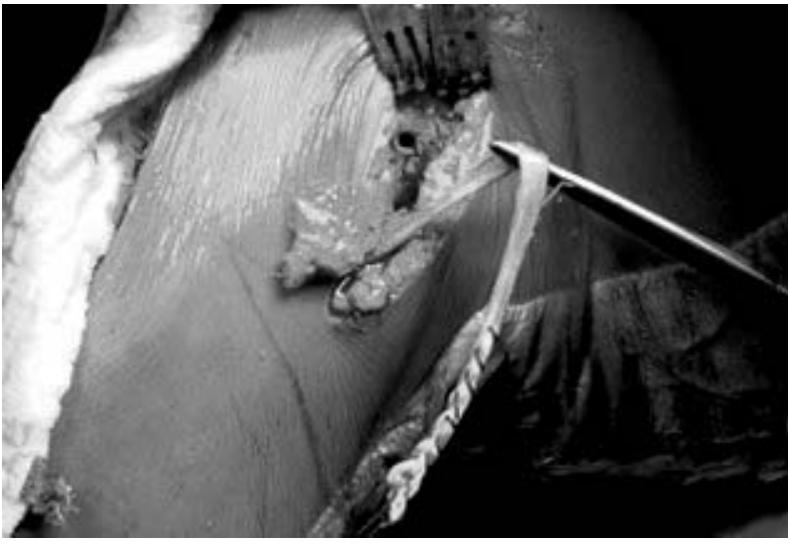

Fig. 3. Strip of semitendinosus tendon used to support the posterior oblique ligament (POL) and the superficial medial collateral ligament (SMCL)

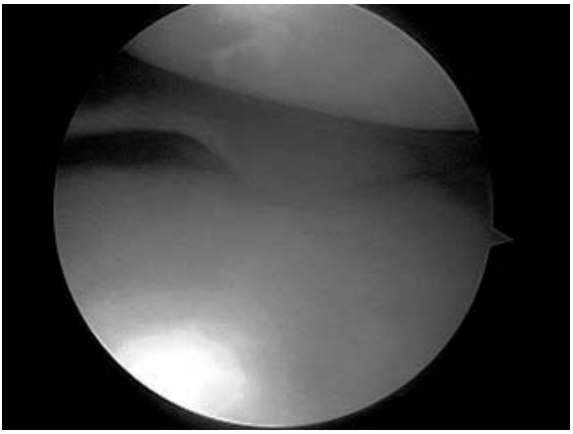

Fig. 4. Meniscal rise during abduction stress at 30° of flexion

and injury-site bleeding. Surgical technique involves a curved incision 5 cm above the epicondyle directed distally towards the medial margin of the patella. Once the fascia has been exposed, it is cut from proximal to distal anterior to the epicondyle and posterior to the medial vastus muscle fibres as far as the insertion of the pes anserinus tendons. This allows the observance of the pes anserinus tendons distally and the posteromedial corner. The superficial MCL that inserts the medial epicondyle is identified. The deep MCL inserts slightly distally, and the PCL and posteromedial capsule insert at the adductor tubercle. It is also necessary to identify the distal insertion of the superficial MCL situated beneath the pes anserinus tendon (Fig. 5) and the semimembranosus tendon, with its expansions. The gastrocnemius insertion is freed from the posteromedial capsule using a Homan retractor to protect the vasculo-nervous structures. A retro-LLI arthrotomy is performed through an oblique incision and the posteromedial capsule and semimembranosus tendon are examined and repaired using bone anchors (Figs. 6, 7), and any meniscal disinsertion is sutured to the capsule. If damaged, the meniscotibial ligament is now repositioned using bones-anchors. A valgus stress test at 30° allows a clearer indication of the superficial MCL rupture site. Bony avulsions are fastened using washers and screws, and ligament avulsions are repaired using bone anchors or screws with toothed washers. The superficial MCL is repaired with the knee flexed. The posteromedial capsule and the PCL are then repaired with the knee extended using bone anchors or detached sutures according to the site of rupture. If necessary, the direct expansion of the semimembranosus at the tibia is repaired. Tension the PCL distally with sutures from proximal to the semimembranosus or tibia or do so proximally if the femoral attachment is loose. The knee should be stable now in extension. Advance and retension of the *posterior extension* of the PMCL (oblique

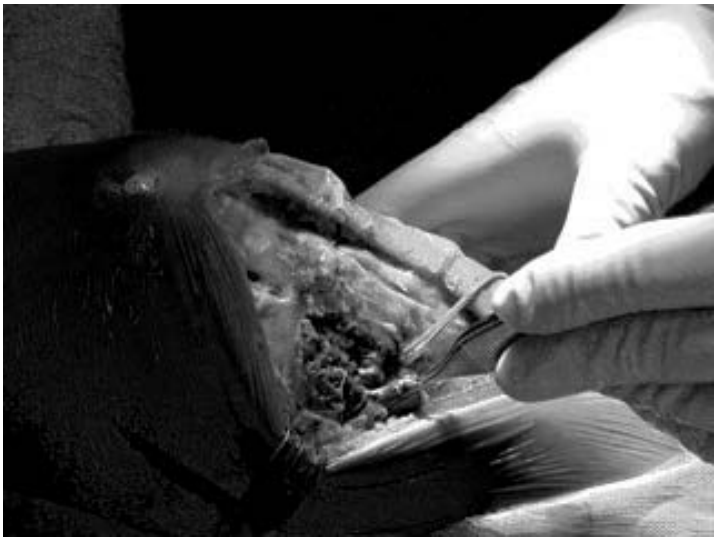

Fig. 5. Superficial medial collateral ligament rupture at the tibial site

Fig. 6. Capsule tear at the tibial junction

popliteal ligament) to the semimembranosus corner by closing the posterior arthrotomy, and use multiple sutures for fixation. Advance and retension of the *capsular arm* to the semimembranosus and *advance the semimembranosus* to the capsule. PCL/PMCL should be tight in extension.

Conclusion

Isolated medial-side knee injuries in athletes are usually treated conservatively with excellent results. However, in particularly severe cases with valgus

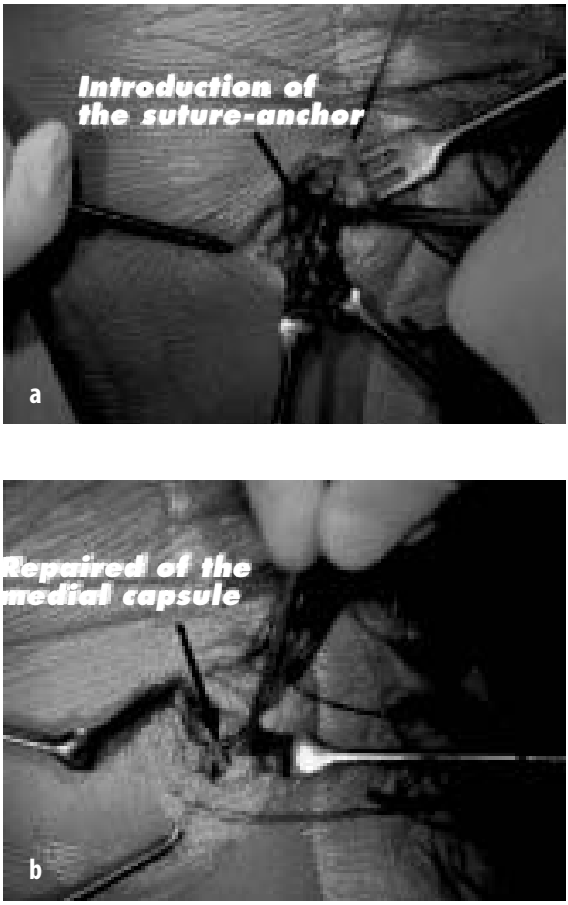

Fig. 7a, b. Introduction of the suture anchor to repair the medial capsule at the tibial junction

laxity of 10–15 mm, surgical treatment is suggested. In such patients, the presence of anteromedial rotatory instability could condition functional results. During the operation, surgeons must examine all the various medial structures involved rather than simply repairing the superficial MCL. The posteromedial corner constituted by the PCL, the posteromedial capsule, and the tendinous expansions of the semimembranosus at the tibia to the capsule and the posterior horn of the medial meniscus constitute an important medial stabiliser and restriction to internal and external rotation. The posterior horn of the medial meniscus is also a secondary stabiliser to anterior tibial translation in the event of ACL insufficiency but not in cases of meniscotibial ligament and capsule rupture posterior to the tibia. A lack of stabilisation of the posteromedial corner and medial meniscus could cause condyle and meniscal damage secondary to the greater mechanical strain placed on the ACL.

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Posterior Cruciate Ligament Injuries

Pier Paolo Mariani, Fabrizio Margheritini

Introduction

Soccer is the world's most popular organised sport with over 200 million males and 21 million females registered with the Fédération Internationale de Football Association [International Football Federation (FIFA)]. Despite the widespread distribution of this sports activity and the fact that the knee is the second most frequent location of injury [1, 2], there is a lack of information concerning the distribution of posterior cruciate ligament (PCL) injuries among those who play this activity even though the PCL is the stronger of the cruciate ligaments and has been considered by some to be the primary stabiliser of the knee. Historically, the athletic population has been considered at risk for PCL injury, but interestingly, the reported incidence of this lesion appears to be relatively low. Some have expressed the concern that PCL injuries are underestimated secondary to subtlety of symptoms and physical exam findings. Furthermore, 50–90% of PCL injuries are combined injuries involving, most commonly, the posterolateral structures (PLS) of the knee [3, 4]. Although athletes, and soccer players specifically, can often function at a high level with an isolated PCL injury, combined injuries and severe isolated injuries with persistent symptomatic instability usually require surgical treatment. It has been suggested that isolated PCL injury, if left untreated, may result in disability years later. The natural history of the PCL-deficient knee, however, remains a matter of debate. Furthermore, clinical evidence that current reconstruction techniques significantly alter the stability and function of the PCL-deficient knee is lacking. The purpose of this article is to provide a current overview of the basic science and clinical aspects of PCL injury, focusing on the soccer player population.

Anatomy and Biomechanics

Understanding the anatomy and biomechanics of the PCL is important in diagnosing and treating its injury. This ligament is a complex structure that

arises from the posterior tibia 1 cm below the joint line and extends anteromedially to the lateral surface of the medial femoral condyle. The PCL averages in length between 32 mm and 38 mm and has a cross-sectional area of 31.2 mm^2 at its mid-substance level, which is 1.5 times that of the anterior cruciate ligament (ACL) cross-sectional area [5–7]. Its femoral and tibial insertion sites are approximately three times larger than the cross-sectional area at the mid-substance level of the ligament. The large ligamentous insertion sites and the lack of isometry within the fibres of the PCL complicate the task of designing a PCL reconstruction technique that adequately re-creates the anatomical and biomechanical properties of the intact PCL.

The ligament consists of two functional components referred to as the anterolateral (AL) and the posteromedial (PM) bundles [7–9]. The AL bundle is two times larger in cross-sectional area than the PM bundle, and they behave differently depending on the degree of knee flexion [5, 8]. During passive flexion and extension of the knee, the AL bundle is more taught in flexion and lax in extension. Conversely, the PM bundle is more taught in extension and lax in flexion. Despite the widespread acceptance of this anatomical division of the PCL, alternate anatomic descriptions of the PCL exist, including three- and four-bundle divisions as well as a continuum of PCL fibre orientation [10]. In addition to the AL and PM bundles, there are two meniscofemoral ligaments (MFL) closely associated with the PCL: the ligament of Humphry (anterior) and the ligament of Wrisberg (posterior) (Fig. 1). They originate from the posterior horn of the lateral meniscus, run along side the PCL, and insert anterior and posterior to the PCL on the medial femoral condyle [11]. The presence of these ligaments is highly variable, and discrepancies exist in the literature regarding their prevalence [8, 12]. The importance of the MFL has not been fully characterised, but they are believed to be significant anatomic and biomechanical structures that provide stability to the lateral meniscus and restrain the posterior translation [7, 12].

The PLS consist of a complex system of static and dynamic stabilisers that function in providing posterolateral stability to the knee. Numerous anatomic studies have focused on defining this complicated region of the knee. Despite the inconsistencies in nomenclature that exist in the literature, it is generally accepted that static stabilisers of the PLS include the lateral collateral ligament (LCL), the arcuate ligament complex, the fabellofibular ligament, the posterior horn of the lateral meniscus, and the lateral part of the posterior capsule; and that the dynamic stabilisers of the PLS include the popliteus complex, the biceps tendon, and the iliotibial tract [13–19].

Biomechanical studies have shown that the PCL is one of the major stabilisers of the knee. It has a primary function in preventing posterior tibial displacement [20–24] and a secondary role in limiting varus and valgus and external rotation [22, 25]. Initial tensile testing reported the tensile strength

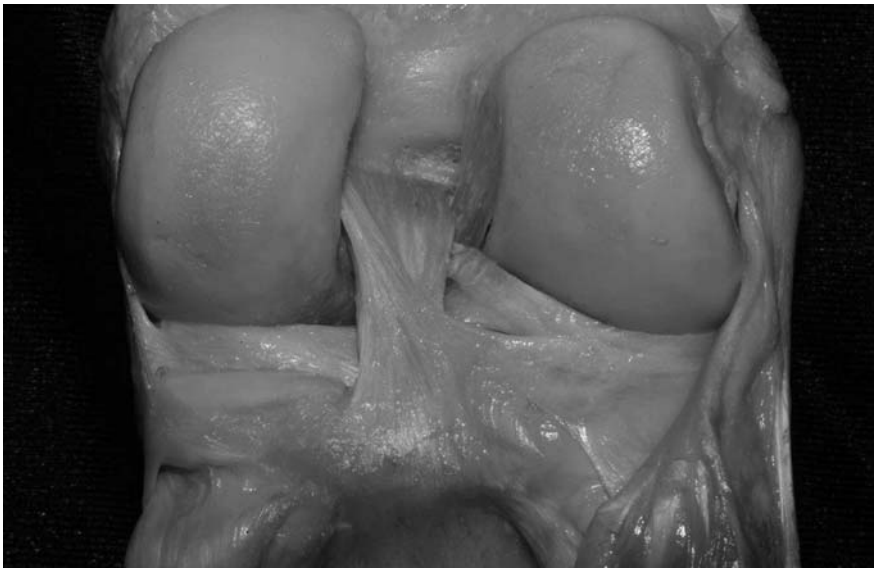

Fig. 1. Anatomic posterior view of posterior cruciate ligament (PCL). The broad insertion on femur and tibia of the PCL and the posterior meniscofemoral ligament is clearly seen (Courtesy of Dr. Pau Golano)

of the excised PCL to be twice that of the ACL [26]. Harner et al. [7] showed the linear stiffness of the AL bundle (120±37 N/m) to be 2.1 times that of the PM bundle and 2.5 times that of the MFL, and the ultimate load of the AL bundle (1120 ± 362 N/m) to be 2.7 times that of the PM bundle and 3.8 times that of the MFL. Several biomechanical cutting studies have demonstrated that isolated section of the PCL increases posterior tibial translation progressively as the knee is flexed from 0° to 90°, with maximal increase in translation occurring at 90° of knee flexion [22–24, 27]. Furthermore, results suggest that a biomechanical interaction exists between the PCL and the PLS in providing stability to the knee. Isolated sectioning of the PCL results in posterior tibial translation by up to 11.4±1.9 mm while isolated sectioning of the PLS increases posterior translation between 1.5 to 4 mm. However, after sectioning of both the PCL and PLS, posterior tibial translation in response to a posterior load is increased by up to 25 mm. Combined PCL and PLS section increases posterior tibial translation at all degrees of knee flexion greater than isolated PCL section. The PLS is a secondary restraint to posterior tibial translation, contributing to posterior stability particularly in the PCL-deficient knee, while it plays a primary role in resisting excessive varus and external rotational forces [20, 23, 28, 29]. Isolated section of the PLS increases varus and external rotation maximally at 30–45° and has little effect on this rotation at 90° of knee flexion [23, 24, 30]. Combined PCL and PLS section

increases varus and external rotation at both 30° and 90° [22, 23]. These findings, in addition to the results pertaining to posterior tibial translation, suggest a synergistic relationship between the PCL and the PLS in providing stability to the knee.

Incidence and Mechanism of Injury

Despite the fact that athletes have historically been considered at high risk for PCL injuries, data regarding the distribution and incidence of sports-related PCL injury are limited, and specifically, data are even more rare when dealing with soccer. While some information is available concerning the increasing incidence of PCL injuries among American football players, rugby players, baseball athletes, and skiers, very little has been written about the incidence of PCL lesions in soccer. Arendt and Dick [31], reporting on knee injuries in collegiate soccer and basketball, found that among all knee injuries, the incidence of PCL tears was 4% and 2%, respectively. More recently Schultz et al. reported an incidence of 24.7 % of soccer-related injuries among a group of 494 studied PCL lesions [32]. Fowler and Messieh [33] reported that hyperflexion, with or without anterior tibial trauma, was the most common mechanism of PCL injury reported by the athletes studied at their institution. A specific example is the goalkeeper who raises a knee to a hyperflexed position while jumping to catch the soccer ball [34]. This goalkeeper is at risk of a PCL injury when an opposing player strikes the knee while it is in the hyperflexed position. In case of more complex injuries, these are more likely caused by a posteriorly directed force applied to the tibia or a valgus or varus force applied to the hyperflexed knee. These injuries are more severe and involve additional structures such as the ACL, the collateral ligaments, or, most commonly, the PLS. Shelbourne et al. [35] described the low-velocity knee dislocation as a relatively common mechanism of combined injury in the athletic population. In our practice, we found that ACL/PCL combined injuries are relatively common among soccer players since, other than for traumatic injury, these lesions can occur even when stumbling over a hole while running on the soccer field [36].

Diagnosis

Regardless of the subjective information provided by the patient, a physical examination of the knee should always include a thorough evaluation for PCL deficiency. Without this essential objective information, the examiner risks

falsely attributing an increase in anterior-posterior laxity to a torn ACL. On physical exam, the most sensitive test for PCL deficiency is the posterior drawer test. Following the application of a posterior tibial load, while maintaining the hip and knee flexed at 45° and 90°, respectively, posterior tibial translation is measured and divided into three grades of injury. A grade I injury is represented by posterior translation between 1 mm and 5 mm, grade II between 5 mm and 10 mm (with the anterior border of the tibial plateau lying flush with the femoral condyles), and grade III greater than 10 mm (with the anterior border of the tibial plateau lying posterior to the femoral condyles). Additional information can be obtained by performing this test with the tibia held in internal and external rotation. Additional tests commonly used to evaluate the PCL include the posterior sag, the quadriceps active tests, and the Whipple. PL structures can be checked using the reverse pivot shift test [37] and the external rotation tight-foot angle (ERTFA or dial clock) test [23]. Much information regarding PCL injury can be obtained from imaging studies, including stress radiographs [38] and magnetic resonance imaging (MRI) [39].

Management

Non-Operative Approach

A non-operative approach to an isolated PCL injury has traditionally been recommended because of the capacity of the ligament to heal. With isolated PCL injury, the athlete is often able to compensate for the change in joint kinematics resulting from ligamentous disruption. This adaptation most likely occurs through compensatory muscle function developed through a carefully guided physical therapy programme. The approach to non-operative treatment revolves around adequate knee stabilisation that facilitates healing and return of the ligament's primary function in resisting excessive posterior tibial translation. A 2- to 4-week period of immobilisation is suggested with the knee in full extension. This position results in reduction of the tibia, prevents posterior sag, and diminishes the effects of gravity and hamstring muscle contraction on tibial translation. Additionally, in this position, the AL component of the PCL is under the least tension, allowing it to heal in a more biomechanically optimal position. During this period, quadriceps muscle strengthening exercises are encouraged whereas the use of the hamstring muscles is prohibited to minimise posterior tibial load. Depending on the severity of the injury, the athlete can usually return to sports activity 1–3 months after injury.

Surgical Reconstruction

Surgical reconstruction of the PCL is recommended in acute injuries that result in severe posterior tibial subluxation and instability, objective criteria that are often met in cases of combined ligamentous injuries. Numerous PCL reconstruction techniques have been described in the literature. Since its first description by Clancy et al. [40] in 1983, single-bundle PCL reconstruction has become a popular surgical option. Its focus is on the reconstruction of the larger, stiffer AL bundle. A recent biomechanical study [41] has shown that the single-bundle reconstruction technique can restore normal knee kinematics from 0° to 60° of knee flexion. The Authors of this study reported that the stability achieved by this reconstruction is adequate for activities that do not require full range of knee flexion. Clinical results support this theory [42, 43]. The same study [41] included a biomechanical evaluation of the double-bundle PCL reconstruction with a splitted patellar tendon graft. The Authors showed that this double-bundle reconstruction restored normal kinematics across the full range of knee flexion. In another recent biomechanical study comparing these two reconstruction techniques, Harner et al. [44] performed a double-bundle reconstruction using an Achilles tendon graft to reconstruct the AL bundle and a semitendinosus tendon graft to reconstruct the PM bundle of the native PCL. These authors showed that this type of double-bundle reconstruction is better able to reproduce normal knee kinematics between 0° and 120° of knee flexion. These current biomechanical data support the use of the double-bundle reconstruction, suggesting that it is able to better restore normal knee kinematics and joint forces when compared with the single-bundle approach. Clinical studies providing long-term results from the single- and double-bundle reconstructions are currently lacking. Tibial inlay reconstruction, involving both an arthroscopic and a posterior open approach, has more recently been presented as an alternative technique to the transtibial tunnel single-bundle technique [45]. Rather than using a tunnel for tibial attachment, the tibial inlay technique uses a bone trough at the tibial site of PCL insertion to which the bone block of the graft is directly fixed. This type of reconstruction has the specific aim of restoring normal knee kinematics by achieving a more anatomic tibial fixation and by avoiding what has been described as the *killer turn*. While first biomechanics studies supported the superiority of the tibial inlay technique versus the transtibial one, more recent studies [46, 47], however, failed to show any significant difference between these two techniques.

Postoperative rehabilitation following PCL reconstruction focuses on avoiding excessive graft stress until adequate healing of the PCL graft has occurred. This is achieved by fixing the patient in extension during early rehabilitation and avoiding exercises, such as open-chain hamstring exercis-

es, which place an excessive posterior force on the tibia. Patients are immobilised in extension for 1 week following surgery. The hinged brace is unlocked during range of motion exercises for the following 6-week period and then can be unlocked continuously as the patient regains a normal gait. Partial weight-bearing status is maintained for 6–8 weeks. Range of motion exercises are essential to regaining full knee flexion. Patients are progressed slowly through passive flexion exercises in the early postoperative period and, in most cases, regain full flexion in 5–7 months. The goal of rehabilitation is to achieve 90% of normal quadriceps and hamstring strength. Closed-chain exercises are begun after 6–8 weeks when the patient is full weight bearing. A structured physical therapy programme is essential to regaining motion, strength, and proprioception. In general, athletes are able to return to full activity 9–12 months following surgery, depending on the demands of the specific sport and the progression of physical therapy.

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Articular Cartilage Lesions in Football Players

Lars Peterson, Christian Ernest

Introduction

The first physician to recognise the clinical problems associated with cartilage injuries is reported to be Hippocrates in 400 BC. His pupil Herodicus was supposed to be the first sports medicine doctor treating Olympic athletes. Trauma or impacts, both occasional and repetitive, can cause damages to the articular cartilage as well as the subchondral bone in a joint. The reparative response to articular cartilage injuries is limited. The cartilage has no vascular supply, so unless the subchondral bone is involved, an injury does not cause bleeding and formation of a blood clot that possibly could fill the cartilage defect. The chondrocytes are unable to migrate into and populate the defect. Local increase of the synthesis of matrix molecules occurs in the neighbourhood of the defect during the acute phase but ends at about 2 weeks for unknown reason. Untreated acute lesions penetrating the articular cartilage of the knee down to bone will likely progress to early post-traumatic osteoarthritis by continued enzymatic degradation activity and mechanical wear over time. Osteoarthritis is an irreversible process and may lead to considerable disability for the player. The challenge is not only to restore activities of daily life but also to allow athletes to go back to top-level sports such as football.

Risk for Cartilage Injuries

Articular cartilage injuries are common in football and may be career ending if not treated properly. In acute and chronic injuries to the knee joint treated surgically, more than 40% of patients were found to have articular cartilage injuries down to bone. Noyes et al. found that acute and chronic anterior cruciate ligament (ACL) injuries were 40–70% associated with articular cartilage injuries [1, 2.] There are also reports that in 40–50% of cases with meniscus injury also have cartilage damage. Hjelle et al. reported that in 1,000 consec-

utive patients with symptoms requiring arthroscopy, chondral or osteochondral lesions of any type were found in 61% [3]. In a study by Drawer and Fuller on retired English professional players, 32% had osteoarthritis [4]. Levy et al. found a higher risk of chondral injuries in highly skilled football players [5]. It is thought that the increased risk of articular cartilage injuries and gonarthrosis in football is due to the high joint stresses associated with repetitive joint impact, rapid acceleration and deceleration, and pivoting as well as contact with other players. ACL and meniscus injuries are often combined with articular cartilage injuries where the cartilage injury is the most serious and hardest to treat.

Symptoms and Diagnosis

As cartilage lesions are likely to progress in size and worsening symptoms and may cause an end to the career for the player, it is important to diagnose and treat cartilage lesions at an early stage. Symptoms often associated with articular cartilage lesions are pain and swelling during or after activity, crepitations, and catching or locking. To diagnose a cartilage lesion, a thorough clinical examination is needed and includes assessment of ligament instability, varus-valgus malalignment, and patella maltracking or malalignment. X-rays, computed tomography (CT), and especially magnetic resonance imaging (MRI) are useful tools to diagnose damage to the articular cartilage. The definite diagnosis is taken during arthroscopy and probing of the articular surfaces.

Surgical Treatments

Treating chondral lesions has been a known difficult clinical challenge since Hippocrates. There are today many different treatment options for cartilage lesions, such as *débridement*, coblation, drilling, abrasion, microfracturing, osteochondral transplantation as in mosaic plasty, and autologous chondrocyte transplantation.

Arthroscopic *débridement* of loose flaps and rugged surfaces gives short-term relieve of symptoms and may delay or stop further deterioration of a cartilage surface. Coblation (vaporisation) may be used with caution and strictly according to instructions. Drilling, abrasion, and micro-fracturing are all treatments that penetrate the subchondral bone plate into the bone marrow, which will cause bleeding, and stem cells and fibroblasts can migrate into the cartilage lesion and produce repair tissue of fibrocartilage [6]. However, the mechanical properties of the fibrous cartilage are inferior to those of hyaline cartilage and are probably not able to withstand the demands of football

over a longer period. Mosaicplasty is a treatment with transplantation of fully developed osteochondral plugs into drill holes in the defect [7]. The technique is limited to smaller defects since there are not so many plugs that can be harvested from the articular surfaces before causing too much damage to the joint.

Autologous chondrocyte transplantation (ACT) is indicated for symptomatic, full-thickness, chondral and osteochondral lesions [including osteochondritis dissecans (OCD)] of the knee with an area between 1 to 2 and 16 cm (Fig. 1). An initial arthroscopy is made, and the cartilage lesion is evaluated and documented regarding size, depth, location, containment, and accessibility for transplantation (Fig. 2). Slices of cartilage are harvested from a minor load-bearing area in the knee, the most common being the proximal medial edge of the femoral trochlea. From the cartilage specimens, chondrocytes are isolated and cultured for a minimum of 2 weeks.

The joint is opened, and the arthrotomy is adjusted so the lesion is accessible (Fig. 3a). The patella may have to be dislocated. All damaged and undermined cartilage is radically excised, and the defect is carefully debrided to vertical edges of healthy cartilage. Care must be taken not to cause any bleeding from the subchondral bone. A periosteal flap, harvested from the upper medial tibia, is placed on top of the defect, with the cambium layer facing the

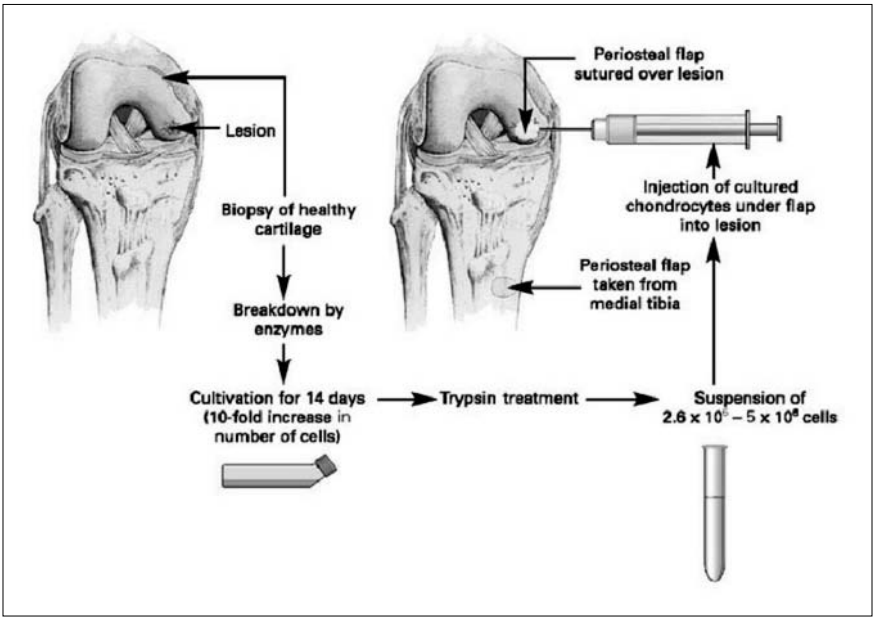

Fig. 1. Schematic drawing of autologous chondrocyte transplantation

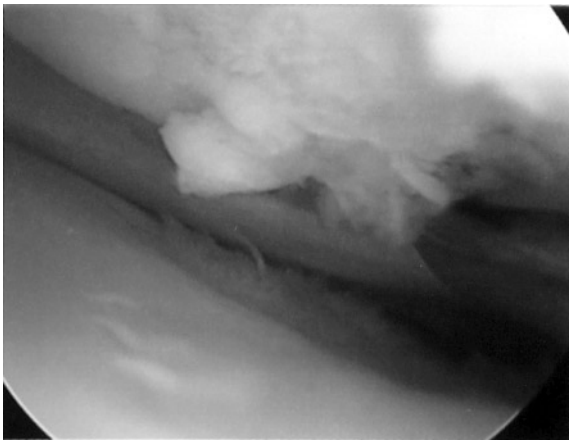

Fig. 2. Arthroscopic assessment of chondral lesion in the knee

defect. The flap is sutured to the cartilage edges with resorbable, interrupted sutures, and the intervals between the sutures are sealed with fibrin glue (Fig. 3b). After checking that the periosteal cover is watertight, the chondrocytes are injected into the defect and the last opening is closed. The arthrotomy is closed in layers.

If there is an instability or malalignment of the knee, it is imperative to surgically treat this ailment either before or at the same time as the ACT. If not treated, the chances for a successful outcome are greatly reduced. If the meniscus has previously been totally removed, a meniscal allograft should be considered.

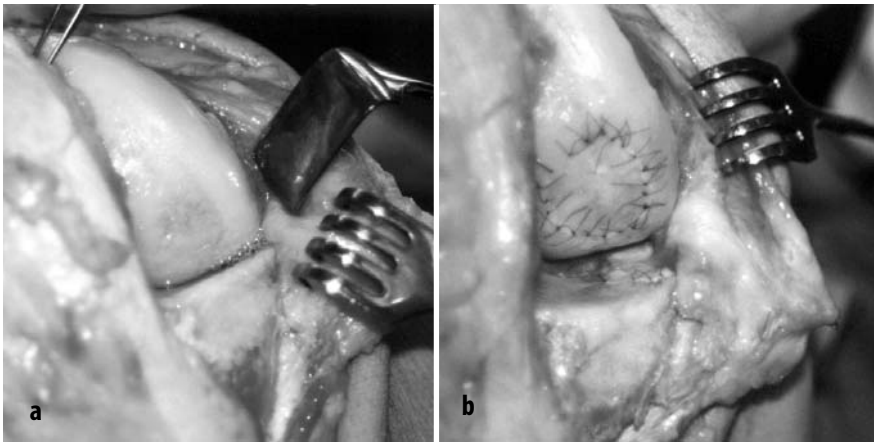

Fig. 3a, b. Chondral lesion before (**a**) and after (**b**) treatment with autologous chondrocyte transplantation

Postoperative Treatment and Rehabilitation

Postoperatively, antibiotics are given for the first 48 h, and intermittent continuous passive motion is started 6–8 h after surgery. Weight bearing is limited to 20 kg for the first 6 weeks, then gradually increased to full weight bearing in the following 6 weeks. The rehabilitation following ACT is long and focuses mainly on functional training and motion training. Return to football is judged on an individual level. Clinical follow-up, including strength and endurance tests as well as arthroscopic evaluation and indentation testing of the stiffness of the repair tissue, are important before allowing the player to go back to football training. MRI enhanced with gadolinium contrast may be useful for assessing the healing. The player starts with individual training and then gradually goes back to individual football training and then to competitive training and matches (Fig. 4).

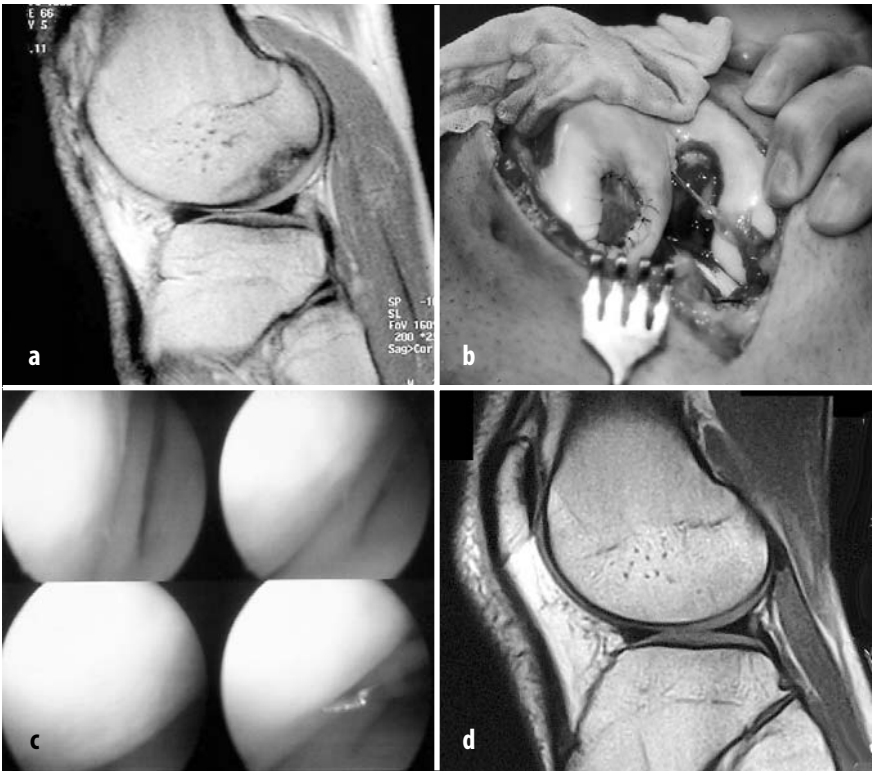

Fig. 4a-d. **a** Magnetic resonance imaging (MRI) showing osteochondritis dissecans. **b** Same lesion after transplantation of autologous chondrocytes. Second-look arthroscopy at 4 years (**c**) and MRI at 9 years (**d**) show complete healing of defect. The player was able to return to international-level football

Results

While other treatments often have failed after 2–4 years, ACT has proven to maintain a good result, even at a long-term follow-up. A recent study comparing osteochondral autologous transplantation with microfracture showed a higher return to sports at the pre-injury level for the osteochondral grafting group compared with only 52% of those treated with microfracture [8]. A study by Mithöfer et al. showed decrease in the functional outcome 24 months after treatment of symptomatic cartilage lesions in the knee with the microfracture technique. Only 44% of the patients could go back to high-impact activities such as football [9]. In a Swedish study of the first 61 patients treated with ACT on femur or patella, 50 were clinically considered good or excellent at a 2-year follow-up. At a second follow-up of the same cohort 5–11 years after surgery, 51 patients were still good or excellent, no patient had deteriorated, and the failures occurred before the first follow-up [10].

Follow-up 2–9 years postoperative of the first 101 consecutive patients with chondral or osteochondral lesions of the knee treated with ACT showed a 77% good to excellent result overall, and 89–96% in OCD and isolated femoral condyle lesions. According to the Tegner/Wallgren score, the patients were also able to have an active life, including football. The modified Cincinnati score was average 9 out of 10, meaning they could return to high-level sports, including football. The 2-year follow-up results seem to be an indicator for the long-term result [11].

In another study by Mithöfer et al., 45 players treated with ACT were followed for a mean of 40 months. Players younger than 26 years, high-skill-level players, and players operated within 12 months after trauma returned to pre-injury level in 83% of cases. Time to return from surgery to football was 12–18 months [12].

Using ACT with scaffolds such as Hyalograft and collagen membranes (MACI) have recently reported promising short-term results but have still to be evaluated for longer follow-up and return to football [13–16].

Conclusion

ACT is a treatment for symptomatic, full-thickness, chondral and osteochondral lesions of the knee, which is able to produce a hyaline-like repair tissue and restore knee function. It is possible to return to football and other high-level competitive sports; 83% of players could return to football if the operation was within 12 months after injury. If the player can comply with the rehabilitation period of 12–15 months, it seems that ACT as the primary treatment gives best options for return to football with durable results.

