**Sports and Traumatology** *Series Editor:* Philippe Landreau

Pieter P.R.N. d'Hooghe Gino M.M.J. Kerkhoffs *Editors* 

# The Ankle in Football





## Sports and Traumatology

Series Editor Philippe Landreau

Pieter P.R.N. d'Hooghe • Gino M.M.J. Kerkhoffs Editors

# The Ankle in Football







*Editors* Pieter P.R.N. d'Hooghe, MD Department of Orthopaedic Surgery Aspetar Hospital Doha Qatar

Series Editor Philippe Landreau, MD Sports Orthopaedic Surgery Aspetar, Orthopaedic and Sports Medicine Hospital Doha Qatar Gino M.M.J. Kerkhoffs, MD, PhD Department of Orthopedic Surgery Academic Medical Centre Amsterdam University of Amsterdam Amsterdam The Netherlands

ISBN 978-2-8178-0522-1 ISBN 978-2-8178-0523-8 (eBook) DOI 10.1007/978-2-8178-0523-8 Springer Paris Heidelberg New York Dordrecht London

Library of Congress Control Number: 2014936635

© Springer-Verlag France 2014

This work is subject to copyright. All rights are reserved by the Publisher, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilms or in any other physical way, and transmission or information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed. Exempted from this legal reservation are brief excerpts in connection with reviews or scholarly analysis or material supplied specifically for the purpose of being entered and executed on a computer system, for exclusive use by the purchaser of the work. Duplication of this publication or parts thereof is permitted only under the provisions of the Copyright Law of the Publisher's location, in its current version, and permission for use must always be obtained from Springer. Permissions for use may be obtained through RightsLink at the Copyright Clearance Center. Violations are liable to prosecution under the respective Copyright Law.

The use of general descriptive names, registered names, trademarks, service marks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.

While the advice and information in this book are believed to be true and accurate at the date of publication, neither the authors nor the editors nor the publisher can accept any legal responsibility for any errors or omissions that may be made. The publisher makes no warranty, express or implied, with respect to the material contained herein.

Printed on acid-free paper

Springer is part of Springer Science+Business Media (www.springer.com)

To my wife Valérie and kids Margaux, Estée and Alix d'Hooghe

Pieter

To my wife Lisette and kids Quirine and Jan Kerkhoffs

Gino

"La vraie générosité envers l'avenir, Consiste à tout donner au présent" Albert Camus

### Foreword



Message from the Honorary President of the International Olympic Committee

Ankle lesions are frequent in football. They represent 14% of the total number of injuries and and lead to a game incapacity of 164 days per season. This has important repercussions on players' careers and their teams' performance. Over recent years, the diagnostic approach to ankle lesions has changed completely thanks to the introduction of new clinical visions and new technical possibilities. This has led to more appropriate therapeutic measures, both conservatively and surgically, achieving much better results and faster resumption of competition.

The purpose of this book is to present these new preventive, diagnostic, and therapeutic strategies.

As an Olympian, orthopaedic surgeon, and IOC Honorary President, I have always promoted a healthy approach to sports practice.

I therefore congratulate the authors for producing what is a very useful and wellwritten book.

Lausanne - Vidy, Switzerland

Jacques Rogge

## Foreword





Football (soccer) is played by more than 300 million people globally. It is the most popular sport in the world, and participation in this dynamic sport continues to grow among men and women exponentially. As a high-impact contact sport, football is associated with significant acute and chronic foot and ankle contact forces with consequent and potential injury to the ligaments, bone, and the joint surfaces. FIFA's "Football Medicine" mission is manifest through the FIFA Medical Assessment and Research Center (F-MARC) and aims to prevent injury, optimize performance of the athlete and provide efficient care for the injured player. It is the aim of FIFA to communicate, educate, and implement the basic principles of this mission. This book The Ankle in Football, as edited by D'Hooghe and Kerkhoffs, systematically depicts critical anatomical and pathoanatomical issues and disorders that are of particular importance to the football athlete's ankle. In addition to identification of the spectrum of injury, the text clearly presents a comprehensive multidisciplinary and algorithmic approach that includes the prevention, diagnosis, treatment, rehabilitation, and return to play progressions. This book also provides a complete but concise reference guide for players, coaches, medical and training staff, and researchers working with football players. Furthermore, it intends to facilitate the advancement of science and education in football and the development of innovative treatment concepts and prevention strategies that help to further reduce the incidence of injury and risk of osteoarthritis in football players of all ages and skill levels.

Zurich, Switzerland Zurich, Switzerland Bert R. Mandelbaum MD, DHL (hon) Jiri Dvorak MD, F-MARC

## Foreword



Continuous education is a current priority of *ESSKA and FIFA*. The current publication is an important part of a strategy to provide sports medicine up-to-date knowledge and facilitate access to recent insights concerning some hot topics in a friendly, clear, and practical way. To achieve such goal we tried to gather contributions from some of the more renowned experts in the field, whom we thank for their precious contributions.

The goal of the authors was to provide high-performing educational sets. You will be joining us in this priceless educational mission. Consequently, it will allow you to keep in touch with the most advanced techniques, with ultimate technologies and under guidance of the best current practice rules. This way we expect to assure that high-quality standards in patient care will be achieved.

FIFA has a mission on Football for Health: "...playing football, protect yourself from injuries and learn how to live a healthy life. Health is key to development and growth. Football, the one passion that unites people of all ages, gender, languages, colour, cultures and religions, may prove a uniquely effective and low-cost tool in the world's fight for health." This is also part of the goals of this book.

In the subsequent pages dedicated to *The Ankle in Football*, the reader will be able to get acquainted with state of the art on that subject. This outstanding and generous share of knowledge conveys a comprehensive resource to education and clinical practice. There are many relevant improvements in the understanding of anatomy, biomechanics, and biology of the ankle and football lesions. In the same way we have changed from classical view of anatomy to the "arthroscopic" joint approach, we are now entering the era of biology and molecular understanding of healing.

Several surgical techniques addressing new problems have gained space from experimental therapies to evidence-based treatments.

The reading you are about to begin consubstantiates a text gathering the new fundamentals of basic science and clear explanation of surgical techniques to provide the best health care to patients. Science and skills brought to you by this book's authors arise from talented and passionate personalities that bring novelty to orthopedics. ESSKA and FIFA's "families" strive to give no less in education.

ESSKA President João Espregueira-Mendes

Porto, Portugal

## Preface





Football is the most popular sport worldwide. People played football from the eighteenth century onwards, and the game has developed tremendously through the years. Nowadays, it's the world's biggest side issue with all (dis)advantages that come with that status.

As football has evolved so importantly, the physical and psychological impact on the players' health, together with the upgraded multidisciplinary care for the players, grew along by becoming a big issue.

In this regard we see that ankle injuries present a high burden for all sports medicine physicians, physiotherapists, players, coaches, and paramedics involved worldwide in and around the football pitch. The challenge for the technical and medical staff in football nowadays is to prevent and treat injuries, according to their best possible standards. Ankle joint injuries present approximately 20 % of all injuries on the football pitch and can therefore put forward an increasing challenge to the player and his/her staff.

The current book aims at creating a unique platform that covers frequent ankle pathologies, specifically related to football. One chapter (on Metatarsal 5 base fractures) goes beyond the anatomical ankle region but has been added because of its ankle-related frequency and importance in football.

Most commonly, injuries are described from trauma mechanism, physical examination findings, and diagnostic and treatment algorithms towards rehabilitation programs and full return to sports. The extra dimension in this book is the "Interview Sections," where the input by elite level players, coaches, team doctor, physical therapist, scout, and referee offers a personal view on ankle-related treatments and rehabilitation in a much broader aspect than just the injury itself.

Therefore, this book is directed to orthopedic surgeons, sports medicine physicians, physiotherapists, general practitioners, club managers, football players, and coaches.

The authors are all scientifically active in the field of sports but are also well known for their great clinical experience in the care of football players, with often more than 20 years of expertise in the field.

The book is structured in a fashion that allows people to use it as a reference manual combining educational material with evidence-based information on ankle injuries in football.

We hope *The Ankle in Football* will help in your clinical practice, nourish your further interest, and improve your treatment strategies with the ultimate goal of protecting the ankles in football players.

Pieter P.R.N. d'Hooghe Gino M.M.J. Kerkhoffs

## Contents

1	Anatomy of the Ankle Pau Golanó, Miquel Dalmau-Pastor, Jordi Vega, and Jorge Pablo Batista	1
2	Interview – Raul. Pieter P.R.N. d'Hooghe and Gino M.M.J. Kerkhoffs	25
3	<b>Epidemiology and Mechanisms of Ankle Pathology in Football</b> Ryan L. Anderson, Lars Engebretsen, Nicholas Kennedy, Robert LaPrade, Adam M. Wegner, and Eric Giza	31
4	Interview – Ricardo Pruna Pieter P.R.N. d'Hooghe and Gino M.M.J. Kerkhoffs	61
5	<b>Prevention of Ankle Injuries</b> Miriam van Reijen and Evert Verhagen	65
6	Interview – Frank de Bleeckere Pieter P.R.N. d'Hooghe and Gino M.M.J. Kerkhoffs	77
7	Ankle Ligament Lesions Gino M.M.J. Kerkhoffs, Peter A.J. de Leeuw, Joshua N. Tennant, and Annunziato Amendola	81
8	Interview – Jan Wouters Pieter P.R.N. d'Hooghe and Gino M.M.J. Kerkhoffs	97
9	<b>Osteochondral Defects in the Ankle Joint</b> Rogier Gerards, Maartje Zengerink, and C. Niek van Dijk	101
10	Interview – Velibor "Bora" Milutinović Pieter P.R.N. d'Hooghe and Gino M.M.J. Kerkhoffs	119
11	Anterior Ankle Impingement Johannes L. Tol, Pieter P.R.N. d'Hooghe, Peter A.J. de Leeuw, Mario Maas, and Gino M.M.J. Kerkhoffs	123

Contents

12	Interview – Eva Blewanus	135
13	<b>Posterior Ankle Impingement</b> Gino M.M.J. Kerkhoffs, Peter A.J. de Leeuw, and Pieter P.R.N. d'Hooghe	141
14	Interview – Ron Spelbos Pieter P.R.N. d'Hooghe and Gino M.M.J. Kerkhoffs	155
15	Ankle Fractures, Including Avulsion Fractures Kyriacos I. Eleftheriou, James D.F. Calder, Peter Kloen, and Pieter P.R.N. d'Hooghe	159
16	Interview – Leonne Stentler Pieter P.R.N. d'Hooghe and Gino M.M.J. Kerkhoffs	187
17	Fifth Metatarsal Stress Fractures in Football Hanneke Weel, Simon Goedegebuure, Pieter P.R.N. d'Hooghe, and Gino M.M.J. Kerkhoffs	193
18	Acute Achilles Tendon Rupture Jon Karlsson, Nicklas Olsson, and Katarina Nilsson Helander	201

	Jon Karlsson, Nicklas Olsson, and Katarina Nilsson Helander	
19	Achilles Tendinopathy	213
	Robert Jan de Vos, Pieter P.R.N. d'Hooghe, Peter de Leeuw, and Gino M.M.J. Kerkhoffs	
20	Peroneal and Posterior Tibial Tendon Pathology	235
	Hélder Pereira, Pedro Luís Ripoll, Joaquim M. Oliveira,	

	Rui L. Reis, João Espregueira-Mendes, and C. Niek van Dijk	
21	<b>The Footballer's Inlay Sole: An Individualised Approach</b> D.P. Francisco Escobar Ruiz and Pieter P.R.N. d'Hooghe	253
22	Rehabilitation After Ankle Football Injuries	269

23	Taping Techniques and Braces in Football	287
	Ruben Zwiers, Leendert Blankevoort, Chris W.A. Swier,	
	A. Claire M. Verheul, and Gino M.M.J. Kerkhoffs	
24	Ankle Osteoarthritis in Former Elite Football	

24	Ankle Osteoarthritis in Former Elite Football	
	Players: What Do We Know?	311
	Vincent Gouttebarge and Monique H.W. Frings-Dresen	

## Chapter 1 Anatomy of the Ankle

Pau Golanó, Miquel Dalmau-Pastor, Jordi Vega, and Jorge Pablo Batista

The best Football referee has the sharpest eyes and the lousiest ears.