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Patellofemoral Problems

Alfredo Schiavone Panni, Mario Tartarone, Alessandro Antonio Patricola, Daniele Santaiti

Introduction

Soccer is performed by men and women, adults, and children and at different levels of expertise. It is estimated that almost 25 million people participate annually [1]. Soccer injuries increase in frequency as the age of the participant increases, and several intrinsic and extrinsic factors play a part. Basically, injuries in the soccer player can occur anywhere in the body like in every sport, but the lower extremities, more specifically the knee and ankle, are the most injured parts [2–4].

Football is a contact sport, and patellofemoral (PF) problems may be related to a trauma or overuse; both are common PF injuries in recreational and competitive soccer players. Pivoting actions combined with kicking and contact with other players leave knees extremely vulnerable to acute injuries. Most common traumatic injuries of PF joints are: acute patellar subluxation or dislocation and, rarely, traumatic osteochondral fracture of the patella [5]. Overuse injuries develop when repetitive stress to bone and musculo-tendinous structures damages tissue at a greater rate than that at which the body can repair them. The pathophysiology of these disorders is unclear but may be related to many factors. A combination of extrinsic factors, such as training errors and environmental factors; and intrinsic or anatomical factors, such as inferior limb alignment and ligamentous laxity, predispose athletes to develop overuse injuries [6]. "PF disorders" is a global term for syndromes that arise from the extensor mechanism (quadriceps muscle, patella, patellar tendon) and its surrounding soft tissue attachments. As we shall see elsewhere in the text, it is useful to separate patellar instability from PF pain syndrome.

Biomechanics of the Patellofemoral Joint

The articular cartilage of the patella is the thickest in the body, a testament to the high forces imparted on it; it can reach 7 mm in the central portion of the patella. The articular surface of the patella features facets that vary in size, orientation, and magnitude from person to person [7]. PF cartilage is similar to any human articular cartilage to the extent that it can be considered a biphasic material, with a freely flowing fluid phase and a porous-permeable, fibre-reinforced solid phase [8]. However, patellar cartilage is more permeable and more pliable than other cartilage, even that of its mating surface on the trochlea [9, 10] and insensate [11].

Patella increases the mechanical advantage of extensor muscles by transmitting forces across the knee at a greater distance (moment) from axis of rotation [12]; in full flexion, when the patella is entirely in the inter-condylar notch, it increases the lever arm of quadriceps by only 10%. As the knee starts to come into extension, the patella's contribution increases until 45° of flexion, at which the patella lengthens the lever arm by 30%.

Another important biomechanical function is the transmission of tensile forces; in fact, at nearly full bend, tension in the extensor mechanism has risen to nearly 150% of force passing through tibiofemoral joints; this explains the need for the large area of thick articular cartilage on normal the patella and the femoral condyle.

The patella is subjected to complex loading, and the maximum contact area is at 45° when both the central and the medial and lateral facets are in contact with the sulcus. With the knee in extension position, it transmits almost all of force of quadriceps contraction and thus is loaded primarily in tension. With the knee flexion, however, its posterior surface contacts the distal aspect of femur and it is subjected to compressive forces. The PF joint is considered to be one of the highest loaded musculo-skeletal components in the human body [12]. Joint reaction forces that are created within the PF joint in compression and tension with normal activities of daily living are on the order of multiples of body weight [13]. These high loads have been estimated from 3.3 times body weight with activities such as climbing up or down stairs, to 7.6 [14] times body weight with squatting, and up to 20 times or more body weight with jumping activities [15].

During a 90-min game, soccer player is required to perform different explosive burst of activity, including jumping, kicking, tackling, turning, sprinting, and sustaining forceful contractions to maintain balance and control of the ball. Within this explosive activity, the player runs about 10 km in a match. Kicking a ball is associated with a high knee-joint velocity [16] that, combined with repetitive pivoting deceleration can place extreme stress on the PF articular cartilage.

Patellofemoral Injuries

Acute and overuse injuries to the knee are seen as a result of participation in football activity. Patella subluxation, dislocation or tracking abnormalities can occur rarely as a result of mechanical predisposition as well as direct or indirect trauma to the knee. Cumulative microtrauma or overuse can lead to PF disorders.

Patellar instability

Patellar instability represents a patella that has normal or abnormal alignment in the femoral groove but is displaced by internal or external forces. This can result from lack of bony restraining forces, as in congenital hypoplasia of the PF joint; laxity of the surrounding tissues from recurrent trauma; and/or a hyper-laxity syndrome, such as Ehlers-Danlos syndrome. Subluxation in this instance is chronic malalignment of the kneecap out of its central position in the trochlear groove without frank dislocation.

Our inability to fully understand and document PF relationships through an arc of active motion suggests that PF pain syndrome (PFPS) without radiographic malalignment may represent subtle malalignment that is not detectable with current imaging techniques.

Traumatic Subluxation/Dislocation

A dislocated patella is typically the result of sudden changes in direction while running or jumping. Any force that causes the knee joint to rotate may displace the patella from its natural position. Injuries may also occur from a direct blow to the knee, but in very rare cases, the patella can dislocate after a powerful muscle contraction by the strong pull of the quadriceps muscles. An isolated study [17], supported by many physicians' personal impressions, reports that patellar dislocations recur more frequently in female athletes, but most studies on acute patellar dislocations, however, continue to show a male preponderance. It is difficult to interpret these findings because most studies that report acute patellar dislocations in men are not prevalence studies and date from a time when men constituted a much larger majority of athletes than they do now. Additionally, the studies do not classify dislocations according to disruption of soft tissue ligament restraints or the character of the individual's bony anatomy, which are likely prognostic indicators.

Adolescent players have a higher risk of patellar subluxation/dislocation than professional soccer players because of less training and balance muscular control and because of relatively hypotrophy of the quadriceps muscle, the most important active stabiliser of the PF joint. Increased Q angle (an angle

between the longitudinal axis of the femoral and tibial shafts), hypoplastic vastus medialis obliquus (VMO), knee recurvatum, and gender differences are advocated as co-factors of patellar dislocations, but further studies are needed to determine it.

Diagnosis is based on clinical and anamnestic findings. The patella may slip outwards and stay there, in which case the knee will lock and the player will not be able to straighten it. Most of the time, the patella will reduce itself spontaneously but sometimes will require manual reduction. Players who have acute patellar dislocation generally have an episode of instability and localised tenderness along the medial extensor retinaculum or possibly at the adductor tubercle, which is the origin of the medial PF ligament. Also, the patient has localised tenderness along the peripheral edge of the lateral femoral condyle where impaction from the patella occurs with flexion of the knee. An effusion is often present. Radiographs should be obtained since up 20% of patients develop an osteochondral loose body secondary to patella dislocation [18–21].

Patellar Osteochondral Fracture

Osteochondral fractures complicate approximately 5% of all acute patellar dislocations [18]. This fracture will usually occur at the medial side of the patella or, less frequently, on the lateral margin of the lateral femoral condyle [19–21]. Osteochondral fracture of the patella or the femoral condyle may occur as the patella slides tangentially over the surface of the lateral femoral condyle with the knee in the flexed position, as in landing from a jump or pivoting movement, or it could be secondary to a direct blow trauma to the frontal region of the knee. In both cases, the characteristic anatomo-pathological finding is the complete separation of the uncalcified articular cartilage from the calcified cartilage of the PF joint; in very rare cases, a direct high-energy trauma can cause the complete rupture of the patella, which has to be confirmed by an x-ray examination.

The player suffering of osteochondral fracture complains of pain, effusion with crepitus, and joint-line tenderness. The preoperative magnetic resonance (MR) image will show chondral lesions; traditional x-ray exam can, eventually, show bone fragment on the supra-patellar pouch. In this case, arthroscopic treatment is required for removal the loose body and to debride to a stable margin the chondral lesion.

Patellofemoral Pain Syndrome

PFPS is pain in the PF joint with or without documented instability, which seems to be caused by damage to the under-surface of the patella or PF articulating surfaces (chondromalacia) (Fig. 1), modification of homeostasis [22],

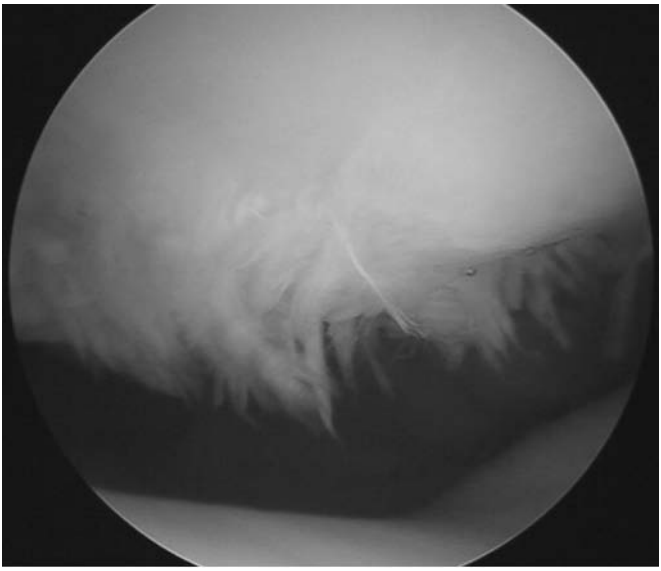

Fig. 1. Patellar chondromalacia of a 32-year-old player

or neural [23] or chemical factors [24]. The etiopathogenesis of PFPS is multifactorial [25, 26]. Causes include overuse and overload, biomechanical problems, and muscular dysfunction. Because bending the knee increases pressure between the patella and its various points of contact with the femur, PFPS is often classified as an overuse injury; however, a more appropriate term may be "overload", because the syndrome can also affect inactive patients.

Players with PF pain represent an important challenge to the orthopaedic surgeon because no single explanation or therapeutic approach has fully clarified this problem yet. Repeated weight-bearing impact may be a contributing factor, particularly in soccer players involved in activities such as running and jumping where as much as seven times one's body weight may be transmitted through the knee. Knee injuries, such as a direct blow to the front of the knee, are significant contributor factors.

No single biomechanical factor has been identified as a primary cause of PF pain although many have been studied: pes planus, pes cavus, Q angle, and muscular causes. Many authors believe that a large Q angle is a predisposing factor for PF pain even if other authors question this concept [27, 28]. Furthermore, normal Q angles vary from 10° to 22°, depending on the study, and measurements of the Q angle in the same patient vary from clinician to clinician and from the supine to the standing position.

Chondromalacia is a degenerative condition of the articular surface of patella. It is not strongly correlated with patellar pain, but pain occurs as the cartilage becomes soft and begins to break down and is no longer able to distribute pressure evenly along the back of the patella. It is probable that the concept [29] of pressure or friction in considerable flexion, such as in squatting, and of compression of the patella over the femoral shaft during quadriceps contraction, comes closest to explaining the aetiology of chondromalacia.

In football players, the amount of potential damage imparted to articular cartilage during exercise is related to the magnitude of the shear and compressive stresses absorbed by the articular surface [30]. Malalignment of the lower extremity, femoral anteversion, lateral tibial torsion, tibia vara, genu varum or valgum, subtalar varus, and excessive pronation are frequently cited as pre-disposing to knee extensor mechanism overuse injuries. Thus, PFPS can be further classified as with or without malalignment on axial x-ray. Malalignment is an abnormal relationship between the patella and the trochlear groove that transmits unusual force to the PF joint, causing pain. This condition is particularly frequent in adolescent players and is often combined with muscle hypotrophy and patellar instability. At this time, PFPS without radiographic malalignment is the most widespread and the most difficult form of PFPS to treat.

The complex causes of PF disorders are most effectively identified through a systematic evaluation of the player's lower-extremity alignment, patellar mobility, muscle flexibility, strength, and an assessment of soft tissue and articular pain. By combining information from such an exam with a careful history and appropriate radiographic studies [dynamic computed tomography (CT) scan, CT measurement of tibial tubercle to trochlea groove (TT-TG), MR imaging] (Figs. 2, 3), the physician can make a specific diagnosis.

Treatment of Patellofemoral disorders

Acute Dislocation

There are still controversies regarding indications and timing for surgical treatment of acute dislocation. In general, after immobilisation in a brace at 30° of knee flexion for at least 2 weeks, a rehabilitation programme for lower-extremity strength and function should be started. Quadriceps strengthening is a universal recommendation for athletes with PF problems. It should initially avoid exercise in the arcs of motion found to be painful during articular compression and should gradually increase.

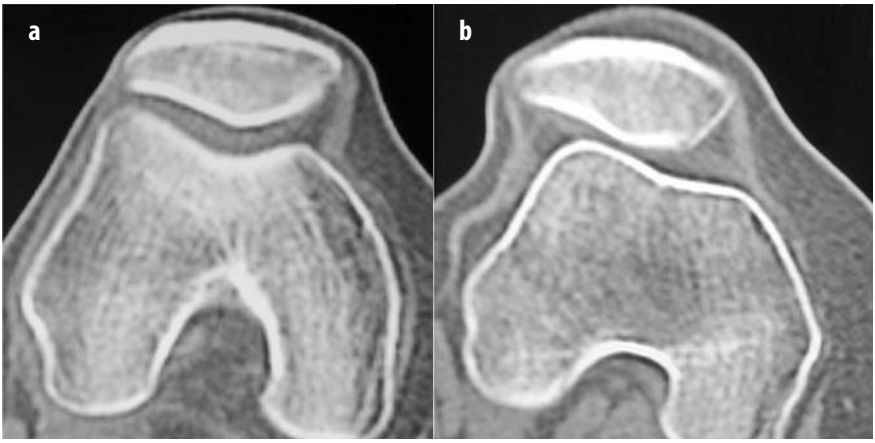

Fig. 2a, b. CT scan of patellofemoral joint without (**a**) and with (**b**) quadriceps contraction is very important to evaluate the degree of patellar instability

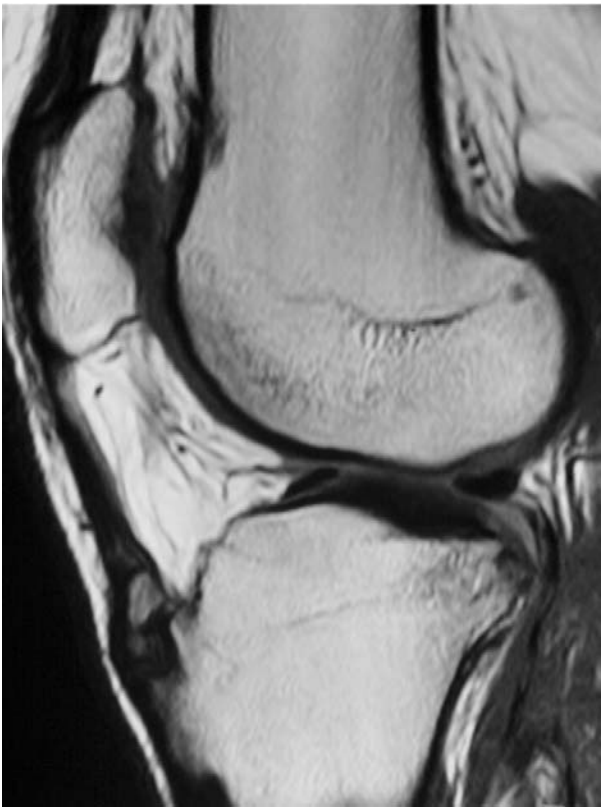

Fig. 3. MRI is the most appropriate exam to evaluate chondral pathology

The advantages of closed- versus open-chain exercises are still debated [30]. Steinkamp et al. found that closed-chain knee extension (leg press) generated less PF joint reaction than open-chain knee extension from 45° to full extension of the knee [31]. As rehabilitation progresses and pain decreases, the player should also include sport exercise in the programme. In case of recurrent dislocation or severe instability, a surgical procedure to stabilise the patella is required.

Historically, the advancement of the VMO, portion of the quadriceps muscle, or proximal and distal re-alignment alone or combined were advocated in order to prevent lateral patellar luxation. A modern approach is to reestablish a medial soft tissue restraint by repairing or re-constructing the medial PF ligament (MPFL). The clinical significance of the MPFL, in fact, has been demonstrated in a variety of recent scientific publications [32–34] where it is believed to be the essential ligament to be restored to a suitable tension or length after acute lateral patella dislocation.

Patellofemoral Pain Syndrome

Non-surgical management continues to be the mainstay of treatment for PFPS. Historically, this has involved strengthening the quadriceps muscle, in particular, the VMO. Currently, a more global approach to optimising function of the lower-extremity kinematic chain is advocated. This can include optimising the strength of the pelvifemoral musculature to help control limb alignment and rotation, balance between quadriceps and hamstring muscle strength, and balance between individual components of the quadriceps muscle bellies, in particular, the medial and lateral dynamic stabilisers of the patella.

An alternative approach would be to recognise that femoral rotation needs to be controlled underneath the patella, and this is largely controlled by the pelvifemoral musculature, in particular, the muscles that control hip rotation. Therefore, an attempt to maximise strength of the pelvifemoral musculature, including hip extensors and hip abductors, is emphasised. In addition, an anteriorly rotated or hemi-rotated pelvis is a common postural habit of many people with PFPS. This can aggravate PF instability by posturing the femur in internal rotation (which is a coupled motion associated with an anteriorly tilted pelvis) and placing the pelvifemoral musculature in a non-optimised biomechanical position.

Knee activity should be reduced, at least relatively, because the theory that PF pain is an overuse and overload syndrome has merit. A relatively inactive patient can benefit from simply strengthening the leg periodically, as needed. If the patient is a football player who insists on continuing the same rigorous activity, swimming (avoiding breast-stroke style) or another non-impact aer-

obic activity is a reasonable recommendation. Ice is the safest anti-inflammatory medication, but its successful use requires discipline. Applying ice for 10–20 min after sport activity is reasonable. Patients with PFPS have not been conclusively shown to benefit from non-steroidal anti-inflammatory drugs (NSAIDs).

The use of knee sleeves and braces in patients with PF pain remains controversial, and their use should not be considered a substitute for exercise. PF braces are an inexpensive, subjectively helpful component of PFPS. Their mechanism of action remains unclear, but most appear to improve patellar tracking through a medially directed force [35]. Changes in regional temperature, neurosensory feedback, or circulation may also contribute to their effects. Overall, PF braces should be used in conjunction with a comprehensive knee rehabilitation programme that includes strengthening, flexibility and technique improvements.

Patella taping, or the McConnell tape technique [36], was originally reported to have a high success rate in reducing PF pain, but subsequent researchers have been unable to reproduce the results of McConnell's original study [37, 38]. However, it is the opinion of most physiotherapists treating PF pain and instability that patella bracing or taping can be helpful adjuvants to a mainstay of PF rehabilitation.

The objective of surgical treatment is to correct the malalignment, if present, and to treat the articular cartilage damage. Damaged articular cartilage can be repaired only partially, as normal hyaline articular cartilage cannot be replaced or reconstructed as yet. Arthroscopic *débridement* and shaving of degenerated cartilage is of unclear benefit while the electrothermal chondroplasty seems to cause significant thermal damage [39, 40] and an unacceptable level of irreversible chondrocyte injury. The microfracture repair technique [41] generates fibrocartilage, and more advanced autologous chondrocyte implantation has the potential to re-surface defects in the patella with hyaline-like tissue.

Conclusion

Knee pain in contact-sport athletes is perhaps the most common complaint to confront trainers and physicians. Chondral injuries of PF joint are believed to occur through two distinct mechanisms. Most commonly, they arise through abrasive wear, which results in superficial fibrillation and can be asymptomatic until erosion progresses to subchondral bone. The second type occurs because of disruption of the deep cartilage ultrastructure by large-shear forces, and it is often painful and function limiting.

Traumatic PF injuries are rare in soccer practice, and they occur mainly

during tackling and jumping. The incidence is higher in teen players and at the lower senior series because of their lower grade of training. PFPS with or without degenerative changes in PF articular cartilage is a common overload condition both in division and series players. If a cartilage lesion is present, arthroscopic *débridement* and microfracture technique combined with a specific rehabilitation programme can offer functional and subjective improvement in athletes that can functionally meet the demand of the sport, but we must inform athletes that other procedures may be indicated in the future. Increased training, as muscle strength and co-ordination, causes a reduction in traumatic injuries while overuse injuries remain constant with a progressive pattern.

In conclusion, the diagnosis and treatment of PF problems still remain the greatest enigma for the sport medical physician and orthopaedic surgeon.

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Footballer's Arthritic Knee

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Introduction

The direct correlation between competitive activity and early osteoarthritis is an assumption held by many individuals, medical authors, and the sport community [1, 2]. Although it has been established in the hip and ankle joints of football players [3, 4], it seems that it cannot be generalized to the non-traumatic knee. Klunder et al. [3] studied 57 retired European football players and a corresponding control group; they found a positive correlation between the development of osteoarthritis of the hip and football participation but no such relationship for the knee.

Football is a sport that puts maximal demands on the knee. The risk of trauma to the knee of a football player is 1.3 per 1,000 exposures, with a particular risk of rupture of the anterior cruciate ligament (ACL) between 0.31 and 0.87 [5]. These traumas and the lesions that result from them can be responsible for the evolution of arthritis [we exclude true articular fractures (patellar or lateral plateau), which are specific problems].

Osteoarthritis Definition

The rheumatologist, the radiologist, and the arthroscopist do not share the same definition of osteoarthritis. Superficial chondral lesions seen through the arthroscope are not osteoarthritis. Osteoarthritis is present when there is wearing away of the two joint surfaces (a mirror lesion) with abrasion of the cartilage and, at least at one site, the subchondral bone is exposed on the two joint surfaces [6].

These lesions always follow a stage of pre-osteoarthritis. In this condition, there are mirror chondral lesions, but the subchondral bone is not exposed and joint-space narrowing is incomplete. An isolated unipolar lesion (traumatic, osteochondritis) is not osteoarthritis. This type of lesion may lead to

osteoarthritis, but the delay between a unipolar chondral lesion and osteoarthritis is often more than 20 years.

Osteoarthritis and Meniscus Lesion

The risk of osteoarthritis of the knee following meniscectomy is well known [7]. It increases with a reported rate of 20–40% within 30 years when compared with normal knees [8–10]. In a study of the knee joints of 81 veteran soccer players between the ages of 40 and 74 [11], all players who had undergone a meniscectomy presented with radiological signs of osteoarthritis. Even if discrete medial femorotibial remodelling can appear quickly after meniscectomy, there will be no evolution during at least 20 or 30 years. By comparison, an associated varus (or valgus) morphotype will be responsible for a hyper-pressure syndrome and faster degenerative change.

Intact versus Non-Intact ACL

A study of 91 knees in a population of soccer players who had undergone a partial meniscectomy, with an average follow-up of the 27 years [12], showed that radiological osteoarthritis was present in only 24% of the *intact ACL* group compared with 77% of the *ruptured ACL* group. Moreover, only 2% of the *intact ACL group* required operation for osteoarthritis compared with 16% of the *ruptured ACL group*. The main prognostic factor in the management of a meniscus lesion seems to be the state of the ACL [13].

Medial versus Lateral Meniscectomy

In knees with an isolated medial meniscal lesion, the prognosis is better if the patient is young, participates in sports, has a vertical tear, has no cartilage damage, and has an intact meniscal rim at the end of the meniscectomy. By comparison, age over 35 years, operative findings of medial compartment degeneration, posterior one-third resection, and meniscal wall resection all predict a poor outcome [14]. In knees with an isolated lateral meniscal lesion, a better prognosis can be predicted if the patient is young and has an intact meniscal rim at the end of the meniscectomy [15].

Clinical Implications

In the management of meniscal tear, it is essential to take into account the age and the level of the football player. In a young, professional player who presents with a medial meniscus lesion, one will propose a meniscectomy while trying to preserve the meniscal rim. Recovery will be faster than for a meniscal repair, and the risk of osteoarthritis is low at short- and middle-term fol-

low-up. In the case of a lesion of the lateral meniscus, if the player is very young (16–18 years), a lateral meniscectomy will risk shortening their football career due to a poor functional result and will also lead to a lateral femorotibial osteoarthritis in the following 10 years. One will consequently propose a meniscal repair even if the time to return to sport is longer and the risk of failure (and thus of re-intervention) is higher.

Anterior Instability and Osteoarthritis

Natural History of Osteoarthritis after ACL Rupture

After the initial accident during which the ligament was ruptured, the functional tolerance is variable. In the case of isolated ACL rupture, if the patient (without lower-limb misalignment) keeps a low level of activity, the tolerance may be excellent. Most such patients will present no disabling osteoarthritis before a mean delay of 35 years. However, if ACL rupture is followed by a subsequent medial meniscectomy, then the duration of tolerance drops to 20 years [13].

Arthrogenic Factors

Factors that can favour the onset of osteoarthritis after an ACL rupture are not well established. Nevertheless, some authors [6, 13] have studied the mechanical factors.

Anterior Tibial Translation

Rupture of the ACL is accompanied by an abnormal anterior tibial translation at the time of contraction of the quadriceps. This translation is very clearly amplified when landing from a jump and when the quadriceps contract violently. These kinetic disturbances involve a loss of balance between rolling and gliding, with an increase in gliding. This repetitive translation–reduction generates shear stresses in articular cartilage and leads to cartilage wear. Anterior translation leads to posterior third lesions of the medial meniscus. Lesions of the medial meniscus, loosening of the posteromedial corner, and an excessive tibial slope contribute to an increase of anterior tibial translation and favour the onset of osteoarthritis [6].

Articular Lesions

Lesions of articular cartilage: These are due either to new episodes of instability (medial compartmental cartilaginous lesions) or to the pivot shift (lateral cartilaginous lesions). True osteochondral or chondral fractures are located on the medial condyle. A recent study including 33 patients with acute

ACL injuries, normal radiographs, and occult osteochondral lesions revealed by MRI (chondral fractures and bone bruise) [16] showed a cartilage thinning adjacent to the site of the initial osteochondral lesion 6 years after ACL reconstruction. Moreover, marrow signal changes persisted in 65% of patients. This suggests that the initial injury resulted in irreversible changes in the knee. Injuries causing marrow signal changes may result in an alteration in the load-bearing properties of subchondral bone, which in turn allows for changes in the overlying cartilage. We do not yet know if this signal modification will lead to arthrosis at long-term follow-up.

Less commonly, lateral chondral lesions may be observed. They occur during the pivot shift or following a previous lateral meniscectomy. These lateral lesions could explain pain and swelling, but they rarely lead to lateral compartment osteoarthritis (except in the case of lateral meniscectomy).

True articular fractures (patellar or lateral plateau) are a different problem with a worse prognosis.

Medial meniscus lesions: These are the most significant factors in the onset of osteoarthritis after ACL rupture. In the event of isolated ACL rupture, the medial meniscus plays a fundamental role in anteroposterior stability. It limits anterior tibial translation and thus shear stresses of the cartilage. Statistics show that with or without a graft of the ACL, loss of the medial meniscus leads in the medium term to medial femorotibial osteoarthritis [6].

Posterolateral Lesions

Associated injury of the lateral collateral ligament (LCL) and/or the popliteus complex are uncommon in ACL rupture (5%). If this diagnosis is missed, however, the following laxity allows deviation of the knee in varus when walking or running. It is a very significant factor in the progression of medial femorotibial osteoarthritis.

Genu Varum

Varus knee is not an arthrogenic factor in itself. However, in the event of meniscus lesion, genu varum and the tibial slope in the sagittal plane can potentiate the onset of osteoarthritis.

Radiological Aspect

We want to emphasize the particular aspect and specificities of osteoarthritis following ACL insufficiency. The development of osteoarthritis takes place in two ways. The most common is one in which there is a medial compartmental osteoarthritis with deviation of the knee into varus. Less frequently, bicompartmental total osteoarthritis is seen without deformity in the frontal plane. Isolated lateral compartmental osteoarthritis is rare. Osteoarthritis due to anterior cruciate laxity presents four characteristic signs:

Tibiofemoral remodeling: It is characterized by osteophytosis developing on the femoral condyles and on the tibial plateau associated with some degree of flattening of the condyles. The feature that is characteristic of anterior cruciate laxity is its localisation in both compartments.
Osteophytes in the intercondylar notch: The tibial spines develop a classic hooked appearance (Fig. 1). A view of the intercondylar notch shows that this tibial osteophytosis is accompanied by osteophyte formation on the inner aspects of both femoral condyles, tending to narrow the intercondylar notch.

Posterior tibial osteophytosis: This can best be seen on lateral radiographs. The osteophyte is a horizontal one that elongates the medial tibial plateau posteriorly. This posterior osteophyte is recognized on lateral radiographs in monopodal stance with the knee at 30° of flexion. The early narrowing of the joint space in the posterior part of the medial tibial plateau seems to be due to an exaggeration of the concavity of the plateau by the posterior tibial osteophyte. In primary osteoarthritis not associated with ACL insufficiency, the osteophyte has a very different vertical disposition.

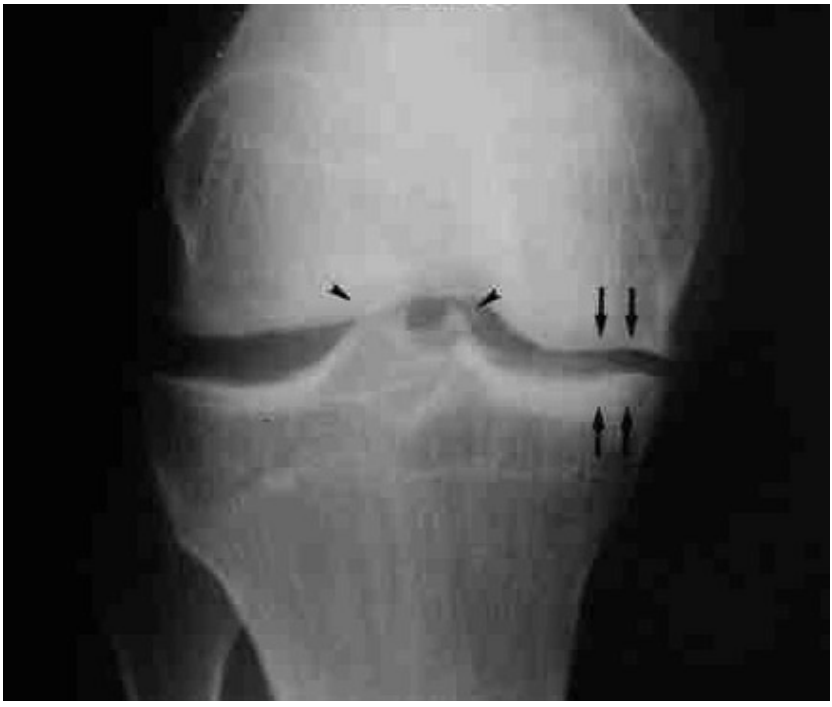

Fig. 1. Medial meniscectomy and anterior cruciate ligament (ACL) lesion with 10 years follow-up. The tibial spines have developed a classic hooked appearance. The medial compartment is at pre-arthrosis stage

Anterior translation of the tibial plateaus: This is the final characteristic element of osteoarthritis, the origin of which lies in ligamentous laxity. On the lateral radiographic view in monopodal stance, one may observe the medial femoral condyle passing into the cupped posterior part of the medial tibial plateau and appearing to rest on the posterior osteophyte.

Degenerative Changes after ACL Surgery

Does ACL reconstruction prevent the onset of osteoarthritis? Numerous clinical studies [13, 17] reported that ACL reconstruction may protect the knee from meniscal tear, particularly medial meniscal lesion. The preservation of the medial meniscus is the key point to protect the knee from osteoarthritis. A recent study [17] of the long-term result after ACL reconstruction gives us some information.

ACL Reconstruction with Intact Medial Meniscus

At 10 years follow-up (Fig. 2), 88% of patients did not develop osteoarthritis, 9% were at the stage of pre-osteoarthritis, and only 2% had femorotibial osteoarthritis.

ACL Graft and Medial Meniscectomy

If a previous medial meniscectomy had been performed before the ACL graft, 26% had osteoarthritis, 33% pre-osteoarthritis, and only 41% had a normal knee at the 10-year follow-up. If the meniscectomy was performed during ACL reconstruction, then 16% had osteoarthritis, 16% pre-osteoarthritis, and 70% a normal knee.

A recent study [17] demonstrated that ACL reconstruction associated with extra-articular tenodesis can provide good functional and radiological results at 17 years mean follow-up for patients with preserved (sound or sutured) menisci. The status of the medial meniscus at long-term follow-up appears to be the key feature determining the low rate of degenerative changes.

Significant factors in the onset of osteoarthritis after ACL reconstruction are: the status of the medial meniscus, age, residual anterior tibial translation, duration since ligament reconstruction, and sporting level after ACL reconstruction (more osteoarthritis among patients playing pivot-contact sports, such as football) [6].

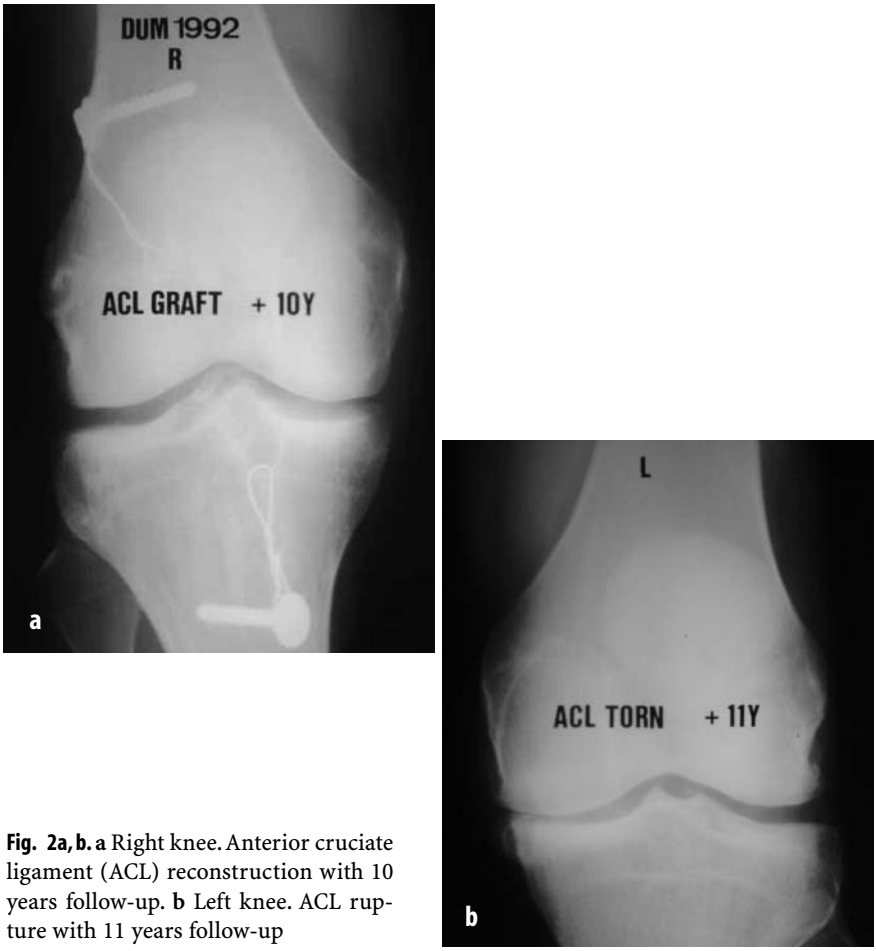

Fig. 2a, b. a Right knee. Anterior cruciate ligament (ACL) reconstruction with 10 years follow-up. **b** Left knee. ACL rupture with 11 years follow-up

Clinical Implications

In the event of rupture of the ACL, the practice of football requires a surgical correction of laxity by an intra-articular graft with or without an extra-articular tenodesis. An isolated extra-articular tenodesis (Lemaire, for example) is not recommended and, we believe, is contra-indicated if a previous preliminary medial meniscectomy has been performed. There is no place nowadays for a ligament reconstruction using synthetic materials.

The age and the competitive level of the football player must be taken into account [18]. In the case of young, non-professional players, one must consider long-term results. An ACL reconstruction (possibly associated with a meniscal repair) is recommended. In the case of a professional player near the end of his or her career, one must take into account the need for a rapid recovery; one will prefer a meniscectomy.

Time between injury and intervention may also influence the decision. If the rupture occurred in a 20-year-old player and the patient is seen at 40 years, pre-osteoarthritis can be present, modifying the indication. In case of pre-osteoarthritis with incomplete joint-space narrowing but presence of cartilage, a combined valgus high tibial osteotomy (Fig. 3) must be discussed [19]. This combined osteotomy allows the player to return to recreational sports, but only a small percentage is able, in our experience, to return to competitive sports. In a high-level, competitive soccer player, we propose an ACL graft immediately and an osteotomy later, despite the risk of early osteoarthritis.

Where there is evolved osteoarthritis with a posteromedial cupping of the tibial plateau, an ACL graft is useless; an isolated valgus high tibial osteotomy

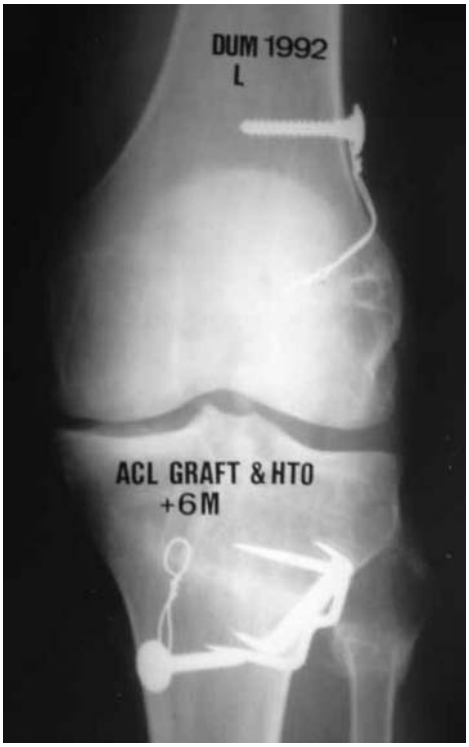

Fig. 3. Same patient, left knee. Anterior cruciate ligament (ACL) graft with high tibial valgisation osteotomy associated

without ACL reconstruction is recommended. Whatever the type of osteotomy, one must not increase the posterior tibial slope.

Posterior Instability and Arthrosis

PCL lesions frequently involve goalkeepers. They generally occur following a direct anteroposterior trauma at the level of the upper extremity of the tibia, with the knee at 90° flexion. After a rehabilitation period, the tolerance is often good, and most patients are able to return to recreational (80%) or even full competitive sport activities (64%) [20]. Over time, PCL insufficiency leads to degenerative changes in the medial femorotibial and femoropatellar joint. This progression of osteoarthritis is accelerated in the event of medial meniscectomy [21].

The majority of isolated PCL ruptures do not require surgical treatment. When there is pain or instability, one can propose a surgical reconstruction of the PCL. Recent results are encouraging. In the event of associated osteoarthritis, one may discuss a tibial osteotomy, either of valgisation in the event of a predominantly medial femorotibial lesion, or of flexion, by anterior opening, to increase the posterior tibial slope.

Bi-Cruciate Lesions, Dislocations and Osteoarthritis

The management of such a lesion is complex. Anatomical repair is the best option if the patient wants to return to sport. In the chronic phase, one can consider ligament reconstruction, but in some cases, multiple ligament injuries are best treated with an osteotomy [22].

Conclusion

It remains unproven that playing football increases the risk of osteoarthritis of the knee. However, injuries caused by playing football are without doubt an important factor in the onset of osteoarthritis. Strenuous sport activities induce the majority of detachments or ruptures of the medial meniscus, especially as a complication of ACL rupture. Direct trauma to joint surfaces can lead to osteoarthritis. In addition, indirect trauma can occur as a consequence of cruciate ligament or meniscal injuries that result in repetitive, abnormal, anterior tibial translation. Such translation generates shear forces in cartilage, leading to premature osteoarthritis.

Acknowledgments to Andrew Davis

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Leg Fractures

Karl P. Benedetto

Epidemiology of Leg Fractures

As participation in football expands, an increase in the number of injuries is inevitable. Contusions, ligament sprains, and muscular strains to the lower extremities are responsible for 50–80% of all soccer injuries. Court-Brown [1] reviewed 523 consecutive tibial fractures in a Scottish trauma centre and classified them according to their epidemiology: 22.3% were isolated tibial shaft fractures and 77% were combined with fibula fracture; 30.9% of all analysed fractures were caused by sports activity, of which 80% were documented as soccer injuries. Lenehan analysed 50 consecutive tibial shaft fractures in adult footballers treated over a 5-year period in Ireland. Although most of the fractures were classified as A injuries (AO classification) with a low incidence of complications, only 54% of all players returned to playing competitive sports[2]. Templeton documented 329 tibial diaphyseal fractures in a 54-month period. Sports injuries in general accounted for 73/329 of these fractures, and soccer was responsible for 79.5% of all sports injuries. All his patients were male, and the age range was 8–48 years. Five children under the age of 15 years were included in this series; 93.9% of the diaphyseal fractures were at the middle or junction of the middle and distal third [3].

In our database, we found 42 tibial diaphyseal fractures related to soccer injuries. In addition to this number, we had documented two stress fractures in professional soccer players in the first and second Austrian division. All patients who sustained a diaphyseal fracture during soccer were male, and their age range was 12–31 years. Four athletes were professionals and playing first- or second-division soccer league, and 38 players were amateurs or semi-professional playing regularly in a low division.

Classification of Fracture Type

All fractures were classified according to AO–ASIF classification, and for soft tissue damage, Gustilo-Anderson classification was used (Tables 1 and 2).

Table 1. Classification of isolated tibial fractures (*n*=6)

AO classification	Number
A1	-
A2	1
A3	4
B1	-
B2	1
B3	-
C1	-
C2	-
C3	-

Table 2. Fracture classification (*n*=42)

AO classification		Gustilo-Anderson classification			
	Tibia plus fibula fractures (*n*=36)	Open (*n*=3)			Closed (*n*=39)
		I	II	III	
A1	2	-	-	-	
A2	7	-	-	-	
A3	18	1	-	-	
B1	-	-	-	-	
B2	8	1	-	-	
B3	-	-	-	-	
C1	-	-	-	-	
C2	-	-	-	-	
C3	1	1	-	-	

Out of 42 fractures, 6 were isolated tibial shaft fractures: 1 was classified A2, 4 were A3, and 1 was B2. There was no C fracture according to the AO classification, and all fractures were closed. Thirty-six were diaphyseal fractures combined with fibula fracture, and all were in the middle third or at the junction of the middle to distal third. Two were classified A1, 7 A2, and 18 A3. There were 8 B2 fractures and 1 C3 fracture. According to the Gustilo-Anderson soft tissue classification, 3 were grade I open fractures: 1 A3, 1 B2, and 1 C3.

Treatment

Operative treatment of diaphyseal tibial fractures has been widely accepted in the German-speaking area over the last 30 years. Open reduction and plate fixation was a common procedure for operative treatment in the 1970s and 1980s. Development of nailing systems and especially outcome research of unreamed interlocking nailing has replaced plate fixation over the last decade. The rationale of unreamed nailing was based on the significantly lower infection rate, incidence of skin necrosis, and re-fracture following removal of implant compared with plate fixation.

Immobilisation and cast fixation for treatment of B3 fractures is widely accepted in the English speaking countries, especially in the United States. Templeton in his series treated 44/57 conservatively and reported significantly fewer complications compared with operative treatment [3]. There were 9/44 complications in conservatively treated patients: 1 re-fracture 11 months later, 3 delayed or non-union that required late operative treatment, and 4 angle deformation more than 10°. Twelve patients were treated operatively by intra-medullary nailing, and 5/9 had delayed or non-union and 2/9 deep infection, which is an unusually high complication rate. Boden reported a 53% major complication rate in 15 diaphyseal tibial fractures combined with fibula fracture; 3/15 primarily treated with cast immobilisation required late operative treatment with intramedullary nailing, and 2/15 developed lower-leg compartment syndrome requiring fasciotomy [4].

In our series of 42 acute tibial fractures, 7 patients were treated conservatively. Plaster cast immobilisation was the treatment option in isolated closed tibia fractures (A3) in patients with open physis (3/6) (Figs. 1, 2). One patient with an isolated B2 tibia fracture, and 2 patients with tibial and fibula frac-

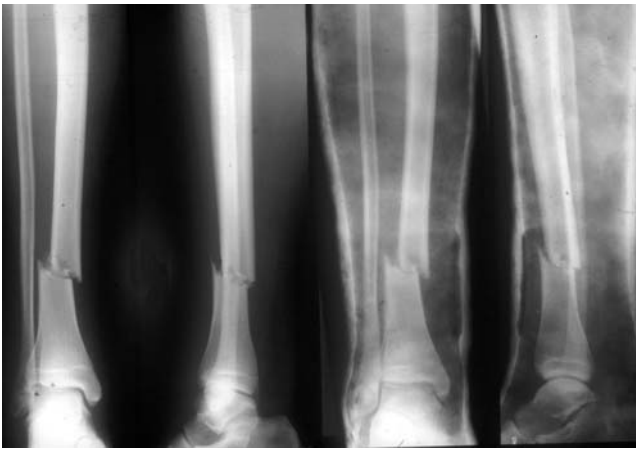

Fig. 1. Isolated tibia fracture in adolescent soccer player (A3). Conservative treatment

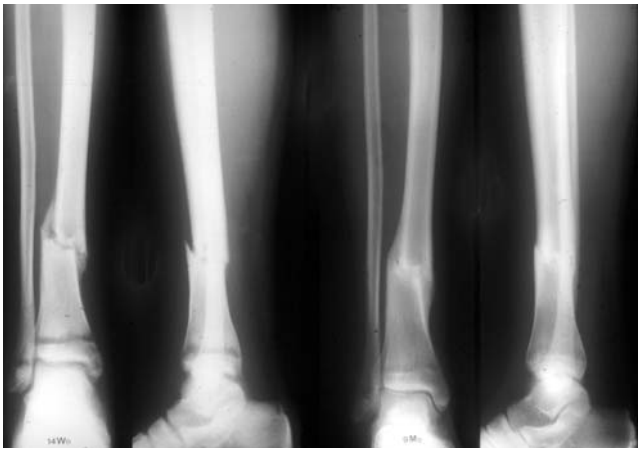

Fig. 2. Consolidation after 14 weeks solid healing, and remodelling after 9 months

ture (B2) were treated conservatively by calcaneus traction for 3 weeks followed by a long-leg plaster and cast immobilisation for 8 weeks. One patient with an A3 fracture was treated immediately by plaster cast for 12 weeks but required 1 manipulation under anaesthesia. Open reduction and plate fixation according to the AO technique with simultaneous fasciotomy of tibialis anterior compartment was performed in 8 patients. There was 1/8 delayed healing with fatigue fracture of the plate, which required revision operation during which plate removal was carried out, and reamed intra-medullary nailing was performed 4 months following primary surgery. In 27 patients, primary intra-medullary nailing was performed. An unreamed interlocking nail was used in 18 patients, and reamed nailing in 9 (Figs. 3–6). Two patients required early revision and fasciotomy due to compartment syndrome, and 2 patients with an unreamed interlocking nail required revision because of delayed healing (Figs. 7, 8). Reamed nailing and fibula osteotomy was carried out in these patients.

Re-fracture rate in the entire series was 2/42. One patient sustained re-fracture of the tibia following conservative treatment 10 weeks following cast removal due to a minor injury. One patient sustained re-fracture of the tibia 4 weeks following plate removal (Figs. 9, 10). Both patients required operative treatment and stabilisation by reamed nailing. Return to sports required rehabilitation of 18–52 weeks; of 3 professional players, one did not return to his previous competitive level in the first division. Two players were treated for a stress fracture of the tibia in the middle third. Diagnosis was made by x-ray and confirmed by MRI. Initial treatment was complete rest and immobilisation for 4 weeks in both players. One returned to his previous level of activity following a prolonged rehabilitation program after 8 weeks. The second

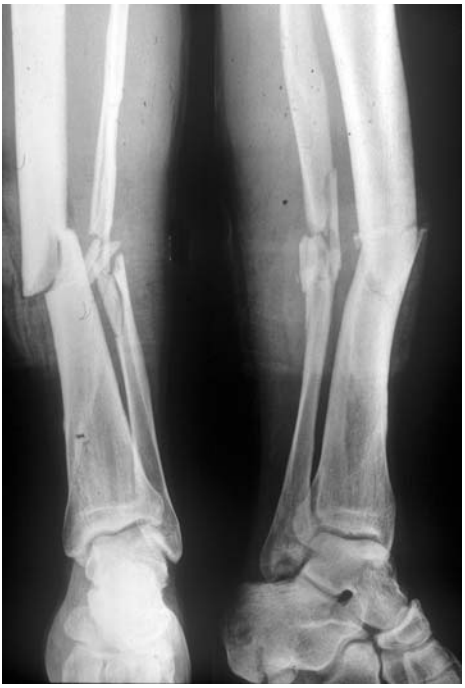

Fig. 3. Tibial shaft fracture (A2) comminuted fibula fracture

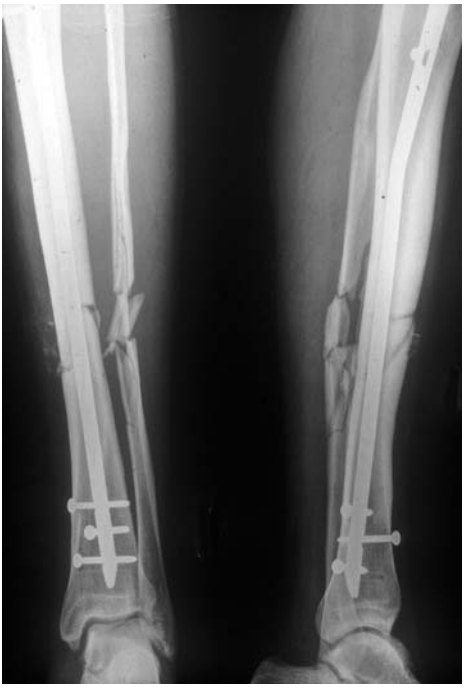

Fig. 4. Closed reduction – unreamed interlocking nail UTN

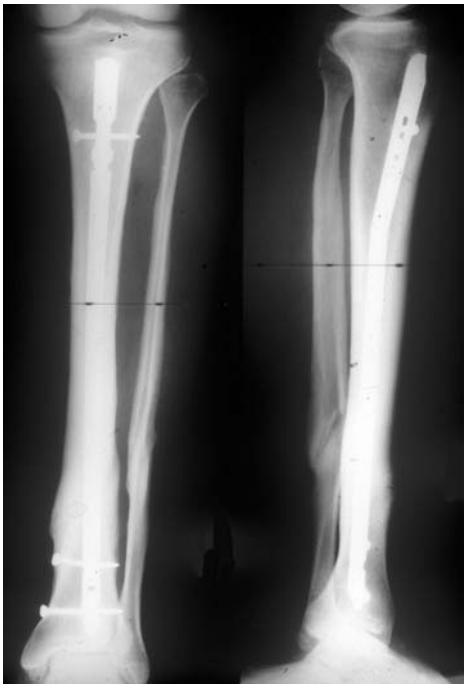

Fig. 5. Healing after 16 weeks – 1 distal interlocking screw broken

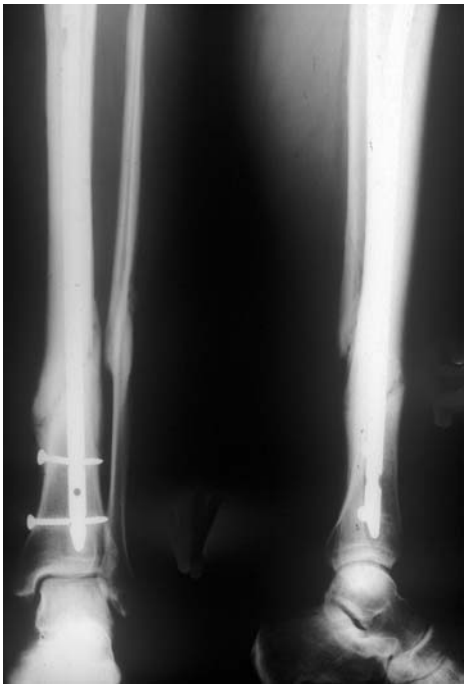

Fig. 6. X-ray before removal after 9 months

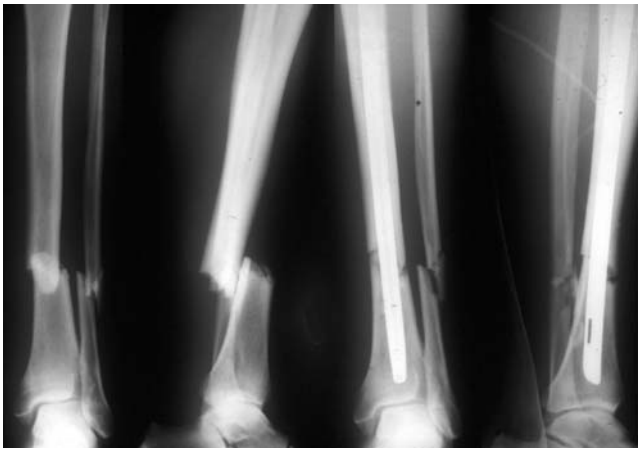

Fig. 7. Diaphyseal fracture (B2). Closed reduction – reamed AO nail + fasciotomy

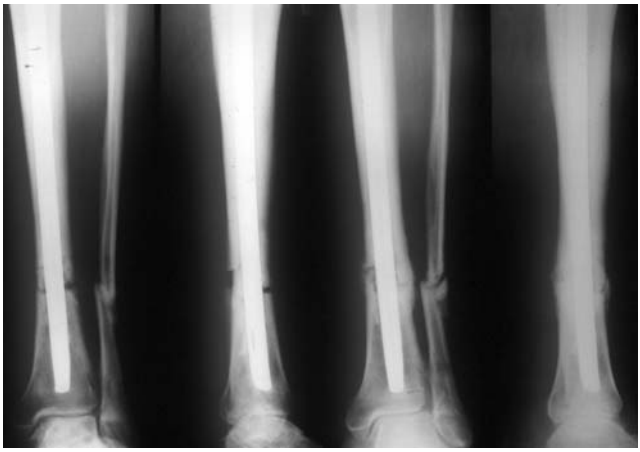

Fig. 8. Solid healing in anatomic position

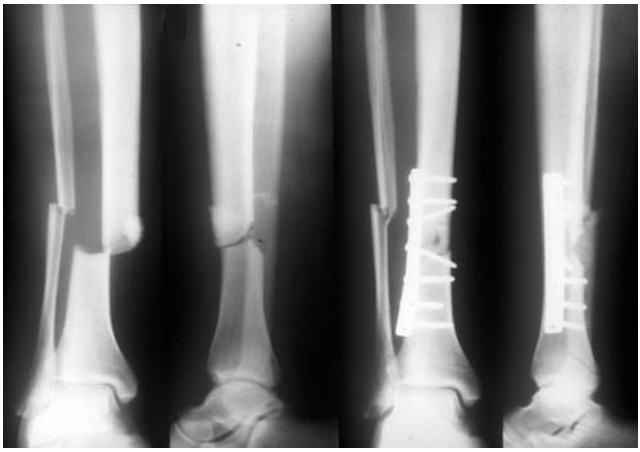

Fig. 9. Diaphyseal shaft fracture (A3) grade I, open. Open reduction, plate fixation

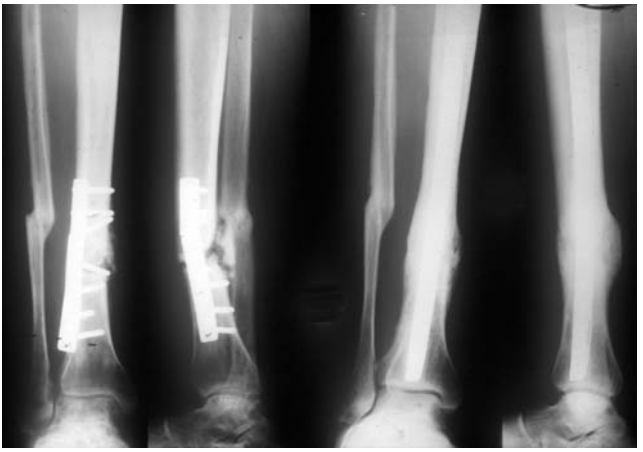

Fig. 10. Fatigue fracture of plate revision – reamed intramedullary nailing and bone grafting healing after 6 months

player required intra-medullary nailing due to persistent pain 8 weeks after diagnosis of the stress fracture.

Table 3. Complications and treatment

	Nailing	Conservative	Plate	Unreamed nail
Compartment syndrome	2/27	-	-	-
Manipulation under anaesthesia	-	1/7	-	-
Delayed union	-	-	1/8	2/18
Re-fracture	-	1	1	-

Prevention

The mechanism of kicking in soccer may be associated with the generation of high kinetic energy. Since soccer is a contact sport, the kicking leg can cause severe injuries. During miskicks or slide tackles, the energy may be transmitted to an opponent's lower leg, resulting in a fracture. Shin guards have become the only protective device for the lower leg in soccer players. Experimental studies by van Laack [5] have shown that shin guards decrease the magnitude of forces by prolonging the amount of contact time. Bir found by testing mechanical properties of commercially available shin guards that the load forces were reduced by 41–77% [6]. Francisco analysed mechanical

properties of different shin guards and found that those with compressed air were most effective [7].

Isolated or combined diaphyseal fracture of the tibia and fibula in soccer players is not a benign fracture although in most cases, they are classified as a simple fracture according to the AO classification. The complication rate following conservative as well as operative treatment is high, and time out of competitive sports is quite long in some players. Prevention is extremely important, and attention should be focused on protection equipment.

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Malleolar Fractures

Filippo Castoldi, Roberto Rossi, Antongiulio Marmotti, Rainero Del Din, Paolo Rossi

Introduction

Soccer is the most popular game in Europe. It accounts for 28.9% of exercise-related injuries. However, malleolar fractures are relatively infrequent in soccer players [1, 2]. Malleolar fractures generally result from rotational forces and rarely involve the horizontal articular surface of the distal tibia and fibula. Most foot and ankle injuries are the consequence of foul play involving direct contact with the injured ankle. Player-to-player contact has been reported to be a contributory factor in 44–74% of soccer injuries [2, 3], with a higher incidence for weight-bearing limb lesions, resulting in ankle sprains or fractures (39%), compared with non-weight-bearing limbs (23%) [2].

The immediate goals of malleolar fracture treatment are to restore overall limb alignment, reconstruct the articular surface with early reduction, and obtain early ankle mobilisation, avoiding major complications. The ability to achieve these objectives is a function of the severity of fracture and associated soft-tissue injuries. In order to determine proper treatment for these injuries, it is essential to classify the fractures: the Danis-Weber/AO and Lauge-Hansen classification systems [4, 5] based on mechanism of injury and fracture pattern are the most widely used.

Initial Evaluation

Clinical examination and radiographic evaluation form the basis of treatment for ankle fractures. A careful neurovascular examination is mandatory, and special attention should be paid to soft-tissue status, noticing wounds, ecchymosis, blistering, skin tension, and massive swelling. The standard radiographic evaluation includes anteroposterior, lateral, and mortise views (Fig. 1). Great attention must be paid to some parameters, such as tibiofibular clear space, tibiofibular overlap (Fig. 2a), medial (tibiotalar) clear space (Fig. 2b),

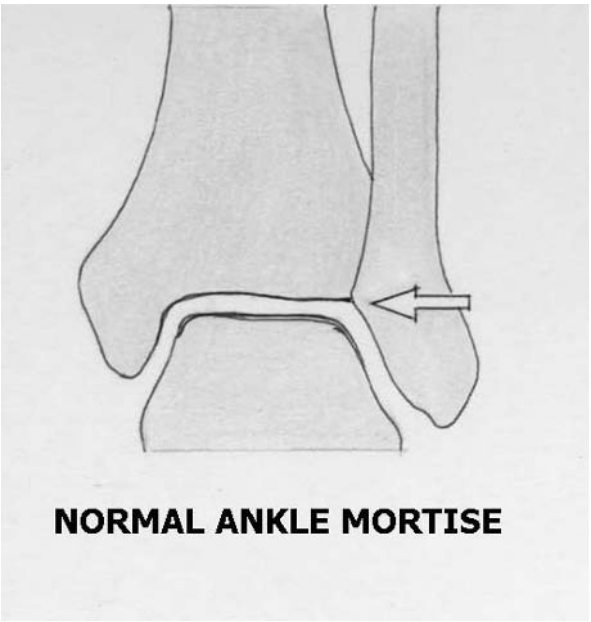


Fig. 1. Normal ankle mortise view

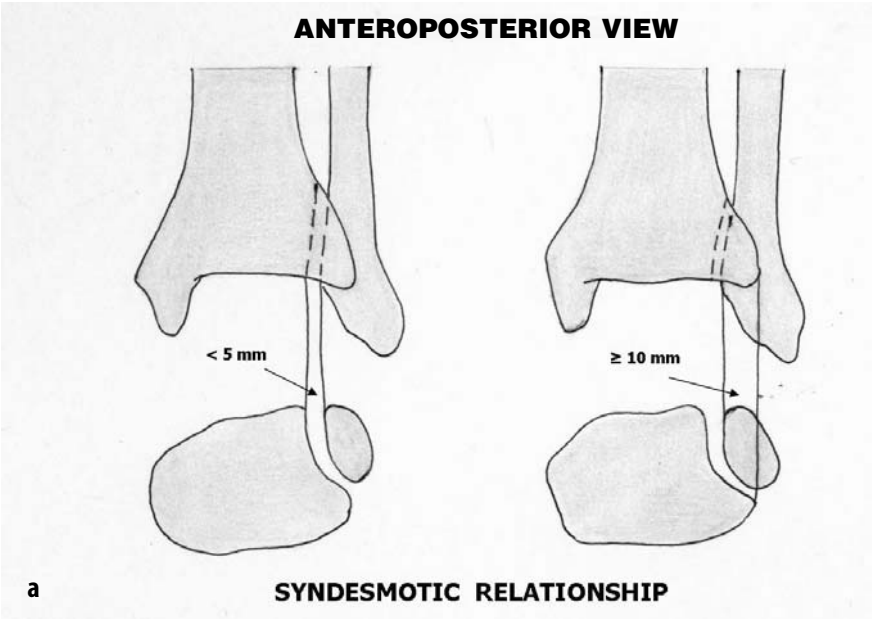
tibiofibular line, talar subluxation, and talar tilt (Fig. 2c, d). Computed tomography (CT) is required only in cases of severe displacement or impaction of the tibial weight-bearing articular surface.

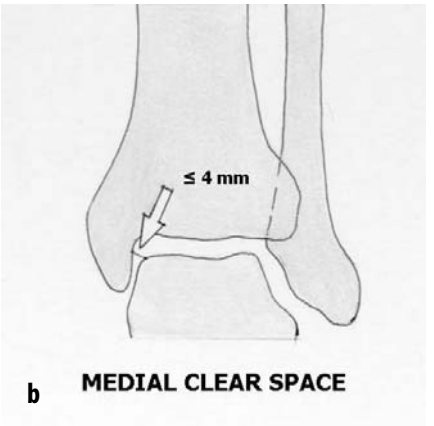
Classification

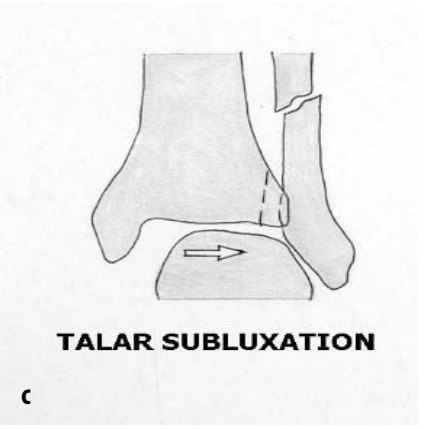
The two most widely used classification systems are the Danis-Weber/AO and the Lauge-Hansen [4, 5], the former based on the level of fibular fracture, the latter on the mechanism of injury. Both systems have poor inter-observer and intra-observer reliability. Further more, associated soft-tissue injuries are not adequately considered, and in this is their limit. A third system has been recently developed by the Orthopaedic Trauma Association (OTA). However, its reliability and usefulness in decision making and prognosis is yet to be evaluated [6].

Timing of Surgery

Many Authors have expressed the opinion that malleolar fractures can be safely treated surgically within a short, early window (from 4 to 48 h) when the swelling is from haematoma rather than oedema; but no convincing evidence exists that early surgery is safe or beneficial [7]. Although it is undoubt-







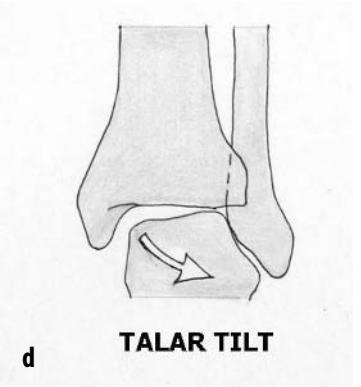


Fig. 2 a-d. **a** X-ray appearance of the syndesmotic relationship. **b** Normal medial clear space must be < 4 mm. **c, d** Talar malalignment is clear if a talar subluxation or a talar tilt is present

edly true that surgery has to be postponed in the presence of soft-tissue injuries (intra-dermal oedema, fracture blisters, subcutaneous oedema), it seems a reasonable early surgical treatment for soccer players. Indeed, the initial swelling is due generally to haematoma formation, and early open reduction internal fixation (ORIF) techniques allow primary closure without tension of the surgical wound, releasing this haematoma.

Treatment Options

The goals of surgical treatment are to reduce the joint surface, restore axial alignment, and obtain enough stability of the distal tibia to allow for early motion of the ankle joint, meanwhile avoiding complications that could severely compromise the outcome.

Isolated Malleolar Fractures

Tip avulsions or small transverse lateral malleolar fractures below the joint are treated non-operatively. In several studies, the outcome of anatomic reduction and internal fixation of lateral malleolar fractures with up to 3 mm of displacement [8] was similar to that of non-surgical treatment. This finding suggests that injuries associated with up to 3 mm of displacement can be addressed by conservative treatment, provided that anatomic reduction of the talus is achieved. In these cases, a short-leg weight-bearing cast or a functional fracture brace should be applied, usually for 4–6 weeks. However, following AO principles of early motion in fracture healing, the indication for operative treatment in soccer players is primary to obtain early ankle mobilisation, even in absence of weight bearing. Isolated medial malleolar fracture is a rare entity: It is usually associated with soft-tissue injury or other malleolar fractures. When faced with this injury, the same principles of lateral malleolar fracture treatment are applied.

Bi-Malleolar Fractures

Bi-malleolar fractures involve both peroneal and tibial malleolus or lateral malleolus with accompanying disruption of the deltoid ligament complex. Although it has been reported that 80–91% of ankle traumas in soccer players involve the lateral compartments [9, 10], it must be considered that the medial side of the foot is frequently used for passing, shooting, and tapping the ball so that the ankle is exposed to extremes of plantar flexion, dorsiflexion, inversion, or pronated foot position. Therefore, soccer players might be more subjected to medial injuries than the general sporting population.

The severity of the fractures often depends on the mode of injury. Another major determining risk factor is the weight-bearing status of the limb at the time of the incident. Hence, the fracture pattern must be understood and classified according to the mode of injury (i.e., supination–external rotation fractures) before deciding the proper treatment [2]. Surgical treatment is recommended for evident instability or articular incongruity and is less straightforward than for isolated malleolar fractures. Furthermore, because of acute and chronic instability of the ankle as a common complication of this injury, surgical repair in soccer players is mandatory.

Anatomically, medial structures have a pivotal role in ankle stability while lateral malleolus determines the talar position by means of contact with the lateral facet of the talus. During the reduction procedure, the talus follows faithfully the lateral malleolus; for this reason, in bi-malleolar fractures, it is fundamental to obtain anatomical reduction of the fibula, and in that, restoring physiological motion in both planes [11].

On the other hand, medial reconstruction restores normal coronal plane motion [11]. Injury to the superficial portion of the deltoid ligament does not always cause instability; on the contrary, a complete rupture associated with a lateral fracture severely compromises ankle stability so that surgical reconstruction is required for a better clinical result. Nevertheless, the deep deltoid ligament usually ruptures in its mid-substance, and it is difficult or impossible to fully restore. So the real amount of stability after surgical repair is not known by the surgeon unless post-operative stress x-rays are obtained [12].

For all these reasons, bi-malleolar fracture in a soccer player's ankle should be surgically addressed with reconstructive procedures and internal fixation.

Tri-Malleolar (Cotton) Fractures

Tri-malleolar fractures include a lateral malleolar fracture; a triangular fracture of the posterolateral corner of the tibial plafond, most of the time displaced; and a horizontal or oblique fracture of the medial malleolus. Sometimes, the medial malleolus may be intact with a tear of the deltoid ligament. Tri-malleolar fractures always require ORIF when more than 25% of the posterior articular surface is involved or the fracture is displaced more than 2 mm [12]. Because of the high level of fracture instability, treatment results are not as predictable as in bi-malleolar fractures.

In facing these complex injuries, particular attention must be paid to restore the length and alignment of the fibula, as it plays a major role in stabilising the talus in the ankle joint complex. Moreover, any medial compartment injury (i.e. deltoid ligament rupture) has to be properly repaired.

Injuries of the Syndesmosis

It is essential that the syndesmosis be anatomically reduced. The syndesmosis will be stable if medial and lateral ligament complexes are anatomically restored; stabilisation of the syndesmosis may be initially accomplished by repairing ruptured ligaments and by fixing the associated fractures of the fibula, avulsed tubercles, and medial malleolus [6, 12]. Whether any further fixation is necessary depends strictly on the stability of the syndesmosis once the fibular length has been restored, the fibula fixed, and the medial side reconstructed. The answer to this controversial orthopaedic dilemma can be found intra-operatively using a simple hook test or taking stress x-rays in external rotation of the ankle: Widening of medial joint space by more than 2 mm suggests residual syndesmotic instability (Fig. 3).

Fixation is usually accomplished by one or two screws inserted in the fibula 3–4 cm above the level of the ankle joint perpendicular to the tibiofibular joint, allowing primary stability for ligament healing. The syndesmotic screw (cortical or malleolar screw) is a positioning screw used to hold and not compress the syndesmosis. The foot is placed in dorsiflexion to bring the widest portion of the talus into the mortise [13–15]. There are also discussions as to whether one or two tibial cortices have to be engaged by the screw threads; drilling the lateral tibial cortex only allows loosening of the screw rather than breaking if movement occurs. Instead, if the fibular fracture is so high that its direct fixation is not possible (i.e. Maisonneuve injury), then both tibial cortices should be engaged in order to increase intrinsic stability of the construct. When to remove the screw remains controversial. We advocate removal before weight bearing to restore the physiological movement of the fibula and prevent fatigue failure of the screw.

Injuries of the Medial Collateral Ligament

An isolated lateral malleolar fracture associated with a medial collateral ligament injury is equivalent to bi-malleolar fracture. Anatomical reduction of the lateral malleolus restores the medial anatomy and allows medial ligamentous structures to heal without the need for operative repair. Surgical indication is mandatory if medial clear space is widened by more than 2 mm after reduction of the fibula or the reduction of the fibula is blocked by interposing tissues (deltoid fibres, posterior tibial tendon).

Reconstruction of the deep deltoid ligament, as stated above, could be difficult because of its transverse position under the malleolus, short length, and the difficulty in getting sutures to hold in the remnant of a ruptured ligament. Keep in mind the possibility of interposition of the deltoid ligament or others soft tissue (posterior tibialis tendon).

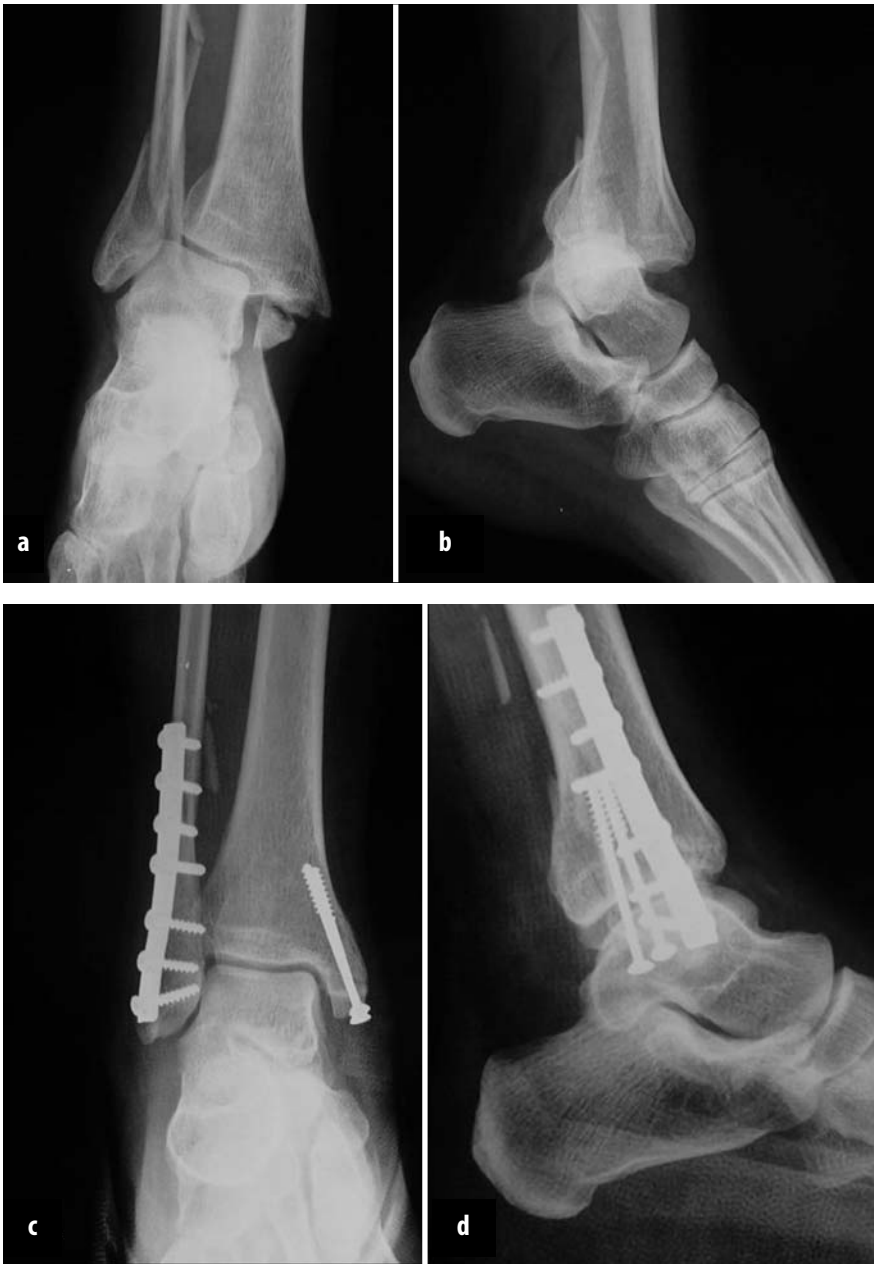


Fig. 3 a-d. a,b AP and lateral view of a tri-malleolar fracture with subluxation of the talus. **c, d** Postoperative x-rays: no screws in the syndesmosis were required

Complications

There are several complications affecting the outcome of the treatment of ankle fractures. These are in common with other articular fractures such as osteochondral fracture, malunion, non-union, wound problems (4–5% and that of secondary surgery, 5%) [6, 7], and post-surgical infection (1–2% for unstable ankle fractures). Loss of motion, degenerative arthritis (greater in the soccer population [16, 17]), development of anterior bone impingement secondary to damage of articular cartilage, and repetitive kicking of the ball, are peculiar, although not exclusive, of a soccer population. Any one of these requires an individualised treatment considering the circumstances, the personality of the fracture, and the athlete.