Mario van der Ende Ex-international Referee

**Abstract** A thorough knowledge of the ankle anatomy is absolutely necessary for diagnosis and adequate treatment of ankle injury. The ankle is among the most prevalent joints injured in sports, the ankle sprain being the most common ankle lesion. The ankle joint is formed by the distal tibia and fibula, and the superior talus. Two ligamentous complexes join the bones that form the ankle: the ligaments of the tibiofibular syndesmosis (anteroinferior tibiofibular, posteroinferior tibiofibular, and the interosseous tibiofibular ligament) and the lateral (anterior talofibular, calcaneo-fibular, and the posterior talofibular ligament) and medial (superficial and deep

P. Golanó, MD (🖂)

Laboratory of Arthroscopic and Surgical Anatomy,

Department of Pathology and Experimental Therapeutics,

Human Anatomy Unit, University of Barcelona, Barcelona, Spain

Department of Orthopaedic Surgery, University of Pittsburgh, Pittsburgh, PA, USA e-mail: paugolano@gmail.com

M. Dalmau-Pastor, PT, POD Laboratory of Arthroscopic and Surgical Anatomy, Department of Pathology and Experimental Therapeutics, Human Anatomy Unit, University of Barcelona, Barcelona, Spain e-mail: mikeldalmau@gmail.com

J. Vega, MD Unit of Foot and Ankle Surgery, Etzelclinic, Pfäffikon, Schwyz, Switzerland e-mail: jordivega@hotmail.com

J.P. Batista, MD Orthopaedic Department, Club Atlético Boca Juniors, Buenos Aires, Argentina

Centro Artroscópico Jorge Batista, Buenos Aires, Argentina e-mail: jbatista20@hotmail.com

P.P.R.N. d'Hooghe, G.M.M.J. Kerkhoffs (eds.), *The Ankle in Football*, Sports and Traumatology, DOI 10.1007/978-2-8178-0523-8\_1, © Springer-Verlag France 2014 component) collateral ligaments. The anterior talofibular ligament is the main stabilizer on the lateral aspect of the ankle, limiting the anterior translation and internal rotation of the talus. Because most ankle sprains occur by inversion, this ligament is the most frequently injured, and it has been involved in the soft-tissue impingement syndrome and the microinstability and the major instability of the ankle.

#### 1.1 Introduction

The talocrural or ankle joint is a load-bearing joint formed by the articular surfaces of the tibial and fibular distal epiphyses and the talus in its superior, lateral, and medial aspects. The talus is firmly gripped between the tibia and the two malleoli, medial and lateral malleoli, respectively, which form a mortise to receive the talus (Fig. 1.1). The morphology of these surfaces forms a ginglymus or hinge-type synovial joint (Fig. 1.2) with a single axis of movement (bimalleolar axis) that allows dorsiflexion (flexion) and plantar flexion (extension) of the ankle and foot in the sagittal plane. The range of ankle motion is variable. The methodology used to measure it (clinical, roentgenographic, or anatomic) accounts for some of the reported discrepancies. The normal range of motion has been reported to be  $13-33^{\circ}$  of dorsiflexion and  $23-56^{\circ}$  of plantar flexion [1].



**Fig. 1.1** Bone components of the ankle joint. *1* Inferior articular surface of the tibia. *2* Articular surface of the medial malleolus. *3* Articular surface of the lateral malleolus. *4* Superior articular surface of the talus. *5* Lateral articular surface of the talus. *6* Medial articular surface of the talus. Cartilage has been colored digitally by Adobe<sup>®</sup> Photoshop<sup>®</sup> software (Figure copyright © Pau Golanó)

Fig. 1.2 Frontal section of the ankle and subtalar joints. 1 Tibia. 2 Medial malleolus. 3 Lateral malleolus. 4 Talus. 5 Calcaneus, 6 Sustentaculum tali, 7 Tibiofibular syndesmosis. 8 Interosseous talocalcaneal ligament. 9 Peroneus brevis tendon. 10 Peroneus longus tendon. 11 Tibialis posterior tendon. 12 Flexor digitorum longus. 13 Flexor hallucis longus. 14 Sural nerve and small saphenous vein. 15 Medial plantar nerve, plantar medial artery and veins. 16 Lateral plantar nerve, lateral plantar artery and veins. The nerves and vessels have been colored digitally with Adobe® Photoshop® software (Figure copyright © Pau Golanó)



The bones are connected by a fibrous capsule which is similar to the capsule of any other joint, with the exception of a singular characteristic: the anterior capsular insertion in the tibia and talus occurs at a distance from the cartilaginous layer (Fig. 1.3). According to Testut and Latarjet [2], the distance is approximately 6–8 mm in the tibia and 8–10 mm in the talus. In a recent study, the distance was found to be 4.3 mm (0.5–9.0 mm) and 2.4 mm (1.8–3.3 mm) in the tibia and talus, respectively [3]. This anatomic detail belies the hypothesis of formation of talotibial spurs, frequently found in athletes, due to recurrent traction of the joint capsule. The repetitive direct (micro) trauma on the ankle cartilage rim by repetitive kicking action in soccer will induce inflammation, the development of scar tissue, calcification, and, subsequently, reactive spur formation [4]. The pain is probably caused by impingement of synovial or posttraumatic scar tissue between the bony surface of the distal tibia and/or malleoli and the bony contours of the talus (Fig. 1.4).

On the other hand, this peculiarity of the ankle capsule insertion determines the existence of a substantial anterior capsular recess that allows the arthroscopist to encounter a working area. Nevertheless, the size of this area depends on the position of the foot.

Together with the elbow, the ankle is one of the most congruous joints in the body. The stability of the ankle is determined by passive and dynamic factors. The passive stability depends on the contour of the articular surfaces, the articular



**Fig. 1.3** (a) A lateral weight-bearing foot x-ray showing a tibial osteophyte. (b) Arthroscopic view of the ankle through anterolateral portal showing tibial and talar osteophytes. *1* Tibial osteophyte. *2* Articular surface of the medial malleolus. *3* Talar osteophyte (Figure copyright © Pau Golanó)

capsule and integrity of the collateral ligaments, and the retinacula around the joint. The dynamic stability is conferred by the muscle action mainly.

Based on its functional relationship to the rest of the body, it is not surprising that ankle injuries are among the most common, particularly in sport such as basketball (45 %), football (31 %), and handball (25 %) [5]. The ankle sprain accounts for 85 % of all ankle lesions [6, 7].

The most common mechanism of injury to the ankle ligament is inversion sprain (Fig. 1.5). This mechanism affects from 75 % [8] to over 90 % [9] of the cases involving the lateral collateral ligament. With this mechanism of injury, the anterior talofibular ligament is the first or only ligament to sustain injury [10]. The anterior talofibular ligament restricts the anterior translation and the internal rotation of the talus [11, 12]. An abnormal anterior drawer sign has been reported in 42 % of patients after an ankle sprain [13]. When the mechanism of injury continues around the lateral aspect of the ankle, rupture of the anterior talofibular ligament is followed by the damage to the calcaneofibular ligament and finally to the posterior talofibular ligament [14].

Injury to the medial collateral ligament is much less frequent, accounting for about 5–15 % of all ankle ligamentous lesions [15, 16]. Medial collateral ligament injury rarely occurs alone; it is more often associated with other ligamentous injuries or fractures. Syndesmotic injuries occur in 1–18 % [17] of patients with an ankle sprain and are more common in collision sports involving a forced ankle dorsiflexion.

Most of these patients are successfully treated conservatively. However, residual symptoms after ankle sprain are reported in 30–40 % of patients [18], including chronic pain, muscular weakness, and recurrent giving way or instability [18, 19].



**Fig. 1.4** Sagittal section of the ankle with joint distraction showing the characteristic insertion of the anterior capsule joint. *1* Ankle anterior joint capsule (and *black arrows*). *2* Inferior articular surface of the tibia. *3* Anterior cartilage rim. *4* Superior articular surface of the talus. *5* Intra-articular but extrasynovial fat pad located in the anterior synovial recess. This structure can be compared to the Hoffa fat pad of the knee. *6* Transverse ligament (deep component of the posterior tibiofibular ligament). *7* Posterior intermalleolar ligament. *8* Posterior talofibular ligament. *9* Posterior talar process. *10* Posterior subtalar joint. *11* Ankle posterior joint capsule (and *black arrows*). *12* Deep fascia (fibular-talocalcaneal ligament). *13* Flexor hallucis longus muscle belly. *14* Ankle joint. Cartilage has been colored digitally by Adobe<sup>®</sup> Photoshop<sup>®</sup> software (Figure copyright © Pau Golanó)

The most frequent cause of chronic pain after an ankle sprain is known as soft tissue impingement syndrome [5] (Fig. 1.6). However, recurrent instability of the ankle is a known sequela following inversion injuries and is estimated to occur in approximately 10-20 % of patients regardless of the type of initial treatment [20, 21].

The primary etiology of all these conditions is injury to the ligamentous structures.

An understanding of the anatomy and biomechanics of the ligamentous complexes is essential for diagnosis and adequate treatment of this condition. Therefore, and because there are relatively few published studies centering on these ligaments [22, 23] as compared to those in other joints, the aim of this chapter is to review the anatomy of ankle ligaments.

In order to simplify the description of the ankle ligaments, we have divided the structures involved into two sections as follows:

- Ligaments that join the distal epiphyses of the bones of the leg (tibia and fibula): the ligaments of the tibiofibular syndesmosis
- Ligaments that join the tibia and fibula to the skeletal structure of the foot: the lateral collateral ligament and medial collateral ligament

**Fig. 1.5** Adobe<sup>®</sup> Photoshop<sup>®</sup> photocomposition of the ankle inversion sprain mechanism. The anterior talofibular ligament is the weakest of three lateral collateral ligaments involved in practically all ankle inversion sprains. *I* Anterior talofibular ligament (Reproduced with permission: Golanó and Vega [24]. Figure copyright © Pau Golanó)





**Fig. 1.6** Arthroscopic view of the ankle impingement through the anteromedial portal (Image courtesy of Dr José Achalandabaso (San Sebastián, Spain))

## **1.2 Ligaments That Join the Distal Epiphyses** of the Tibia and Fibula

The distal epiphyses of the tibia and fibula are firmly joined by ligaments, creating a moveable joint system that encompasses the talus, thus forming the talocrural joint.

The articular surfaces of the tibia and the fibula form a triangular configuration with a distal base. The surface provided by both the fibula and the tibia, called the tibial and fibular notch, respectively, is rough in the proximal region since it is the insertion site for one of the syndesmotic ligaments, the interosseous tibiofibular ligament, which is simply the continuation of the interosseous membrane at this level. Distal to the insertion site of this ligament, the remaining anterior surface corresponds to the tibiofibular synovial recess of the ankle joint, and at the posterior surface there is a small bundle of adipose tissue called the fatty synovial fringe. This structure has been implicated as a cause of chronic pain following ankle sprain in the condition known as syndesmotic impingement [25, 26] (Fig. 1.7).

As can be deduced from the above description, the distal tibiofibular joint has no articular cartilage (Fig. 1.2). It is a syndesmotic articulation that allows the tibia-fibula as a whole to adapt to the varying width of the upper articular surface of the



**Fig. 1.7** (a) Arthroscopic view of the normal syndesmotic area through the anteromedial portal. (b) Typical appearance of a syndesmotic ankle impingement showing the erythematous synovial fringe during arthroscopic resection. *I* Synovial fringe. *2* Inferior articular surface of the tibia. *3* Articular surface of the lateral malleolus. *4* Superior articular surface of the talus. *5* Transverse ligament (deep component of the posterior tibiofibular ligament) (Image courtesy of Dr José Achalandabaso (San Sebastián, Spain))

talus; it does so through the slight ascending and medial rotation movements of the fibula during extreme dorsiflexion (maximum width) and by inverse movements during plantar flexion (minimum width) [27].

Three ligaments join the distal tibial and fibular epiphyses: the anterior or anteroinferior tibiofibular ligament, the posterior or posteroinferior tibiofibular ligament, and the interosseous tibiofibular ligament. The inferior segment of the interosseous membrane also helps stabilize the tibiofibular syndesmosis.

#### 1.2.1 Anterior or Anteroinferior Tibiofibular Ligament

The anterior tibiofibular ligament is the weakest of all the syndesmotic ligaments and the first to yield when the fibula is turned outward over its longitudinal axis [28]. The ligament originates in the anterior tubercle of the tibia, and its fibers extend in a distal and lateral direction to the insertion site in the anterior margin of the lateral malleolus. Upon examination, the ligament is seen to be divided into several fascicles, which gives it a multifascicular morphology (Fig. 1.8a). This multifascicular appearance is probably due to its relationship with the perforating branch of the peroneal artery, which runs along the surface of the ligament, providing small vessels that penetrate through the interfascicular spaces [22].

The importance of this ligament lies in its distal portion, which has been associated with anterolateral pain after ankle sprain [29]. Knowledge of its configuration is important to understand the anatomic bases for anterolateral soft tissue impingement [30], since abrasion between the distal fascicle of the anterior tibiofibular ligament and the talus will lead to pain.