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Ankle Ligaments Injuries

Alberto Ventura, Albino Lanzetta

General Features

Ankle ligament injuries represent one of those pathologies considered the most common in athletes. According to Perlman, they cause from 10% to 30% of sport injuries in young athletes [1]. It is considered that the ankle area causes up to 10% of medical treatment, of which capsular ankle ligament lesions constitute 75% [2]. Certain epidemiological studies have estimated that approximately 10,000 persons are affected by sprain each day (equivalent to 27,000 capsular ankle ligament lesions a day) [3]. Athletics and jumping sports are frequent causes: Almost 25% of injuries in such sports are caused by ankle sprains [4].

As revealed by Lanzetta [5] (Table 1), of sports that cause ankle ligament injuries, basketball is said to cause 31.5% of capsular ankle ligament lesions in the Milan area while basketball, soccer, volleyball, and all combined track-and-field sports constitute 80%. Kujala [6] confirms that after basketball (31.4%) and volleyball (31.1%), soccer (20.8%) causes most cases of such lesions (Table 1). The mechanism that accounts for 85% of cases seems to be an injury in inversion [7]. According to Yeung, the ankle of the most predominantly used leg undergoes ligament injury at a frequency 2.4 times higher than the ankle of the less dominant leg [8]. That study shows that 59% of ankle ligament injuries are followed by residual symptoms such as pain, swelling, rigidity, and a sense of instability. The persistence of such symptomatic features in athletes leads to limitation of their level of athletic performance, thereby in most cases reducing their level of competitive sport activity compared with before the injury, or rather, limiting them in their individual life activities. Also, almost 40% of particularly serious cases lead to a state of chronic instability [9–11].

Table 1. Frequency of ankle ligament injuries in sports (reproduced from [5])

Sport	Percent
Basket	31.5
Soccer	19.2
Volleyball	13.7
Track and field	13.4
Gymnastic	7.9
Tennis	3.3
Skiing	0.3
Rugby	1.9
Grass hockey	1.6
Judo	0.5
Fencing	0.5
Karate	0.5
Motocross	0.5
Swimming and water polo	0.5
Hockey	0.3
Body building	0.3
Savate	0.3

Anatomy

The ankle is an unstable, articular complex, particularly when in an upright position and during walking. It is said to be constituted by components characterised by a relative incongruence represented by the so-called mortar-shaped fibular-tibial structure and talus-shaped pulley, which acts as a support for the underlying heel. Two articular structures are consequently formed – the fibular-talus-tibial articulation and the lower talus, all characterised by a condition of precarious stability and to which the ankle ligament system offers an outstanding contribution.

The ankle ligament system is subdivided into medial and lateral components: (1) The medial compartment is represented by the internal lateral ligament (ILL), or deltoid ligament; (2) the lateral compartment is represented by the external lateral ligament (ELL), which is divided into the anterior talofibular (ATFL), the calcaneofibular (CFL), and posterior talofibular (PTFL) ligaments. Moreover, the tibiofibular interosseous membrane, the ATFL and PTFL, the CFL, and the interosseous ligaments all form part of the ankle ligament structure.

In addition to these structures, we can include the control functions of the long fibular malleolus (especially during the monopodalic support) and the so-called active stabilisers, which are represented by the muscular structure and subdivided into: (1) eversion movement (brevis and longus peroneus muscles) and (2) inversion movement (anterior and posterior tibial, extensor digitorum longus, and extensor hallucis longus). The role of the receptor structure in contributing to ankle articulation is not, however, of little importance: It has to do with capsular ligament proprioceptors (mechanoceptors), which transmit important information for the neuromuscular control of articulation.

Biomechanics

The anatomic characteristics of the ankle enable us to interpret the epidemiological results that show an almost total involvement of the external section (95%) as opposed to the internal one (5%) [12]. In fact, the peroneal malleolus avoids a real medial ligament lesion when injury is in the form of a sprain, which causes a greater destructive force. Thus, the sprain first fractures the lateral malleolus rather than the deltoid ligament. The rupture of the deltoid ligament is therefore not frequent, and when it occurs, it is associated with a malleolus fracture or bi-compartmental capsular ligament lesions. In the other cases, a distractive or elongation type of lesion is noticed without true interruption of the fibres.

In the lateral compartment, the ligament structure that always and before all the others endures the injurious force application is the distal tibioperoneal articulation since the reverse movement causes an aperture of the malleolus pincers as well as the consequent distraction or rupture of both the anterior and posterior distal tibiofibular ligaments. What follows is that the injurious force successively involves the ATFL and the CFL. According to Fallat et al. [13], 85% of sprains cause rupture of the ATFL, and in 20% of such cases, rupture of the CFL takes place. It is extremely rare for rupture of the CFL only to take place [8]. Rupture of the PTFL can occur only in case of dislocation of the ankle [8]. Two explanations exist in support of the principal reaction of the ATFL towards the CFL. On the basis of studies by Attarian [14], the CFL rupture load ranges from 2 to 3.5 times as much as the ATFL (345.7 vs. 197 Newton). Moreover, every single ankle ligament is in the position to maintain articular stability at a precise angle of articular excursion or foot movement. The ATFL gets stretched when the ankle is in plantar-flexion; it is so with the CFL when in a neutral position and with the PTFL when the foot is in dorsi-flexion. Since it is well known that most sprains take place in the plantar-flexion position, it is easy to understand the motive behind the ATFL's main reaction.

Clinical and Instrumental Examination, and Classification

Treatment of capsular ankle ligament lesions, as usual, depends on a correct clinical organisation, which starts with the anamnesis data collection. It is fundamental in this phase to ascertain whether it is a question of the very first lesion or whether there have been other cases, and if so, which treatments have been carried out. Such a process enables us to get to the anamnesis framework within the classification, as affirmed by Lanzetta [5], who names three types of lesion: (1) recent lesion; (2) recent lesion over previous ones; (3) inveterate lesions or chronic instability. At present, it is preferable to consider the recent lesions over previous ones as the first level of the chronic lesions since there is a rather common opinion that recent, untreated lesions or those inconsistently treated doubtlessly lead into instability.

For this first subdivision, in order to ensure a more precise classification according to the four grades of gravity (Table 2), there should follow other, further detailed, data. In the first place, a correct objective test involving the injured area must be carried out (to detect swelling and the area of ecchymosis) as well as evaluation of the articular mobility and the execution of functional tests (anterior drawer test and talar tilt test). X-ray evaluations will then follow in both standard and dynamic projections. Dynamic x-rays, essential for correct diagnostic planning or framework [15], are carried out with the use of appropriate equipment or apparatus (Telos, or *diadistorsio*) [5] in a forced inversion position (talar tilt) [16] as well as in plantar-flexion with a forced anteropulsion of the foot (anterior drawer test). The use of other diagnostic methods can in some occasions be useful. While the CT does not seem to have the necessary capacity to identify the involved structure, the MRI is more valid and frequently used; it allows well-defined morphology of tendon, cartilage, and ligament injuries although it does not enable the degree of instability to be measured and cannot be a substitute for x-ray projections [17]. It does, however prove to be useful by confirming ligament injuries and detecting other related pathologies.

On obtaining these data, it is possible to classify the injury into one of the 4 grades and thus evaluate the correct therapeutic treatment, taking into considering whether it is a recent injury or an instability (Table 2).

Related Lesions

Injuries associated with traumatic ankle sprain are numerous. It is necessary to consider that in almost all cases there is a chondral or osteochondral injury. Taga et al. [18] detected the presence of tibiotalar chondropathy in 85% of acute injuries and in 93% of chronic injuries. Among bone injuries,

Table 2. Classification of ankle ligament lesions (reproduced from [12])

Grade	Clinical pathology	X-ray: dynamic in varus	X-ray: dynamic in anteropulsion	Anatomical pathology
0	- Ordinary swelling - Sometimes lateral haematoma - Perimalleolar pain	10°	5 mm	No rupture of ligament
1	- Lateral swelling with crepitus and haematoma - Tibiofibular syndesmosis pain - Pain on weight bearing	10–15°	8 mm	ATF isolated rupture
2	- Medial lateral haematoma - Pre-malleolus pain - Lateral mobility increase - Limp	20–25°	10–15 mm	ATF + CF rupture
3	- Oedema + haematoma - Anterior tibiofibular syndesmosis - Pain in varus position - Anterior talus drawer test - Unable to place foot	30°	15 mm	ATF + CF + PT+ interosseus ligament rupture

ATF, anterior talofibular; *CF*, calcaneofibular; *PTF*, posterior talofibular

the most frequent is the fracture of the base of the fifth metatarsus while among tendons injuries, the most frequent are rupture of the lateral retinaculum with subdislocation or dislocation of the peroneal tendons, followed by rupture of the peroneus brevis.

Treatment

The choice of the most appropriate treatment regarding an ankle capsular ligament injury is inevitably conditioned by whether it is a recent injury or a chronic instability. While in the case of instability numerous scientific research works have been published to show the advantages of surgical treatment with respect to the functional one, the same cannot be said about the treatment of acute injuries. Discussion about what is considered to be the right choice of treatment follows.

Recent Injuries

We want to achieve two objectives with appropriate therapy: (1) first, reducing painful symptoms and oedema; (2) second, restoration or recovery of anatomic integrity within the least possible time. We have at our disposal two types of treatment: conservative and surgical. A meta-analysis of 70 research works published by Kerkhoffs et al. in 2002 [19] established that there was no difference in results while other research works [20] demonstrated that it is possible to obtain satisfactory subjective and clinical stability with the use of conservative treatment. Kannus and Renstrom [20] have, in addition, shown how functional treatment with immobilisation in a plaster cast or splint is doubtlessly superior. Functional treatment is based on the concepts that constitute the basis of the cure of the injury, thus the different grades, degrees, or levels of recovery:

- Phase 1: immediately post-trauma (oedema hemorrhage)
- Phase 2: 1–3 weeks (proliferation phase)
- Phase 3: 3–6 weeks (maturing phase)
- Phase 4: 6–8 weeks (remodelling phase)

It is fundamental to protect the joint during phases 1 and 2 of ankle sprains with anatomic lesions.

Grade 0 injuries are treated typically by following the acronym RICE (rest, ice, compression, elevation). *Grade 1* injuries are treated with: (1) adhesive elastic bandaging for 18–20 days and (2) semi-rigid ankle support for 15 days (to be worn over the adhesive elastic bandage). As an alternative, a plaster cast or splint for 10 days and an adhesive, elastic bandage for an additional 10 days. *Grade 2* injuries need to be immobilised for a longer period and be free from weight bearing, as a result of swelling of the perimalleolar, for at least

8–10 days. Anti-thromboembolic drugs are prescribed in this case. Semi-rigid ankle support is required for 8–10 days without weight bearing, with adhesive elastic bandage and orthosis for 10–15 days. As an alternative, splint without weight bearing for 10 days followed by a plaster cast with weight bearing for an additional 10–15 days. *Grade 3* injuries are surgically treated by re-building the capsular ligament. The objective is to repair the ligament by suture with a non-reabsorbable thread, followed by synovial and articular capsule reconstruction. Immobilisation with the use of a splint is effected after the operation. Sport activities can begin roughly at the beginning of the third month after the operation.

Chronic Instability

In *grade 1* instability, the advisable treatment is the functional type together with proprioceptive gymnastics to be carried out with the help of the prioceptive table. In recent times, a close look at the achieved results indicates that the use of thermal capsular retention is becoming more common in addition to making it possible to obtain a satisfactory stabilisation within the grade 1 stability through the shrinkage of the anterolateral capsule [21]. The principle upon which the *shrinkage* technique is based was clinically used for the first time in the early 1990s to correct relaxation of the capsular tissue in shoulder articulation. The first instruments utilised for thermal energy supply were lasers, which were later abandoned and replaced by radio-frequency equipment that produces heat through electro-thermal energy. This proves to be less expensive, easier to use, and, importantly, characterised by fewer complications, probably due to the interaction modality with the tissue of the photo-thermal energy produced by laser instruments. In the application of arthroscopic shrinkage carried out by us, we used the 2000 system (ArthroCare Corporation) as the heat-producing instrument, thus a bipolar, electrothermal generator with radio frequency. The instrument provides more thermal electrodes, according to the required needs of shrinkage, with each one having a different depth of penetration. Control of this last parameter is particularly important to reduce the risk of damage to the surrounding tissue.

The choice of establishing the cases of ankle instability by surgical means is linked with the almost constant arthroscopic verification of cartilaginous injury present in cases of light laxity as well, referring to those of grade 1, which are almost always associated with fibrous impingement. The use of the radio-frequency in arthroscopy, besides serving for elimination of the impingement, enables us to localise the ligament injury and perform shrinkage alongside the ATFL direction since it is the only intra-capsular ligament. In case of major instability, the advisable treatment is capsular retention and

external ligament plasty [5]. External capsular retention, which presents many analogies with that of Broström [9], is carried out through a small pre-malleolus incision to gain access to the anterolateral capsular level. The procedure continues up to the reduction of the articular space, with separate stitches that are applied along the ATFL and CFL directions. In Broström's type of surgery, we can at this point, use the lower retinaculum to increase stabilisation of the capsular retention. External ligament plasty is performed with half of the tendon of the peroneal brevis that once dissected at its proximal insertion is successively made to take a back-to-front movement in a tunnel created in the peroneal malleolus and is sutured to itself under tension (Fig. 1).

After each of these operations, the ankle is immobilised in a protective ankle brace or plaster cast for 20 days followed by progressive weight bearing with the use of 2 sticks. The final step deals with recovery of complete articular movement and muscular strength. The estimated period until re-entering sports activities is 3 months.

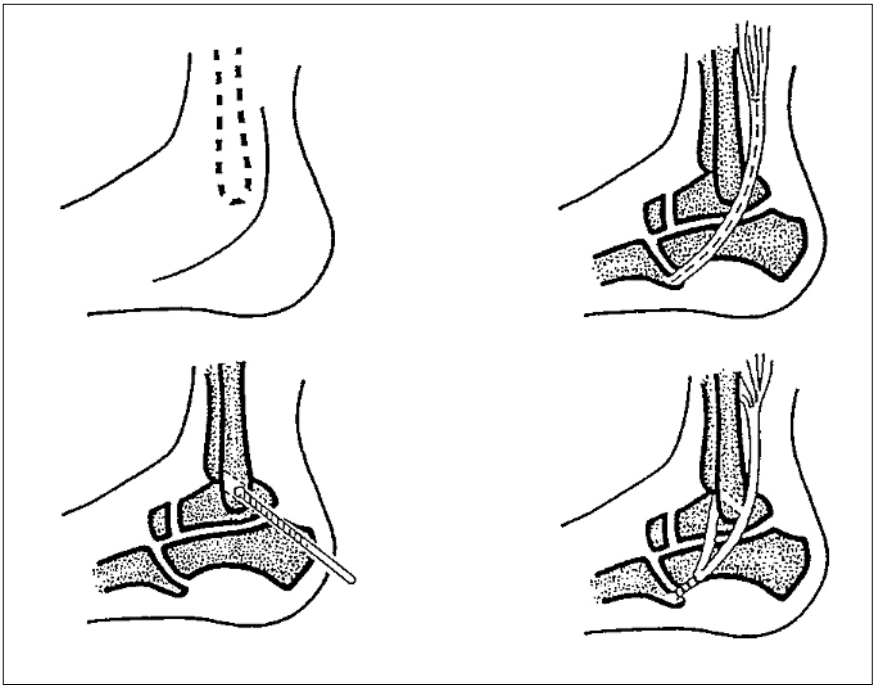

Fig. 1. External ankle ligament plasty (reproduced from [5])

Joint Degeneration Lesions as a Consequence of Ankle Instability

Lesions resulting from post-traumatic articular instability predominantly affect the anterior compartment and are mainly to be considered as hypertrophy of both soft and cartilaginous tissues. All these forms of pathology have similar clinical aspects, as well as a linking aspect that progressively evolves by starting as a reactive synovitis and transforms into either a fibrous type of structure (fibrous impingement) or a reaction of the sub-chondral bone (bone impingement). These structures ultimately reach a state of arthrosis characterised by a shrinking of the rima as the articular cartilage disappears. Symptoms include diffused pain, limitation of active and passive mobility, and, in most severe cases, de-ambulation or walking difficulties. Through an objective examination, it is possible to detect swelling and pain sensitivity in the talus dome-shaped area and articular tibial surface, and symptoms in the adjacent articular structure. Initial treatment is based on taking non-steroidal anti-inflammatory drugs (NSAIDs) over a short period but most especially on physiokinesitherapy. In case of persisting or increasing symptoms, it becomes necessary to employ surgical treatment.

We now consider three aspects of the degenerating pathology of the ankle: (1) fibrous (soft tissue) impingement; (2) bone impingement; (3) ankle arthrosis.

Impingement

Fibrous impingement involves especially the anterolateral compartment and is mostly caused by synovitic irritating processes that can lead to formation of hypertrophy of the synovia and fibrotic tissue. In most cases, the *primum movens* is represented by a congenital or post-traumatic ligament laxity that induces instability [22]. Another cause of synovitic hypertrophy is represented by the meniscoid lesion, which is a post-traumatic lesion of the soft tissue lying between the fibula and the talus [23]. In the same way, swelling of the distal bundle of the lower anterior tibiofibular ligament causes it to come into contact with the articular bone structures and becomes responsible for fibrous impingement and chronic ankle pain.

Bone Impingement

Bone impingement, which has the highest frequency among soccer players and dancers, is – on the contrary – mostly due to osteochondral or osteochondrotic types of lesions and to pseudo-cystic formations of the tibial mortar and the talus surface, and to malleolar bone projections [24].

Ankle Arthrosis

Ankle arthrosis represents the last stage of the degenerative pathology. Very rarely, it is primary; much more frequently, it is due to secondary causes. On x-rays, osteophytic formations and reduction of the joint rima are visible while clinically, chronic pain and progressive articular rigidity associated in flexion-extension movement is evident. These degenerative processes manifest as a rigid ankle. In recent years, surgical treatment of degenerative pathology has changed.

Arthroscopy is now being used more frequently, particularly in complicated situations or where open treatment can lead to an unsatisfactory results. The young age of the patient, non-acceptance of the most invasive surgical treatments, and some particularly discouraging data on prosthetic ankle implants have brought about frequent use of ankle arthroscopy. This technique presents few contraindications. The optics are inserted into the articulation by an anterolateral portal at 30° with respect to the walking level of the leg axis, and with anteromedial introduction of motorised equipment, it is possible to remove the necrotic cartilage and the osteophytic formation, debride synovial hypertrophy, extract free, loose bodies, and reduce the bone impingement.

In cases of serious rigidity, it is possible to associate the chondroplasty with the arthrodiastasis. This technique is executed by applying the external fixator, simultaneously maintaining diastasis of the ankle and flexion-extension movement [25]. The arthroscopic technique is also used for certain procedures, such as arthrodesis, that some years ago were exclusively executed by open surgery. It is also often indicated in patients with severe forms of rheumatoid arthritis, previous osteomyelitic processes, bone necrosis, and in degenerative arthrosis.

Until roughly a decade ago, arthrodesis was being carried out with an open technique through an anterolateral or medial incision; posterior incision was at times preferable for its better cosmetic effect [26]. There are various methods of performing ankle arthrodesis; however, the most common is based on the denudation of the articular surface of both the distal tibia and the talus, with exposure of the subchondral bone and successive fusion by posterior or anterior bone graft and then fixation by a rigid synthesis or Kirschner's wire. Currently, arthroscopy is preferred for this type of surgical operation and is performed through anterolateral and anteromedial incisions. Motorised instruments are used for debridement and cartilage shaving right up to the exposure of the subchondral bone. Reduction is then carried out with two or three canula screws positioned according to the guidelines of the x-ray scope [27]. After the procedure, articulation is immobilised in a plaster cast for 6 weeks and without a cast for over 2 weeks, always without

weight bearing. Complete fusion is obtained after 7–20 weeks.

Various studies demonstrate that the percentage of good results in arthroscopic arthrodesis ranges between 84% and 100% [28] as against the 64–81% of arthrodesis in open surgery [29]. In fact, the only complications that occur in this type of surgical approach are due to painful *symptomatic screws* and screw penetration at the talus level (9.5% of such cases). Arthroscopic treatment offers the patient with articular ankle problems a rapid improvement of symptoms with a fast recovery of functionality, thereby postponing the more invasive surgical procedure. Also, results show that the younger the patient, the more satisfactory the results, which include a recovery that is almost complete with regards to previously practiced activities.

Several studies [30] have produced results comparing arthroscopic and open *débridement*. Arthroscopic surgery produced excellent results in 88% of cases, with the hospital admission period and functional recovery time being doubtlessly shorter than in open surgery.

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Osteochondral Ankle Defects

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Introduction

An osteochondral ankle defect is a lesion involving talar articular cartilage and subchondral bone, mostly caused by a single or multiple traumatic events leading to partial or complete detachment of the osteochondral fragment with or without osteonecrosis. Many terms are in use to describe this defect, including osteochondral fracture, osteochondral lesion, osteochondritis dissecans, transchondral fracture, flake fracture, and intra-articular fracture. Osteochondral defects (OCDs) can occur in any joint; however, the most common location is the knee.

OCDs of the ankle comprise approximately 4% of the total number of OCDs [1]. It occurs most frequently in 20- to 30-year-old men [2]. Defects can be found on the medial and lateral sides of the talar dome and occasionally are located centrally. Ankle sprains are accepted as being the most common cause of osteochondral ankle defects. Ankle injuries are also among the most common injuries seen in sports medicine. It is estimated that one ankle injury per 10,000 people per day occurs [3]. Ankle injuries comprise 45% of basketball injuries, 25% of volleyball injuries, and 31% of football injuries [4]. The percentage of osteochondral lesions associated with lateral ankle ligament rupture has been determined by three Authors who routinely inspected the lateral talar dome in a consecutive series of patients operated for lateral ankle ligament rupture. These Authors reported 5%, 6%, and 9%, respectively, of lateral talar dome lesions [5–7]. The percentage of medial dome lesions is unknown but is estimated to be as high as lateral talar dome lesions [6].

After an athlete sustains an ankle sprain, treatment is directed at returning him or her to the previous level of competition as soon as possible. However, after standard treatment for acute ankle sprains, residual symptoms are reported in 33–40% of patients [5]. If symptoms persist after an ankle sprain, the possibility of an OCD needs to be considered.

The aim of this chapter is to provide an overview of osteochondral ankle defects, their symptoms, and specific treatment indications in football players.

History

In 1856, Monro first reported the presence of cartilaginous bodies in the (ankle) joint [8]. In 1870, Paget further described the defects [9]; and in 1888, König first used the term *osteochondritis dissecans* for loose bodies in the knee joint [10]. He suggested that these were the result of spontaneous osteonecrosis secondary to vascular occlusion of the subchondral bone. He used the term *osteochondritis* to refer to an inflammatory process, and *dissecans*, derived from the Latin word *dissecare*, to separate. An inflammatory process, however, has never been proved to be involved in the pathology. In 1959, Berndt and Harty were the first to mention trauma as the main etiologic factor of osteochondral ankle defects. They used the term transchondral fracture of the talus to describe the defect and presented a classification system and guidelines for indications for surgery [8]. Since Berndt and Harty's classic paper, indications for surgical treatment have changed, and nowadays, a large variety of treatment options exist for the different forms of osteochondral ankle defects.

Aetiology

In the ankle, traumatic insult is more widely accepted as the aetiology of talar OCDs although not without controversy. Both trauma and ischemia are probably involved in the pathology. As not all patients report a history of ankle injury, a subdivision can be made in aetiology of non-traumatic and traumatic defects. The non-traumatic aetiology concerns idiopathic OCDs. In these, ischemia, subsequent necrosis, and possibly genetics are aetiologic factors. OCDs in identical twins and in siblings have been described [12–14]. In 10–25% of patients, the occurrence of the defect is bilateral [11, 15].

In the etiology of traumatic OCDs, ankle sprains play the largest role. A severe ankle sprain can cause a small fracture and subsequent impaired vascularity, leading to the formation of an OCD. Besides, microtraumas caused by repetitive articular cartilage surface loading or excessive stress can lead to cellular degeneration or death by the disruption of collagen fibril ultra-structure and thickening of the subchondral bone [16]. In lateral lesions, trauma is described in 98% of cases; in medial lesions, this is 70% [17].

Mechanism of Injury

When the talus twists inside its box-like housing during an ankle sprain, the cartilage lining can be damaged. This may lead to a bruise and subsequent softening of the cartilage or worse: a crack in the cartilage or delamination. Separation of the cartilage can occur in the upper layer as a result of shearing

forces. Alternatively, separation may occur in the subchondral bone, giving rise to a subchondral lesion. Fragments can break off and float loose in the ankle joint or they can remain partially attached and in position. Progression may result in increased joint pressure, resulting in the forcing of synovial fluid into the epiphysis, creating a subchondral cyst. The subchondral cyst and increased joint pressure may prevent healing. The subchondral fracture has no soft tissue attachments and is highly susceptible to subsequent avascular necrosis.

In cadaver ankles, Berndt and Harty could reproduce lateral defects by strong inversion of a dorsi-flexed ankle [11]. As the foot was inverted on the leg, the lateral border of the talar dome was compressed against the face of the fibula. When the lateral ligament ruptured, avulsion of the chip began. This chip could be completely detached but remain in place or be displaced by supination. With the use of an excessive inverting force, the talus within the mortise was rotated laterally in the frontal plain, impacting and compressing the lateral talar margin against the articular surface of the fibula. Then, a portion of the talar margin was sheared off from the main body of the talus. This caused a lateral OCD. They were able to reproduce a medial lesion by plantar flexing the ankle, slight anterior displacement of the talus upon the tibia, inversion, and internal rotation of the talus on the tibia.

Lateral osteochondral lesions are usually located in the anterior third of the talar dome. Medial lesions are mostly located in the posterior half (Fig. 1). There are exceptions, however, and anteromedial, posterolateral, as well as

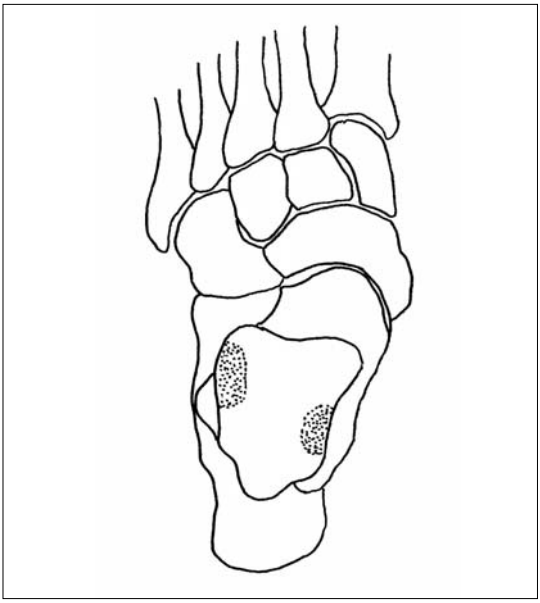

Fig. 1. Main locations of osteochondral ankle defects

centrally located lesions do occur after trauma. Multiple lesions in one patient can be present. Lateral lesions are typically shallow and wafer shaped, indicating a shear mechanism of injury (Fig. 2). In contrast, medial lesions are generally deep and cup-shaped, indicating a mechanism of torsional impaction. Medial lesions are usually asymmetric whereas lateral lesions are symmetric. Because of their shape, lateral lesions are more often displaced than medial lesions.

Clinical Presentation

A differentiation has to be made between the acute and chronic situation. In the acute situation, symptoms of osteochondral ankle defects compare to those of acute ankle injuries. They include lateral or medial ankle pain, functio laesa, and swelling. In patients with an isolated ligamentous ankle injury, these symptoms usually resolve after functional treatment within 2–3 weeks. If symptoms do not resolve after 3–6 weeks, an OCD of the talus should be suspected. These patients typically present with persisting symptoms and limited range of motion. Locking and catching are symptoms of a displaced fragment. In most patients with a non-displaced lesion after supination trauma, the symptoms in the acute situation cannot be distinguished from the soft tissue damage.

Chronic lesions classically present as deep lateral or medial ankle pain

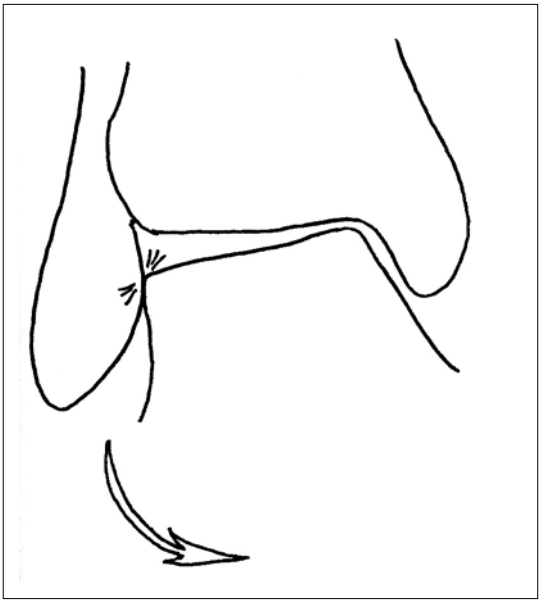

Fig. 2. Shear mechanism of injury in lateral osteochondral ankle defects

associated with weight bearing. Reactive swelling and stiffness can be present, but absence of swelling, locking, or catching does not rule out an OCD. Recognisable pain on palpation is typically not present in these patients. Some patients have a diminished range of motion.

Possible symptoms are:
- Lateral or medial ankle pain;
- Functio laesa;
- Swelling;
- Locking;
- Catching;
- Deep pain on weightbearing;
- Stiffness;
- Diminished range of motion.

Differential diagnoses are:
- Posttraumatic synovitis;
- OCD of tibial plafond;
- Sinus tarsi syndrome;
- Os trigonum;
- Ligament laxity;
- Peritendinitis
- Osteoarthritis;
- Osteoid osteoma;
- Avascular necrosis of the talus.

Damaged talar cartilage is responsible for pain during weight bearing. This is probably the result of edge loading by the tibia on the cartilage rim of the defect and the subchondral bone underneath. Due to the convex nature of the talus, the edges of the mainly circular defect are more heavily loaded than usual. Part of the healthy cartilage is gone, and the remaining cartilage has to carry the weight transmitted by the tibia. Nerve endings in the subchondral bone in the rim of the defect or underneath the defect are excitated by the increased loading. The purpose of treatment is to diminish the edge loading, destroying the mechanism responsible for increased local hydraulic pressure onto the subchondral area below the defect.

Diagnosis

After careful history taking and physical examination of the ankle, routine radiographs are made consisting of weight-bearing anteroposterior, mortise, and lateral views of both ankles. The radiographs may show an area of detached bone surrounded by radiolucency (Fig. 3). Initially, the damage

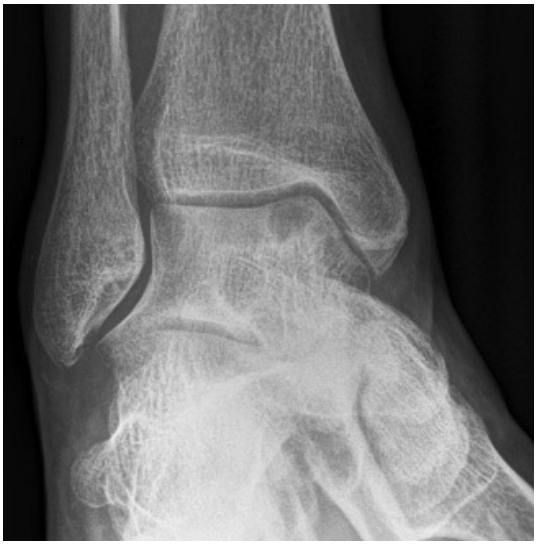

Fig. 3. Radiolucency medial talar dome indicating an osteochondral defect (x-ray)

may be too small to be visualised on a routine x-ray. By repeating the imaging studies at later stage, the abnormality sometimes becomes apparent. A heel-rise view with the ankle in a plantar-flexed position may reveal a posteromedial or posterolateral defect [18]. A bone scan can differentiate between a symptomatic and asymptomatic lesion. Magnetic resonance imaging (MRI) is often used for detection of these lesions. Computed tomography (CT) is useful for better defining the exact size and location of the lesion and is therefore more valuable for preoperative planning (Figs. 4, 5). In diagnosing an OCD, CT has proven to be just as valuable as MRI [18].

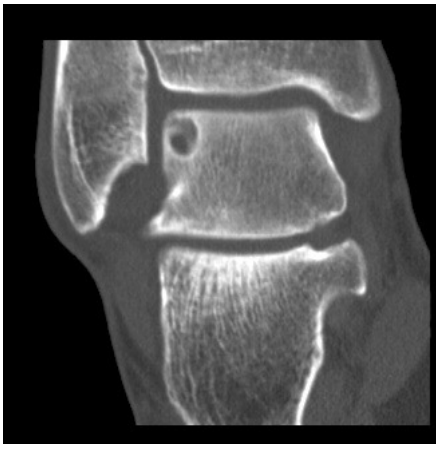

Fig. 4. Computed tomography (CT) scan of a lateral osteochondral defect, coronal reconstruction

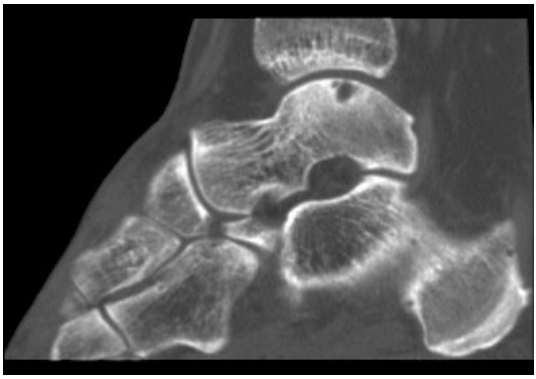

Fig. 5. Computed tomography (CT) scan of a medial osteochondral defect, sagittal reconstruction

Classification and Staging

In 1959, Berndt and Harty suggested a classification system for staging the lesions at the time of surgery based on plain radiographs of the ankle [8]. In grade I, there is local compression of the cartilage and subchondral bone, and usually there are no radiographic findings. In grade II, there is avulsion or partial detachment of the osteochondral fragment, but the main part is still attached to the talus. In grade III, there is complete avulsion of an osteochondral fragment without any displacement. In grade IV, the osteochondral fragment is completely detached and displaced inside the ankle joint (Table 1 and Fig. 6). Later, classification systems based on MRI, CT, and arthroscopic findings were made [19–21]. The use of these classification systems is questionable since none of the systems are dually related to the current treatment options [22].

Table 1. Classification and staging of lesions according to Berndt and Harty (adapted form [11])

Stage	Description
I	Small compression fracture
II	Incomplete avulsion of a fragment
III	Complete avulsion of a fragment without displacement
IV	Displaced fragment

Current Treatment Options

There are widely published non-surgical and surgical techniques for treatments of symptomatic osteochondral lesions.

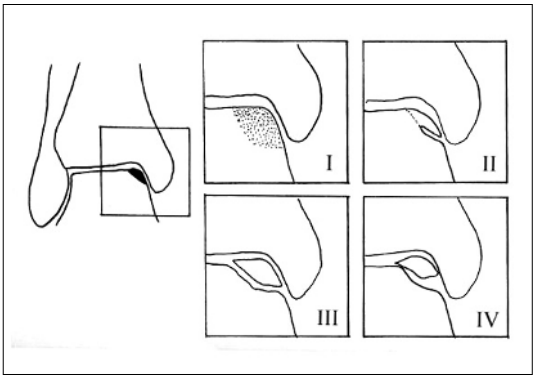

Fig. 6. Classification of osteochondral ankle defects (adapted from [11])

Non-operative Treatment

This may be rest and/or restriction of (sporting) activities with or without treatment with non-steroidal anti-inflammatory drugs (NSAIDs) or cast immobilisation for at least 3 weeks and up to 4 months. The aim is to give the bruised talus rest so oedema can resolve and necrosis prevented or for the (partly) detached fragment to become re-attached to the surrounding bone.

Bone Marrow Stimulation (BMS)

This is the treatment mostly indicated. After *débridement*, multiple connections with the subchondral bone are created. They can be accomplished by drilling or micro-fracturing. The objective is to partially destroy the calcified zone that is most often present and to create multiple openings into the subchondral bone. Intra-osseous blood vessels are disrupted, and the release of growth factors leads to the formation of a fibrin clot. The formation of local new blood vessels is stimulated, marrow cells are introduced in the OCD, and fibro-cartilaginous tissue is formed. In the case of large defects, a cancellous bone graft can be placed.

Retrograde Drilling

Retrograde drilling is done for primary OCDs when there is more or less intact cartilage with a large subchondral cyst. The aim is to induce subchondral bone revascularisation and subsequently to accomplish new bone formation. A cancellous graft may be placed to fill the gap.

Autologous Chondrocyte Implantation/Transplantation (ACI/ACT)

Autologous chondrocyte implantation (ACI) attempts to regenerate tissue with a high percentage of hyaline-like cartilage. By means of an arthroscopic

approach, a region of healthy articular cartilage is identified and a biopsy is taken. The tissue is minced and enzymatically digested. Chondrocytes are separated by filtration, and the isolated chondrocytes are cultivated in culture medium for 11–21 days. An arthrotomy is performed, and the chondral lesion is excised up to the healthy surrounding cartilage. A periosteal flap is removed from the tibia and is sutured to the surrounding rim of normal cartilage. The cultured chondrocytes are then injected beneath the periosteal flap. This treatment is not widely available as yet, and more results need to be published.

Osteochondral Transplantation

Osteochondral autografts have been examined as an alternative to allografts for the treatment of OCDs. Two related procedures have been developed: mosaicplasty and osteochondral autograft transfer system (OATS). Both are reconstructive bone grafting techniques that use one or more cylindrical osteochondral grafts from the less-weight bearing periphery of the ipsilateral knee and transplant them into the prepared defect site on the talus. Its goal is to reproduce the mechanical, structural, and biochemical properties of the original hyaline articular cartilage that has become damaged. It is carried out either by an open approach or by an arthroscopic procedure.

Fixation

Large fragments are treated surgically with reduction and fixation of the osteochondral fragment. Several types of internal fixation have been reported, including Herbert screws, Kirschner wires, absorbable fixation, and fibrin glue.

Management and Prognosis

The choice of treatment for osteochondral ankle defects depends on symptomatology, duration of complaints, size of defect, and whether a primary or secondary OCD. None of the current grading systems are sufficient to direct the choice of treatment [22]. Pure cartilage lesions, asymptomatic, and low symptomatic lesions are treated conservatively with rest, ice, temporarily reduced weight bearing and, in case of giving way, an orthosis. Consideration for surgical treatment is failure of non-operative treatment or continuing symptoms after previous surgical treatment (secondary OCD). In three recent reviews of the literature, the best treatment currently available for primary osteochondral ankle defects is excision, *débridement*, and drilling [22–24].

According to the recent International Society of Arthroscopy, Knee Surgery, and Orthopaedic Sports Medicine and the International Federation

of Sports Medicine (ISAKOS–FIMS) consensus, *débridement* and drilling/micro-fracturing is the first step in the treatment of symptomatic osteochondral lesions that are too small to consider fixation [25]. Fixation with 1 or 2 leg screws is preferred in (semi)acute lesions in which the fragment is 15 mm or larger. In adolescents, re-fixation of an OCD should always be considered even in fragments that are smaller than 15 mm. Large talar cystic lesions can be treated by retrograde drilling and filling the gap with a bone graft. In the case of failed primary treatment, an osteochondral transplant can be considered. The place of cultured chondrocyte transplant still needs to be determined. Treatment by means of excision, *débridement*, and bone marrow stimulation is the first choice and results in 86% good/excellent results [22].

Preoperatively, we have to decide how to approach the defect. Depending on the preference of the surgeon and the location of the lesion, the approach can be from the front, the back, or by means of a medial malleolar osteotomy. In the case of arthroscopic treatment, we have to decide whether to use mechanical distraction in combination with a 2.7-mm arthroscope or to use a 4.0-mm arthroscope and treat the OCDs in the anterior working area by full plantar-flexion of the ankle. In patients with unlimited plantar flexion, all defects in the anterior half of the talus as well as lesions located in the anterior part of the posterior half can thus be reached and treated.

The procedure is started without distraction. Introduction of the instruments is with the ankle in the fully dorsiflexed position using the standard anteromedial and anterolateral portal. The standard anteromedial and anterolateral approaches are created as described [26]. A 4.0-mm scoop and a 4.5-mm or 5.5-mm shaver are introduced. If the OCD is located anteromedially, the 4.0-mm arthroscope is moved over to the anterolateral portal, and the instruments are introduced through the anteromedial portal. For an anterolateral defect, the arthroscope remains in the anteromedial portal, and the instruments are introduced through the anterolateral portal. If osteophytes are present, they are removed first by chisel, burr, or aggressive full-radius resector (Bone Cutter). Synovitis located anterolaterally (in case of an anterolateral defect) or anteromedially (in case of an anteromedial defect) is removed first by a 4.5-mm or 5.5-mm full-radius resector with the ankle in the dorsiflexed position. The completeness of removal of osteophytes and synovitis is checked by bringing the ankle into plantarflexion. It should now be possible to palpate and visualise the OCD without disturbance of the synovium or overlying osteophyte. If this is not the case, then a further synovectomy is performed with the ankle in the dorsiflexed position. After sufficient synovectomy, it should be possible to identify the lesion with the ankle in the forced plantarflexed position by palpating the cartilage with a probe.

In the case of a posteriorly located osteochondral lesion, this demands a full plantar flexion. A little joint laxity helps to open up the joint. During this

part of the procedure, we apply a soft tissue distractor [26]. Not only can the lesion be palpated with a probe, but it should also be possible to visualise at least the anterior part of the lesion. If possible, the 4.5-mm or 5.5-mm aggressive, full-radius resector is now introduced into the defect. In doubtful cases, before introduction of the resector, it can be useful to identify the defect by introducing a spinal needle, thereby penetrating the defect area. If there is any doubt about the direction and the extent of the defect, the arthroscope is moved over to the portal opposite the defect (the anteromedial portal in case of an anteromedial OCD), and the completeness of the *débridement* is assessed. The scope is then brought back to the opposite portal, and further *débridement* is performed by means of the aggressive full-radius resector or a small closed cup curette. It is important to remove all dead bone and overlying unsupported, unstable cartilage.

Every step in the *débridement* procedure is checked by regularly switching portals. A precise and complete *débridement* with removal of all loose fragments can thus be performed. Introduction of the instruments and the arthroscope is performed with the ankle in the fully dorsiflexed position, thus preventing iatrogenic cartilage damage. After full *débridement*, the sclerotic zone is drilled by multiple drill holes using a 2-mm burr or a 1.4-mm K-wire. A K-wire has the advantage of flexibility whereas a 2-mm drill can break more easily if the position of the ankle is changed during drilling. When a 2-mm drill is used, a drill sleeve is necessary to protect the tissue. Micro-fracturing by means of a microfracture probe offers the possibility to work "around the corner". Make sure that the calcified area is penetrated.

After treatment depends on the type of surgical treatment. After arthroscopic *débridement* and drilling, patients are encouraged to make active plantar-flexed and dorsi-flexed ankle movements. Partial weight-bearing is allowed. Full weight-bearing is dependent on the size and location of the lesion. A lesion of up to 1 cm is allowed to progress to full weight bearing within 2 weeks. Larger lesions and anteriorly located lesions require partial weight bearing of up to 6 weeks. Running on even ground is permitted after 12 weeks. If a fragment is fixed, the period of non-weight bearing is 6 weeks followed by another 4–6 weeks of controlled weight bearing. After medial malleolar osteotomy, weight-bearing is dependent on the surgical treatment of the osteochondral lesion. After OATS, running is not permitted until the graft has been incorporated. Full return to normal and sporting activities is usually possible 3–6 months postoperatively.

The guidelines we propose for treatment of osteochondral talar lesions are presented in Table 2. In spite of the recommended considerations for types 3–6, *débridement* and bone marrow stimulation can always be considered as a first treatment option.

Table 2. Proposed treatment guidelines for osteochondral talar lesions

Type	Treatment
1. Asymptomatic lesions, low symptomatic lesions	Conservative
2. Symptomatic lesions ≤10 mm	*Débridement* and drilling/micro-fracturing
3. Symptomatic lesions 11–14 mm	Consider *débridement* and drilling, fixation, osteochondral graft, or chondrocyte transplant
4. Symptomatic lesions ≥15 mm	Consider fixation or graft
5. Large talar cystic lesions	Consider retrograde drilling with/without bone transplant
6. Secondary lesions	Consider osteochondral transplant

Important Notes Concerning Football Players

The major cause of osteochondral ankle defects is supination trauma. Prevention should therefore be aimed at preventing ankle sprains. A 2001 Cochrane systematic database review provides good evidence for the beneficial effect of ankle supports in the form of semi-rigid orthoses or air-cast braces to prevent ankle sprains during high-risk sporting activities. Athletes with a history of previous sprain can be advised that wearing such supports may reduce the risk of incurring a future sprain. However, any potential prophylactic effect should be balanced against the baseline risk of the activity, the cost of the particular device, and – for some – the possible or perceived loss of performance [24]. Proprioceptive training has also been shown to be effective for prevention of ankle sprain recurrences [28].

Concerning treatment, arthroscopic intervention affords a quicker return to play than an open technique. The majority of lesions can be treated arthroscopically. Many posteromedial lesions do not have to be treated by malleolar osteotomy but can be treated arthroscopically by bringing the foot in hyperplantar-flexion although skill and experience are required. Advantages of arthroscopic treatment are: low morbidity, low cost, fast recovery, and fast mobilisation.

Possible disadvantages of a medial malleolar osteotomy in case of a posteromedial osteochondral ankle defect are persisting ankle stiffness, use of fixation screws, mal-union, non-union, and degenerative changes due to the osteotomy over the long term [19]. Morbidity at the donor site in OATS is seen in up to 50% of cases [30, 31]. In an athlete, this is a concern since knee pain

may prevent the patient from returning to competitive play.

Rehabilitation in athletes is not only directed at progressing the patient from protected mobilisation to partial and full weight bearing but also at strengthening and proprioceptive activity.

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Chronic Footballer's Ankle

Eric Giza, Bert Mandelbaum

Summary

Football places high demands on the foot and ankle, which inevitably lead to chronic changes that are likely related to exposure time to football. Chronic footballer's ankle (CFA) comprises a spectrum of pathology from mechanical instability to functional instability (Fig. 1) and includes ligament instability, tendinopathy, articular cartilage damage, and tibiotalar osteophytes. Recognition of the individual facets of CFA is necessary for effective diagnosis and treatment.

Introduction

A review of 20 epidemiological studies showed that the incidence of injuries during men's elite football matches ranges from 13 to 35 injuries per 1,000 h of competition [1]. An audit of English football clubs showed that the thigh (23%–24%), ankle (18%–19%), and knee (15%–17%) were the most common injury locations [2, 3]. It is estimated that the incidence of foot/ankle injuries in elite football during competition is between 3 and 9 injuries per 1,000 h.

Football places high impact on the ankle and foot, and players must maintain a balance between strength and agility in a joint that has multiplanar ranges of motion. The most common mechanism for ankle injury in football is an inversion/plantar-flexion mechanism [4, 5]; however, the aggressive cutting and direction changes can place large demands on the anterior, posterior, and medial ankle [6, 7]. Video analysis studies have provided information regarding the mechanisms of ankle injuries during football matches. Giza et al. reviewed injuries from four International Football Federation (FIFA) football competitions and found that direct contact between players occurred in 72 of 76 injuries [5]. They also demonstrated a significantly higher risk of time lost from play if the footballer is weight bearing at the time of injury. Andersen et al. reviewed 26 match injuries and found that lateral ankle

injuries are most commonly a result of a tackle that places a laterally directed force on the medial aspect of the leg of the injured player, leading to an inversion injury [8].

Chronic footballer's ankle (CFA) involves a spectrum of injuries that are accrued over the course of the footballer's career. In separate studies, Larsen et al. [9] and Drawer and Fuller [10] found that the risk of knee and ankle osteoarthritis in professional footballers is significantly greater than in the general population. Others have found that the incidence of degenerative arthritis after chronic lateral ankle instability ranges from 13% to 78% [7]; therefore, it is important to be aware of the factors that lead to chronic footballer's ankle. Chronic problems associated with laxity of the lateral or medial ligamentous structures of the ankle are called *mechanical instability*. In other cases, however, the pain or sensation of instability is a result of intra- or peri-articular changes without ligamentous laxity, which is called *functional instability*. Many footballers have a combination of the two conditions and are in the middle of the spectrum of pathology (Fig. 1).

The condition can therefore be grouped into two large categories: CFA with mechanical instability and CFA with functional instability. This chapter will first discuss CFA with mechanical instability and then focus on those conditions that can exist both with and without instability, such as bony impingement, soft-tissue impingement, tendinopathy, and chondral injury.

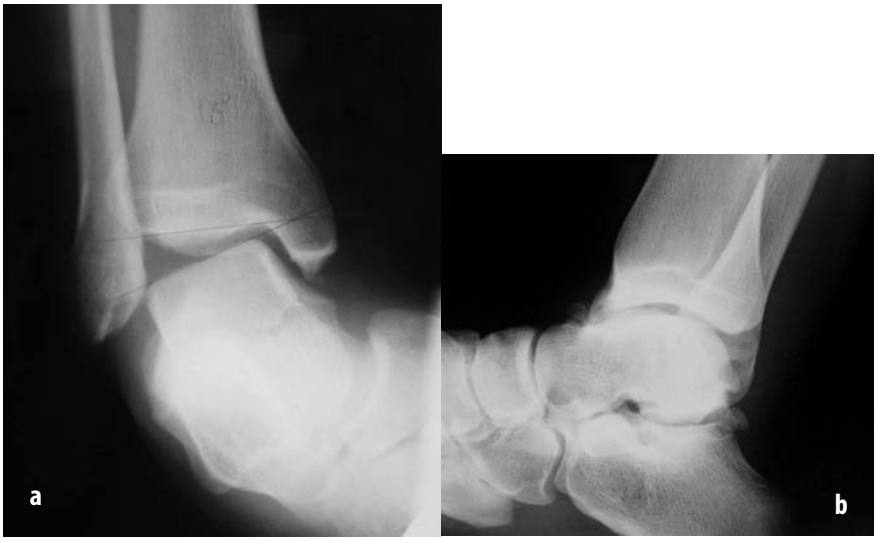


Fig. 1a, b. The spectrum of pathology found with chronic footballer's ankle (CFA)

Chronic Footballer's Ankle with Mechanical Instability

Ankle instability is a result of repeated lateral or medial ligamentous injury. A two-season survey of 91 English football clubs showed that 11% of all injuries were ankle sprains and that 77% of sprains involved the lateral ligament complex [6]. Forty-eight percent of footballers with a first-time sprain will have another sprain, and up to 26% will have recurrent sprains [11]. Many ankle sprains in football are a result of unavoidable, traumatic situations [5, 8]; however, the sports medicine professional should be aware that risk factors such as ligamentous laxity, posterior position of the fibula relative to the tibia, and peroneal muscle weakness can lead to chronic instability [12–14].

Anatomy

There are four important ligaments of the lateral ankle complex. The anterior talofibular ligament (ATFL) restricts internal rotation of the talus in mortise, is elongated in plantar-flexion, and can undergo greater plastic deformation than the calcaneofibular ligament (CFL) [15]. The CFL is a diarthrodial ligament that prevents hindfoot adduction, is stiffer than the ATFL and is elongated in dorsi-flexion [15]. The posterior talofibular ligament (PTFL) restricts external rotation when the ankle is dorsiflexed [15]. The inferior extensor retinaculum (IER) plays an important role in stability by linking the lateral ligament complex with the subtalar joint [15].

Conservative Treatment

Numerous systems have been used to grade acute lateral ankle sprains [13]; however, treatment can be guided by an understanding of the structures injured. A grade 1 injury involves only a tear in the ATFL. In grade 2 injuries, the CFL is involved, and in grade 3 injuries, the entire lateral complex is disrupted (Fig. 2). Operative treatment is rarely, if ever, indicated for acute lateral ligament injuries, and the mainstay of treatment is rest, ice, compression, and elevation (RICE). Players with excessive swelling or tenderness of the distal fibula should have a radiograph done to exclude ankle fracture [16]. Return to play ranges from 1 to 8 weeks depending on the severity [11], and players should undergo a systematic rehabilitation program that includes peroneal strengthening and proprioceptive exercises in order to prevent future injury and progression to chronic laxity [17, 18]. Most footballers will be completely pain-free 8 weeks after the injury, and those who have continued pain should be referred for an MRI to evaluate the possibility of other ankle pathology.

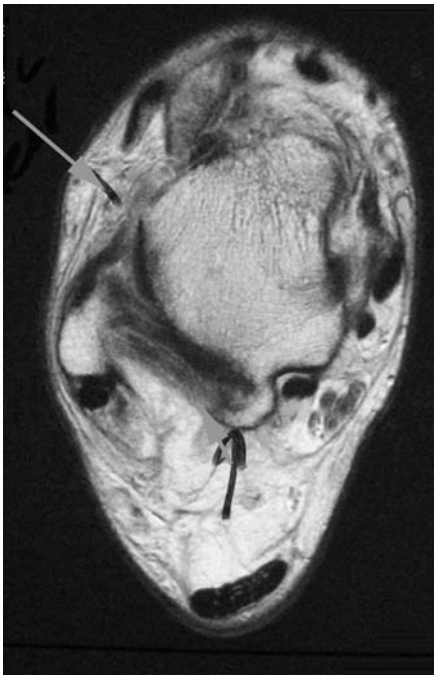


Fig. 2. T2-weighted MRI demonstrating a tear of the anterior talofibular ligament (ATFL) (*white arrow*) with an intact posterior talofibular ligament (PTFL) (*dark arrow*)

Diagnosis

The hallmark of mechanical instability is mobility beyond the physiological range of motion, which has been traditionally characterised by a positive talar tilt and/or anterior drawer test [11]. There is some variability in the radiographic criteria for mechanical instability due to differing amounts of laxity among patients; however, Mann et al. showed that 81% of patients with radiographic instability had recurrent sprains [19]. Mechanical instability is considered present if there is more than 10 mm or anterior translation on the effected side or more than 3 mm on side-to-side difference, or if talar tilt is more than 9° or more than 3° on side-to-side difference [11] (Fig. 3).

Another method to determine instability is to place the patient prone on the exam table and allow both ankles to hang over the edge of the table. This relaxes the peroneals, which are often in spasm to compensate for the instability. The distal leg is stabilised with one hand, and the calcaneous is translated medially to reveal the amount of lateral stability. The anterior drawer can also be easily measured in this fashion. Scranton and McDermott have described a method by which the distance between the centre of the Achilles and the tip of lateral and medial malleoli are measured. A ratio of Achilles/lateral tip to Achilles/medial tip of greater than 1.3 is indicative of lateral instability [20].

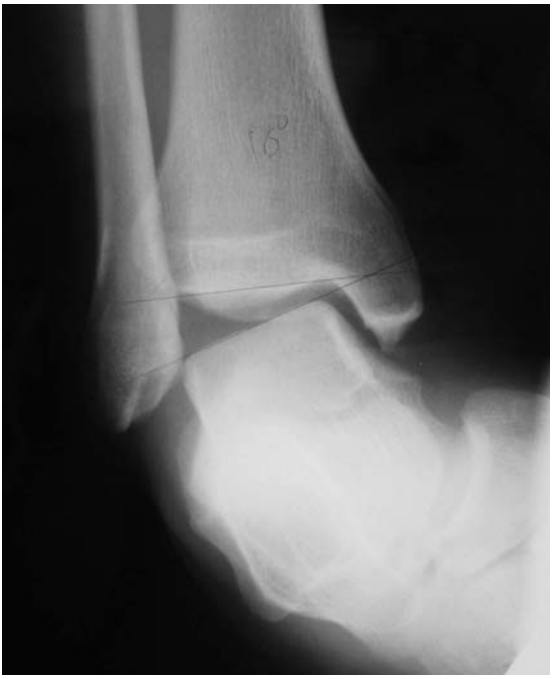
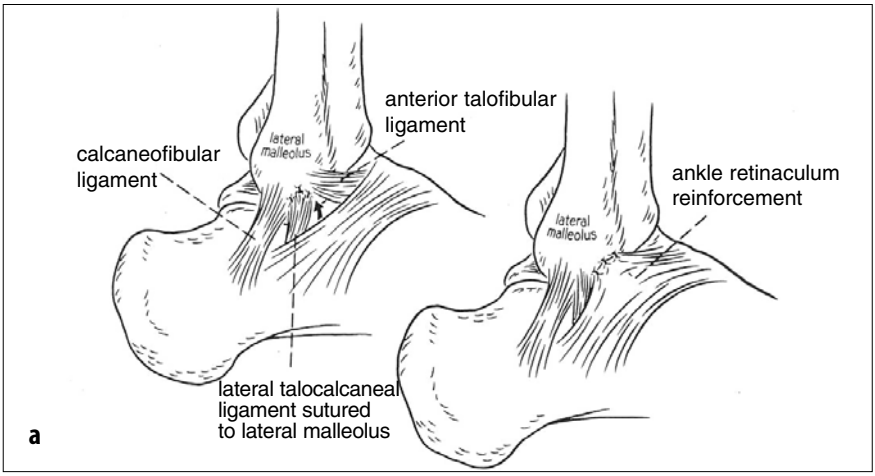


Fig. 3. AP x-ray of the ankle demonstrating a positive talar tilt test

Operative Treatment

Operative treatment of CFA with mechanical instability is necessary for footballers who have had multiple sprains and have continued episodes of instability despite taping and rehabilitation. Many different procedures have been described and fall into the categories of anatomic or non-anatomic reconstruction. Non-anatomic reconstructions utilise a graft from the peroneal tendons, Achilles, or allograft and can lead to excessive tightness of the lateral ankle and subtalar joint [21–23]. While the ATFL spans only one joint, the CFL and IER play an important role in subtalar stability [15, 24]. Footballers need to preserve as much ankle and subtalar motion as possible to maintain a *touch* on the ball; therefore, the anatomic reconstruction described by Broström is the preferred treatment for CFA with mechanical instability [22, 25]. The procedure involves exposure of the attenuated ATFL, CFL, and IER with advancement of the ligaments back to their anatomic insertions on the fibula using bone tunnels or suture implants (Fig. 4). Krips et al. compared the functional outcome and sports activity level of 41 patients with an anatomic reconstruction to 36 patients with a non-anatomic reconstruction and found superior results in return to play in the anatomic reconstruction group [26]. Karlsson et al. found good to excellent results in 132 of 152 ankles



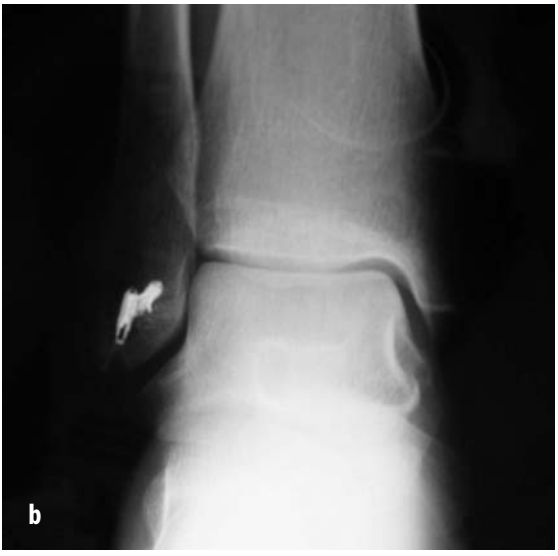


Fig. 4a, b. a Diagram of the modified Broström lateral ankle ligament reconstruction (reproduced from [25]). **b** Postoperative mortise x-ray of the ankle showing suture anchor ligament repair

that were operated on for chronic lateral instability, and the 20 with poor results were found to have generalised ligamentous laxity or multiple surgeries [27].

The immediate postoperative protocol includes immediate weight bearing, stabilisation in an Aircast® (Aircast, Inc., Summit, NI, USA) brace, and range of motion exercises. After 4 weeks, the brace is removed and the patient begins peroneal strengthening and an ankle exercise program [28, 29]. Jogging begins at 6 weeks, and return to football is often possible between 12 to 16 weeks.

Association of CFA with Mechanical Instability and Chronic Ankle Pathology

CFA with mechanical instability can often have associated intra-articular pathology. Van Dijk et al. found acute injuries to the tibia and talus in 20 out of 30 patients who had arthroscopy after acute ankle sprain [30]. In a study of 61 patients who underwent stabilisation for lateral ankle laxity, DiGiovanni et al. found no patients with isolated instability [31]. Takao et al. found that patients who had continued disability for more than 2 months after ankle sprain had intra-articular pathology. Moreover, they found that arthroscopy was particularly useful for the diagnosis of osteochondral lesions since the pre-arthroscopic sensitivity with MRI and physical exam was only 82.4% [32].

In contrast, Hintermann et al. in 2002 found that 66% of 148 patients with mechanical instability had articular cartilage damage, showing that ligament damage does not always correlate with articular lesions. Their study was limited to 22.2 months of follow-up and indicates that chronic laxity in the absence of an acute articular cartilage injury may not lead to further damage [7]. Okuda et al. also found cartilage damage in only 63% of 30 patients with chronic instability [33]. They concluded that the presence of cartilage injuries did not increase with instability if the length of instability was less than 7 years. These results should be interpreted with caution since neither study exclusively included footballers who may be more likely to create cartilage injuries in the presence of ligamentous laxity.

The superior peroneal retinaculum (SPR) spans from the posterior and distal aspect of the fibula to the Achilles fascia and os calcis. Along with the retromalleolar sulcus and cartilage rim, the SPR provides stability for the peroneal tendons [13]. Chronic lateral laxity and stretching of the SPR can lead to subluxation of the peroneus brevis and longus. Recognition of SPR laxity and tendon pathology associated with lateral ligament laxity is paramount, as failure to do so can lead to chronic pain after a lateral stabilisation procedure [34–36].

Prevention of Mechanical Ankle Instability

Recurrent inversion episodes can lead to chronic mechanical instability. Thacker et al. performed a meta-analysis of 113 ankle sprain studies and found that the most common risk factor for an ankle sprain is a history of prior sprains [37]. Surve et al. compared 258 soccer players without a history of sprains to 246 players with a history of sprains and found the use of a semi-rigid orthosis led to a significant reduction in sprains only for the group with a history of sprains [38]. Verhagen et al., performed a prospective study on 116 male and female volleyball teams that were randomly divided into control

and intervention groups [18]. The intervention groups performed 5 min of proprioception or ankle strengthening exercises before each practice session during a 36-week season. A significant risk in reduction of ankle sprain was found only for players with a history of previous sprain [18]. Footballers with an ankle sprain should therefore undergo a full rehabilitation program and use protective bracing or taping until they are fully rehabilitated.

Medial Ankle Instability

Although injuries to the medial ligament complex are less common than lateral sprains, Woods et al. found that 14% of all ankle injuries in English professional football were medial ligament injuries [6]. Medial instability can coexist with lateral instability. In a series of 52 patients with medial ankle instability, 40 (77%) were found to have concomitant lateral instability [39].

The deltoid ligament complex is comprised of 6 separate ligaments and has both a deep and superficial layer [40]. The deltoid prevents external rotation of the talus in the mortise and prohibits abduction of the ankle [13, 41]. Hintermann et al. found that deltoid ligament instability is often missed on exam and is demonstrable on arthroscopic evaluation of the ankle. Moreover, they found that cartilage damage was present in 98% of cases with medial instability but only in 66% of cases with lateral instability, indicating that pathology to the medial ligaments may result from a more traumatic injury [7].

The clinical characteristics of medial ankle instability are a feeling of giving way and a valgus and pronation deformity of the foot that can be actively corrected by firing the posterior tibial muscle [39, 42]. Often, the footballer will have failed some weeks of treatment for a lateral ligament injury but have continued pain. Exam may reveal tenderness of the medial gutter of the ankle and a valgus/pronation deformity of the foot [42]. Testing anterior drawer of the ankle in plantar-flexion and internal rotation may reveal a slight increase in translation compared with testing in plantar-flexion and external rotation.

X-rays may reveal a disruption of the mortise or bony avulsion (Fig. 5) but are not always reliable. MRI may aide in the diagnosis, but history and clinical exam are the key to diagnosis [39]. Surgical reconstruction of the deltoid complex can include imbrication of the ligament or advancement of the attenuated structures to the medial malleolus [39, 43]. Hintermann et al. found good to excellent results in 46 of 52 deltoid ligament reconstructions [39].

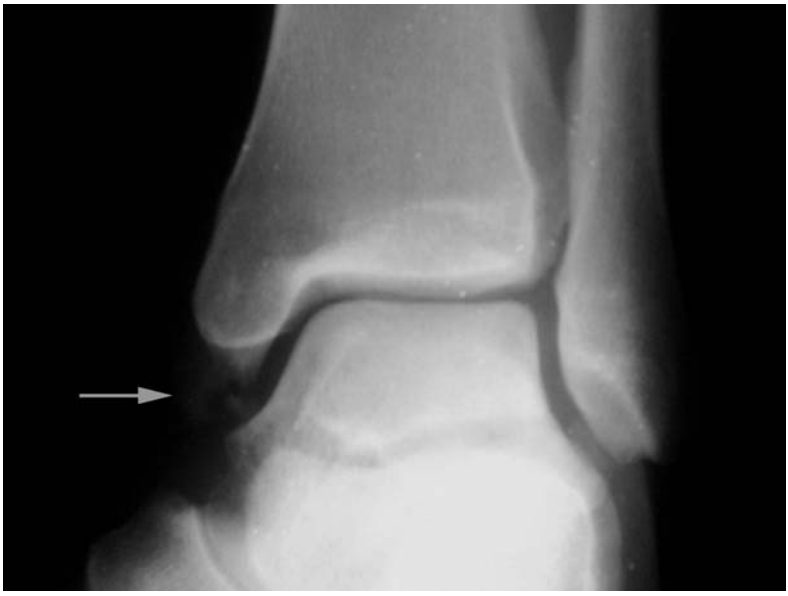


Fig. 5. AP x-ray of the ankle in a football player demonstrating an avulsion of the anterior colliculus of the medial malleolus and deltoid ligament (*arrow*)

Chronic Footballer's Ankle with Functional Instability

Numerous pathologies about the ankle can create a sensation of catching or giving way that lead to *functional* instability in a football player. The pathologies discussed below can also be found in conjunction with mechanical instability. Furthermore, many non-symptomatic changes can also occur that do not necessarily need treatment (Fig. 6).

Osteochondral Lesions of the Talus

Chronic sprains or acute injury can lead to damage of the articular surface. Sprains are the most common injury to the ankle in sport, and it is estimated that injuries to the talus occur in 6.5% of all ankle sprains [37, 44].

Footballers with cartilage damage to the talus will complain of swelling and medial or lateral ankle pain associated with training. Reports of an ankle sprain that has failed to completely heal by 8 weeks should raise suspicion of a talus cartilage injury. The patient may have a normal exam without evi-

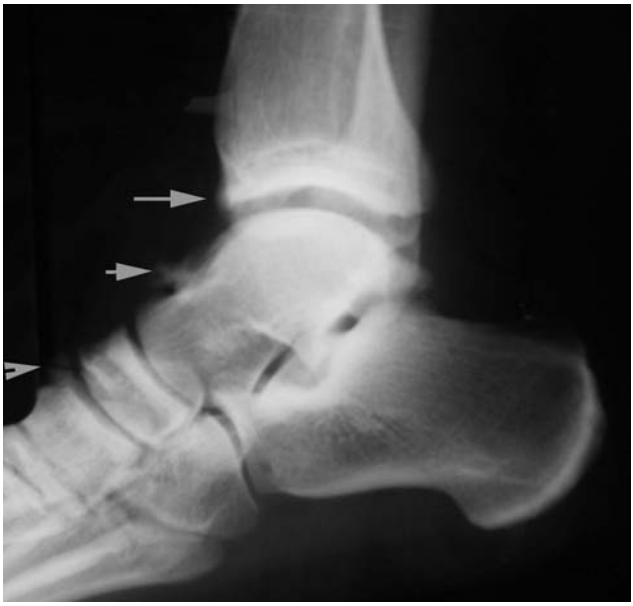


Fig. 6. Lateral ankle x-ray demonstrating chronic changes in the ankle of an asymptomatic 28-year-old national team football player. Talus osteophytes (*small arrow*), tibial osteophytes (*large arrow*), enlarged posterior tubercle (*large open arrow*), avulsion of the first cuneiform (*small open arrow*)

dence of instability; however, direct pressure over the talus with the ankle in plantar-flexion will elicit tenderness. Plain x-rays of the ankle can only identify 50–66% of lesions [45], and the only evidence may be an inconsistency in the trabecular bone of the talus (Fig. 7a). MRI is superior to x-rays to determine the location and size of the lesion (Fig. 7b). Bone oedema and potential stability of the fragment can be determined on MRI, which is useful for operative planning [46].

Robinson et al. demonstrated that traumatic lesions occur on both the lateral and medial aspects of the talar dome [47]. At arthroscopy, 20 of 65 patients had lateral dome injuries. The lateral injuries were more often delamination type injuries, and the average time to presentation was 1.5 years. Medial lesions occurred in 45 of 65 patients, 35 of the 45 were associated with a single traumatic event, and the average time to presentation was 3 years. This study underscores the importance of prompt MRI in cases with suspected talus lesions.

Conservative management of a stable lesion can be attempted with limited weight bearing in a walking boot for 3 months [44]. For élite or competitive footballers, ankle arthroscopy is recommended and is an effective means

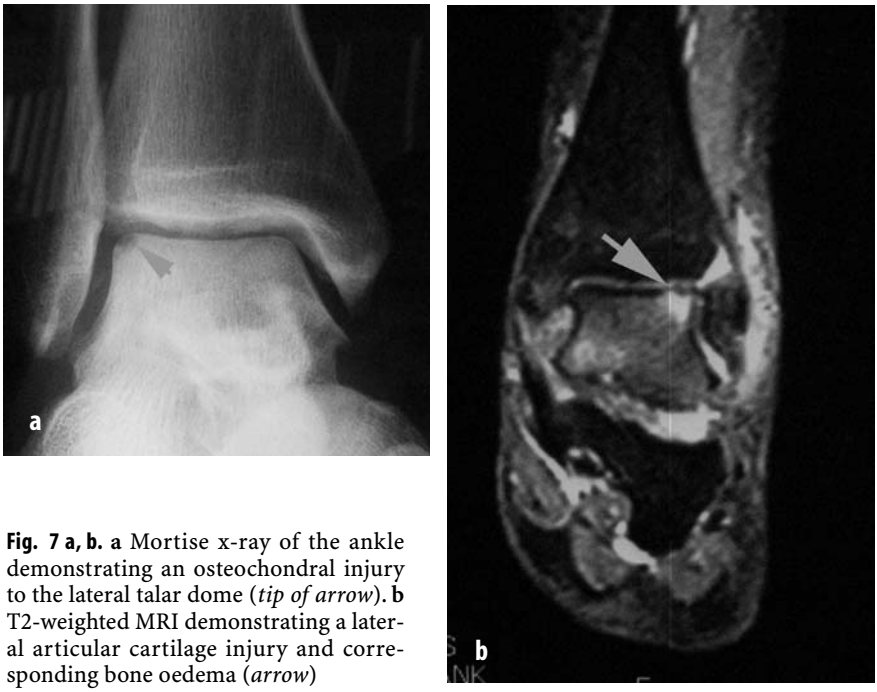


Fig. 7 a, b. **a** Mortise x-ray of the ankle demonstrating an osteochondral injury to the lateral talar dome (*tip of arrow*). **b** T2-weighted MRI demonstrating a lateral articular cartilage injury and corresponding bone oedema (*arrow*)

for diagnosis and treatment of lesions of the talus, with up to 85% of patients improving after arthroscopic drilling or curettage [48]. Takao et al. have shown improved outcomes if the damaged cartilage is removed and microfracture is performed compared with indirect, subchondral drilling [49]. Larger lesions that fail to improve 4 months after arthroscopy should be considered for osteochondral grafting or autologous chondrocyte implantation (ACI). Osteochondral grafting of defects have yielded 91–94% good to excellent results [50, 51]; however, arthroscopic ACI procedures using collagen or hyaluronic membranes have favourable results and will likely become the standard of care for these lesions [52].

Anterior Bony Impingement

The term *footballer's ankle* was first used by Morris and then McMurray to describe the debilitating osteophytes of the distal tibia and talar neck found in up to 60% of footballers [53–55]. The characteristic locations are the medial aspect of the talar neck and lateral to mid-line of the anterior distal tibia [56]. In 1992, Scranton and McDermott devised a classification system (Table 1) that aides in both clinical and operative decision making [57]. Footballers will have a palpable ridge of bone at the medial or central distal tibia, pain

Table 1. Classification of anterior ankle impingement (adapted from [57])

Grade	Radiographic and clinical findings
1	Synovial impingement & inflammation. Tibial spurs <3 mm
2	Grade 1 with tibial spurs >3mm. No talus spurs
3	Grade 2 with spurs on talus
4	Tibial and talar spurs with pantalocural arthritis

with dorsiflexion, and difficulty with cutting and push-off.

Lateral x-rays of the ankle may show the osteophytes but are often insufficient. Tol et al., demonstrated the effectiveness of an oblique anteromedial impingement radiograph, which should be performed when anterior bony impingement is suspected [58]. Chronic changes of the talar surface can occur from the osteophytes. Raikin et al. described a *divot sign* of the talar neck, and Kim et al. noted a *tram-track* fissure of the talar dome articular cartilage surface corresponding to the offending spurs [59, 60].

The aetiology of anterior impingement is likely a time-dependent exposure to football and is represented by two theories. First, the spur formation could be due to excessive stretching of the anterior capsule, resulting microtrauma, scar formation, and then ossification [57]. The second, and more likely, theory is that direct trauma causes the inflammation and resultant spurring. Tol et al. studied 150 kicking actions by 15 élite footballers in the laboratory and found that maximal plantar-flexion and stretching of the capsule occurred in only 39% of the kicks but that direct trauma to the anteromedial tibia occurred in 76% [61]. A follow-up cadaver study showed that the anterior capsule inserts on average 1.5 cm from the distal tibia rather than the area where the spurring is located [62]. Lateral instability can also contribute to spur formation, as Scranton et al. found that spurs were 3.4 times more prevalent in patients undergoing a Broström procedure [63].