On careful inspection, the most distal fascicle of the anterior tibiofibular ligament appears to be independent from the rest of the structure. It is separated by a septum of fibroadipose tissue and may be slightly deeper than the rest of the ligament. This relative independence of the distal fascicle from the anterior tibiofibular ligament led authors such as Nikolopoulos [31] to give it a separate name: the accessory anteroinferior tibiofibular ligament. This term was refuted years later by Basset et al. [29] which led to its designation as distal fascicle of the anteroinferior tibiofibular ligament. Due to its intracapsular and extrasynovial location, the distal fascicle of the anteroinferior tibiofibular ligament can be seen by arthroscopy (Fig. 1.8b, c).

**Fig. 1.8** Osteoarticular dissection of the ankle joint. (**a**) Anterolateral view. (**b**) Anterior view. (**c**) Arthroscopic correlation view of the distal fascicle of the anterior tibiofibular ligament through anteromedial portal. *1* Anterior tibiofibular ligament. *2* Distal fascicle of the anterior tibiofibular ligament. *3* Anterior tubercle of the tibia. *4* Interosseous membrane. *5* Interosseous membrane perforation for the passage of the anterior peroneal artery branch (*red arrow*). *6* Anterior talofibular ligament. *7* Calcaneofibular ligament. *8* Dorsal talonavicular ligament. *9* Interosseous talocalcaneal ligament. *10* Inferior extensor retinaculum (*cut*). *11* Bifurcate ligament. *12* Cuboid bone. *13* Peroneus brevis tendon. *14* Peroneus longus tendon. *15* Calcaneal or Achilles tendon. *16* Superior peroneal retinaculum (*cut*). *17* Superior extensor retinaculum (*cut*) (Figure copyright © Pau Golanó)





In its oblique course toward insertion in the fibula, the distal fascicle covers the angle formed by the tibia and fibula and comes into contact with the dorsolateral border of the talus during flexion of the ankle. Basset et al. [29] measured the dimensions and the degrees of dorsiflexion needed for the distal fascicle to come into contact with the talus (mean 12°). The author also published a series of seven case studies in which resection of the distal fascicle of the anterior tibiofibular ligament satisfactorily resolved the symptoms of patients with chronic ankle pain who had a history of inversion sprains of the ankle. Thickening of the ligament was seen in all these cases, and five patients additionally showed abrasion of the joint cartilage in the region where the ligament came into contact with the talus. Resection of the distal fascicle as a therapeutic approach, whether by open surgery or arthroscopy, does not produce noticeable changes in the stability of the ankle [31-33] and leads to clinical improvement. However, impingement of the distal fascicle of the anterior tibiofibular ligament seems to depend on changes in the ankle mechanics [29] and on the distal fascicle obliquity [34]. An injury to the lateral collateral ligament, specially to the anterior talofibular ligament, would increase anteroposterior laxity of the ankle [11, 12]. This, in turn, would result in increased anterior extrusion of the talus and cause the distal fascicle to have greater contact and pressure on the talus [29]. Another factor related to distal fascicle impingement is its obliquity or the level at which the anterior tibiofibular ligament is inserted in the fibula with respect to the joint line. More distal insertion of the ligament could lead to increased contact in the neutral position of the ankle and a higher potential for ligamentous pathology [34].

Our observations in the dissection room have allowed us to identify contact between the distal fascicle and the talus in the neutral position [22]. Contact decreases with joint distraction [34] or dorsal flexion of the ankle, which should be taken into account during arthroscopy. These findings should be considered as a normal feature [22, 34]. In cases of anatomic variation or ankle instability, the feature may be pathological. The lateral ankle instability is a direct factor in the anterior tibiofibular ligament pathology, and therefore, its impingement should be considered a consequence of the ankle instability. In fact, Kim and Ha [35] considered that isolated impingement by this entity is uncommon, the condition usually being associated with anterior talofibular ligament pathology (ankle sprain).

#### 1.2.2 Posterior or Posteroinferior Tibiofibular Ligament

According to the classic descriptions, the posterior or posteroinferior tibiofibular ligament is formed by two components, one superficial and the other deep [36], although this subdivision is not accepted by all authors (Fig. 1.9). On the basis of its position, Nikolopoulos et al. [32] compared this ligament to the anterior tibiofibular ligament and its distal fascicle. The terminology used for this ligament and its components is controversial [37], and this fact is particularly evident in the arthroscopic literature [38].

#### 1 Anatomy of the Ankle



Fig. 1.9 Posterior view of an osteoarticular dissection of the ankle joint. *1* Posterior tibiofibular ligament, retracted by surgical instrument. 2 Posterior tibial tubercle. *3* Transverse ligament (deep component of the posterior tibiofibular ligament). *4* Fibular malleolar fossa. *5* Posterior talofibular ligament. *6* Calcaneofibular ligament. *7* Posterior intermalleolar ligament (and *gray arrows*). 8 Lateral talar tubercle. *9* Medial talar tubercle. *10* Medial collateral ligament. *11*. Inferior peroneal retinaculum (and *black arrows*). *12* Peroneal tendons path. *13* Tibial malleolar sulcus. *14* Flexor retinaculum (and *black arrows*). *15* Tibialis posterior tendon path. *18* Flexor hallucis longus tendon path. *19* Flexor hallucis longus tendon retinaculum (Figure copyright © Pau Golanó)

The superficial component originates at the posterior tubercle and runs distally and laterally toward the fibula, where it inserts in the posterior edge of the lateral malleolus. This component would be homologous to the anterior tibiofibular ligament. The term posterior or posteroinferior tibiofibular ligament is usually used to refer to the superficial component.

Sarrafian coined the term transverse ligament to refer to the deep component [36]. This is a cone-shaped structure, with its fibers turning on during its course. It originates at the posterior edge of the tibia, immediately posterior to the cartilaginous covering of the inferior tibial articular surface, and runs toward the fibula, with insertion at the proximal area of the malleolar fossa. The transverse ligament extends beyond the osseous margin in a distal direction, conforming a true labrum [36] dependent on the inferior articular surface of the tibia. By increasing the size and concavity of the tibial joint surface, it provides talocrural joint stability and prevents posterior talar translation [39].

On arthroscopic examination through anterior portals, many authors report that the posterior tibiofibular ligament (superficial component) is readily visualized,



**Fig. 1.10** Arthroscopic view of the normal ankle posterior area through the anteromedial portal. On arthroscopic examination through anterior portals, only the deep component of the posterior syndesmotic ligaments is visible arthroscopically. The superficial component cannot be seen. *I* Synovial fringe. 2 Inferior articular surface of the tibia. *3* Articular surface of the lateral malleolus. *4* Superior articular surface of the talus. *5* Transverse ligament (deep component of the posterior tibiofibular ligament). *6* Posterior intermalleolar ligament (Image courtesy of Dr José Achalandabaso (San Sebastián, Spain))

but there is some controversy regarding the transverse ligament (deep component). We have shown that only the deep component is visible arthroscopically and that the superficial component cannot be seen, thus helping to clarify previous descriptions of this ligament that might be confusing [38] (Fig. 1.10). The superficial component can be seen during ankle posterior endoscopy.

#### 1.2.3 Interosseous Tibiofibular Ligament

The interosseous tibiofibular ligament is a dense mass of short fibers, which, together with adipose tissue and small branching vessels from the peroneal artery, span the tibia to the fibula. It can be considered a distal continuation of the interosseous membrane at the level of the tibiofibular syndesmosis [2, 36].

#### **1.3** Ligaments That Join the Leg Bones to the Skeletal Structure of the Foot

The ligaments that join the tibia and fibula to the skeletal structure of the foot have been grouped into two main ligamentous complexes, the lateral collateral ligaments and the medial collateral ligaments.

#### **1.3.1** Lateral Collateral Ligaments

Located on the lateral part of the joint, the lateral collateral ligament (LCL) is comprised of three fascicles or ligaments, entirely independent from each other: the anterior talofibular, the calcaneofibular, and the posterior talofibular ligaments.

#### 1.3.1.1 Anterior Talofibular Ligament

The anterior talofibular ligament (ATFL) is the most frequently injured ligament of the ankle. This ligament is the main stabilizer on the lateral aspect of the ankle [40]. Due to the fact that most ankle injuries occur by inversion with the foot in plantar flexion, the ATFL is the most vulnerable to injuries.

This flat, quadrilateral ligament is in close contact with the capsule and is considered an intrinsic ligament reinforcing the joint capsule. The ATFL is typically composed of two bands separated by an interval that allows penetration of the vascular branches from the perforating peroneal artery and its anastomosis with the lateral malleolar artery; the upper band is larger than the lower one (Fig. 1.11). Occasionally, there are three bands [36], although we have never observed this morphology in our dissections [22]. Milner and Soames [41] found that the ligament had a single band in 38 % of cases, two bands in 50 % of cases, and three bands in 12 %, in contrast with Sarrafian's observations [36]. Nevertheless, it is notable that subsequent anatomic studies [42, 43] to determine the anatomy and dimensions of the parts of the lateral collateral ligament do not stress this aspect. In a study using MRI to investigate 22 patients with no history of ankle sprains, Delfaut [44] found that the anterior talofibular ligament had a single band in 9 % of cases, two bands in 55 %, and a striated appearance in 36 %. Our dissections [22] have shown that this ligament most commonly has a double-band morphology, as described by Sarrafian [36]. The upper band reaches the insertion on the fibula of the anterior tibiofibular ligament. This anatomic fact is critical to locate the anchor position during the new arthroscopic lateral ligament repair techniques [45]. The inferior band reaches the origin of the calcaneofibular ligament. In many specimens, these latter ligaments (ATFL and calcaneofibular ligament) are joined by arciform fibers at the malleolar origin [36].

Fig. 1.11 Lateral view of the osteoarticular dissection of the ankle joint showing the typical morphology of the anterior talofibular ligament. 1 Anterior talofibular ligament. 2 Calcaneofibular ligament. 3 Anterior tibiofibular ligament. 4 Distal fascicle of the anterior tibiofibular ligament. 5 Anterior tubercle of the tibia. 6 Lateral talocalcaneal ligament. 7 Interosseous talocalcaneal ligament. 8 Cervical ligament. 9 Bifurcate ligament. 10 Dorsal talonavicular ligament. 11 Superior peroneal retinaculum (cut). 12 Long plantar ligament (Figure copyright © Pau Golanó)



In its entirety, the ATFL originates at the anterior margin of the lateral malleolus. The center of this fibular insertion is an average 10 mm proximal to the tip of the fibula as measured along the axis of the fibula [42]. At this level, it is important to remind that the fibular origin (inferior band) shares the fibular origin of the CFL as recently demonstrated [46]. From its origin, the ATFL runs anteromedially to the insertion points at the talus body, immediately anterior to the joint surface occupied by the lateral malleolus, consisting of two small tubercles visible in osseous anatomic preparations and corresponding to the insertion sites of each of the bands. The ligament is virtually horizontal to the ankle in the neutral position but inclines upward in dorsiflexion and downward in plantar flexion. This condition can be seen during ankle arthroscopy to be located in the floor of the lateral recess of the ankle [45].

Ferkel [47, 48] has cited this ligament as a cause of anterolateral soft tissue impingement. An inadequate treatment of the ankle sprain could lead to an inflammatory process in the area of the injury, followed by synovitis and the formation of prominent scar tissue. This mass of tissue would occupy the lateral recess of the joint, possibly causing pain and irritation that would lead to the development of

additional inflammatory tissue and eventually to chronic pain. Chronic progression of this inflammatory tissue, together with remnant scar tissue, would produce the so-called meniscoid tissue described by Wolin [47–49].

Although anterolateral soft tissue impingement syndrome has been well described by Ferkel, patients can continue to experience symptoms and instability even after an operative procedure in which the pathological soft tissue has been successfully resected [48, 50-58]. The possibility of lesion to other regions and the presence of an injured lateral ligament should also be considered. We must remember that the bands of this ATFL have a different behavior during the ankle movements. In plantar flexion, the inferior band of the ligament remains relaxed, while the superior band becomes tight. In dorsiflexion, the superior band remains relaxed, and the inferior band becomes tight. Since the superior band of the ATFL restricts inversion in plantarflexion, it may be the most important of the two bundles. Injury of this superior band of the ATFL is always present after an inversion ankle sprain. Although an injury to the ATFL may not be severe enough to cause a major instability, as we have observed in our arthroscopic experience in treating patients with anterolateral chronic pain, partial injuries affecting the superior band are present in most of these patients. In fact, we consider that isolated impingement, as Ferkel referred in its original studies [47, 48], is uncommon and that the condition is usually associated with an injury of the superior band of the ATFL, resulting in a minor instability or microinstability of the ankle. This partial injury of the ATFL is not severe enough to cause an instability detected in stress explorations. However, these patients describe complaints similar to those to the ankle instability. Repairing the partial injury of the ATFL should be performed to treat ankle microinstability. A complete or nearly complete injury of the ATFL results in a major instability.