Initial treatment can include rest, ice, and range-of-motion exercises for acute grade 1 cases; however, arthroscopic treatment has been shown to be effective [57]. Olesen et al. showed that removal of spurs correlated with decreased pain but only improved range of motion in 59% of patients [64]. Van Dijk et al. demonstrated that 90% of patients without joint space narrowing (grades 1–3) and 73% of patients with less than 2 years of pain improved significantly [65]. Therefore, it is recommended that players with new complaints and a positive exam undergo arthroscopic excision [66].

Posterior Impingement Syndrome

Chronic changes in the posterior aspect of the ankle can occur medially or laterally and include bony or soft-tissue pathology. Irritation or fracture of the os trigonum or enlarged talar tubercle can occur from a hyper-plantar-flexion injury where the structures are pinched between the tibia and os calcis (Fig. 8). Fracture of the os or disruption of the synchondrosis can lead to chronic pain when kicking [66, 67]. Initial treatment with rest and prevention of plantar-flexion with bracing will be effective in 60% of footballers; however, continued symptoms respond well to open or arthroscopic excision [68, 69].

Impingement of the soft tissues can occur both anterior and posteromedially. Stenosing tenosynovitis of the flexor hallucis longus, hypertrophy of the posterior capsule, and enlargement of the posterior inter-malleolar ligament can lead to chronic posterior pain that necessitates surgical intervention [70–72]. Rarely, anomalous muscles such as an accessory soleus or peroneus quartus can cause continued pain after a sprain. Best et al. found that excision is usually curative and allows a return to play [73].

Anterior and posteromedial impingement can be the result of chronic scarring of the medial gutter of the ankle where the deep fibres of the deltoid and capsule are pinched between the talus and the medial malleolus (Fig. 9). Calcification of the capsule can also develop and responds to arthroscopic excision [74, 75].

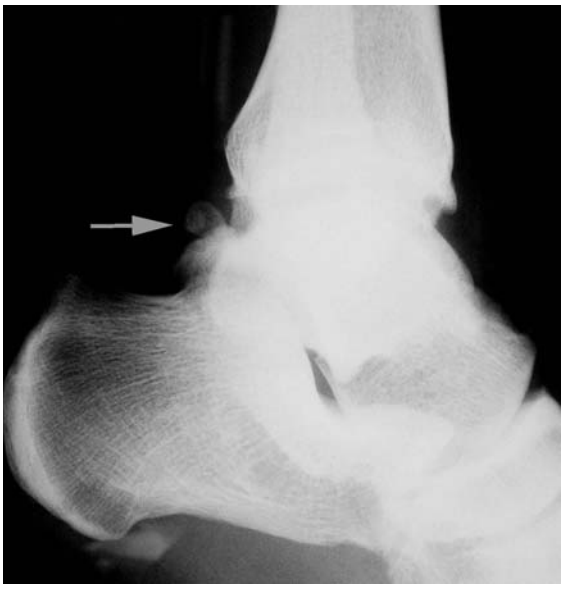


Fig. 8. Lateral x-ray of the ankle demonstrating an os trigonum in an élite football player (*arrow*)

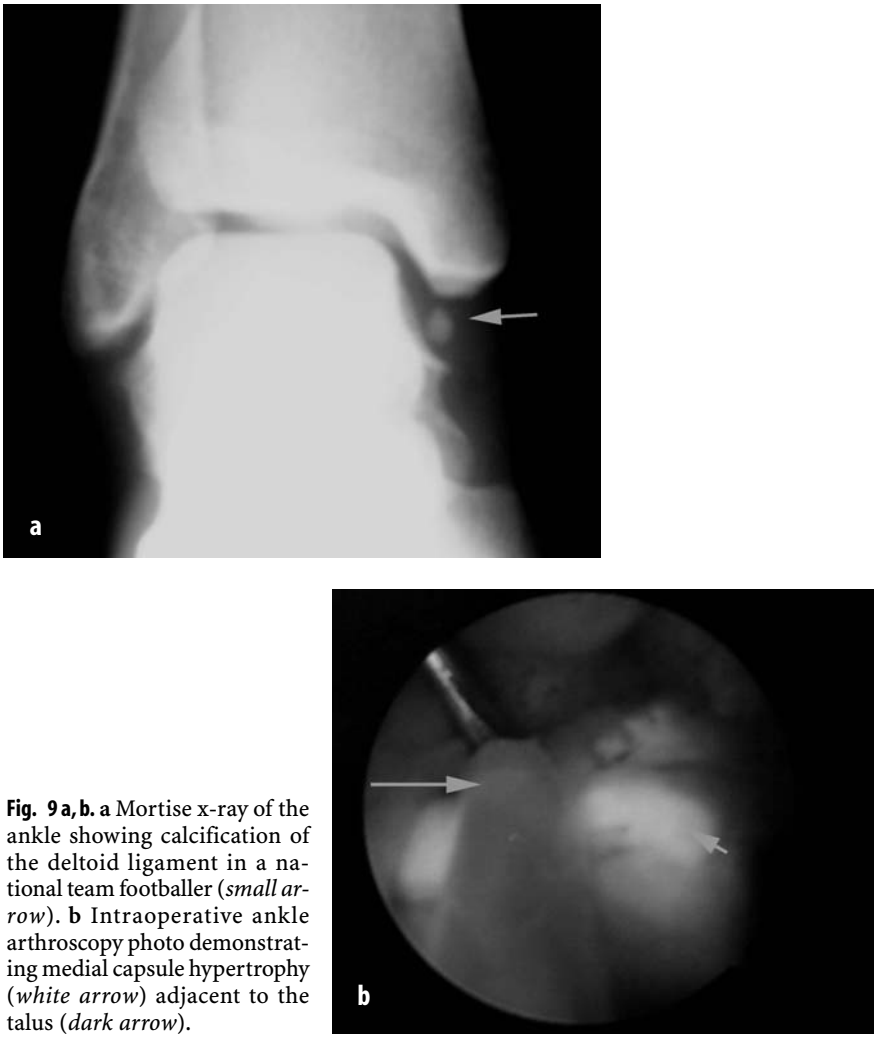


Fig. 9 a, b. a Mortise x-ray of the ankle showing calcification of the deltoid ligament in a national team footballer (*small arrow*). **b** Intraoperative ankle arthroscopy photo demonstrating medial capsule hypertrophy (*white arrow*) adjacent to the talus (*dark arrow*).

Anterior Soft-Tissue Impingement

Following repeated inversion injury, the anterolateral tissues, including the ATFL, Bassett's ligament, and capsule, can elongate and hypertrophy. In 1997, initial reports of MRI identification of the lesions reported low sensitivity and specificity [76, 77]; however, improvement to nearly 100% accuracy has been more recently reported [78–81]. McCarroll et al. described the *meniscoid lesion* in footballers as a mass of hyalinised tissue causing trapping between the fibula and talus, which responded to arthroscopic excision [82] (Fig. 10). Meislin et al. evaluated 29 cases of arthroscopy for synovial impingement and found good to excellent results in 26 patients at an average of 25 months [83].

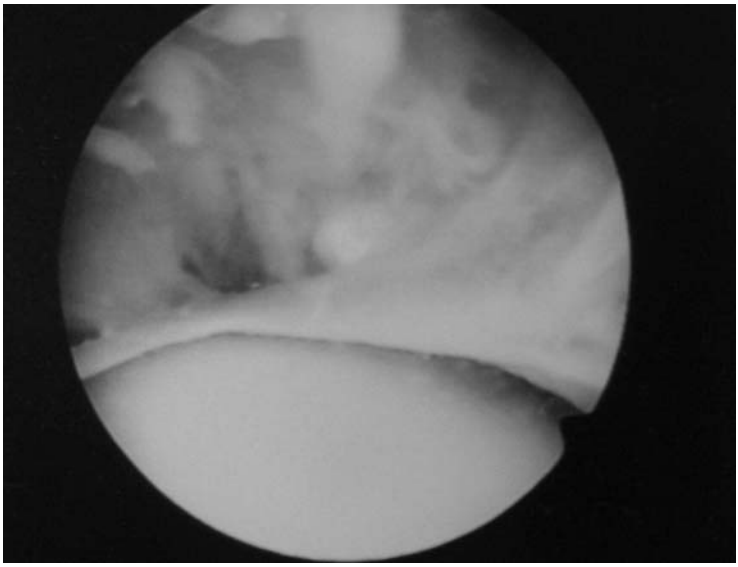


Fig. 10. Arthroscopic view of the lateral talus showing a large meniscoid lesion in a footballer adjacent to the talus

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Section II
Specific Injuries

Foot Problems

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Introduction

Injury frequency has drastically increased over the last few years in parallel with the development of sports activities; the growing popularity of sports and exercise is focusing attention on the injuries that may occur [1, 2]. Several epidemiologic studies have shown that among football players, strains, sprains, and contusions are the most common type of lesions, with an incidence respectively of 35–37%, 20–21%, and 16–24% of all traumatic disorders. The thigh (23–24%), the ankle (18–19%), and the knee (15–17%) represent the most common sites of injuries in football, while foot lesions are reported in 6–8% of cases [3, 4].

Ligament Injuries

Ankle Sprains

Ankle sprains are very common in football and usually result in lateral ligament injuries (anterior talofibular, calcaneofibular, and posterior talofibular ligaments). Clinically, ankle sprains are classified as mild (grade 1), moderate (grade 2), and severe (grade 3). Grade 1 lesion is characterised by ligament stretch without macroscopic tear, minimal swelling and tenderness without functional loss, and joint instability. Grade 2 injury presents partial ligament tear with moderate pain, swelling and tenderness, mild loss of function, and joint instability. Grade 3 lesion is a complete ligament rupture with severe pain, swelling, and marked functional loss and instability [5]. Pain at weight bearing and *giving way* feelings of the ankle are also reliable signs of ligament tear. Clinical inspection includes tests for ankle instability: with the anterior drawer test, we can evaluate a rupture of the anterior talofibular ligament; with the talar tilt it is possible to recognise a lesion of the calcaneofibular ligament. Radiologic evaluation includes x-ray, CT scan, and MRI. Standard

radiographs [anteroposterior (AP) and laterolateral (LL) views] and CT scans are useful to exclude fractures and osteochondral lesions; MRI gives excellent informations about soft-tissue damage. The treatment of grades 1 and 2 lesions is conservative and consists in a short period of immobilisation in a functional bandage followed by early motion and weight bearing exercises and proprioceptive training. Also, grade 3 acute sprain of the lateral ligaments of the ankle can be treated conservatively, and a secondary surgical repair is performed in case of persistence of symptoms.

Lateral ligament injuries, treated either conservatively or surgically, can cause chronic symptoms in 10–30% of patients. They include pain, persistent synovitis, swelling, and giving way feelings and are mostly related to ankle instability. In these cases, surgical repair of the ruptured ligament eventually associated with reinforcement with a periosteal flap from the lateral malleolus is carried out. Postoperatively, the ankle is immobilised with a cast for 4 weeks. At removal, motion and progressive weight bearing exercises are started with proprioceptive training. Sports activity is allowed about 3 months after surgery with an ankle brace [6].

Subtalar Sprains

Subtalar sprains are uncommon in the general population, but particular care must be paid in athletes playing indoor activities or playing football on artificial surfaces due to a strict relationship in these injuries between playing surfaces and footwear. The structures involved are the calcaneofibular ligament, the cervical ligament, and the inter-osseous talocalcaneal ligament. Injury mechanisms are forceful supination of the hindfoot with a plantar-flexed ankle leading to lesions of the cervical and inter-osseous talocalcaneal ligaments, forceful supination of the hindfoot with a dorsi-flexed ankle (calcaneofibular, cervical, and inter-osseous talocalcaneal ligaments lesions), or forceful supination of the hindfoot with a dorsi- and then plantar-flexed ankle causing a severe ankle/subtalar sprain with a complete rupture of the medial and lateral capsuloligamentous structures. Clinically, pain, tenderness, swelling with loss of function, and joint instability are observed, with a strict correlation to the entity of ligament tear [5]. Usually, subtalar ligamentous injuries occur associated to lesions of the lateral ligaments of the ankle. Symptoms of both lesions are similar, so clinical examination of a subtalar joint instability is very difficult and unreliable. Radiologic evaluation includes x-rays, CT scans, and MRIs. Treatment of lesions with partial ligament tears and mild subtalar instability is conservative and consists of a short period of immobilisation with a functional bandage followed by early motion exercises and proprioceptive training. In cases of complete ligament tears with severe joint instability, a surgical repair of the ruptured ligaments is per-

formed. The ankle is then immobilised in a below-knee, non-weight-bearing cast for 4 weeks. Sports activity is resumed at least at 3 months after surgery, always with an ankle brace [5].

Midfoot Sprains

Midfoot sprains represent a range of injuries of the Lisfranc ligament complex from a partial tear without displacement to a complete rupture with diastasis between the first and second metatarsal. In the literature, few cases in football players are reported [7]. In athletes, the lesion of the Lisfranc complex usually occurs when an axial load is applied to the foot that is plantar-flexed and slightly rotated. According to clinical examination, weight-bearing radiographs (AP and LL), and bone scan, Nunley and Vertullo [7] proposed a classification in three grades: Grade 1 lesion is characterised by pain in the Lisfranc area, inability to do sports activities, absence of displacement on weight-bearing radiographs, and a positive uptake on bone scan. In grade 2 injuries, there is a diastasis between the first and second metatarsal of 1 to 5 mm on AP radiograph without loss of plantar arch height on LL view. Grade 3 lesions present diastasis between the first and second metatarsal greater than 5 mm on AP radiograph and loss of plantar arch height on LL view. CT scan and MRI are helpful in patients with a displacement but are unable to recognise undisplaced lesions. In these cases, a bone scan is extremely useful because it can shows minor metabolic and blood-flow changes when the findings of other imaging techniques are negative. Grade 1 injuries are treated conservatively with a non-weight-bearing cast for 4 weeks, followed by an orthosis for other 4 weeks during which movement and proprioceptive exercises are started for a gradual return to sport activity. Grades 2 and 3 lesions are managed with open reduction and internal fixation with screws or K-wires in order to restore and maintain anatomic alignment of the Lisfranc joint.

Tendon Injuries

Achilles Tendon

Achilles tendinopathies and rupture are very common among football players at all levels.

Tendinopathies

With the term tendinopathies, we can include tendinitis (inflammation of the tendon) and tendinosis (inflammation associated with degeneration, calcification, and partial rupture of the tendon). In the literature, a rate of 15% of Achilles tendinopathies is reported in football, at an average age of 36 years

[8]. Usually, they are caused by an overuse during inappropriate or excessive training; calcaneal-Achilles-plantar impingement in the cavus foot or, in case of pronation, of the hindfoot; and mechanical conflict with footwear, especially in patients with an excessive posterior calcaneal prominence. Symptoms include pain that may limit even daily activities, evoked also by contraction of the triceps against resistance and a swollen nodular tendon in case of tendinosis. Treatment is either non-operative or surgical. Conservative management consists of rest, non-steroidal anti-inflammatory drugs (NSAIDs), physical therapy (ultrasound and/or laser therapy), rehabilitative therapy, and insoles to correct excessive pronation or supination of the foot and to raise the heel for distension of the tendon. Shock waves are effective, especially in cases of insertion disorders with ossification. In inflammatory disorders, non-operative management gives good results in 70–90% of cases, while the outcome is less predictable in patients with tendinosis. If conservative treatment fails, surgery is indicated and consists of tenolysis with removal of all the peritendinous and inflammatory tissue. In distal insertion disorders, resection of the calcaneal prominence and removal of the bursa are associated. In patients with inflammation with only slight degeneration of the tendon, a quick recovery without immobilisation and a return to sport activity is obtained in 90% of cases. In degenerative cases, non-weight-bearing immobilisation is required, and recovery may take up to 4 months. To achieve good results it is essential to treat any foot deformity: In anterolateral cavus foot, an uplift osteotomy of the first metatarsus is performed while pronation of the hindfoot is corrected by a calcaneal osteotomy.

Subcutaneous Rupture

Football has the highest rate of subcutaneous ruptures of the Achilles tendon (34%), followed by tracking-field and basketball. The cause of these ruptures is still discussed while several pre-disposing and determinant factors are recognised. The pre-disposing factors include hyperuricemia, systemic or local administration of steroids, and pre-existing tendon disorders. The determinant factors are contusion of the taut tendon in thrusting movements, sudden spurt, leaping and landing with the tendon elongated, excessive or improper training with high muscular power, or failure of the neuromuscular protection mechanism. Patients usually hear a snapping noise, which is associated with pain and walking inability. Examination reveals a dip at the site of the rupture; plantar-flexion against slight resistance is maintained by the action of the posterior tibialis and long flexors tendons. Thompson's squeeze test is always positive. Standard x-rays are carried out to detect calcaneous injuries while echography and MRI are useful only in doubtful cases since the diagnosis is mainly clinical. In athletes and active young persons, surgical treatment is preferred because it ensures an anatomical healing with less risk of recurrence and a

quicker recovery after a short period of immobilisation. Usually, a percutaneous suture technique is performed. Postoperatively, a non-weight-bearing brace at 20° of plantar-flexion is applied for 4 weeks, then partial weight bearing and progressive motion exercises are started; complete rehabilitation and return to sport is achieved at the end of the fourth month [8].

Peroneal Tendons

Peroneal tendon injuries include ruptures and acute/chronic subluxations. Subcutaneous ruptures are very rare; the literature reports only a few cases of peroneus longus rupture in footballers as a consequence of an inversion ankle sprain [9]. Diagnosis is often delayed and suspected after a long period characterized by painful and weaker eversion and tenosynovitis. Treatment consists in an end-to-end suture and tenodesis with the adjacent peroneus brevis tendon.

Acute traumatic or chronic subluxation of the peroneal tendon is uncommon. Usually, it occurs in skiing practice, but cases in football and basketball players are also reported [9]. The mechanism of injury is a forceful dorsi-flexion of the ankle with the foot in eversion associated with a forceful contraction of the peroneal muscles. In acute trauma, the athlete feels a snap sensation on the posterolateral aspect of the ankle, with subsequent pain and swelling. Symptoms quickly become mild and difficult to differentiate from those of a lateral ankle sprain, so the injury tends to become chronic. In recurrent subluxations, there is pain with a snapping sensation in the ankle during activities such as walking or running on uneven ground. In acute trauma, non-operative treatment often leads to recurrent dislocation, so open reduction and repair of the peroneal retinaculum is recommended [10]. In chronic dislocations, surgery consists of deepening of the fibular groove associated to soft-tissue repair with the deep fascia or periosteum. If the tissue is insufficient, a posterior bone block procedure can be performed. After surgery, a non-weight-bearing brace at the neutral position is applied for 4 weeks, followed by progressive weight-bearing with a brace and motion exercises for other 3 weeks [11].

Posterior Tibial Tendon

Posterior tibial tendon dysfunction is sometimes observed in footballers. Tenosynovitis is far more frequent than rupture of the tendon. The dysfunction is usually related to repeated minor traumas and overuse. Symptoms are pain and tenderness behind the medial malleolus caused by active inversion of the foot. Treatment is conservative and consists of rest, ice, NSAIDs, and physical therapy. In the less-frequent cases of tendon rupture, patients present a painful flat foot, valgus of the hindfoot, and progressive loss of function.

Positive *single-heel rise test* and *too many toes sign* are useful for diagnosis. Treatment is surgical with an end-to-end suture and, eventually, plasty with the flexor digitorum longus or the flexor hallucis longus tendons is performed [9].

Osteochondral Lesions

Osteochondral lesions of the talus are common in sports injuries and are reported in 6.5% of all ankle sprains [12]. A trauma in forced inversion or eversion of the foot causing a compression of the lateral or medial talar dome against the lateral or medial malleolus can damage not only the capsuloligamentous structures but also the joint cartilage and the subchondral bone. Symptoms include pain, persistent effusion, locking or giving way of the joint, and synovitis. Standard x-ray views, CT scan, and MRI must be carried out for correct diagnosis and staging of the lesions. Over the years, several classifications of osteochondral lesions have been proposed according to radiological, arthroscopic, or instrumental data. The Berndt and Harry classification in 4 stages [13] gives a good correlation with the different treatment techniques:

- stage I: compression of the subchondral bone;
- stage II: a partially detached osteochondral fragment;
- stage III: a complete detached osteochondral fragment without displacement;
- stage IV: a displaced osteochondral fragment.

Stage I lesions can be treated conservatively. Stage II, III, and IV lesions require a surgical procedure consisting of arthroscopic removal of the fragment and repair of the defect. The methods of bone marrow stimulation (chondroabrasion, shaving, perforation, and micro-fractures) lead to the formation of a repair tissue constituted by fibrocartilage that does not possess the same biological and mechanical features of the hyaline cartilage. These techniques are indicated in the treatment of painful stage I or stage II lesions, always less than 1.5 cm^2. In these selected cases, satisfactory results are reported in a good percentage of patients.

Larger lesions do not respond as well to these procedure; in fact, because of the poor mechanical resistance of the repair tissue, a secondary osteoarthritis of the joint in the mid- to long-term can easily develop. In lesions more than 1.5 cm^2 or with failed previous surgery, two techniques are able to restore the hyaline cartilage surface: mosaicplasty and autologous chondrocyte transplantation. Mosaicplasty consists in the transplantation of at least one autologous osteochondral plug harvested from a non-weight-bearing surface of the knee and then press-fitted into the articular prepared defect of the ankle. Excellent results are reported in 94% of patients at 2–7 years follow-up [14]. The problems and limits of this procedure are related to the pos-

sible secondary pathology at the donor site, to the technical difficulties of filling an irregular-shaped defect with round osteochondral plugs, and to the formation of fibrocartilage repair tissue between the transplanted cylinders. Furthermore, the integration of donor and recipient hyaline cartilage can be difficult due to the different mechanical properties and varying thickness of the transplanted and recipient tissues [12].

The autologous chondrocyte transplantation procedure, developed and proposed by Brittberg et al. for the treatment of osteochondral lesions of the knee [15], is also effective for treating the lesions of the talus with excellent clinical results and hyaline cartilage formation in the transplanted area [12]. This technique is a two-step procedure. The first step consists of harvesting cartilage from the lesion and is performed arthroscopically. The tissue is sent to the laboratory for chondrocyte culture. After approximately 30 days, autologous chondrocytes can be transplanted. Initially, the chondrocytes were injected in suspension under a periosteal flap sutured to the healthy margins of the lesion. This procedure was complex and often difficult to carry out and always required an open trans-malleolar approach medial or lateral in relation to the location of the defect. Research in tissue engineering in the field of biomaterials has allowed these difficulties to be overcome; in fact, now it is possible to develop a line of autologous chondrocyte on three-dimensional scaffolds. In this way, it is possible to perform all the procedure arthroscopically, reducing complications and difficulties with early functional recovery. Postoperatively, weight bearing is contraindicated for 4 weeks, then partial weight bearing and progressive motion exercises are begun. Return to sport activity is usually achieved after 1 year from the transplantation.

Osteochondral lesions in the other joint of the foot are very rare. Kinoshita [16] reported two cases of osteochondral lesions of the proximal phalanx of the great toe in young footballers. Both cases were treated with removal of the free fragment and cancellous bone chips grafts to fill the defect with complete recovery in both cases.

Impingement Syndrome

Impingement syndrome is a condition in which additional bone or soft tissue is entrapped between the anterior aspect of the distal tibia and the talus during dorsi-flexion of the ankle (anterior impingement) or between the posterior rim of the distal tibia and the talus in plantar-flexion of the ankle (posterior impingement). This increasing conflict induces pain, swelling, chronic effusion with progressive reduction of the range of motion, and sport inability [17].

Anterior tibiotalar impingement was first reported by Morris in foot-

ballers and called *athlete's ankle* [18]. Then McMurray defined it as *football ankle* because it was always observed in football players [19]. They described osseous exostoses of the anterior rim of the tibia and the dorsal talus. These spurs may be secondary to traction injuries of the anterior joint capsule in repeated forced plantar-flexion, or they may be the consequence of direct osseous impingement in forced dorsi-flexion of the ankle or of post-traumatic calcification [17]. X-ray weight-bearing AP and LL views associated with LL radiographs at maximal plantar and dorsal flexion of the ankle and CT scan are carried out for correct diagnosis and staging of the lesion. Scranton and McDermott [20] reported a classification in four grades based on the radiographic aspects of the lesion on a standard LL view. Grade 1 impingement presents a tibial bone spur of less than 3 mm; grade 2 shows a spur larger than 3 mm with a sulcus (divot sign) in the talus; grade 3 lesion presents severe exostosis, often fragmented, associated with a spur in the dorsal talus; in grade 4 impingement, there is a complete severe arthritic degeneration of all the joint. Arthroscopy represents the primary treatment of these lesions. Grade 1 and 2 lesions can be successfully treated with arthroscopic shavers and burs. In grade 3 impingement, after arthroscopy, it is often necessary to perform a mini-arthrotomy in order to remove the larger spurs with chisels and the loose bodies. In grade 4 lesions, an arthroscopic shaving may represent the option to delay more appropriate but also more aggressive and invalidating therapeutic techniques such as ankle arthrodesis, prosthesis, or massive articular allograft (bipolar tibiotalar osteochondral shell allograft).

A less frequent cause of the anterior tibiotalar impingement in footballers is the so-called *meniscoid lesion* described by Wolin [21]. This is the main cause of an anterior fibrous impingement and is often the result of an inversion sprain of the ankle with tear of the anterior talofibular ligament and migration of the torn fragment in the articular space between the lateral malleolus and the talus. At arthroscopy, we can observe a band of fibrous tissue mimicking a meniscus. Arthroscopic shaving represents the treatment of choice with excellent results [17].

Repeated traumas in plantar-flexion of the ankle can lead to a posterior talotibial impingement. Several lesions are described as being responsible for this syndrome: calcification or fracture of the os trigonum or of the posterior tubercle of the talus and synovitis of the flexor hallux longus. If the conservative treatment fails, surgical removal of the os trigonum or tenolysis of the flexor hallux longus must be performed [13].

Stress Fractures

Stress fractures are partial or complete fractures caused by repeated, cyclic, non-violent forces acting on a normal bone. The continuous and excessive strains applied to the bone can induce a predominant osteoclastic activity of tissue resorption compared with the osteoblastic phase of bone formation, with a progressive weakening of the tissue. Change or increase in training or change in surfaces, shoes, or local mechanics of the foot, are possible pre-disposing factors for stress fractures. Clinically, there is pain that arises after a period of strenuous activity. Initially, the pain can decrease with rest but if not recognized becomes chronic and exacerbated by weight bearing. Standard radiographs are often negative in the initial phase and are capable of recognising only late alterations of bone while bone scan, CT scan, and MRI can confirm the clinical suspect of stress fractures in the early phase (Fig. 1). In footballers, the metatarsals represent the most common site of stress fractures while lesions are less frequent in the navicular, calcaneus, and malleoli and are rare in the proximal phalanx of the big toe [13, 22]. Treatment consists of rest, ice, NSAIDs, and immobilisation for a short period followed by non-weight-bearing exercises. Closed reduction and fixation with K-wire or screw is recommended when conservative treatment fails and for stress fractures at the base of the fifth metatarsus and at the navicular because they tend to heal over a long time and have a high rate of non-union due to poor vascularization [13].

Fractures

In footballers, foot and ankle fractures are uncommon; however, fractures of the base of the fifth metatarsal are quite frequent. These fractures can be divided into two groups: fracture/avulsion of the apophysis, and fracture at the metaphyseal junction (Jones's fracture). The mechanism of the lesion is a trauma in the vertical direction with the foot in inversion and plantar flexion and a reflex contraction of the peroneus brevis, as it occurs in falling after a jump on uneven ground [23]. The treatment is conservative, consisting of a cast for 4 weeks in the fracture/avulsion of the apophysis while in the Jones's fracture, a surgical procedure with reduction and fixation with K-wire or screw is recommended (Fig. 2).

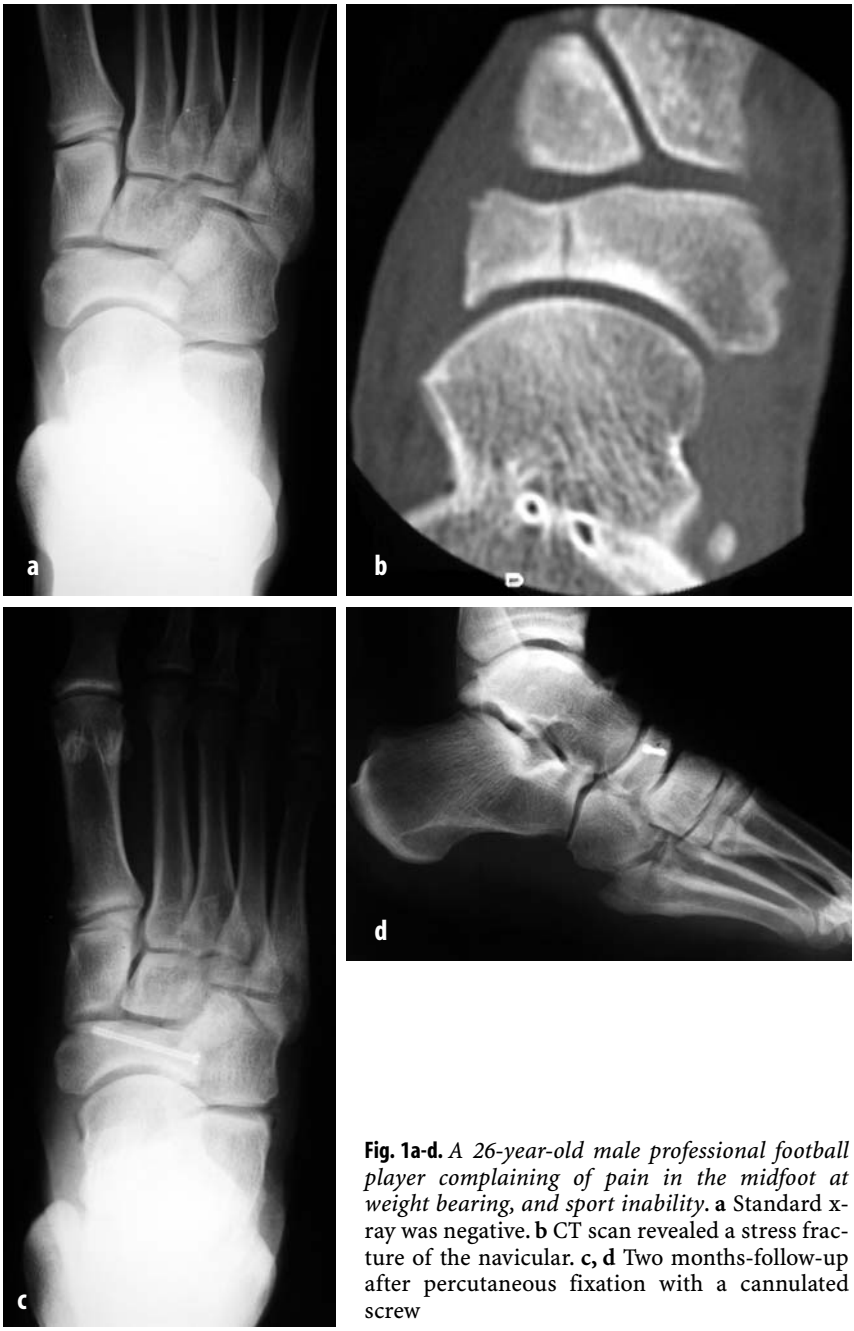

Fig. 1a-d. *A 26-year-old male professional football player complaining of pain in the midfoot at weight bearing, and sport inability*. **a** Standard x-ray was negative. **b** CT scan revealed a stress fracture of the navicular. **c, d** Two months-follow-up after percutaneous fixation with a cannulated screw

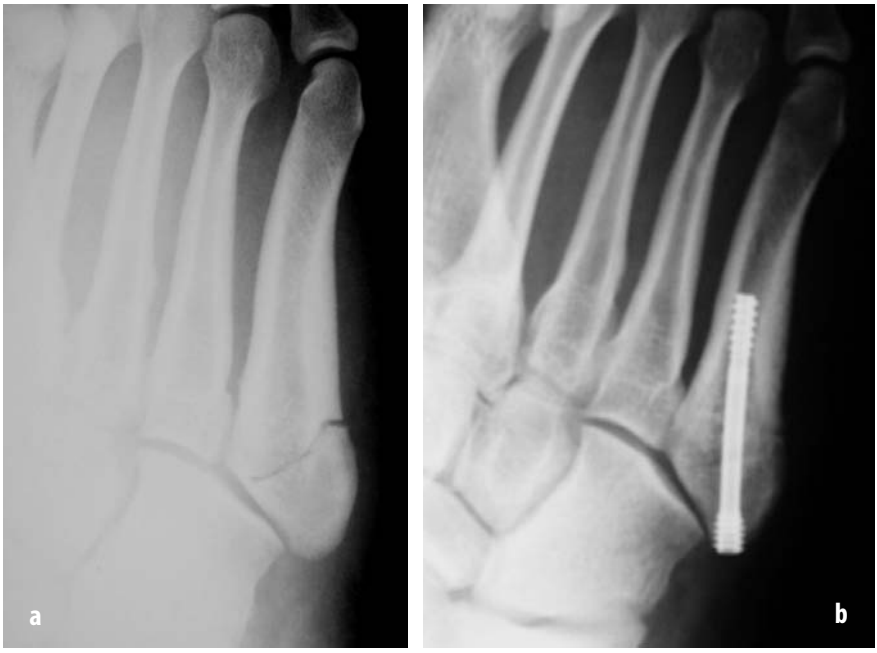

Fig. 2a, b. a A 27-year-old male professional football player with fracture of the proximal fifth metatarsus. **b** Two months-follow-up after percutaneous fixation with a cannulated screw

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Stress Fractures

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Introduction

Overuse injuries develop when repetitive stresses to bone and musculo-tendinous structures are applied and cause changes that damage tissue at a greater rate than that at which the body can repair itself. A combination of extrinsic factors, such as training errors and environmental factors; and intrinsic or anatomical factors, such as bony alignment of the extremities, flexibility deficits, and ligamentous laxity, predisposes athletes to overuse injuries [1]. Lower-extremity stress fractures are common injuries most often associated with participation in sports involving running, jumping, or repetitive stress, such as in soccer [2].

Stress fracture is the consequence of bone structure failure after repeated micro-traumas. The effect of repeated injuries is stronger than bone remodelling potential. Different populations can be affected, and different patterns of insufficiency fracture can be seen related. Also among athletes, differences in terms of sport specialty, morphotype of the athlete, age, and race, must be considered [3, 4]. Many different factors are involved in generating a stress fracture [5]:

- Running is the most important factor in stress fracture of the inferior limb; 84% of inferior-limb stress fracture in athletes is related to running. Furthermore, a sport-specificity of injury must be considered. Specific actions involved in a particular sport determine site and type of fracture. As described, the specific way of running in soccer influence the typical stress fracture related to this sport (of the metatarsal bone).
- Frequency, speed, and amount of load, as well as recovery time between load phases, are important. In athletes, risk of stress fracture increases proportionally to load increase. This risk is related to muscular fatigue: when the buffer action of muscle fails, the impact of injuring force on bone increases. The buffer action of muscles is related to load application on bone, including control of piezo-electric activity and distribution of electrical changes. A concentration of positive changing has been consid-

ered a trigger factor due to stimulation of osteoclastic activity. However, muscular strength can be a risk factor, as well. For instance, in case of traction force at the muscle insertion site, the stronger the muscle, the higher the risk of stress injuries (olecranon in throwers, fifth metatarsal base in soccer players).

- Vectors of load application related to bone structure are also involved. Individual anatomic features (limb length, load axis) can condition biomechanics and kinematics of sport. Abnormalities in alignment can also be important in stress-fracture pathogenesis (flat foot, valgus knee, hyperpronation).
- Quality of bone related to age and gender (hormonal influence) [6]. Stress fractures are overall more frequent in women, basically related to nutritional habits and menstrual abnormalities. Furthermore, adolescent age is a risk factor for the following reasons: articular cartilage in growing subjects (especially in elbow, knee and ankle) is particularly vulnerable; overload injuries at this level are quite frequent. Long bones and tendon-muscle unit growing speeds are not the same. This fact can lead to muscular imbalance and consequently to joint stiffness and high-traction tension at apophysis [4].
- Ground conditions are extremely important in soccer, especially in lower series where terrain can be uneven and re-covered with uniform grass. Terrain is also subjected to changes due to weather conditions (harder surfaces in winter time) [7].
- Other controversial issues concern materials and shoes and particularly their shock-absorbing power, which could protect the skeleton from injuring stresses.
- Malalignment of the lower extremity, including excess femoral anteversion, increased Q angle, lateral tibial torsion, tibia vara, genu varum or valgum, subtalar varus, and excessive pronation, are frequently cited as predisposing to lower-limb overuse injuries [8].

Diagnosis

Diagnosis of a stress fracture may be easy if the clinical suspicion is high in case of bony pain secondary to high physical performance. The only symptoms may be pain and functional impairment, both having different clinical aspects.

Pain

Pain can be progressive and insidious, increasing with sport activity, especially if the athlete has an increase in:

- Length of sport activity;
- Intensity;
- Frequency in training;
- Equipment;
- Training playground.

Pain is generally localised and increases during sport activity, decreasing with rest, and progressively presenting over 2–3 weeks. In the complete phase of evolution of the fracture, pain may be so intense as to prevent every kind of sport participation where terrain can be uneven and uncovered with uniform grass or on harder winter surfaces.

Imaging

Conventional x-rays can help in diagnosing the stress fracture if the following radiological patterns are seen:
- Parosteal bony formation;
- Sclerotic area;
- Bone callus;
- Fracture line;
- Cancellous bone sclerosis.

The standard radiological aspect is quite typical: sclerosis and parosteal bony callus and the dreaded black line – a cortical radiolucency line at the side of the bone submitted to tension. The fracture line cannot be considered the "must be present" sign. Although pathognomonic, these radiological patterns are not frequent; they can appear either late after the clinical suspicion or not at all [9].

Bone Scan

In case of high clinical suspicion, when stress fracture is not visible on x-rays, a bone scan with 99m Tc-MDP or EDP is useful because of its sensitivity in spotting high bone remodelling areas (Fig. 1). Some factors affect the radionuclide concentration, such as blood flow, extra-cellular compartment, local enzymatic activity, and quality of osteoid matrix. Nowadays, the triphasic method is used. The first phase of vascularisation shows the blood supply, the second shows the soft tissue and extra-vascular bone tissue activities, and the third – the metabolic phase – detects the bony formation activity. Comparison of the three allows a dynamic evaluation of stress injuries that would otherwise be impossible in a static mode. The main limitation of this method is non-specificity: a concentration of radionuclide can be seen in painless areas. These are areas of "stress reaction", a positive compensatory reaction to overload [10].

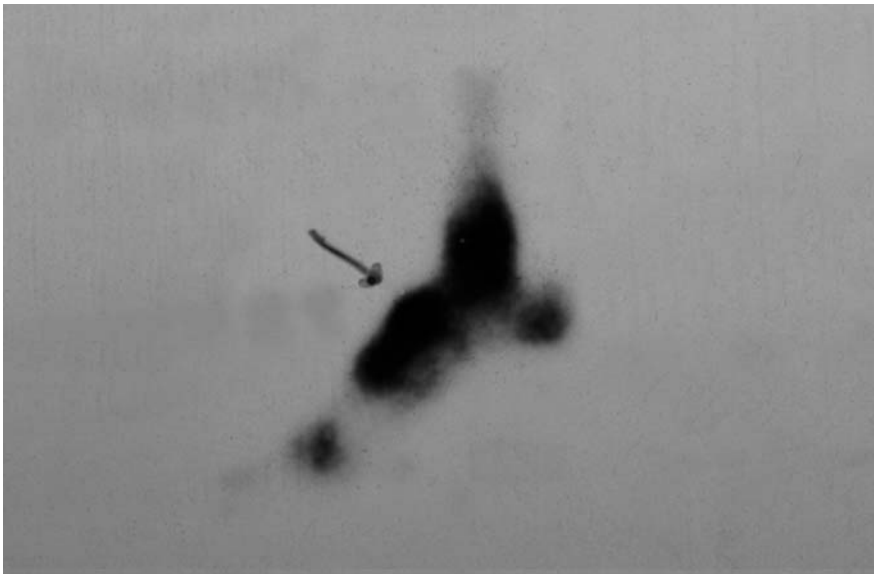

Fig. 1. In bone scan, the radionuclide accumulation is the expression of the sensitivity of this examination in bone remodelling areas

Computed Tomography (CT)

Once the area is identified in which the metabolic increase is present, the lack of specificity left by the bone scan can be fulfilled by computed tomography (CT). Some pathognomonic radiological alterations that can be detected by CT are:

- Sclerosis;
- Periosteal and endosteal reaction;
- Trabecular reaction;
- Fracture line (if present).

Thus, CT must be performed in the positive bone-scan hyper-captation areas only if painful. Otherwise, in stress reaction painless areas, it would mean needless x-ray exposure. CT is diagnostic especially in navicular tarsal bone, talus, and vertebral lamina fractures [11].

Magnetic Resonance Imaging (MRI)

Magnetic resonance imaging (MRI) can now be considered the gold standard in stress-fracture diagnosis. MRI can spot bone marrow abnormalities, soft tissue oedema, as well as any blooding phenomenon and for these reasons is useful in diagnosis of even very small stress reactions [12]. Stress fractures are spotted early by decreased bone marrow intensity in T1- weighted images.

Intensity is increased in T2-weighted images after gadolinium injection and in short tau inversion recovery (STIR) sequences. Periosteal oedema can be also detected, as well as the fracture line. The latter appears as a low-density intramedullary line that prolongs in cortex bone. When the bone repair phase begins, a decrease of intensity is visible in T2-weighted sequences, as well as the bony callus phase [13].

Tibia and "Shin Splints"

In the tibia, pain may present in an acute or subacute manner accompanied by swelling, oedema, and clear dolorability that may be extended to a very large area, with peak pain intensity in the actual fracture sight. The differential diagnosis between shin splint, tibial medial syndrome, and compartmental syndrome may be difficult. The tibia has 3 different areas that are most commonly involved:

- Anterior and anteromedial cortical bone;
- Posteromedial cortical bone at the superior third;
- Medial malleolus.

In soccer, the most commonly involved area is the medial shaft, even though the proximal metaphysis or the medial malleolus may be rarely involved [3]. The pathogenesis is directly connected to the shape of the tibia, the distribution of muscular forces, and the kind of functional weight-bearing of the athlete. Muscular groups occupy the anterolateral and the posterior district of the tibia while the anteromedial cortical has no muscles. Furthermore, the tibia has an anteroexternal curvature, evident as more varus is present. In addition to the arch principle (the tibia) stretched by its chord (the muscles), loading forces and muscular contraction generate compression vectors in the concave side (posteromedial cortical) and distraction forces in the convex side (anterior cortical). This situation is increased in those athletes with great flexor strength with triceps suralis hypertrophy.

On the anterior cortical, "tensile" fractures are produced, with transversal rim fracture, cortical hypertrophy, and difficult healing. "Compressive" fractures in the posterior cortex (especially proximally) are produced, with hypertrophy cortico-spongeous, difficult visualisation of the fracture rim, and easier healing.

The stress fracture of the medial malleolus (Fig. 2) is connected to the bearing: pes cavus without pronation stresses this area of the tibia with astragalus impingement [14].

There is another situation connected to overuse injury of the tibia with similar pathogenesis and clinical aspects. The "shin splint" is a situation characterised by pain and oedema at the distal two third of the tibia in differential diagnosis with stress fractures, insertional tendinopathies, and compart-

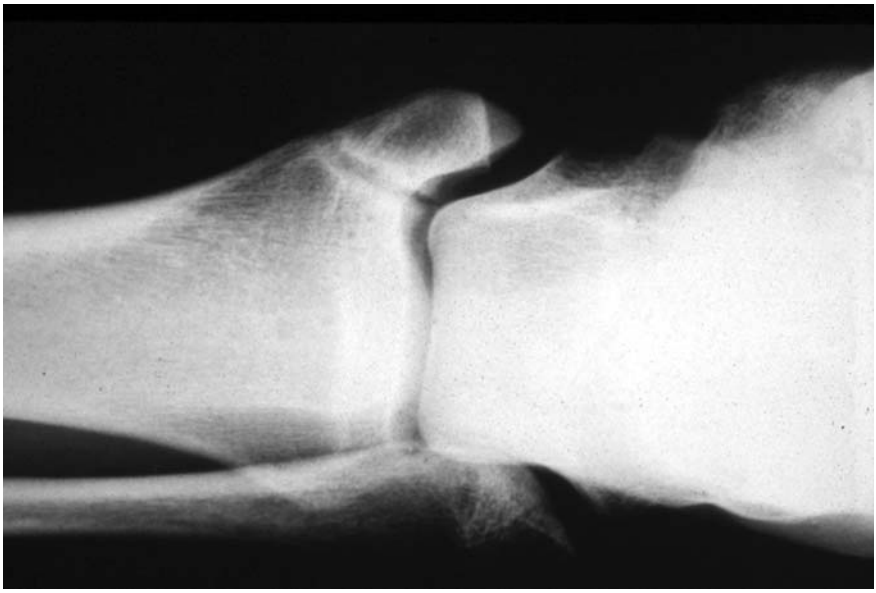

Fig. 2. Stress fracture of medial malleolus occurs mainly in pes cavus without pronation

mental syndrome. The American Medical Association (AMA) defines shin splints as "leg pain derived either from running on hard ground, or from excessive dorsiflexion of the foot". The AMA recommends that this definition be reserved to muscle-tendinous problems as opposed to fractures or ischemic pathologies. In the shin splint definition, we may find anterior overuse pathologies such as anterior tibialis tendinopathies, but many authors suggest that this definition should be indicated for posterior tibialis tendinopathy, the soleus, and finger flexors: they prefer to define this situation as "medial tibial syndrome", which includes:

- Periostitis and insertional tendinopathies;
- Compartmental syndrome;
- Stress fractures.

Actual general agreement is to consider periostitis and posteromedial insertional tendinopathies as the only cause of tibial medial syndrome, just for the differences in prognosis and therapy management.

Metatarsal

In the metatarsal bone, pain may start in a sub-acute way or may be acute after a functional overload (up-hill running): in any case, the fracture is secondary to previous alteration of the bone and may be either in one side on the cortical or circumferentially.

Second, Third, and Fourth Metatarsals

Stress fractures of the metatarsal bones occur in all sports that have running as the fundamental athletic activity. Functional overload caused by various activities required by soccer players (running, change of direction, kicking) favours the onset of these fractures, which are frequently localised in the neck and distal metaphysis of the second and third metatarsal. The second is the longest metatarsal bone, through which the entire propulsion force is generated, particularly if its difference in length with the first metatarsal is remarkable. (The metatarsus brevis, the metatarsus adductus; it should be noted that these anatomical situations are also correlated with the fractures of the navicular bone).

As a consequence of the repetitive application of forces generated and unloaded through it, the second metatarsal can positively respond with a reactive cortical hypertrophy from remodelling or sustain a duration lesion of the distal metaepiphysis, with a callus that appears after 2–3 weeks from the beginning of symptoms.

A pronated foot, associated with calcaneous valgus, is responsible for prolonged support of the metatarsal heads, with increased quantity of strength of load that they must absorb. Similarly, a foot cable constitutes the anatomical sub-stratum for an ineffective amortisation and adaptation of the foot to the ground, with increase of mechanical solicitations on the metatarsal.

Fifth Metatarsal

The pathogenesis of a fracture of the fifth metatarsal base as well as clinical features are different from fracture of the metatarsal bones (Fig. 3). This type of fracture has been described by Jones [13], and as such, it is now defined. In soccer players, this type of fracture is particularly frequent because of jumping, repetitive tackles, and changing of directions. It is important to note that the Jones fracture, which occurs in the proximal metaphysis, must not be confused with more proximal partial separations: in the past, this has generated confusion around their natural history and the best method of treatment. Besides, this fracture may be presented in acute form, which happens when the foot is bent in plantar-flexion and inversion and in a chronic form when traction of a smaller entity working on the brief peroneus with inversion of the foot provokes a rim of fracture in an area affected by pain from previous functional overload. An acute fracture does not have radiological signs of previous injury, as intramedullary sclerosis and cortical hypertrophy, with narrowing of the channel or even obliteration of the same, situation leading to pseudoarthrosis.

Torg et al. classified these lesions into 3 categories: acute (type I), late of consolidation (type II), and pseudoarthrosis (type III) [16]. Type I lesions are

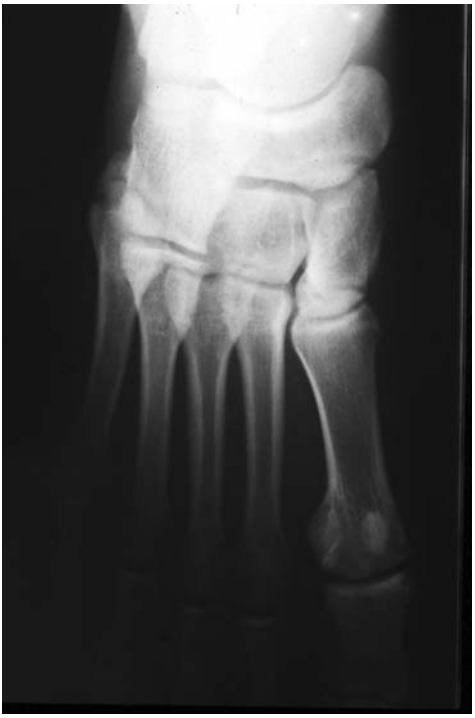

Fig. 3. Base of fifth metatarsal fracture may be acute when the foot is bent in plantar-flexion and inversion or chronic when traction of a smaller entity works on the brief peroneus with inversion of the foot

considered stress fractures in the precocious phase; type II, in phase of delay, a widening of the fracture rim is underlined with medullar sclerosis; type III have the characteristics of pseudoarthrosis. This classification was formulated in accordance with treatment, as we shall see later.

Treatment

Stress-fracture treatment may be either conservative or surgical, depending on the involved bone, the sight of the fracture, length of injury time, or the athlete's activity level. In a tibia stress fracture, in case of limited dimensions, therapy is generally conservative. A cast may be useful in resting, controlling pain, and favouring the bone remodelling processes. Bone electrostimulation treatments (capacitive fields) stimulate neo-angiogenesis processes, especially in the early phases [15]. Sport activity suspension is generally 6–12 weeks.

Conservative treatment may be different, as we said before, and depends on the fracture sight. In the posterior proximal cortex, rest and muscular reconditioning is important, with exercises of triceps stretching, anterior reinforcement, and proprioceptive exercises. If the proximal anterior cortex is

involved, an orthesis must be added, which may correct the dynamic contact with the ground that may be altered and be the cause of the fracture. In soccer players, it is generally the medium shaft of the tibia that may more easily sustain a stress fracture. In this case, conservative treatment, as suggested before, may be ineffective, and the fracture may not heal. Surgical treatment has different options:

- Bone perforation (Fig. 4); removal of fracture rim with bone transplantation; cast for 4 weeks
- Endomedullary blocked nailing.

When sclerosis and cortical hypertrophy are present, it may be necessary to use both surgical procedures in order to obtain the biological effect (endosteal healing, angiogenesis) and the mechanical effect (stiffness of the system without using a cast).

In case of a metatarsal fracture, localisation is typically at the medium shaft, with callus hypertrophy and easy and speedy healing. Treatment is generally conservative (cast). It may, however, be different in case of fracture of the base of the fifth metatarsal (Jones fracture). According to Torg et al.'s classification [16], we suggest that therapy may be:

- Conservative: type I fracture
- Surgical: types II and III fractures.

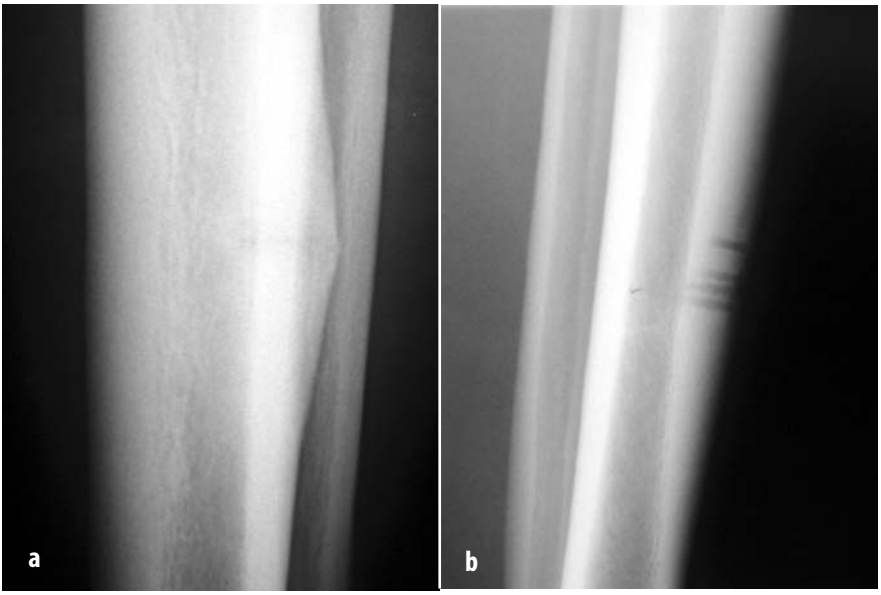

Fig. 4a, b. Surgical treatment of tibia stress fracture with bone perforation

A cast and bearing extension is recommended until healing is obtained – approximately 6 weeks. Considering this and the athlete's activity level, we prefer a surgical approach with endomedullary screw or compression plate because of the possibility of praecox full weight bearing and easier return to sport activities – generally in 6–8 weeks [16].

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Rehabilitation after Football Injuries

Suzanne Werner

Sports Rehabilitation in General

Physically active individuals expose themselves to the risk of considerably higher loads on their bodies compared with physically inactive individuals. Therefore, high demands are put on the rehabilitation of an active individual after injuries or surgery. Sports rehabilitation should strive to obtain not solely the same physical condition as before the injury but, rather, a better one in order to try to prevent re-injury or a new injury. Exercises for improving range of motion, muscle strength, muscle flexibility, agility, proprioception, balance, co-ordination, and conditioning should be included in the rehabilitation, irrespective of type of sports injury. Furthermore, the rehabilitation programme should be tailored to each individual based on both physical conditioning and his or her specific needs. The rehabilitation protocol design should include the specific demands that the individual athlete is exposed to in his or her sport.

The main goal of rehabilitation after sports-related injuries is to safely return the athletes as soon as possible to their pre-injury level of physical activity. Sports activity puts heavy demands on physical fitness and conditioning, and it is especially important to pay attention to them when a recently injured athlete is returning to sport. Rehabilitation should lead to normal function before the athlete is permitted to return to sporting activities. To make this possible, fast and correct acute care as well as optimal treatment and rehabilitation is required. This means concerted effort to eliminate pain and obtain full range of motion, muscle strength, co-ordination, and sports-specific performance.

The rehabilitation protocol should preferably be divided into different phases, such as the acute phase, the rehabilitation phase, and the sport-specific phase. These phases are closely linked, each with a variety of specific goals. For successful clinical outcome and decreased risk of re-injury, it is important that these goals are attained before the athlete is permitted to move to the next phase.

Rehabilitation after Common Football-Related Injuries

The definition of football injuries means an injury that occurs during football and leads to absence from practice or games. The most common injuries are ankle sprains, knee sprains, and muscle strains.

Lateral Ankle Sprain

The lateral ankle sprain is one of the most common injuries sustained in sport and physical activities, accounting for 11–25% of all acute injuries [1–6]. The most common mechanism of injury is with the ankle joint in a plantar-flexed and inverted position. The anterior talofibular ligament that most often is ruptured in lateral ankle sprains has been found to be the weakest ligament in tensile strength when compared with other ankle-joint ligaments [7]. Ankle-sprain recurrence is common, and among adolescent female football players, it appears as a re-injury in 56% of cases [8].

Lateral ankle sprain should usually be treated with non-operative functional rehabilitation, most often leaving only the most serious cases and repeat recurrences for consideration of surgical treatment [1, 9, 10].

Acute Treatment

It is generally agreed that the acute phase should include swelling limitation (compression), pain reduction (local cooling), and range of motion maintenance. Compression bandaging during the first 2 days and crutches when needed are recommended. When pain and swelling have subsided, bracing or taping could be considered. The ankle joint should initially be kept in as much dorsi-flexion as possible in order to stabilise the joint and minimise capsular distension.

Subacute Treatment and Rehabilitation

During the first 3 weeks, precaution should be undertaken to prevent ankle-joint inversion in order to decrease the risk of ligament elongation [10]. Absence of pain and swelling direct rehabilitation towards weight-bearing exercises, improvement of full range of motion, balance and proprioceptive training, and muscle strengthening, starting with the use of, for instance, elastic bands or tubing exercises. Strength training should focus on the peroneal, anterior, and posterior muscles, as well as intrinsic foot muscles. In order to avoid recurrence, a rehabilitation period of 12 weeks is recommended, focusing on muscle-strengthening exercises and balance and proprioceptive training. Return to football might, however, be possible as early as 1–2 weeks after the injury if the ankle joint is supported with an ankle orthosis or possibly with taping [11]. Exercises in all planes are most likely to be of the highest benefit and are therefore recommended to be included in the rehabilitation programme.

Proprioceptive training has been found to be an important factor in ankle-joint rehabilitation [12–14]. A normalised proprioceptive ankle-joint function has been found after 10 weeks of proprioceptive training [15]. Additionally, exercises for balance and co-ordination have been reported to reduce perceived ankle-joint instability and to improve proprioception [16, 17]. Training on balance boards has been reported to improve proprioception and balance [1, 10, 18] and should be performed with gradually increased levels of difficulty. Standing on one leg on a trampoline while throwing a ball against a wall is one example of a high level of balance activity.

Functional training, which usually means activities performed during weight bearing, is important for improving ankle-joint function following ankle sprain. Activities in a weight-bearing position have been reported to be of great benefit in regaining functional stability of the ankle joint [19]. Functional exercises, such as figure-of-eight runs, single-leg hops, carioca crossovers, and shuttle runs could be used for functional stability training. Furthermore, in a controlled investigation, we found that the figure-of-eight hop test [20] was the most sensitive test for functional evaluation of lateral ankle sprain patients when compared with a number of other sports-related functional tests and could therefore be recommended when evaluating ankle-joint function [Thoruid M and Werner S (2005) unpublished data].

Knee Sprains

Football players put great demands on their knee joints. The sudden changes of direction, hard cutting and pivoting, acceleration and deceleration, and occasionally violent collisions often put the player's knees at great risk of injury. The anterior cruciate ligament (ACL) is particularly at risk [21], and ACL injury is one of the most serious injuries in football. An ACL injury leads to a long absence from football and is also the injury that leads to the highest risk of sustaining arthrosis later in life [11].

In order to regain good knee-joint stability, most orthopaedic surgeons agree that ACL injuries should be treated with an ACL reconstruction. Today, no consensus regarding the optimal rehabilitation programme after ACL reconstruction exists. We know about the importance of early motion and weight bearing [22, 23]. However, only little is known about how much activity will promote adequate rehabilitation of an injured knee without permanently elongating the graft, producing graft failure, or creating damage to articular cartilage. More than 30 years of research at the Karolinska Institute into ACL rehabilitation has led to the rehabilitation protocol we use today. Based on new research, we are continuously updating our rehabilitation programme. The goals of rehabilitation after ACL reconstruction are presented in Table 1 and the rehabilitation protocol in Table 2.

Table 1. Goals of rehabilitation after anterior cruciate ligament (ACL) reconstruction

Time	Goals
0–5 weeks	Reduce pain and swelling Improve range of motion Achieve full knee extension Achieve ≥90° of knee flexion Regain quadriceps and hamstring muscle control Improve proprioception and balance of the lower extremity Achieve normal gait
When the goals above are achieved	
6–11 weeks	No swelling Achieve full range of motion Further improve muscle strength of the lower extremity Further improve proprioception and balance of the lower extremity
When the goals above are achieved	
3–4 months	Improve thigh muscle strength, power and endurance *without* pain Gradually return to functional activity and/or sport-specific training Achieve a normal running pattern
When the goals above are achieved	
5–6 months	No pain or swelling during football specific exercises Maximal muscle strength and endurance Good neuromuscular co-ordination Return to football, training, and gradually to games

Return to Football

The following criteria are universally suggested for allowing the ACL-reconstructed patient to return to pivoting sports such as football:

- Full range of motion of the knee joint;
- Stable knee joint and absence of giving way;
- Thigh-muscle strength ≥90% compared with the contra-lateral leg;
- Good results in physical performance evaluated with knee-related functional tests;
- No pain or swelling in connection with playing football.

Table 2. Rehabilitation protocol after anterior cruciate ligament (ACL) reconstruction

Time	Exercises
0–2 weeks	Pain control; cold and compression Passive knee extension exercises to 0° Patellar mobilisation – when needed Active knee-flexion exercises Electrical muscle stimulation – when needed Closed kinetic chain exercises Two-leg calf raises Balance and proprioception exercises Gait training - preferably in front of a mirror
3–5 weeks, add	Stationary bicycling – when 110° knee flexion attained Open kinetic chain exercises
6–8 weeks, add	Eccentric quadriceps training Step-up and step-down exercises Lunges with weights One-leg calf raises Two-leg trampoline exercises Slide-board exercises Stair-master exercises Gait training with different types of steps Overall stretching exercises
9–11 weeks, add	One-leg trampoline exercises Functional training, different jumps on the floor, skip the rope Jogging and running on even surfaces – in a straight line
3–4 months, add	Quadriceps training, in open and closed kinetic chain, concentric and eccentric actions in full range of motion Increased intensity of strength training in general Balance drills and coordination exercises Plyometric training Sport-specific exercises with emphasis on thigh-muscle training
5–6 months, add	Jogging and running on uneven surfaces Jogging with turns 90°, 180°, 360° Cutting with 45° changes of direction Acceleration and deceleration exercises Football-specific exercises with increased intensity

Muscle Strains

Too much stretch or tension in the muscle leads to an indirect muscle injury, a muscle strain [24], that may be graded into mild (grade I), moderate (grade II), and/or severe (grade III). Strains occur in muscles that are being stretched while undergoing strong activation to decelerate a motion, that is, an eccentric action [25, 26]. However, as well a sudden acceleration for extra speed during running, a sudden deceleration might result in a muscle strain. These situations are common in football when sprinting, kicking the ball, and stretching the leg to trap or tackle [24]. The athlete experiences local pain and tenderness and, a grade III strain will also appear with swelling and bruising. The healing time is 2–12 weeks, depending on the grade (mild, moderate, or severe).

Basic Concept of Acute Management

- Encourage rest, which means temporary cessation of sports activity;
- Apply immediate compression, a tightly drawn elastic bandage tied as firmly as possible for approximately 15 min, in order to limit the amount of muscle bleeding and thereby minimise the range of injury. Continue with compression, bandaged half as hard, for another 1–3 days;
- Keep the injured extremity immobile during the first minutes;
- Cool the affected area in order to limit pain. However, do not apply the cold pack (or ice) directly on the skin;
- Keep the injured extremity elevated for 1–3 days;
- Relieve load, especially if the injury is moderate or severe. Crutches can be used until a definite diagnosis has been made.

One should pay attention to the following questions 48–72 h after a muscle injury:

- Has the swelling resolved?
- Has the bleeding spread and caused bruising at some distance from the injured area?
- Has the ability of muscle contraction returned or improved?

If the answers are “no” to these questions, an intra-muscular bleeding is most probably present, and the patient should therefore be referred to an orthopaedic surgeon.

Subacute Treatment and Rehabilitation

Activity improves durability in healing muscle tissue. Progressive physical therapy is therefore recommended as soon as possible after the injury. As motion is restored, gentle muscle strengthening is started, with isometric training and preferably also with electrical muscle stimulation, isotonic dynamic training, and isokinetic training (if available) at low intensity.

Concentric exercises should be introduced before eccentric ones. Gradually, running exercises such as sprinting, cutting, and ball drills can be added when tolerable (Table 3).

Table 3. Treatment protocol for muscle strains

Mild strains	
Days 1–3	Compression, ice, elevation, active range of motion, electrical muscle stimulation, and isometric training
Day 4, add	Pool training, pain-free stretching, isotonic training (from light weights to heavier ones, from concentric to eccentric actions), bicycle training, and functional exercises
Day 7, add	Isokinetic training (from fast to slow angular velocities, from concentric to eccentric actions), plyometric training, and sports-specific exercises
Moderate strains	
Days 1–3	Compression, ice, elevation, pain-free active range of motion, electrical muscle stimulation, and crutch walking
Day 4, add	Pain-free isometric training
Day 7, add	Pool training, pain-free stretching, isotonic training (from light weights to heavier ones, from concentric to eccentric actions), bicycle training, and functional exercises
Week 2, add	Isokinetic training (from fast to slow angular velocities, from concentric to eccentric actions), plyometric training, and sport-specific exercises
Severe strains	
Days 1–3	Compression, ice, elevation, and crutch walking
Day 4, add	Electrical muscle stimulation
Day 7, add	Pain-free active range of motion and pain-free isometric training
Week 2, add	Pool training, pain-free stretching, isotonic training (from light weights to heavier ones, from concentric to eccentric actions), bicycle training, and functional exercises
Week 3, add	Isokinetic training (from fast to slow angular velocities, from concentric to eccentric actions), plyometric training, and sport-specific exercises

Muscle strains have a high rate of recurrence when competition is resumed [24]. Forty percent of muscle strains appeared as a re-injury in adolescent female football players [8]. Therefore, it is important that complete recovery of full range of motion, good muscle strength (at least 90% of the contralateral healthy side), and functional ability be attained before returning the athlete to competition.

Return to Football

It is recommended that the following criteria be fulfilled before allowing the athlete with a thigh-muscle injury to return to sports such as football:

- Full range of motion of both the knee and hip joints;
- Good muscle flexibility of the lower extremity, especially the thigh muscles;
- Good thigh-muscle strength, <10% side-to-side differences;
- Good muscle balance hamstring/quadriceps ratio 55%;
- Sports-related functional tests (most likely tailored for football), including running tests with acceleration and deceleration, sprint, and hop tests, performed at full speed without residual symptoms.

Hamstring Strain

In football, hamstring strains are the most common muscle injuries [24]. The biceps femoris is the most commonly injured muscle of the hamstring group [27, 28]. The hamstring muscles have a relatively high proportion of fast-twitch fibres [24]. The hamstring group crosses two joints, leading to greater changes in length when compared with muscles that cross only one joint. Therefore, the high levels of intrinsic tension, produced by the fast-twitch fibres, combined with the extrinsic stretch involved with length changes over two joints, might make the hamstrings prone to injury during high-intensity sprinting and jumping activities. The athlete usually experiences a sudden onset of pain in the posterior thigh during rapid activity. Occasionally, there might be an audible "pop", identifying a grade II or III strain. The muscle area involved and a local tenderness can be identified by palpation with the athlete lying prone with the knee slightly flexed against resistance.

Hamstring flexibility and muscle strength, as well as hamstring-to-hamstring and hamstring-to-quadriceps imbalances, appear to be the variables most often considered when dealing with hamstring strains. Subsequently, tests attempting to stimulate conditions in which hamstring strains occur appear to be the most valid indicators of predisposition to hamstring injury.

Quadriceps Strain

The second most commonly strained muscle group is the quadriceps, where the rectus femoris muscle that crosses two joints is the most frequently

injured muscle [24]. Quadriceps muscle fibres are predominantly type II and therefore best suited to rapid, forceful activity. Typically, the athlete feels the injury as a sudden pain in the anterior thigh when the quadriceps muscle requires a vigorous explosive contraction. For a football player, these situations are common during sprinting when accelerating towards the goal, jumping when heading the ball against the goal, and/or when kicking the ball into the goal, for instance.

Strain of the rectus femoris can be confirmed by eliciting pain when the hip joint is extended and the knee joint is flexed. Because the muscle is subcutaneous and overlies the remainder of the quadriceps, localised swelling or defect is rapidly apparent. This can be confirmed with the athlete lying supine and flexing the hip with the knee held in approximately 45° of flexion.

Adductor-Muscle Strain

In football, the incidence of groin pain is 10–18% per year in men [29–31] but less frequent in women [8, 6]. Adductor-related pain is a frequent cause of groin pain [33] and can result in long-standing problems [33]. However, it is not known whether these adductor-related groin injuries are due to tendon overload or whether the strength of the tendon is impaired. The patient usually complains of distinct tenderness at the pubic bone over the origin of the muscle. The pain can be triggered by applying resistance against adduction. Reduced adductor muscle strength and groin pain at full passive abduction, often with a reduced range of motion of abduction, are frequent signs. The pain often decreases after a warm-up period and can disappear completely during sport. However, the pain usually returns after physical activity, with even greater intensity.

The goal of the rehabilitation protocol is to regain a normalised movement pattern by improving muscle strength, balance, and coordination as well as stability of the pelvis and the hip joint. Focus should be put on strengthening the adductor muscles. Rehabilitation should be performed with a gradual increase of intensity during a period of 2–3 months, 3 times weekly. It should be stopped when the athlete manages to carry out the different exercises without pain. Hölmich et al. suggest dividing the treatment/rehabilitation into a first phase consisting of the first 2 weeks and a second phase starting from the 3rd week and onwards until the player is rid of symptoms. Sport participation should be avoided during the treatment period [34].

Treatment/Rehabilitation: First Phase

The initial treatment should be based on the cause of symptoms. This means, for instance, that if an excessive pronation exists, a wedge in the shoe could

be suggested. The majority of patients with adductor-related pain have difficulties activating the adductor muscles due to pain inhibition. Therefore, the first 2 weeks of rehabilitation should consist of careful static and dynamic exercises in order to teach the patient to re-activate the adductor muscles. Addition of transcutaneous electrical muscle stimulation could also be of potential value for selective muscle activity. Core stability, including good strength of the abdominal muscles, is important for football players when, for instance, kicking the ball. Therefore, exercises of the abdominal muscles, such as sit ups in both a straight forward and an oblique direction, is advisable. Training on a balance board could also be carried out in the first phase to aid in promoting core stability. Furthermore, one-foot exercises in abduction – adduction with parallel feet using a slide board – could be carefully performed.

Treatment/Rehabilitation: Second Phase

From week 3, a more demanding rehabilitation protocol is introduced. This includes heavier resistance in terms of dynamic leg abduction and adduction exercises in side-lying and standing positions. The patient is also encouraged to perform more challenging balance and co-ordination exercises. Skating movements on a slide board could be performed, with longer intervals for endurance muscle training. When these exercises can be performed at a high intensity without pain, the athlete is permitted to start running, with a gradual increase of speed. Finally, running with sudden changes of direction, cutting, and sprinting can be permitted, as well as sports-specific exercises.

Muscle Contusions: Quadriceps

A muscle contusion is likely to be the result of a direct blow to the muscle. Upon this direct impact, the contracted muscle is compressed against the underlying bone, often causing a deep rupture and bleeding. It may be graded in severity by restriction in range of motion of the subtended joints. A mild contusion causes loss of less than one third of the normal range of motion whereas a severe contusion causes limitations to less than one third of normal excursion. A muscle contusion can lead to the more serious intramuscular bleeding, which requires surgical treatment, or an inter-muscular bleeding, which can be treated with physical therapy (Table 4). Quadriceps contusion is the most frequent form of muscle contusion [35]. Kicking the ball in football makes football players vulnerable to quadriceps contusion.

Table 4. Treatment protocol for muscle contusions

Mild contusions	
Day 1	Compression, ice, and gentle stretching
Day 2, add	Strengthening exercises progressing from isometric training, through isotonic training (from light weights to heavier ones), to isokinetic training (from fast to slow angular velocities, from concentric to eccentric actions)
Moderate contusions	
Day 1	Compression, ice, electrical muscle stimulation, and crutch walking
Day 3, add	Pain-free isometric training and pain-free active range of motion
Day 5, add	Pool training, isotonic training (from light weights to heavier ones, from concentric to eccentric actions) and bicycle training
Days 7–9, add	Isokinetic training (from fast to slow angular velocities, from concentric to eccentric actions)
Days 10–14, add	Stretching exercises
Severe contusions	
Days 1–3	Compression, ice, rest, electrical muscle stimulation, and crutch walking
Days 5–7, add	Pain-free isometric training and pain-free active range of motion
Day 10, add	Pool training, isotonic training (from light weights to heavier ones, from concentric to eccentric actions), and bicycle training
Day 14, add	Isokinetic training (from fast to slow angular velocities, from concentric to eccentric actions) and stretching exercises

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Return to Play

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Introduction

Resumption of sport after an accident or surgery is an important step for every athlete. The eager wish to return, the fear of another accident, and the haunting doubt that previous levels of performance may no longer be attainable are all weighty features that must be taken into consideration by those engaged in rehabilitation. Sports in general and football in particular are generally seen as a form of recreation and distraction that cannot be readily surrendered. An accident is thus a dramatic event that may deprive both professionals and amateurs of the possibility of resuming sport and often has an adverse effect on their social and emotional lives. The rapid, unending development of therapeutic methods, therefore, has endowed functional recovery with a new and specific role in the handling of sports disorders.

The consequences of any accident are graded at three levels. The initial level is solely concerned with the *physical deficit* induced and the extent to which one or more of the specific characteristics that may be involved, such as force, strength, range of motion (ROM), and flexibility, has been impaired. The second level assesses *functional disability,* in other words, the extent to which the deficit has affected a player's movements in the form of reduced ability to run, jump, etc. The third level relates to what may be called a *sport-derived handicap* and hence examines the psychological and social consequences of the deficit. All too often, however, rehabilitation does not set out to achieve complete functional re-instatement. Instead, attention is simply devoted to reduction of the deficit or, even worse and even more frequently, the alleviation of pain. This attitude obviously delays healing. It also exposes a player to the risk of recurrences or other disorders. If healing is confined to the resolution of symptoms, in fact, the player may even start playing again but not at optimum level and certainly not at maximum safety. Treatment of even top-flight players, indeed, is far too frequently founded on irrational casuality rather than on rehabilitation correctly planned in the light of what has emerged from the application of the basic sciences to sport. The objective

of such a plan must be re-instatement of the subject's prior psychological and physical condition through rehabilitation based on the evidence and centred on the subject. Even if our knowledge of the biological aspects of soft tissue healing is not complete, there can be no excuse for failing to understand the processes involved and the biomechanical and physiological demands of a given sport, or for applying irrational therapeutic and rehabilitative procedures. Treatment must thus take account of the biological healing of tissues and the performance required from the player when sport is resumed, in the elaboration of re-educating and re-conditioning exercises and in modulation of the load to be applied.

Load modulation is a concept that approximates rehabilitation to the more classic forms of medical management. Application of a load, in fact, can be likened to the administration of a drug insofar as the benefits hoped for may be accompanied by unwanted side-effects. Gradual application of increasing loads is the basis of a correct approach to the resumption of sport. Overloading, in fact, must be the subject of maximum attention since it can readily result in muscle and tendon disorders.