#### 1.3.1.2 Calcaneofibular Ligament

The calcaneofibular ligament (CFL) is a thick, cord-like ligament that originates at the anterior edge of the lateral malleolus, right below the origin of the lower band of the ATFL, to which it can be joined by arciform fibers. It is important to note that the origin of this ligament does not reach the tip of the malleolus, which remains free from ligamentous insertions (Fig. 1.11). In the neutral position, the ligament courses backward, downward, and medially and inserts in a small tubercle in the posterior region of the lateral calcaneus, posterior to the peroneal tubercle.

This ligament is superficially crossed by the peroneal tendons and sheaths, which can leave a concavity over the ligament; only about 1 cm of the ligament is uncovered [36]. In addition, the CFL is separated from the subtalar (talocalcaneal) joint by the lateral talocalcaneal ligament and is separated from this ligament by adipose tissue.

The CFL controls two joints, the talocrural joint and the subtalar joint, unlike the other two elements comprising the LCL, which only affect the talocrural. Little attention has been given to this ligament as compared to the other ligaments of the LCL, although variants in the orientation of the structure have been studied by

Ruth [59]. The CFL becomes horizontal during extension and vertical in flexion, remaining tense throughout its entire arc of motion. A valgus or varus position of the talus considerably changes the angle formed by the ligament and the longitudinal axis of the fibula. The ligament is relaxed in the valgus position and tensed in the varus position. This would explain the potential for injury even without dorsi-plantar flexion movement in the ankle.

Isolated rupture of the CFL remains very rare and usually is combined with a rupture of the ATFL [40].

#### 1.3.1.3 Posterior Talofibular Ligament

The posterior talofibular ligament (PTFL) is a strong, thick, fascicled, trapezoidal ligament in an intracapsular but extrasynovial location, found in an almost horizontal plane (Fig. 1.12). It originates on the medial surface of the lateral malleolus in the malleolar fossa and courses toward the posterolateral talus. In the posterior view, the overall structure is triangular in shape, with the vertex located laterally and

Fig. 1.12 Posterior view of the osteoarticular dissection of the ankle joint. 1 Posterior talofibular ligament. 2 Lateral talar tubercle. 3 Posterior intermalleolar ligament (and gray arrows). 4 Calcaneofibular ligament. 5 Posterior tibiofibular ligament (superficial component). 6 Transverse ligament (deep component of the posterior tibiofibular ligament). 7 Posterior tibial tubercle. 8 Interosseous membrane. 9 Interosseous tibiofibular ligament. 10 Medial talar tubercle. 11 Peroneal tendons path. 12 Tibialis posterior tendon path. 13 Flexor digitorum longus tendon path. 14 Flexor hallucis longus tendon path. 15 Flexor hallucis longus tendon retinaculum. 16 Calcaneal or Achilles tendon (Figure copyright © Pau Golanó)



the base medially. The fibers of the ligament are inserted along the lateral aspect of the talus, on a rough, grooved surface situated along the posteroinferior border of the talar lateral malleolar surface. Other fibers are inserted in the posterior surface of the talus and may reach the lateral talar tubercle, trigonal process, or os trigonum by expansion. These fibers can be seen during posterior endoscopy and must be disinserted in order to achieve a successful lateral prominent tubercle or os trigonum excision during posterior ankle impingement treatment. The PTFL is tight during dorsiflexion and relaxed during plantarflexion of the ankle and is usually not injured after an ankle sprain, unless a true dislocation of the ankle joint occurs [40].

Furthermore, fibers from the PTFL can contribute to form the tunnel of the flexor hallucis longus tendon. Another group of fibers originates at the upper edge of the ligament near its origin and courses in an upward medial direction to the insertion site at the posterior edge of the tibia. These fibers fuse with the deep component of the posterior tibiofibular ligament (or transverse ligament), reaching the posterior surface of the medial malleolus and helping to form the existing labrum in the posterior margin of the tibia. This group of fibers has been given several names (capsular reinforcement bundle [60], ascending or tibial bundle of the posterior talofibular ligament [2]) although we prefer the one proposed by Paturet [61]: posterior intermalleolar ligament (Fig. 1.12). This ligament has been designated the tibial slip by Chen [62] and Ikeuchi [63] in several articles on arthroscopy.

The posterior intermalleolar ligament has been the subject of several studies because of its involvement in the posterior soft tissue impingement syndrome of the ankle [64, 65]. Rosenberg et al. [66] observed this ligament in 56 % of the dissections he performed and 19 % of patients in an MRI study, attributing the difference in frequency to the limited spatial resolution of MRI. In an MRI study of 23 classical ballet dancers with symptoms of posterior ankle impingement syndrome, Peace et al. [67] detected the posterior intermalleolar in 48 % of the cases, a higher incidence than found by Rosenberg. The author ascribed the difference to the possibility that among these patients the ligament would be more obvious on MRI because of scar thickening and inflammation resulting from repeated trauma. In the recent study of Oh et al. [68] on the morphology of the posterior intermalleolar ligament and its correlation with MR images, the posterior intermalleolar ligament was observed in 81.8 % of the specimens.

In their anatomic study, Milner and Soames [43] reported the presence of the posterior intermalleolar ligament in 72 % of the specimens. Golanó et al. [38] identified this ligament in all his dissections as well as in the arthroscopic study performed. In our opinion, the difference in frequency is probably due to the small size of the posterior intermalleolar ligament (mean 2.3 mm, range 1–5 mm), interracial variations [68], and the fact that it is difficult to dissect [38]. In addition, the ligament may be divided (20 % [66]–100 % [68]) into 2 or 3 different bands, any of which may not achieve bony insertion and become inserted in the joint capsule of the ankle. Upon examination by arthroscopy, the division of this structure into bands should not be confused with an injury.

In the posterior view, the posterior intermalleolar ligament is situated between the transverse ligament and the posterior talofibular ligament and runs obliquely from lateral to medial and from downward to upward. The posterior intermalleolar ligament tenses during dorsiflexion and relaxes during plantar flexion [38], and therefore trauma that causes forced dorsiflexion of the ankle can be assumed to produce either injury or rupture of this ligament or osteochondral avulsion [69]. Plantar flexion would cause it to relax and become susceptible to entrapment between the tibia and the talus, leading to impingement. The possibility of impingement is accentuated by the presence of predisposing factors such as an os trigonum or a prominent lateral tubercle of the os talus (Stieda process). Debridement of this ligament or resection of the os trigonum or lateral tubercle through an endoscopic technique [70] has been reported with excellent results [71–79].

#### 1.3.2 Medial Collateral Ligament

The medial collateral ligament (MCL) is a strong, broad ligament with a multifascicular appearance that spans out from the medial malleolus toward the navicular, talus, and calcaneus (Fig. 1.13a). Because the origins and insertions of the various fascicles or components of the MCL are contiguous and poorly defined, the anatomic descriptions show several interpretations, with artificial division being common. However, most authors appear to agree that the MCL has two components, one superficial and another deep, separated by a fat pad, each one formed by multiple components [36, 80–82]. The ligaments that comprise the superficial plane cross over two articulations, the ankle and the subtalar joints, whereas those comprising the deep plane only cross the ankle joint [81]. Nevertheless, this differentiation is not entirely clear [82].

Most of the MCL is covered by tendons as it extends down the leg to the bony insertions in the foot. The anterior region, in continuation with the joint capsule, is covered by the tendon of the posterior tibial muscle, and the middle and posterior area is covered by the tendons of the posterior tibial and long flexor muscles of the toes. The floor of the fibrosynovial sheaths covering these tendons is frequently composed of fibrocartilaginous tissue that is firmly adhered to the MCL. Precise dissection is needed to separate the fibrous sheath from the MCL. In the posterior region, the MCL continues with the posterior capsule of the ankle joint.

The most commonly accepted description of the MCL is the one proposed by Milner and Soames [81] and later corroborated by Boss and Hintermann [82] (Table 1.1). According with their attachments, six bands or components have been described for the MCL: four components or fascicles belong to the superficial layer and two to the deep. Three of them are always present (the tibiospring ligament, tibionavicular ligament, and deep posterior tibiotalar ligament), whereas the presence of the other three may vary (superficial posterior tibiotalar ligament, tibiocal-caneal ligament, and deep anterior tibiotalar ligament) (Fig. 1.13a, b).

The superficial layer is composed of four fascicles. However, only two of these are found in every instance, the tibiospring and tibionavicular ligaments. The other two, superficial tibiotalar and tibiocalcaneal ligaments, may vary. The deep layer is formed by two components, one posterior and one anterior. The deep posterior

#### 1 Anatomy of the Ankle



**Fig. 1.13** Medial view of the osteoarticular dissection of the ankle joint showing the morphology of the major components of the medial collateral ligaments. (**a**) Plantar flexion. (**b**) Dorsiflexion. *I* Medial malleolus. 2 Tibiospring ligament. 3 Spring ligament (superomedial calcaneonavicular ligament). 4 Tibionavicular ligament. 5 Deep posterior tibiotalar ligament. 6 Medial talar tubercle. 7 Navicular tuberosity. 8 Tibialis posterior tendon insertion (*cut*). 9 Plantar calcaneonavicular ligament. *10* Long plantar ligament. *11* Sustentaculum tali. *12* Anterior tibialis tendon (*cut*). *13* Calcaneal or Achilles tendon. *14* Medial talocalcaneal ligament
Sarrafian [36]	Milner and Soames [81]
Superficial layer	
Tibioligamentous fascicle	Tibiospring ligament (major component)
Tibionavicular fascicle and anterior superficial tibiotalar fascicle	Tibionavicular ligament (major component)
Superficial posterior tibiotalar ligament	Superficial tibiotalar ligament (additional band)
Tibiocalcaneal ligament	Tibiocalcaneal ligament (additional band)
Deep layer	
Deep posterior tibiotalar ligament	Deep posterior tibiotalar ligament (major component)
Deep anterior tibiotalar ligament	Anterior deep tibiotalar ligament (additional band)

 Table 1.1 Comparison of the nomenclature used for the medial collateral ligament components, as suggested by Sarrafian [36] and Milner and Soames [81]

tibiotalar ligament always is present, while the deep anterior tibiotalar ligament is nonconstant. The deep plane of the MCL can be seen during ankle arthroscopy.

Although the description proposed by Milner and Soames [81] has been accepted, the anatomy of this ligament and its elements is still confusing, partly because differentiation between the components during dissection is difficult and probably artificial since its origins and insertions are complex. Furthermore, the nomenclature used still has not been reviewed and accepted by the Federative Committee on Anatomical Terminology [83]. Moreover, the images, sketches, and diagrams shown in the literature are also imprecise [82].

The MCL is a primary medial stabilizer of the ankle and restrains against valgus tilting and anterior translation of the talus [84]. It is also the most prominent restraint against lateral translation. Functionally, the superficial component helps to maintain the alignment of the talus and medial malleolus, as well as to resist external rotation of the talus to the tibia and valgus stress [85]. The deep component prevents lateral displacement and external rotation of the talus specially in ankle plantarflexion; it is also the primary restraint to medial opening [86, 87].

The injury to the MCL is far more uncommon than LCL injury. The mechanism of injury to this ligamentous complex is controversial. Although some authors report that the most frequent mechanism is inversion [88, 89], the primary mechanism of acute ligament injury is eversion or external rotation of the ankle [90–92]. Such injuries often occur while walking or running on uneven ground or jumping and landing on uneven surfaces [93]. However, a minority of ankle injuries are medial sprains affecting only the MCL, and these are often associated with complex ligamentous injuries [94].

The soft tissue impingement syndrome related to the MCL has also been described. According to the location of the fibrous tissue mass at the level of the medial recess and relative to the medial malleolus, the condition is differentiated into anteromedial soft tissue impingement, as described by Egol and Parisien [95], or posteromedial soft tissue impingement, as described by Liu and Mirzayan [91].