Investigation into and clarification of the way injured structures heal and the time required to do so have enabled treatment to be founded on these parameters rather than empirical notions. It is thus more appropriate to speak of a functional recovery project as opposed to a rehabilitation project since the former comprises all the pertinent branches of sports medicine. The time required for biological tissue healing is obviously dependent on the type and extent of a lesion as well as the patient's ability to respond. Constant clinical and instrumental monitoring is thus required to guide and adjust the course of treatment during rehabilitation. The parameters best suited to the monitoring of rehabilitation and to determination of the best moment for resumption of the activity are the subject of extensive research. Clinical studies, however, are few and of poor quality. Their results, in fact, nearly always express personal experiences and opinions rather than conclusions drawn from sound scientific bases, and even less so from so-called "medicine based on the evidence". For these reasons, therefore, it is particularly difficult to decide whether a player should resume sport activity. The intricacy that has always been a feature of such a decision has continued to make it the weak link in the functional recovery process. Sport resumption is the critical end point of functional recovery. Its superficial or hurried attainment carries the risk of recurrences, the consequences of which are often devastating.

Functional Recovery after an Accident

Biological soft tissue healing must be taken into account when treating the five stages [1–3] of sports disorders: inflammation, proliferation, maturation

and remodelling, functional recovery, and prevention of recurrences. As already indicated, the temporal sequence of these stages is dependent on the type and extent of a lesion as well as the patient's ability to respond, and constant clinical and instrumental monitoring is thus required to guide and adjust the course of treatment during the functional recovery project. The six aims of a such a project are:

1. Reduction of pain
2. Reduction of inflammation
3. Reduction of swelling
4. Reestablishment of ROM
5. Maintenance and/or reestablishment of prior muscle and organic (force, strength, velocity) levels
6. Complete functional reestablishment and resumption of sport [1–3].

Understanding the biomechanical requirements of a sport enables the design of rehabilitation in keeping with the functions needed. A recovery project can thus be tailored to a player's sport or even the particular role in that sport.

The progress of such a project is determined by a series of activities simulating the movements a sport requires. It ensures the gradual re-acquisition of qualities lost due to injury and needed for the resumption of effective and, above all, safe performance. The first thing to be done in the functional recovery process is to provide the patient with an intelligible explanation of the disorder, treatment objectives and how they are to be reached, difficulties that may arise, and the importance of the patient's co-operation. The aim is not just to restore functional efficiency of the damaged structure as quickly as possible but also to prevent post-traumatic degeneration.

The period devoted to sport constitutes a limited period of a person's life. What is needed, therefore, is a personalised series of steps that follow the principles of biological tissue healing and guide and direct it without forcing the pace. Understanding and observance of healing times are perhaps the most important outcomes of scientific progress in recent years. Technological developments serve to optimise attainment of the objectives that are set but must not constitute the foundation of the healing project.

Treatment objectives and the ways of reaching them are now the subject of general agreement among those concerned with the rehabilitation of sports persons. A similar clarity and consensus, however, have not emerged with regard to the progression times of a recovery project. This state of affairs often stems from both a lack of communication between surgeons, rehabilitators, trainers, and players and the paucity and non-generalisability of the statistical validation and scientific investigation of the procedures proposed. It is these concepts that underlie modern evidence-medicine.

Resumption of sport is the natural consequence of the functional stage

of rehabilitation. This begins when the subject has reached four clinical objectives (disappearance of pain and swelling, substantial recovery of ROM, normal gait, and force and strength levels in the injured area 70% of those of the contra-lateral limb) and concentrates on recovery of running, motor control, muscle concentric and eccentric force and strength, and aerobic capacity.

Resumption Assessment Tests

Functional assessment of a sports person provides a quantitative and qualitative picture of the extent of the functional deficit induced by an accident and serves as a guide when choosing the process of recovery and deciding whether sport can be resumed. It is difficult to reach an objective decision, and a full evaluation of the subject's psychological and physical condition is required. Very often, the type of assessment adopted, such as isokinetic tests; or parameter values regarded as decisive, such as the attainment of 80% of the force capacity of the contra-lateral limb, measurement of thigh circumference, or joint mobility, as such do not correspond to the subject's functional requirements, prevent the formation of an appropriate judgement, and carry the risk of recurrences.

We believe that such tests must be supplemented by functional evaluation tests that furnish a more accurate representation of the dynamic situations inherent in football. Isokinetic evaluation is widely employed to investigate neuro-muscular function [4–9]. Within the sphere of functional assessment, however, it provides limited, small-scale information that has little to do with the real functional requirements imposed by a match or during training [10–12]. It must be remembered that a constant speed is an inherent feature of isokinetic work whereas this is rare in real life. All sport movements and actions, in fact, are conducted in antigravity and acceleration conditions and hence with changes in velocity. Their assessment cannot be undertaken with instruments incapable of weighing these parameters.

It is none the less customary to use isokinetic dynamometry to determine muscle force. Concentric evaluation of the peak moment of force of agonists and antagonists is the most frequent approach. The isokinetic dynamometer evaluates muscle force expressed in open chain and in the absence of loading and thus cannot reproduce joint kinematics in a closed chain in the presence of loading. It is thus unsuitable for evaluation of the role of the musculature as a dynamic joint stabiliser. Aagard et al. [13] have suggested interpolation of the hamstring eccentric peak force with the concentric peak of the quadriceps to produce what they call the "extensor functional ratio of the knee". In this case, the peal force of the hamstrings is directly proportional to the angular

velocity at which the exercise is performed and inversely proportional to the degree of knee flexion. This evaluation is, in effect, more functional for assessment of the state of the muscles responsible for dynamic stabilisation of the knee, with biomechanical implications for kicking a football, which requires explosive, concentric contraction of the quadriceps and eccentric braking on the part of the flexors. This form of "functional" isokinetic assessment must always be used during both pre-season evaluation and when deciding whether an injured player should resume playing.

In the case of recovery after a lower-limb accident, we advise a concentric isokinetic test to evaluate the force moment peak of the quadriceps and flexors at both slow and medium-to-high velocities (60° and 180–240°s/-1, respectively) and a "functional" test (eccentric for the flexors and concentric for the quadriceps) at 60° and 180°/s-1. Scales with scores have often been advanced as criteria for the resumption of sport and assigned a worth they cannot have since they express the extent of a disability and not the functional ability attained by the subject.

Daniel et al. [14], followed by Barber et al. [15] proposed one leg-hop forward and three leg-hops forward as tests for the dynamic evaluation of lower-limb disorders. The patient takes off and lands on the same foot and tries to go as far as possible. Both tests are obviously simple and require little space. They combine explosive force, co-ordination, and balance and indicate both the recovery of force and the ability to land after a leap. The contra-lateral limb serves as reference. This, however, presents the drawbacks described below. The result is classed as normal when the difference between the hopping distances reached by the two legs is not more than 10% whereas a higher percentage is interpreted as an indicator of instability and hence the likelihood of "giving way" if sport is resumed.

We believe these tests are certainly indicated for the functional assessment of a footballer since they allow investigation of three main requirements, namely, explosive force, co-ordination, and ability to adapt. They are, however, unable to evaluate a common component of athletic and technical movements, namely, the shear and rotatory forces imposed on leg joints during swerves and other changes of direction.

Augustsson et al. [16] observed differences between the single-leg-hop test of reconstructed anterior cruciate ligament (ACL) patients before and after strenuous exercise. All subjects had normal results under baseline conditions whereas 68% displayed abnormalities after leg extension to the point of exhaustion. This study shows that "normality" may be deceptive and hence that the test should be performed in a state of fatigue. It also emphasises the need for thorough investigation of a player's physical characteristics, including resistance to prolonged effort.

Since there are no tests that specifically reproduce such characteristics,

some workers [17] have validated more complete dynamic tests that reflect the manoeuvres typical of football. Lephart et al. [18] have elaborated the "functional performance test" (FPT). This consists of three tests that impose rotational forces on the knee to evaluate the dynamic stresses elicited by spurts, braking, sudden stops, swerves, starts, and squatting. These are timed tests with scores; high scores are correlated with resumption of sport at the pre-accident level.

1. The first test is called the "co-contraction" test. The subject moves five times and as quickly as possible along the perimeter of a semi-circle painted on the floor, against the forward pull of an elastic cord 122-cm long and 2.5-cm in diameter (Sportcord, Pro Orthopedic Devices, Inc., Tucson, AZ, USA) around the waist. This cord is attached to a wall at a height of 154 cm, and the radius of the semi-circle from the wall is 244 cm. The test requires the use of the leg muscles for sideways movement and knee stability.
2. The second test is called the "carioca" test. The subject moves sideways, crossing the legs and as quickly as possible, to the end of a 12-m path and back.
3. This third test is called the "shuttle-run" test. In this classic test, the subject sprints four times as quickly as possible to the end of a 6-m path and back.

The FPT score is the sum of the best times in the three tests. A test can only be regarded as valid if it can be used to obtain a sensitive, specific, and accurate diagnosis [19]. Those tests that do not meet this requirement provide data that are correct but not reliable. The most sensitive assessments are those that faithfully reproduce the actions and movements that take place during a match.

Some fundamental features of evaluation tests must be borne in mind to determine their appropriateness with respect to the purpose of measurements and the parameter being measured. The validity of a test defines its ability to measure what is intended to be measured [20–22] and takes three forms:

1. *Construct validity*, which indicates the degree to which an instrument, such as a dynamometer, provides measurements consonant with the theoretical model, for example, a physiological/biomechanical model, from which it was devised
2. *Concurrent validity*, namely, how closely the result of a test correlates with that of another test regarded as the gold standard
3. *Predictive validity*, that is, a test's prognostic value.

Also, four factors must be measurable for a test to be valid:

1. *Reliability*: the ability of a test to give analogous results when repeated either by another operator (inter-operator reliability or concordance) or

by the same operator at different times (intra-operator reliability or concordance)
2. *Sensitivity*: the ability to provide "true positives", namely, those that possess the parameter concerned and are assigned it by the test
3. *Specificity*: the ability to provide "true negatives", namely, those that do not possess the parameter concerned and are not assigned it by the test
4. *Accuracy*: the overall ability to identify true positives and true negatives.

Functional evaluation must also take into account a feature that is often underestimated, namely, the reference parameter with which the results of a test are compared. "Normal" or, in the case of limbs, "contra-lateral" values are generally employed for this purpose. They are not the best, however, and should be replaced by reference values. What, in fact, is meant by normal and contra-lateral values, and why are they not the best? Normal values usually reflect the extraction of two fringe values (bracketing virtually the whole of the population) from the series of values within which all values for a given parameter for a supposedly healthy and hence "normal" population are distributed. It is obvious that a population of injured athletes is neither normal nor healthy. Contralateral values, too, assume that the other limb is healthy and hence normal. This assumption, however, requires further consideration. The contra-lateral limb of an injured athlete, especially if the injury is not recent or the "unhealthy" limb is afflicted by a chronic disorder, cannot be regarded as "normal" since it will have undergone adaptation to offset the defective function of its fellow, or become involuted owing to the lack of training signals due to the athlete's immobility. To treat a contra-lateral limb as automatically normal and use it as a term of comparison is obviously likely to result in an incorrect evaluation.

It would thus be clearly preferable to draw upon reference values, namely, extracted in the same way as "normal" values but from a selected population – sports persons, in our case, or, better still, those engaged in the same sport and, as far as team sports are concerned, in the same role as the subject of our tests. Moreover, the optimum solution would be to have reference values acquired when the injured subject was in a state of good psychological and physical health.

This, then, is why it is essential to gather data during pre-season training by means of both clinical and functional tests. This, of course, is for high-level athletes served by a multi-disciplinary staff. Cacchio et al. have suggested that this end can be reached by coupling the "field-test" coaches' use for functional evaluation [23] with anthropometric parameter values [24, 25], blood chemistry tests [26], imaging [27], and instrumental assessment of muscle qualities [28] and the entire neuro-muscular system [23, 29]. Periodic functional evaluation furnishes a series of physiological baselines for the creation of a database upon which decisions concerning the attainment/improvement

of high-performance levels and their recovery after an accident can be founded [23]. It is evident, in fact, that a player's physiological baselines will serve as excellent reference values for the determination of work loads and attainment of an objective decision concerning return to sport when planning functional recovery after an accident [23].

The aim of a multidisciplinary *équipe* is to elaborate an objective grid for evaluation of a player's performance from that player's field and functional laboratory test scores. When combined with the clinical, imaging, and blood chemistry results, this evaluation constitutes a functional profile of the player at a given moment in time. More or less sport-specific guidelines have been devised to facilitate the planning of a recovery programme. These tests have been constructed with an eye to the fine balance between practicability and inexpensiveness and their reliability. Field tests are obviously easier to apply and execute, especially when many players are involved as in team sports, but are not in themselves sufficient to provide exhaustive information about the functional efficiency of certain mechanisms. When resumption of sport is involved, such tests must be supplemented with more analytical methods. This does not necessarily require the use of highly specialised and expensive laboratory equipment. The literature describes the following field and laboratory tests:

- Squat;
- Squat jump;
- Counter-movement jump;
- Twenty-five metre shuttle;
- Standing long jump;
- Sprint with timing at 10, 30, 40 and 50 m;
- Fatigue index derived from six 70-m shuttles, the running time of which (total running/recovery time: 1 min) is calculated as mean running time x (max/min time) x 6 divided by the number of completed shuttles [30, 31];
- Construction of tension-time curves from the following parameters: maximum voluntary isometric contraction (MVIC), T30, T50, and T90, and rate of force development (RFD) 30, 50 and 90 (i.e., at 30%, 50%, and 90% of the maximum voluntary contraction corresponding to the mean gradient of the segment of the tension-time examined: this gives both the absolute muscle force capacity and the time taken to reach it).

When using isometric measurements, the selection of correlated time parameters for the evaluation of injured players lies in the observation that the patterns of motor recruitment and the more qualitative expressions of force in general are those that should be of most interest with regard to resumption of play. The neuro-muscular mechanical feature of all power

sports, in fact, is apparent in the form of rapid force peaks that rarely exceed sub-maximal values. It is therefore considered that, while the maximum force is a value to which due attention must be paid when evaluating an injured player, only dynamic parameters, such as the RFD, are in a position to indicate the level of protection the neuro-muscular system is able to provide for the dynamic stability of a joint [32]. It has also been shown that the RFD, like the T25–50 and other time-dependent parameters, is significantly correlated with explosive force and vertical jumping expressions [33–35], which are an excellent illustration of complex ballistic movements that are a good reference for sport expressions. The decision to not electively indicate unilateral jumping tests, e.g., the leg-hop test, as indices of correct recovery stems from the observation that the execution of a unilateral vertical jumping test is more difficult to standardise and also presents greater psychological complications when performed by an injured athlete. The persistence of altered RFD values suggests that whereas the maximum expression of force is unchanged, neuromuscular motor unit recruitment patterns, quality of muscle fibres, and muscle stiffness values have been modified by the injury and require more time and better targeted interventions to secure their optimum re-instatement, as already illustrated by studies employing other investigation methods [36].

We thus believe that the RFD in its various forms is a good indicator for handling the return to sport. It has been employed to create an evaluation grid called the "sport recovery score" [23] in which the minimum and maximum values to be reached in the various aptitude tests are indicated. This grid is founded on a database comprising 850 national- and international-level athletes [23]. Resumption is approved when the clinical, imaging, blood chemistry, and instrumental parameters are met and the subject's functional test values are equal to or more than 80% better than those reached in the same tests prior to his accident. If the athlete has no previous records, the mean values obtained by athletes in the same sport or engaged in the same role can be used as reference values [23].

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Protective Equipment

Donald T. Kirkendall

Introduction

Law 4 of the International Football Federation (FIFA) describes the compulsory equipment for the game (Table 1), and FIFA has responded to selected questions regarding the interpretation of that law (Table 2). The laws and subsequent interpretations limit the equipment that a player may wear in order to prevent injury.

Table 1. International Football Federation (FIFA) Law 4: player equipment (reproduced from [1])

Safety	• a player must not use equipment or wear any thing that is dangerous to himself or another player (including any kind of jewellery)
Basic compulsory equipment	• jersey or shirt • shorts – if thermal under-shorts are worn, they are of the same main colour as the shorts • stockings • shin guards • footwear
Shin guards	• are covered entirely by the stockings • are made of a suitable material (rubber, plastic, or similar substances) • provide a reasonable degree of protection

Injury in football might be considered to be of two types: contact and non-contact injuries. Non-contact injuries include overuse injuries (e.g. stress fractures), muscle-tendon strains (mostly to the hamstrings and thigh), and ligament injuries that might be due to field hazards (ankle sprains) or motor control [anterior cruciate ligament injuries (ACL)]. Contact injuries are mostly contusions, lacerations, joint sprains, cartilage injury, concussion, and more.

Table 2. International Football Federation (FIFA) posted interpretations on Law 4 (reproduced from [2])

1. If the colour of the shirts of the two goalkeepers is the same, what should the referee do if neither have another shirt to change into?	The referee allows play to begin
2. According to Law 4, the players of each team and their goalkeepers must wear jerseys or shirts of different colours to distinguish them from the other players. Must the referee and the assistant referees wear clothes with different colours to the players?	No. The players and goalkeepers must wear clothing that distinguishes them from the referee and assistant referees
3. What action should the referee take if a player removes his shirt to reveal a similar shirt underneath?	The referee must caution the player for un-sporting behaviour
4. May players wear a one-piece playing suit in place of shirts and shorts?	No
5. May a player wear equipment designed to protect him against injury during a match?	Players may wear protective equipment such as knee or arm pads, face masks or padded headbands provided the equipment meets the requirements of Law 4 – The Players' Equipment (i.e. it is not dangerous to either the player himself or to other players)
6. May a player wear spectacles during a match?	Modern sports spectacles, made of plastic or similar material, are not normally considered to be dangerous and referees would in such circumstances be expected to allow them to be worn
7. The referee requests a player to remove jewellery. After a number of minutes, the referee realises that the player is still wearing the jewellery. What actions should the referee take?	The player must be cautioned for unsporting behaviour. The player is instructed by the referee to leave the field of play to remove the jewellery
8. Are players allowed to use tape to cover jewellery that is considered to be dangerous?	No

continue →

Table 2, *continue*

9. A player accidentally loses his footwear and immediately scores a goal. Is this permitted?	Yes. The player did not intentionally play barefoot because he lost his footwear by accident
10. Is radio communication between player and/or technical staff permitted?	No
11. Are member associations allowed to introduce modifications to Law 4?	No. Law 4 is not included in the permitted modifications described in the Notes on the Laws of the Game

There is the opportunity to use protective equipment within the laws of the game for primary prevention or for prevention against re-injury, yet some players elect not to use, or to misuse, allowable equipment.

Football Injuries

In order to know how equipment can minimise football injuries, a brief review of injuries seems appropriate; just what injuries might equipment protect? A main limitation when comparing injury reports is the research methodology and definitions of injury. It is difficult to compare injury statistics between different research groups because each has its own definitions, protocols, and biases [3]. When one group is able to study multiple ages, levels of ability, competitions, and sports using a common set of criteria, reasonable comparisons are possible. Since 1994, FIFA has sponsored a research program with its charge being to make football a safe, health-enhancing activity. FIFA President Mr. Sepp Blatter asked the question: How can the frequency of football-related injuries and symptoms be reduced at all levels of play? This simple question led to the development of the FIFA Medical Assessment Research Centre (F-MARC). This prolific group has published over 50 papers on many aspects of football injuries using a common methodology making comparisons easy and understandable.

Table 3 summarises football-related complaints. What is most obvious is that about 70–75% or more of all players questioned had some measure of a physical complaint related to the game, a number of which might not be considered to be related to a specific incident (e.g. back pain) [4]. Rates of injury per 1,000 h of training or competition are valuable figures for epidemiologic and risk assessment research but can sometimes be hard for others to interpret. A review of the injury literature shows values from 25 to over 100

Table 3. Frequency (percent) of football-related complaints (modified from [4])

	Professionals	Lower-skilled 14- to 16-year-old youth
Headache	10	39
Neck pain	30	11
Low back pain	28	46
Muscle/tendon	38	38
Joint pain	33	44
Any complaint	78	84

injuries per 1,000 h [3]. Within F-MARC studies, the injury rates range between about 25 and 75 injuries per 1,000 match hours, depending on age and gender [5]. A more simple description of injury rate is injuries per player or per match. Table 4 lists the injuries per match for a variety of FIFA-sponsored tournament shows a range of 1.3–3.7 injuries per match [5]. During a comparison of all Olympic team sports at the 2004 Olympics, football was, with team handball, the sport with the highest injury rate [5]. In a direct comparison with rugby, the training injury rate was 45% higher for rugby (15.4 vs. 22.4 injuries/1,000 h for football and rugby, respectively). The

Table 4. Injuries per match in International Football Federation (FIFA)-sponsored tournaments (modified from [5])

Tournament	Injuries per match
2001 U20 men	4.7
2000 U23 men (Olympics)	3.7
1999 U20 men	3.6
2000 World Club Championship	3.2
2001 U17 men	2.8
1998 World Cup	2.6
2000 Olympics women	2.1
2001 Confederations Cup	2.1
1999 Confederations Cup	1.7
1999 U17 men	1.7
1999 Women's World Cup	1.3
2000 Futsal	1.3

match injury rate for rugby was 173% greater than the injury rate for football (47.5 vs. 129.6 injuries/1,000 h for football and rugby, respectively) [6].

Occupational injuries can be classified as trivial (no time loss), minor (<1 week time loss), moderate (1–4 weeks time loss) and severe (>4 weeks time loss). In football, the majority of injuries are classified as minor. At the 2002 FIFA World Cup, 33% of the injuries led to no time loss. The majority of injuries (54%) led to 1–7 days lost, 11% to 1–4 weeks lost, and only 2% were severe, meaning over 4 weeks lost. Thus, a total of 87% of all World Cup injuries resulted in less than 1 week of time lost [7]. Severe injuries were varied and were different according to age (Table 5) [8]. One might assume that

Table 5. Percent of severe injuries by diagnosis and age in male football players (modified from [8])

	14- to 16-year-old players	25- to 41-year-old players
Sprain	22	7.5
Fracture	12	7.5
Strain	7	26
Ligament rupture	6	25
Meniscal injury	7	25
Contusion	23	8

the most severe injuries would be due to player contact, yet many joint sprains (e.g. ACL sprains) were non-contact in nature. But by far, the mechanism most responsible for injury in football is from tackling, with nearly 75% of all contact injuries happening during tackling [9]. Two considerations follow. First, is there a skill issue with respect to tackling? Or second, are the injuries from tackling a natural consequence of player-to-player challenges or from some sacrifice of the concept of fair play based on game circumstances? A disturbing finding from the F-MARC work was that 92% of players were willing to commit the "professional foul", and 50% had responded physically when provoked by an opponent. Finally, 60% were willing to "pay back" what they considered an un-penalised foul. Thus, while skill might be an issue, attitude may also be a factor in tackling-related injuries [10].

It is no surprise that the majority of football injuries are to the lower extremity. Depending on the study, the ankle and knee are the sites of most injuries, followed by the shin, thigh, and head. The most common injury is a contusion, followed by muscle strains, joint sprains, cartilage damage, lacerations, and concussions. Prevention of football injuries is a primary focus of

people involved in football medicine and continues to undergo intense scrutiny on the field as well as in the literature. Many injury prevention strategies involve general and specific training activities. For example, specific warm-up and cool-down activities are considered effective for preventing muscle strains. Non-contact ACL injury rates (especially in young women) can be reduced with specific neuro-muscular warm-up routines that teach motor control of the knee during landing and cutting, which are so common in team sports [11]. Some head injuries (e.g. those occurring while heading the ball backwards) can be prevented by coaching. Some injuries can be prevented by good field maintenance, as an uneven field has been implicated in selected ankle injuries. There are, however, specific injuries where protective equipment can be effective in injury prevention.

Goalkeeper

Goalkeeper Gloves

The evolution of goalkeeper gloves is mostly about performance and may also offer some protective benefits. For example, equipment manufacturers asked goalkeepers about what they want in their gloves. The players said that sometimes they get their hands on a hard shot, but the force of a shot may hyper-extend their fingers, and this could lead to joint sprains. The glove makers responded with a glove with supporting ribs on the posterior aspect of the glove to limit the amount of hyper-extension on the fingers (Fig. 1). Some models also have support for the thumb. Goalkeepers have far more serious injuries to be concerned about (collisions with on-rushing attackers), yet a

Fig. 1. Model goalkeeperglove, 2005

secondary benefit to the goalkeeper might be a reduction in hyper-extension injuries to their fingers.

Goalkeeper Padding

The goalkeeper's uniform has one requirement in the rules: it must be a contrasting color to their teammates. Their uniforms have evolved over time, with padding being added to protect areas of their bodies susceptible to contusions, especially around the elbows, knees, and hips to protect during falls, dives, and other collisions. While head injuries are rare in football in general, and to goalkeepers specifically, the head is very exposed.

Outfield Players

Law 4 says a player's uniform must consist of a shirt, shorts, socks, shin guards, and footwear (Table 1). This does not leave many options for allowable protective equipment. FIFA's interpretation of this law (Table 2) does state that "players may wear protective equipment such as knee or arm pads, face masks or padded headbands provided the equipment meets the requirements of Law 4 – The Players' Equipment" [2].

Ankle Sprains

There are a couple of possible mechanisms for a sprained ankle. A common mechanism is stepping on an uneven surface such as a defect in the field or an opponent's foot [12]. The Oslo group has postulated that a tackle from the side, with a raised foot, can contact the medical side of the opponent's leg (Fig. 2). While this contact itself does not cause the sprain, the attempt to maintain balance to resist a strong lateral impact leads to the foot landing in such a manner that it inverts, leading to a sprain [12].

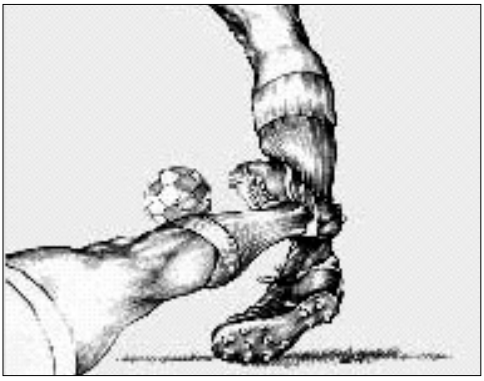

Fig. 2. Hypothesized mechanism of ankle injury. Illustration by Tommy Bolic (reproduced from [13])

Many football players view an ankle sprain as a nuisance where a knee sprain of similar severity would be viewed as far more serious; a few days off the injured foot, regular icing, maybe miss one match, and then return to play. Unfortunately, an incompletely healed minor injury such as an ankle sprain often precedes a major injury either to the same location or another location [14]. The inability to avoid an opponent might lead to a serious collision, or a player with chronic instability from a history of sprains might land in a manner that puts the knee at risk. Thus, protection of a previously sprained ankle is important to ensure continued, safe play.

Medical research is frequently contradictory on many topics, but it is clear on prevention of ankle sprains: no protective device – taping, lace-up braces, air supports – will protect an ankle from the first sprain. Randomised trials show no difference in the rate of first ankle injury whether some sort of protection was worn or was not worn [15]. This is clear. Prevention is focused not on the first sprain but on the next sprain. Here again, the research is clear: protective devices (Fig. 3) drastically reduce the rate of subsequent sprain. The risk of a second ankle sprain increases by a factor of 6 in players with a history of ankle sprain [16].

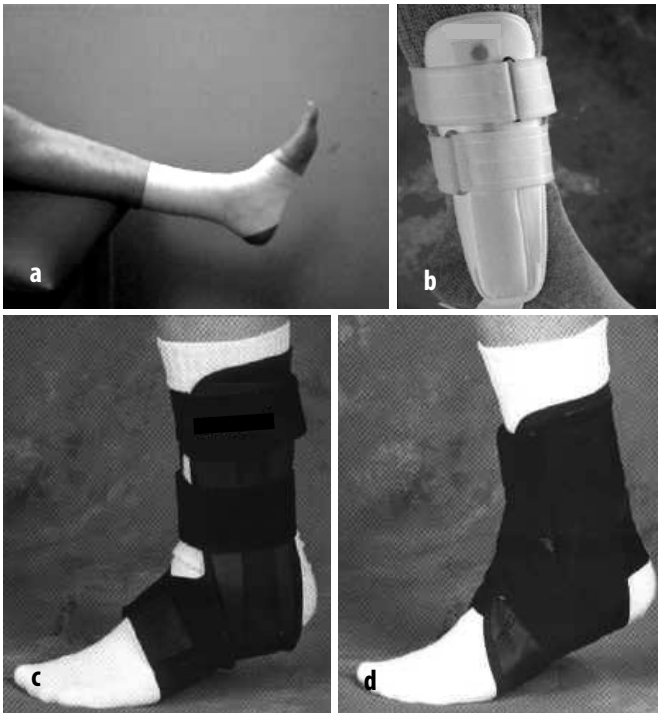

Fig. 3a-d. Examples of external ankle support

What most players do not realise is that to prevent that next sprain, sports medicine doctors recommend wearing external support for 6–12 months after the injury. Football players resist wearing external support, saying that a brace reduces their "touch" on the ball (external supports cover the heel to the arch, well behind where a player's touch would be affected) or that their agility will be affected (basketball has a far greater agility demand, and ankle support is a common sight in basketball).

Taping is often used as a protective measure against ankle sprain. The problem with taping is that tape stretches with ankle movement, and most of the support supplied by the tape is lost after about 20 min of movement [17]. This means that most or all the benefits of taping are gone by the end of a warm up. Yet a superior restriction in range of motion has been shown not to be indicative of superior prevention of ankle sprains [18].

Leg Contusions

Probably the most common injury in soccer is a contusion to the leg after being kicked during a tackle. Shin guards are a required piece of equipment, the purpose of which is to protect the leg from injury. Law 4 simply states shin-guard requirement with no statements as to size or quality of the protective pad. Consumer protection standards have been discussed in Europe and the United States. In the United States, the National Operative Committee on Standards for Athletic Equipment has published procedures for certifying shin guards. The wide variety of types and styles on the market might suggest that universal standards have not been adopted.

Shin guards must fit fully under the socks, and each player is visually inspected before a match to ensure shin protection is being worn. These products should protect the areas of the leg most often injured. Contusions and fractures in football are confined to the lower half of the leg [19]. Yet a casual observation of most photographs of a professional footballer shows a small shin guard, mostly worn on the upper half of the leg, well above the location of most impacts (Fig. 4). Many professional footballers wear a child's shin guard – the smallest possible protection to pass the referee's inspection.

There have been three studies published comparing the impact reduction of various types of shin guards [20–22]. The biomechanical studies were conducted in a laboratory using a standard impact force applied to an instrumented "leg" protected by a variety of products. Shin-guard products typically include plastic, fibreglass, heat-molded cast material, air/foam cells, or Kevlar. All shin guards reduce the force of impact by 10–15% and strain on the bone by about 50% [22]. The thicker products absorb more impact, and at the greatest levels of impact, the thickest and heaviest shin guards (air/foam cell) were the most protective [22]. The challenge for product manufacturers

Fig. 4. Small shin guard on an adult leg

is to produce a shin guard that affords the greatest protection with the smallest total mass while covering more of the leg.

Head Protection

There is probably no topic of greater concern to the soccer community than head injuries. Football is unique because of the purposeful use of the unprotected head to control and advance the ball. Reports of cognitive dysfunction in a small fraction of football players were blamed initially on heading [23]. The media and other lay observers of the game have for many years been concerned about the role of purposeful heading on the cognitive issues in this limited group of players.

The mechanism of head injury is the contact of two hard surfaces: head-head, head-elbow, head-knee, head-ground, and head-goal post [24]. In males, head–elbow is most common and in females, head–head is the most common. Recent F-MARC research suggests that most head injuries (concussion injury) happen in the middle third of the field when two players coming from opposite directions both jump up, and into each other, for the ball [24]. Collisions in the penalty area during corners or crosses are a secondary location, but not as common as players who are not generally coming at each other from opposite directions or with as much closing velocity.

The medical literature continues to pursue this topic aggressively by studying two distinct but related topics: the impact of purposeful heading and the results of concussion injury. The most recent literature from laboratory investigations and computer simulations suggest that the impact of purposeful heading (as opposed to accidental head-ball impact) is well below the level necessary to cause a concussion and that the ball speed needed to reach

the estimated concussion threshold for purposeful heading is far above the speed that a player can achieve [25, 26]. Thus, the general opinion is that purposeful heading will not lead to a concussion. It is not known, however, whether there are any long-term consequences of sub-concussive impacts of purposeful heading, but current studies underway show little effect over time.

The mechanics of head-head or head-elbow impact have recently been reported [27]. When two players approach from opposite directions, the closing speed can be high, with the players focusing their attention on the ball. The collision of heads leads to either a linear or angular impact that can be of sufficient force to cause a concussion. In purposeful heading, the head is controlled by contracting the neck muscles, giving the body a large mass with which to overcome the mass and closing speed of the smaller and softer ball [28]. When two heads collide, neither player is prepared for the coming impact. A primary focus of research in football medicine concentrates on recognition of concussion injury and return to play guidelines [29].

Head injuries can be prevented partly through coaching and educating players as to just what activities are dangerous. Most coaches stress that the most important ball contact on a clear or a punt to the middle of the field is not the first contact but the second contact, so an attempt at heading a punt is not as important to the team as is controlling the ball after that heading opportunity. Players should be taught that a back header (because they have no other choice) is a wasted pass and may place either player at risk of a head injury. Jumping straight up keeping the arms closer to the body is safer for the opponent, yet many players jump with elbows out to keep an opponent at a distance.

In recent years, there have been a number of products developed that attempt to protect the head (Fig. 5). Initially, these products were promoted

Fig. 5. Two types of soft head protection

as protection for heading. The reality is that when three surfaces collide – head, headgear, and the ball – the softest object will absorb the force of the impact and the softest surface is the ball, so headgear offers little protection for heading [29]. More recently, the focus has been on protection against concussion injury. In the collision between head, headgear, and head, the softest surface is the headgear. Recent data has shown that commercial headgear does reduce the acceleration from impact (Table 6) [30]. Whether these reductions are sufficient to protect against concussion injury is unknown. Also unknown is whether this level of protection will protect the head in subsequent impacts. Finally, until there is a randomised trial of protective headgear, there is no data to conclusively state whether soft, protective headgear is effective at lowering the rate of concussive injury. The challenge of such a study is the number of subjects necessary (concussion injury is not a very common injury) and the difficulty of injury recognition with multiple medical personnel across a large number of teams.

Table 6. Percent reduction in linear and angular accelerations with headgear use (modified from [30])

Head (linear)	Temple (angular)	Forehead (linear)	Back of head (angular)
19–22%	14–26%	18–28%	17–32%

The Shoe as Protective Equipment

After the ball, the most basic piece of equipment is the shoe, and the shoe has undergone dramatic changes in the last 25 years. The basic 14-stud molded sole boot has given way to new stud configurations and advancement in materials, especially the use of materials with high strength-to-weight ratio, allowing new shoes to be structurally sound and lighter than earlier shoe designs. When players are surveyed, their primary concern is for fit and comfort in a lightweight shoe that allows for a good feel and touch on the ball. Performance factors (e.g. stability, traction) are secondary, but still important, to players.

It is unrealistic for a shoe to be expected to protect against the contact injuries of football that come mostly from attempted tackles (Fig. 6). Traumatic contact injuries leading to surface, or deeper, contusions and metatarsal or phalangeal fractures would only be reduced with added padding that would lead to a reduced sensitivity (touch) on the ball.

Overuse injures of the foot (particularly metatarsal and navicular stress fractures) have multi-factorial causes, including training errors, hard-surface conditions, and many others. Changes in shoe design have made it possible

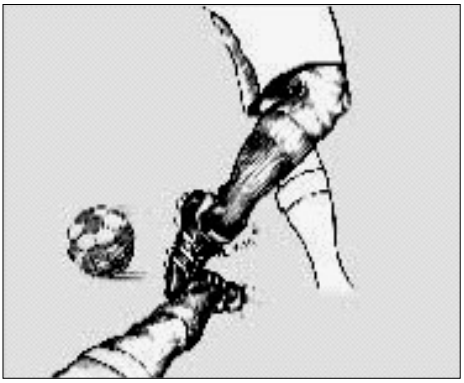

Fig. 6. Foot-foot contact in football. Illustration by Tommy Bolic (reproduced from [13])

for a custom orthotic to be inserted to improve support of the foot and arch better than can be supplied by the manufacturer's insert. A training consideration that sometimes is used, especially in youth football, is to restrict the time in studded shoes. Fitness, skill, and non-competitive activities can be performed in running shoes that have better support, cushioning, and comfort before moving to studded shoes for the more demanding competitive play.

Foot injuries are an uncommon injury in football even though logic might state otherwise. Foot complaints, on the other hand, are more common [1]. The interaction of the foot-shoe-surface interface can lead to many complaints. While a number of professionals feel they need a few steps each morning to "loosen up" their feet, shoe manufacturers have made great strides in trying to modify their shoes to minimise complaints about footballer's feet.

The traditional molded-sole shoe has been a common design standard for decades. As player ability, field conditions, and match demands have changed, an early design change was a shoe with six screw-in studs for use on soft or wet ground. The complaint was with the pressure on the first metatarsal head from the underlying stud. Shoe companies then made numerous attempts at modifying the location of the stud (including some with an adjustable location), how the stud is attached to the shoe, padding above the stud and other ways to distribute the pressure over a wider area. Newer stud designs incorporate what is called a "bladed" design for increased traction. Shoe design to minimise foot complaints will continue to evolve.

Conclusion

The simplicity of football is a major reason for the interest in the game. While there are many injuries in the sport, those injuries are usually minor. Yet

many injuries can be prevented by proper coaching, training, and protective equipment. Proper selection and use of allowable equipment should be beneficial in keeping football a safe, health-enhancing activity.

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