#### 1 Anatomy of the Ankle

#### References

- 1. Castro MD (2002) Ankle biomechanics. Foot Ankle Clin 7(2):679-693
- Testut L, Latarjet A (1985) Tratado de anatomía humana. Salvat Editores, S.A., Barcelona, pp 704–719 [in Spanish]
- Tol JL, van Dijk CN (2004) Etiology of the anterior ankle impingement syndrome: A descriptive anatomical study. Foot Ankle Int 25(6):382–386
- van Dijk CN, Tol JL, Verheyen CC (1997) A prospective study of prognostic factors concerning the outcome of arthroscopic surgery for anterior ankle impingement. Am J Sports Med 25(6):737–745
- 5. Ferkel RD (1996) Soft-tissue lesions of the ankle. In: Whipple TL (ed) Arthroscopic surgery: the foot and ankle. Lippincott-Raven, Philadelphia, pp 121–143
- 6. Garrick JG (1977) The frequency of injury, mechanism of injury, and epidemiology of ankle sprains. Am J Sports Med 5(6):241–242
- 7. Ferran NA, Oliva F, Maffulli N (2009) Ankle instability. Sports Med Arthrosc 17(2): 139–145
- 8. Renström AFH, Lynch SA (1999) Acute injuries of the ankle. Foot Ankle Clin 4:697-711
- 9. Balduini FC, Tetzlaff J (1982) Historical perspectives on injuries of the ligaments of the ankle. Clin Sports Med 1(1):3–12
- van Dijk CN (1994) On diagnostic strategies in patients with severe ankle sprain. Thesis, University of Amsterdam, Amsterdam
- Johnson EE, Markolf KL (1983) The contribution of the anterior talofibular ligament to ankle laxity. J Bone Joint Surg Am 65(1):81–88
- Caputo AM, Lee JY, Spritzer CE et al (2009) In vivo kinematics of the tibiotalar joint after lateral ankle instability. Am J Sports Med 37(11):2241–2248
- Pijnenburg ACM, Bogaard K, Krips R et al (2003) Operative and functional treatment of rupture of the lateral ligament of the ankle: A randomised, prospective trial. J Bone Joint Surg Br 85(4):525–530
- 14. Baumhauer JF, O'Brien T (2002) Surgical considerations in the treatment of ankle instability. J Athl Train 37(4):458–462
- Anderson K, Lecoq JF (1954) Operative treatment of injury to the fibular collateral ligament of the ankle. J Bone Joint Surg Am 36(4):825–832
- Waterman BR, Belmont PJ Jr, Cameron KL et al (2011) Risk factors for syndesmotic and medial ankle sprain: Role of sex, sport, and level of competition. Am J Sports Med 39(5):992–998
- 17. Clanton TO, Paul P (2002) Syndesmosis injuries in athletes. Foot Ankle Clin 7(3):529-549
- Ferran NA, Maffulli N (2006) Epidemiology of sprains of the lateral ankle ligament complex. Foot Ankle Clin 11(3):659–662
- Gerber JP, Williams GN, Scoville CR et al (1998) Persistent disability associated with ankle sprains: a prospective examination of an athletic population. Foot Ankle Int 19(10):653–660
- Brostrom L (1966) Sprained ankles. VI. Surgical treatment of 'chronic' ligament ruptures. Acta Chir Scand 132(5):551–565
- Karlsson J, Bergsten T, Lansinger O et al (1988) Lateral instability of the ankle treated by the Evans procedure: A long-term clinical and radiological follow-up. J Bone Joint Surg Br 70(3):476–480
- Golanó P, Vega J, Pérez–Carro L, Götzens V (2006) Ankle anatomy for the arthroscopist. Part II: Role of the ankle ligaments in soft tissue impingement. Foot Ankle Clin 11(2):275–296
- Golanó P, Vega J, de Leeuw PAJ et al (2010) Anatomy of the ankle ligaments: A pictorial essay. Knee Surg Sports Traumatol Arthrosc 18(5):557–569
- Golanó P, Vega J (2013) Ankle sprain: Diagnosis and therapy starts with knowledge of anatomy. Aspetar Sports Med J 2(2):234–42

- Ferkel RD, Scranton PE (1993) Current concepts review: Arthroscopy of the ankle and foot. J Bone Joint Surg Am 75(8):1233–1245
- 26. Shaffler GJ, Tirman PFJ, Stoller DW et al (2003) Impingement syndrome of the ankle following supination external rotation trauma: MR imaging findings with arthroscopic correlation. Eur Radiol 13(6):1357–1362
- 27. Kapanji IA (1982) Cuadernos de fisiología articular. Cuaderno III. Masson, S.A., Barcelona [in Spanish]
- Kelikian AS, Rinella AS (1999) Ankle fractures. In: Kelikian AS (ed) Operative treatment of the foot and ankle. Appleton & Lange, Stamford, pp 255–283
- Bassett FH III, Gates HS, Billys JB et al (1990) Talar impingement by the anteroinferior tibiofibular ligament: A cause of chronic pain in the ankle after inversion sprain. J Bone Joint Surg Am 72(1):55–59
- 30. Akseki D, Pinar H, Bozkurt M et al (1999) The distal fascicle of the anterior inferior tibiofibular ligament as a cause of anterolateral ankle impingement: Results of arthroscopic resection. Acta Orthop Scand 70(5):478–482
- Nikolopoulos CE (1982) Anterolateral instability of the ankle joint: An anatomical, experimental and clinical study. Thesis, University of Athenes, Athenes
- Nikolopoulos CE, Tsirikos AI, Sourmelis S, Papachristou G (2004) The accessory anteroinferior tibiofibular ligament as a cause of talar impingement: A cadaveric study. Am J Sports Med 32(2):389–395
- Rasmussen O, Tovborg-Jensen I, Boe S (1982) Distal tibiofibular ligaments: Analysis of function. Acta Orthop Scand 53(4):681–686
- 34. Akseki D, Pinar H, Yaldiz K et al (2002) The anterior inferior tibiofibular ligament and talar impingement: A cadaveric study. Knee Surg Sports Traumatol Arthrosc 10(5):321–326
- Kim S-H, Ha K-I (2000) Arthroscopic treatment for impingement of the anterolateral soft tissues of the ankle. J Bone Joint Surg Br 82(7):1019–1021
- Sarrafian SK (1993) Anatomy of the foot and ankle. Descriptive, topographic, functional, 2nd edn. J.B. Lippincott Company, Philadelphia, pp 159–217
- Bartonícek J (2003) Anatomy of the tibiofibular syndesmosis and its clinical relevance. Surg Radiol Anat 25(5–6):379–386
- Golanó P, Mariani PP, Rodríguez-Niedenfuhr M (2002) Arthroscopic anatomy of the posterior ankle ligaments. Arthroscopy 18(4):353–358
- Taylor DC, Englehardt DL, Bassett FH (1992) Syndesmosis sprains of the ankle: The influence of heterotopic ossification. Am J Sport Med 20(2):146–150
- Broström L (1966) Sprained ankles. V. Treatment and prognosis in recent ligament ruptures. Acta Chir Scand 132(5):537–550
- Milner CE, Soames RW (1997) Anatomical variations of the anterior talofibular ligament of the human ankle joint. J Anat 191(3):457–458
- 42. Burks RT, Morgan J (1994) Anatomy of the lateral ankle ligaments. Am J Sport Med 22(1):72–77
- Milner CE, Soames RW (1998) Anatomy of the collateral ligaments of the human ankle joint. Foot Ankle Int 19(11):757–760
- 44. Delfaut EM, Demondion X, Boutry N et al (2003) Multi-fasciculated anterior talo-fibular ligament: Reassessment of normal findings. Eur Radiol 13(8):1836–1842
- 45. Vega J, Golanó P, Pellegrino A et al (2013) All-inside arthroscopic lateral collateral ligament repair for ankle instability with a knotless suture anchor technique. Foot Ankle Int 34(12):1701–1709
- Neuschwander TB, Indresano AA, Hughes TH, Smith BW (2013) Footprint of the lateral ligament complex of the ankle. Foot Ankel Int 34(4):582–586
- 47. Ferkel RD, Fisher SP (1989) Progress in ankle arthroscopy. Clin Orthop Relat Res 240:210–220
- 48. Ferkel RD, Karzel RP, Del Pizzo W et al (1991) Arthroscopic treatment of anterolateral impingement of the ankle. Am J Sports Med 19(5):440–446

- 1 Anatomy of the Ankle
- Wolin I, Glassman F, Siderman S (1950) Internal derangement of the talofibular component of the ankle. Surg Gynecol Obstet 91(2):193–200
- 50. Meislin RJ, Rose DJ, Parisien JS, Springer S (1993) Arthroscopic treatment of synovial impingement of the ankle. Am J Sports Med 21(2):186–189
- Liu SH, Raskin A, Osti L et al (1994) Arthroscopic treatment of anterolateral ankle impingement. Arthroscopy 10(2):215–218
- Rasmussen S, Jensen CH (2002) Arthroscopic treatment of impingement of the ankle reduces pain and enhances function. Scand J Med Sci Sports 12(2):69–72
- Gulish HA, Sullivan RJ, Aronow M (2005) Arthroscopic treatment of soft-tissue impingement lesions of the ankle in adolescents. Foot Ankle Int 26(3):204–207
- Urgüden M, Söyüncü Y, Ozdemir H et al (2005) Arthroscopic treatment of anterolateral soft tissue impingement of the ankle: Evaluation of factors affecting outcome. Arthroscopy 21(3):317–322
- 55. Hassan AH (2007) Treatment of anterolateral impingements of the ankle joint by arthroscopy. Knee Surg Sports Traumatol Arthrosc 15(9):1150–1154
- Villarreal JM, Cerecedo RB, Cal y Mayor FF et al (2008) Arthroscopic treatment for anterolateral ankle impingement of athletes. Acta Ortop Mex 22(2):103–106
- 57. Franco R, Vega J, Pérez M et al (2010) Resultados del tratamiento artroscópico del pinzamiento blando de tobillo. Rev Pie Tobillo 24(2):24–29
- Moustafa El–Sayed AM (2010) Arthroscopic treatment of anterolateral impingement of the ankle. J Foot Ankle Surg 49(3):219–223
- Ruth CJ (1961) The surgical treatment of injuries of the fibular collateral ligaments of the ankle. J Bone Joint Surg Am 43(2):229–239
- Rouvière H, Delmas A (1999) Anatomía Humana descriptiva, topográfica y funcional. Tomo III, 10ath edn. Masson, S.A., Barcelona, pp 362–367 [in Spanish]
- 61. Paturet G (1951) Traité d'anatomie humaine. Masson, París, pp 704–711 [in French]
- 62. Chen Y (1985) Arthroscopy of the ankle joint. In: Watanabe M (ed) Arthroscopy of small joints. Igaku–Shoin, New York, pp 104–127
- Ikeuchi H (1988) Personal communication. Cited by Guhl JF. Soft tissue (synovial) pathology. In: Guhl JF (ed) Ankle arthroscopy. Slack, Thorofare, pp 79–94
- 64. Hamilton WC (1988) Foot and ankle injuries in dancers. Clin Sports Med 7(1):143-173
- 65. Hamilton WG, Gepper MJ, Thompson FM (1996) Pain in the posterior aspect of the ankle in dancers: Differential diagnosis and operative treatment. J Bone Joint Surg Am 78(10):1491–1500
- 66. Rosenberg ZS, Cheung YY, Beltran J et al (1995) Posterior intermalleolar ligament of the ankle: normal anatomy and MR imaging features. AJR Am J Roentgenol 165(2):387–390
- 67. Peace KAL, Hillier JC, Hulme A et al (2004) MRI features of posterior ankle impingement syndrome in ballet dances: a review of 25 cases. Clin Radiol 59(11):1025–1033
- Oh CS, Won HS, Chung IH et al (2006) Anatomic variations and MRI of intermalleolar ligament. AJR Am J Roentgenol 186(4):943–947
- Loren GJ, Ferkel RD (1999) Arthroscopic strategies in fracture management of the ankle. In: Chow JCY (ed) Advanced arthroscopy. Springer, New York, pp 635–653
- van Dijk CN, Scholten PE, Krips R (2000) A 2-portal endoscopic approach for diagnosis and treatment of posterior ankle pathology: Technical note. Arthroscopy 16(8):871–876
- Lohrer H, Arentz S (2004) Posterior approach for arthroscopic treatment of posterolateral impingement syndrome of the ankle in a top-level field hokey player. Arthroscopy 20(4):e15–e21
- 72. Tey M, Monllau JC, Centenera JM et al (2007) Benefits of arthroscopic tuberculoplasty in posterior ankle impingement syndrome. Knee Surg Sports Traumatol Arthrosc 15(10):1235–1239
- Smith WB, Berlet GC (2009) Posterior ankle impingement: The role of posterior ankle arthroscopy. Tech Foot Ankle Surg 8(2):94–98
- Van Dijk CN, de Leeuw PA, Scholten PE (2009) Hindfoot endoscopy for posterior ankle impingement: Surgical technique. J Bone Joint Surg Am 91(Suppl 2):287–298

- 75. Calder JD, Sexton SA, Pearce CJ (2010) Return to training and playing after posterior ankle arthroscopy for posterior impingement in elite professional soccer. Am J Sports Med 38(1):120–124
- Guo QW, Hu YL, Jiao C (2010) Open versus endoscopic excision of a symptomatic os trigonum: a comparative study of 41 cases. Arthroscopy 26(3):384–390
- Noguchi H, Ishii Y, Takeda M (2010) Arthroscopic excision of posterior ankle bony impingement for early return to the field: Short-term results. Foot Ankle Int 31(5):398–403
- Galla M, Lobenhoffer P (2011) Technique and results of arthroscopic treatment of posterior ankle impingement. Foot Ankle Surg 17(2):79–84
- 79. Ogut T, Ayhan E (2011) Hindfoot endoscopy for accessory flexor digitorum longus and flexor hallucis longus tenosynovitis. Foot Ankle Surg 17(1):e7–e9
- Pankovich AM, Shivaram MS (1979) Anatomical basis of variability in injuries of the medial malleolus and the deltoid ligament. I: Anatomical studies. Acta Orthop Scand 50(2):217–223
- Milner CE, Soames RW (1998) The medial collateral ligaments of the human ankle joint: Anatomical variations. Foot Ankle Int 19(5):289–292
- Boss AP, Hintermann B (2002) Anatomical study of the medial ankle ligament complex. Foot Ankle Int 23(6):547–553
- Federative Committee on Anatomical Terminology (1998) Anatomical terminology. Thieme, Stuttgart
- Michelson JD, Hamel AJ, Buczek FL, Sharkey NA (2004) The effect of ankle injury on subtalar motion. Foot Ankle Int 25(9):639–646
- Beals TC, Crim J, Nickisch F (2010) Deltoid ligament injuries in athletes: Techniques of repair and reconstruction. Oper Tech Sports Med 18(1):11–17
- Michelson JD, Waldman B (1996) An axially loaded model of the ankle after pronation external rotation injury. Clin Orthop Relat Res 328:285–293
- Jelinek JA, Porter DA (2009) Management of unstable ankle fractures and syndesmosis injuries in athletes. Foot Ankle Clin 14(2):277–298
- Mosier–LaClair S, Monroe MT, Manoli A (2000) Medial impingement syndrome of the anterior tibiotalar fascicle of deltoid ligament of the talus. Foot Ankle Int 21(5):385–391
- Paterson RS, Brown JN, Roberts SNJ (2001) The posteromedial impingement lesion of the ankle: A series of six cases. Am J Sports Med 29(5):550–557
- 90. Harper MC (1987) Deltoid ligament: An anatomical evaluation of function. Foot Ankle Int 8(1):19–22
- 91. Liu SH, Mirzayan R (1993) Posteromedial ankle impingement: Case report. Arthroscopy 9(6):709–711
- 92. O'Loughlin P, Murawski CD, Egan C et al (2009) Ankle instability in sports. Phys Sportsmed 37(2):93–103
- 93. Hintermann B (2003) Medial ankle instability. Foot Ankle Clin 8(4):723-738
- 94. Savage–Elliott I, Murawski CD, Smyth NA et al (2013) The deltoid ligament: An in–depth review of anatomy, function, and treatment strategies. Knee Surg Sports Traumatol Arthrosc 21(6):1316–1327
- Egol KA, Parisien JS (1997) Impingement syndrome of the ankle caused by a medial meniscoid lesion. Arthroscopy 13(4):522–525

# Chapter 2 Interview – Raul

Pieter P.R.N. d'Hooghe and Gino M.M.J. Kerkhoffs

**Abstract** At the age of 17, Raul debuted in the first team of Real Madrid. With 'Los Merengues', he won 6 Spanish Liga titles, 3 Champions League titles, 1 UEFA Supercup, 4 Spanish Supercopa's and 2 World Cups for teams. He is the ultimate European top scorer of all times with 77 goals, vaporized the 307 Real Madrid goals record of legendary Alfredo Di Stéfano and was captain of 'Los Galacticos' for many years. In the autumn of his professional career, he also won the German Supercup and DFB-Pokal with Schalke 04 in Germany and is now currently playing and winning league titles with Al Sadd Football Club in Qatar.

P.P.R.N. d'Hooghe, MD (🖂)

G.M.M.J. Kerkhoffs, MD, PhD Department of Orthopedic Surgery, Academic Medical Centre Amsterdam, University of Amsterdam, Meibergdreef 9, Amsterdam 1105 AZ, The Netherlands e-mail: g.m.kerkhoffs@amc.uva.nl

P.P.R.N. d'Hooghe, G.M.M.J. Kerkhoffs (eds.), *The Ankle in Football*, Sports and Traumatology, DOI 10.1007/978-2-8178-0523-8\_2, © Springer-Verlag France 2014

Department of Orthopaedic Surgery, Aspetar Hospital, Aspire Zone, Doha, Qatar e-mail: pieter.orthopedie@gmail.com



• What is the most important for an ankle-injured player to experience from his staff to be able to cope with his rehabilitation? What advice can you give to injured players?

That really depends on the injury itself and how long the expected rehab will take. The most important is that the player needs to know all the information about the ankle injury and the expected time frame to recover. Coping with an ankle injury is like with all other types of injury: very individual and depending on the coping mechanisms of the player. Sometimes it's better for a player to give him/her some space the first 3–4 days after a sustained injury if no quick surgery is needed. The family is often of upmost important as a heaven for the player in order to start his/her coping process after an injury.

With the ankle injury that I personally suffered from, I had huge confidence in the physio and doctor. Checking my ankle daily and touching it, see the evolution and work along a clear and well-defined program gave me the confidence to believe in a good outcome. Confidence is very important and is what a doctor should offer his players. I cannot stress more the importance of the doctor and physio also in this regard since they will see you for hours and hours, day in, day out.

What's your philosophy on ankle injury prevention?

As an athlete, you know the signals of your body very well. When you have a good relation with your doctor and physio, you can determine easier the protocol when medical attention injuries should occur. Sometimes there's no magic to be expected, but with mutual respect and close communication, the player can feel very supported by the preventative strategies from its medical staff.

When I suffered from a severe contact lesion – after long meetings with the medical staff – we decided as a team to go for conservative treatment, and it took us 7 months to fully recover from the pain. Since then, I perform extra exercises in my everyday workout to prevent it from coming back. That's hard work. It's a big part of the duty of the medical staff – especially towards young players – to stress the importance of injury prevention.

• Are the new generation of artificial grass, newer football shoe designs and new training strategies beneficial to avoid ankle injuries in your opinion? *Artificial grass is in my opinion very bad for your body and especially for your* 

ankles. I played from my 17 years old in the first team of Real Madrid, and I was lucky only to have two injuries in my whole career until now, but every time I played or trained on artificial grass, my body suffered a lot.

I realize that many efforts are taken with the new generations of artificial grass to improve this, and in many parts of the world, it's the only solution to play the game anyway. Still ... as a football player and along with the opinion of many of my team players, nothing beats playing on natural grass.

The new-generation football shoes have evolved in the most fantastic and comfortable way, and this is a great example of how the industry helped in the development of the game. When comparing shoes from 15 years ago and now, a huge improvement has been established. The new-generation shoes offer the player a nice natural skin feeling, and along with that improved comfort goes the playing performance.

In the beginning of my professional career, some players even didn't visit the gym and just did some running for training and prior to the game. The top coaches that I had the privilege to work with, captured very much the new medical input on injury prevention and implemented them fully into their training sessions. The physical exercise nowadays is individually tailored, very specific and not anymore focussed on endurance only. In the morning – when arriving at the club – we are now offered a program that takes into account our nutrition and hydration strategies, along with extra exercises on injury prevention, prior to the general training sessions.

- What is a good team doctor/medical staff for you and what do you expect from them?
  - I believe for a team it is really crucial to have a good team doctor and staff.

For me there are two important people that need to take care of the daily checkup: the physiotherapist and the doctor. It's your first contact before every training. It's also important to have this on a daily basis so that you can establish a professional friendship and that they know how your body responds to training, games, physiological tests and injuries. If 1 day a decision has to be made on playing or not, this close interaction helps to do the right thing. The physio is somebody that you see at least 4 h per day in the club and is your first line of contact. Nevertheless, for me the doctor has the last decision, and to be able to do that well, he/she needs to first know the injury and second know the player on a very individual basis.

- How did you experience your rehab after ankle injury, and what were the positive and negative moments that you distil out of it retrospectively? *Much of this depends on the mentality of the player. If the player's mentality is professional and compliance is not an issue, fast recovery can be expected more easily. Sometimes sport-psychological support can be very helpful, but communication, motivation and courage are the three pillars that one needs in that situation. Younger players can fear the end of their career in case of an ankle injury, and the medical staff needs to make sure that the player's entourage gives him/ her what he/she needs.*
- Did the medical setup in your club changed the last 10 years, and what do you think about the evolution seen?

There has been a mind-boggling evolution in the medical staff of clubs since the last 15 years. In my early professional years, we had one club physio and three masseurs. Nowadays, next to the physiotherapists, we have recovery trainers, physiologists, osteopaths, nutritionists, podologists, chiropractors, acupuncturists, yoga and mindfulness experts.

Also the evolution of clothes, socks and test technologies are of great added value in the team's performance. The greatest evolution of all for me is the individual approach that the players now can have. Every player now has one daily program, completely tailored to his/her needs. The era where in a club you had one coach, one doctor and one physio lies miles behind us now.

• Do you think you are performing enough injury prevention strategies for your ankle in your daily workout?

I always consider three parts of training: before, during and after.

'Before' concerns the prevention protocol based upon your testing results. 'During' is about the general football training sessions with the team.

'After' is the individual extra that your body needs and is aimed to improve in your individual field and to avoid recurrence of previous problems.

- 2 Interview Raul
- Do you believe that your sport will cause early joint problems (in your ankle) after your career?

That's a difficult one. I agree that the overload in daily training sessions and games also overloads the (ankle) joints. On the other hand, you have to feel fit 100 % in order to perform at a certain level, and that requires a player to push his/her body to the limit and sometimes over it. But it's the whole staff that has to give 100 %.

In the long term, it's better to base your protocols upon cure and care for the player.

Nevertheless, sometimes you have to take a risk that is not always well defined. It's the staff's responsibility to communicate well with the player. The most important thing is the honesty of the doctor in his/her relation with the player. The emotional intensity and pressure can be very high in certain circumstances. Having the physio and the doctor as trustworthy companions is a great feeling to encounter as player.

• Who will win WC 2014 in Brazil? *Spain* 

# Chapter 3 Epidemiology and Mechanisms of Ankle Pathology in Football

Ryan L. Anderson, Lars Engebretsen, Nicholas Kennedy, Robert LaPrade, Adam M. Wegner, and Eric Giza

> A defender should always focus on the opponent's lower legs. I've never seen a ball score on its own...

> > Co Adriaanse Dutch coach

**Abstract** The ankle is one of the most commonly injured joints in football and represents a significant cost to the healthcare system due to time lost from play. The ligaments that stabilize the ankle joint determine its biomechanics, and alterations of those normal biomechanics result from various football-related injuries. Acute sprains are generally treated conservatively, with emphasis placed on secondary prevention to reduce the risk of future sprains and progression to chronic ankle stability. Repetitive ankle injuries in footballers may cause chronic ankle instability, which include both mechanical ligamentous laxity and functional changes. Chronic ankle pathology often requires surgery to repair ligamentous damage and remove

R.L. Anderson, MS

L. Engebretsen Oslo Sports Trauma Research Center, Oslo, Norway

N. Kennedy (🖂) • R. LaPrade Department of Orthopaedic Surgery, University of California at Davis Sacramento, CA 95817, USA

E. Giza, MD (⊠) Department of Orthopaedics Surgery, University of California at Davis, Sacramento, CA 95817, USA

Foot and Ankle Surgery, University of California at Davis, 4860 Y Street, Suite 3800, Sacramento, CA 95817, USA e-mail: eric.giza@ucdmc.ucdavis.edu

A.M. Wegner, MD, PhD Department of Orthopaedics Surgery, University of California at Davis, Sacramento, CA 95817, USA

P.P.R.N. d'Hooghe, G.M.M.J. Kerkhoffs (eds.), *The Ankle in Football*, Sports and Traumatology, DOI 10.1007/978-2-8178-0523-8\_3, © Springer-Verlag France 2014

School of Medicine, University of California at Davis, Sacramento, CA 95817, USA

soft tissue or osseous impingement. Proper initial treatment, rehabilitation, and secondary prevention of ankle injuries can limit the amount of time lost from play and avoid negative long-term sequelae.

Keywords Sport • Football • Injuries • Ankle • Traumatology • Epidemiology

# 3.1 Epidemiology of Football

Nicholas Kennedy, Robert LaPrade, and Lars Engebretsen

Including players and refereeing officials, there are over 270 million participants in football, according to FIFA's 2006 Big Count. Football is widely growing in popularity with high school participation in the United States increasing fivefold in the last 30 years [25]. In a sport where games are 90+ minutes long, with only one halftime and no time-outs to stop the clock and catch your breath, one can expect to find a heavy occurrence of injuries. Due to the repetitive kicking motion, constant changing of direction, and frequent acceleration and deceleration, one of the joints that is particularly susceptible to fatigue and injury is the ankle joint.

Ankle sprains specifically are quite a common occurrence in football [3, 4, 6, 7, 9, 11, 13, 18]. As with most sports, due to common ankle anatomy, the lateral aspect of the ankle is most susceptible and experiences the most sprains [1, 9, 18]. However, Morgan et al. note that football players are far more susceptible to medial ankle injuries than other sports due to the use of the medial aspect of the foot and ankle to kick, and pass the ball, which puts the ankle in an abducted and externally rotated position [18]. Because of the sport's overall worldwide popularity, and vast participation, it becomes important to evaluate the overall occurrence and mechanisms of ankle injuries in soccer and also the treatment options and patient outcomes.

#### 3.2 Prevalence

Ankle injuries are extremely common in football and usually are the first or second most prevalent injury reported overall. They have been reported to account for between 13 and 36 % of all football-related injuries [2–4, 6, 7, 9, 11, 13, 18, 21]. The dominant leg has been reported to be the more common site of injury [20]. Ankle injuries occurrence per 1,000/h of activity has been reported between 342 and 9 [3, 8, 10]. This occurrence has great variation and has been found to be much greater for match time, or games, when compared to practice time. Ankle injuries are oftentimes up to a factor of ten more prevalent in games compared to practices [1, 6, 11, 19, 25]. Also, there has been a correlation reported between the occurrence of ankle injuries and the age of participants, with older players experiencing a higher occurrence. This trend was especially evident in the adolescent age range [4]. Of the injuries that are associated with the ankle, ankle sprains have been found to be the far and away most common injury of all soccer-related injuries [2, 4, 11]. Ankle sprains have been found to account for between 42 and 98 % of all soccer-related ankle injuries [2, 7]. Ekstrand reported a higher incidence of ankle sprains on the dominant leg and hypothesized it was caused by increased inversion strain due to repetitive jumping and kicking actions [7]. Other common injuries associated with football injuries are dislocations and fractures, especially of the lateral malleolus, although these injuries are much less frequent in occurrence [2, 7]. Also, there are injuries found to be more specific and relatively unique to football, such as anterior ankle impingement. These and other injuries have specific injury mechanisms, which will be explained.

#### 3.3 Mechanism of Injury

As has previously been mentioned, there is a great diversity of football-related ankle injuries: fractures, ligament sprains/ruptures, anterior ankle impingement syndrome, tendonitis, bone spurs, dislocations, muscle ruptures, and other pathology. We have chosen to explain the mechanism of two of the most prevalent injuries in football: anterior ankle impingement syndrome and ligament sprains/ruptures. The reasoning behind choosing these two injuries are mechanisms that have been found to be specific to football activities and movements. Other common injuries such as contusions and fractures have generic mechanisms similar to other sports, such as blunt force trauma for contusions.

Anterior ankle impingement syndrome, previously known as footballer's ankle, is when talotibial osteophytes form at the anterior part of the ankle and cause chronic anterior ankle pain [15, 24]. Massada reported that osteophytes of this nature can be found in up to 60 % of football players [16]. The mechanism of this injury is somewhat controversial. To date, there are three main accepted mechanisms. Massada, Andersen, and others have reported that the osteophytes are traction spurs, caused by traction due to the repeated hyperplantar flexion of the foot in the kicking action [1, 16]. Petersen et al. reported an alternative theory, which stated that it was repeated dorsiflexion, as opposed to plantar flexion, which caused anterior ankle impingement. The repeated dorsiflexion caused impact between the talus and tibia, which leads to minor fractures that then caused the formation of the osteophytes on the anterior edge of tibia and talus [21]. Tol et al. performed a study which analyzed the kicking action of elite soccer players. In this study, they measured the angle created during kicking and the contact force between the foot and the ball during kicking. They reported that maximum plantar flexion was met in only a minority of kicking actions, meaning it could only cause the osteophytes in a minority of cases. On the other hand, they found ample evidence to support the third prevailing hypothesis that the osteophytes are caused by the repetitive microtrauma to the anteromedial aspect of the foot when you kick the ball [24]. Their data on impact location and force of kicking the soccer ball demonstrated that direct damage of the articular cartilage rim could lead to osteophyte formation and anterior ankle impingement syndrome [24].

In regard to sprains, there are numerous reported mechanisms of injury. Ankle sprain mechanisms include the most prevalent actions involved in football, including cutting, kicking, running, tackling, being tackled, and landing [1, 7].

Ankle sprains can preliminarily be broken into two categories: lateral ankle sprains and medial ankle sprains. As with most sports, the lateral aspect of the ankle is far more susceptible to injury [1, 2, 7, 18]. However as previously mentioned, Morgan and others have found that the movements involved with football put the medial ankle at higher risk than other popular sports, with the occurrence rate being closer to 3 lateral to 1 medial sprain [1, 7, 18].

Lateral and medial ankle sprains can further be divided into contact and noncontact ankle sprains. Contact ankle injuries are quite common and have been reported to comprise over 70 % of ankle sprains [10, 21]. Andersen found that of 23 ankle sprains recorded on video, 21 occurred during some form of contact. Regarding contact injuries, Anderson reported two common mechanisms specific to football actions that lead to ankle sprains: contact between two players where a collision on the medial side of one of the player's legs before or at foot strike leads to a laterally directed force which causes the player to land in an inverted position and contact of an injured player and an opponent when the injured player is trying to kick the ball and this leads to forced plantar flexion [1].

The first common mechanism of contact injuries was found in 13 out of 14 ankle injuries involving one player tackling or trying to dispossess the opponent of the ball. Nine of the 13 tackling injuries caused by the first common mechanism lead to injury of the player being tackled, eight of those being ankle sprains, while four lead to injury by the tackler, and all four being ankle sprains. There was also one eversion sprain caused by a similar mechanism but a blow from the lateral side causing a medially directed force. The second common mechanism was found in three out of four injuries that occurred while one player made contact with another attempting to kick the ball. All three lead to ankle sprains for the player kicking the ball [1].

Noncontact ankle sprains have a much wider range of reported injury mechanisms. The mechanisms were not noticeably different or unique for football when compared to other athletic activities. All four ankle injuries that occurred while running were inversion injuries, with three resulting in ankle sprains. One of the two injuries that resulted from a player landing after going up for a header was the result of inversion mechanism [1].

#### 3.4 Acute Injury Treatment

The most common acute injuries in football are ankle sprains and contusions. While fractures do occur occasionally, they are far less prevalent.

The treatment of contusions is fairly simple in most cases. The most common treatment would be the RICE principle: rest, ice, compression, and elevation. This treatment is utilized to reduce swelling and inflammation immediately post-injury. After using RICE principle, the athlete can attempt to return to competition, making

sure to continue to monitor pain and swelling. Sometimes, that return can be as soon as immediately to 24-48 h after the injury; other times, the return can take longer in the 7–14 days range.

Ankle sprains, on the other hand, have a very wide range of treatment options for acute injuries. Treatment of acute ankle sprains, for example, differs depending on whether the injury is to the medial or lateral ligaments. For lateral ankle sprains, there are three grades: grade I, grade II, and grade III. A grade I injury is a stretching of the lateral ligaments, with little swelling, little to no functional loss, no mechanical instability, and no macroscopic tearing [22]. Grade II sprains do have slight macroscopic tearing, with moderate symptoms such as pain, stiffness, and swelling. A grade III sprain is a complete tear or rupture of the lateral ligaments and has more severe symptoms that accompany the tear. Grades I and II lateral acute ankle sprains are the most common and have the best prognosis. Usually, grades I and II lateral sprains are first treated with the RICE principle for 48 h following the injury. Then, they are usually treated functionally with the use of a brace, which allows early motion and weight bearing. Taping and strapping are not widely utilized in the longterm treatment of ankle sprains in sports [15]. The reason for this is because Shapiro and others have reported that the mechanical effect of tape wears off with activity, whereas a brace retains its effect throughout activity [23].

Grade III lateral acute ankle sprain treatment is far more controversial than grades I and II. Immediately after the injury, the RICE principle is again employed; however, the next step in treatment is highly debated. The three most common treatment options are surgical repair, cast immobilization, and early controlled mobilization. All three have been shown to restore mechanical stability and produce satisfactory subjective results [22]. However, Kannus and Renstrom demonstrated in their review of numerous studies, regarding the treatment of grade III lateral ankle sprains, that early controlled mobilization is superior [14, 22]. The studies looked at numerous subjective outcomes such as functional instability, pain, swelling, stiffness, mobility, and return to work. While all three treatments were shown to prevent late residual problems, like functional instability, early mobilization allowed for the quickest return to work, which is especially important for a professional athlete such as a football player [14, 22].

It is the opinion of those writing this chapter that this is an area of study which should be further pursued. Lateral ankle sprains, being one of, if not the most common football injury, should have a set acute injury treatment protocol. While a quicker return to gameplay should be a target in treatment, it must also be taken into account that residual disability of ankle sprains is often caused by inadequate rehabilitation and a potentially overly hurried return to play [22]. Therefore, studies which look at long-term subjective and objective outcomes of football players returning to play after specific treatment protocols for acute ankle injuries would be very helpful for future treatment and hopefully decrease the recurrent nature of this injury.

Medial ankle sprains are less frequent and generally are more severe in nature than lateral ankle sprains [5, 18]. A tear of the medial deltoid ligament caused by an eversion mechanism sprain is quite serious because oftentimes the interosseous

membrane is torn and there is a potential for diastasis of the tibia and fibula as well. Because this injury is much more complex than the more common lateral ankle sprain, the recovery time and healing process are much longer [5]. Similar to lateral ankle sprains, the first step of treatment is again the RICE principle for 48 h. Some less severe injuries can be casted for 6 weeks following the RICE principle. However, if the diastasis is severe, the ankle needs to be treated surgically to reduce the diastasis and also to repair the medial deltoid ligament.

#### **3.5** Chronic Injury Treatment

Many acute injuries, such as ankle sprains or contusions, can lead to residual ankle weakness and susceptibility to future reinjury. Therefore, many acute injuries can turn into chronic injuries. In the case of ankle sprains and contusions, the treatment options for a chronic injury are usually the more aggressive option for treating the acute version of the injury. For example, chronic ankle instability and continued ankle sprains will more likely have to be treated surgically or casted as opposed to early controlled mobilization.

In regard to chronic injuries specific to football, one that has been discussed frequently is anterior ankle impingement syndrome. What can sometimes happen with lateral ankle sprains is that over time a mass of hyalinized tissue will form over the anterior aspect of the ankle, which causes both pain and swelling. Usually, a more conservative treatment is first attempted, with up to 6 months of RICE. However, the patients in McCarroll et al.'s study did not experience any reduction in symptoms with the conservative treatment. The next treatment option is arthroscopic surgery, in which the fibrous meniscus-like tissue found between the talus and the fibula is surgically removed. This treatment has been reported to successfully reduce and eliminate the symptoms and allow players to return to play [17].

#### 3.6 Rehabilitation

As it was mentioned earlier, the primary cause of chronic ankle injuries and continuing ankle pain and disability is insufficient rehabilitation [22]. This means that sufficient rehabilitation is extremely important not only to short-term outcomes but also to long-term outcomes and overall career and physical success of football players. Each injury again has a specific rehabilitation program. We have chosen to again describe the two of the most common and football-specific injuries: anterior ankle impingement syndrome and ankle sprains.

McCarroll et al. reported on a postoperative rehabilitation program for the removal of meniscoid lesions that form in anterior ankle impingement syndrome. The patients followed a protocol which included cryotherapy, range of motion, and strengthening exercises for a minimum of 3 weeks. At that time, McCarroll et al. reported that patients had a 90 % return of strength on Cybex testing and passed functional testing [17].

Renstrom et al. summarized an ideal rehabilitation schedule for acute severe ankle ligament injuries complied from numerous different studies [22]. The first part of the rehabilitation program was the RICE principle. This part of the rehabilitation stressed prevention of swelling, inflammation, and other setbacks that could prolong the healing process. The next phase was proliferation phase where the injured ligament was protected by some sort of bracing or support. This protection allows for fibroblasts to migrate into the injured area and stimulate the production of collagen fibers. Also, this phase helped prevent injury to secondary structures, which were more susceptible to further injury at this time. At about 3 weeks, collagen fiber formation was in the maturation phase when final scar tissue was forming. At this point, mobilization was initiated with repeated exercises such as stretching, and controlled movements, because prolonged immobilization at this phase could lead to atrophy. During the final phase, the athlete was 4–8 weeks postinjury and the new collagen fibers were healing to the point that they could withstand almost normal stresses. The next goal was to ease the athlete back into competition, while at the same time ensuring not to rush the process which could lead to long-term recurring disability [22].

#### 3.7 Conclusion

What has been stressed throughout this chapter is that there is a very high incidence rate of ankle injuries in football, especially ankle sprains. These injuries have mechanisms that are very specific to football-specific movements and activities. However, for as prevalent and specified as these ankle injuries are, there is a lack of research and studies done to design treatment and rehabilitation protocols specific to returning athletes to football competition. With the popularity of the sport, and the widespread participation, it is now important to pursue more sport-specific treatment and rehabilitation protocols for ankle injuries in football.

#### 3.8 Ankle Pathology in Football

Ryan L. Anderson, Adam M. Wegner, and Eric Giza

The most popular team sport worldwide is football [26]. During the 2010 FIFA World Cup, nearly half of the world's population watched the tournament on television [27]. It is estimated that 265 million men, women, and children actively participate in the game, with continued growth in popularity expected, particularly among women and at the youth level [28].

The risk of injury to the professional footballer is considerable and well documented in the literature [29–32]. The lower extremity is most commonly affected [12, 18, 19, 29, 33–38], with ankle sprains comprising approximately one-quarter of all football injuries [12, 29, 35, 39-42]. Recent injury data collected during FIFA tournaments and Olympic Games from 1998 to 2012 (totaling 1,681 matches, equivalent to 55,473 player-hours) found the incidence of injury to be 77.3 injuries per 1,000 player-hours or 2.6 injuries per match [43]. Seventy percent of the reported injuries were of the lower extremities, with contusions (55 %), sprains (15 %), and strains (10 %) most commonly diagnosed. Eighty percent of injuries occurred during player-player contact, with nearly half caused by a foul based on the judgment of the team physician. The average incidence of time-loss injuries was 32.8 per 1,000 player-hours or 47 % of all injuries. Other studies have found time-loss injuries to represent 40–84 % of total match injuries [6, 12, 44], with variability most likely due to different definitions of injury and populations used in epidemiologic studies [12, 32]. These findings on injury incidence differ from the 12 to 35 injuries per 1,000 player-hours reported in a meta-analysis of 20 epidemiologic studies [45] and a recent prospective study covering 15 consecutive seasons (1993-2007) of Japanese professional football [37]. From a socioeconomic standpoint, treatment of football injuries is substantial, as the primary medical costs associated with one football injury are estimated to be \$150 US dollars [45]. With 265 million participants, this equates to an annual cost of nearly \$40 billion US dollars.

#### 3.9 Anatomy

## 3.9.1 Joints

Knowledge of the biomechanics of the foot and ankle joints is essential in understanding football injuries. The ankle joint (talocrural articulation) is comprised of the distal ends of the tibia and fibula, which form the mortise, and the superior aspect of the talar dome [46]. As a hinge joint, the ankle provides 20° of dorsiflexion (DF) and 50° of plantar flexion (PF) [47], with stabilization mediated by lateral, medial, and superior ligament complexes. The superior articular surface of the talus (trochlea) is narrower posteriorly, which creates a looser fit within the mortise during plantar flexion [46]. This decreased stability could help explain why the most common injury in football involves a plantar flexion mechanism [48, 49]. Inferiorly, the talus articulates with the calcaneus to form the subtalar joint. It is at this site that the majority of foot inversion and eversion occur. The transverse tarsal joint separates the hindfoot from the midfoot. Movement of this joint depends on the relative alignment of its two articulations: the talonavicular and calcaneocuboid. During foot eversion, these two joints are aligned in parallel allowing supple motion and aiding in shock absorption during the heel strike phase of the gait cycle. With foot inversion, the joints are nonparallel creating a stiff transverse tarsal joint necessary for push-off [46, 47].



#### 3.9.2 Lateral Ligaments

The ankle joint is stabilized laterally by a ligament complex consisting of three individual ligaments, all originating at the lateral malleolus: (1) the anterior talofibular ligament (ATFL), (2) the posterior talofibular ligament (PTFL), and (3) the calcaneofibular ligament (CFL) [46, 47, 50]. The ATFL is a primary resistor of plantar flexion and internal rotation of the talus within the mortise. It works in synergy with the CFL, a resistor to hindfoot adduction and guide for subtalar axis motion. The PTFL restricts external rotation when the ankle is in dorsiflexion [47, 48, 51]. In addition, the Y-shaped inferior extensor reticulum (IER), which runs from the lateral anterosuperior surface of the calcaneus to the medial malleolus and navicular, covers the tendons of the anterior compartment muscles and plays an important role in stability by linking the lateral ligament complex with the subtalar joint [46–48] (Fig. 3.1).

#### 3.9.3 Deltoid Ligament

The deltoid ligament complex consists of six continuous adjacent superficial and deep ligaments that function synergistically to resist valgus and pronation forces, as well as external rotation of the talus in the mortise [46–48, 52]. The superficial deltoid is periarticular. It originates at the anterior colliculus of the medial malleolus (MM), crossing both the ankle and subtalar joints. The deep layer of the deltoid is intra-articular and covered by synovium. It originates at the posterior



**Fig. 3.2** Medical sketch of the superficial and deep layers of the deltoid ligament (Reprinted from Penner [144]: Fig. 48.2; with permission)

colliculus of the MM and intercollicular groove, crossing only the ankle joint [47, 52, 53] (Fig. 3.2).

#### 3.9.4 Syndesmosis

The ankle syndesmosis, or inferior tibiofibular joint, is the distal articulation between the tibia and fibula. The syndesmosis contributes to ankle mortise integrity through its firm fixation of the lateral malleolus against the lateral surface of the talus [46]. Ligaments comprising the ankle syndesmosis include (1) the anterior inferior tibiofibular ligament (AITFL), (2) the posterior inferior tibiofibular ligament (PITFL), (3) the inferior transverse ligament (ITL), and (4) the interosseous ligament (IOL) [47].

#### 3.10 Acute Sprains

Injury to the lateral ligaments of the ankle represents 77-91 % of all ankle sprains in football [7, 40]. Less commonly affected are the medial and syndesmotic ligamentous complexes, with reports of injury in footballers of 14 % [40] and 3–4 % [40, 51], respectively. This trend extends to the general athlete population, where the incidence of isolated syndesmosis sprains is low, responsible for only 10–20 % of all ankle sprains [54–56]. The greatest risk factor for ankle sprain in the footballer is a history of prior sprain [57]. Also implicated are increasing age, player-toplayer contact, condition of the pitch, weight-bearing status of the injured limb at the time of injury, and joint instability or laxity [10, 58].

Evaluation of an ankle sprain is focused on eliciting severity of the injury, and a proper history and physical exam can also identify involved structures. The anterior drawer and talar tilt tests evaluate ankle instability [42, 59]. They are most reliable 4–7 days after initial injury when pain and swelling have diminished [60]. The talar tilt test aids in identifying CFL instability, while the anterior drawer test detects excessive anterior displacement of the talus relative to the tibia, indicative of ATFL laxity [42].

Diagnostic studies may also be required to rule out fracture in ankle injuries. The Ottawa Ankle Rules were developed in an effort to reduce medical costs associated with unnecessary imaging [61], and they have since been verified as highly predictive of those patients with ankle injury who likely need and should receive an x-ray [62]. Accordingly, ankle radiographs should be obtained if ankle pain is reported near the malleoli and one or more of the following: (1) inability to bear weight immediately after the injury and for four steps in the emergency department and (2) bony tenderness at the tip or posterior edge of the malleolus. A radiographic series of the foot is also necessary if the patient has hindfoot pain or bony tenderness at the navicular, cuboid, or base of the 5th metatarsal [63]. Stress radiographs are generally not indicated in acute ankle sprains because the results will not change the treatment protocol; however, they can be useful in differentiating between mechanical and functional instability in chronic ankle instability [60].

A grading system has been devised to aid in guiding the treatment of ankle injuries [42, 59, 60]. Grade I (mild) sprains involve minimal swelling or tenderness with ligaments remaining intact and no functional loss or mechanical joint instability; grade II (moderate) sprains are defined as partial ligament tears associated with moderate pain, swelling, and tenderness; grade III (severe) sprains include complete ligament tears with marked swelling, hemorrhage, tenderness, loss of function, and abnormal joint motion and instability [60].

Initial treatment for all ankle sprains is nonoperative and should follow a conservative functional treatment program [64], as a previous systematic literature review has shown this to be superior to immobilization alone [65]. Other studies have confirmed the superiority of early mobilization as compared to cast immobilization [66–68] or surgery [69]. The functional treatment program consists of three phases that correspond to the biological healing process. The RICE (rest, ice, compression, elevation) method is applied for the first 4–5 days to avoid excess tissue swelling and injury occurring during inflammation. This is followed by a short period of 5–7 days (maximum 10 days) of immobilization with supportive taping, bandaging, or bracing to protect the ligaments during the proliferation phase as fibroblasts invade the injured area and form collagen fibers. Functional treatment concludes with early active range of motion exercises, weight bearing, and neuromuscular ankle training to promote proper collagen fiber orientation and prevent stiffness during the remodeling or maturation phase [42, 60, 70, 71].

Long-term outcome of treatment choice for acute ankle injury is also important to consider, especially in the elite footballer. A meta-analysis of 12 prospective, randomized studies revealed earlier return to work and quicker recovery in ankle mobility after functional treatment compared to surgery or cast immobilization, with no difference in the incidence of chronic functional instability [14]. In contrast, a more recent prospective, randomized study found that after 8 years, patients who received surgery versus functional treatment reported less residual pain, less chronic ankle instability, and fewer recurrent sprains [72]. Given that secondary surgical repair has comparable results to those achieved with primary repair and functional treatment seems to yield similar outcomes, surgical intervention is rarely indicated for acute ankle sprains [42, 71]. An exception could include footballers with severe ruptures associated with other pathology requiring acute intervention such as osteochondral fracture of the ankle joint.

Nonsteroidal anti-inflammatory drugs (NSAIDs) have been shown in randomized controlled trials to be more effective when compared to placebo in controlling pain, lessening swelling, and reducing the time needed for return to activity [73, 74]. Other prospective, randomized double-blind trials have also shown efficacy for NSAID treatment in terms of decreased time for improvement in ankle function, tenderness, swelling, and pain; however, the differences were small, short-term, and not significant in the long-term outcome [75–77]. Animal studies demonstrate conflicting results for NSAIDs on ligament healing [78–80]. Based on available evidence, a short course (3–7 days) of NSAIDs for ligament sprain is likely to be beneficial [81].

### 3.11 Prevention

Since ankle injuries are so common in football, and sports in general, many studies have attempted to look at both primary and secondary prevention of injuries in numerous populations. Ankle instability can arise from several factors including decreased sensorimotor control due to poor muscular reaction time or decreased proprioception, mechanical instability as a result of ligament damage, or compromised muscular strength, power, or endurance. Modes of prevention include orthoses, taping, neuromuscular/proprioception training, and muscular strengthening.

Several studies have identified specific factors worthy of focus in football injury prevention. A review of the literature by Holmes et al. [82] concluded that strengthening protocols may be more effective in addressing invertor muscle deficits, in contrast to evertor muscle deficits, since these were more frequently present in subjects with chronic ankle instability. Footballer age may also be an important area of prevention focus, as a phone survey of amateur Dutch footballers found the highest rate of ankle injuries in senior players (17–34 years old) versus juniors (4–16 years old) [83]. In addition, up to 32–69 % of all ankle sprains are a result of reinjury [2, 7, 9, 12, 19, 35], with one study showing radiographically documented instability in



**Fig. 3.3** Example of a low-profile ankle brace designed specifically for the footballer

80 % of patients with recurrent sprains [51]. This highlights the importance of secondary prevention in minimizing future ankle injuries.

Taping restricts inversion of the ankle, but it requires expertise and assistance to perform. It loses much of its supportive effect after as little as 30 min of play [84] and has minimal effects on primary prevention of ankle injuries [85]. Studies have shown potential for ankle taping in the prevention of reinjury [85–87], although not as effective as a lace-up ankle orthosis [86]. Nonetheless, the low profile of taping allows easy accommodation into boots and under shin guards, making it an attractive option for the footballer.

Application of an orthosis has been shown to be ineffective for primary prevention of ankle injuries [88], yet in football players with previous ankle sprains, these external support devices had a significant effect in reducing the incidence of ankle reinjury [87, 88]. In contrast to tape, orthoses do not require expertise or assistance for application and may be retightened during play. Furthermore, they have been shown to slow the speed of ankle inversion, while maintaining correct anatomic alignment. A majority of studies have found minimal, if any, effect of orthoses on athletic performance [57]. A low-profile brace for football (Fig. 3.3) has been developed which allows for minimal disruption of the player's boot and space proximally to insert the shin guard.

Neuromuscular/proprioception training such as balance exercises including single leg stance, tilt board, or ankle disc training has been shown to reduce the risk of ankle reinjury. Position sense of the ankle is significantly decreased at both 1 week and 12 weeks after ankle injury in the general population [89], indicating that decreased proprioception could contribute to reinjury. A study of first division men's league footballers in Iran showed a significant decrease in reinjury rates with proprioceptive training, but not with strengthening or orthoses [90]. It has also been shown that balance training can improve overall postural alignment of the body and decrease stress on the ankle through the hip and knee joints, potentially leading to decreased rates of reinjury [82].

It is currently unclear why prophylactic taping or orthosis use does not decrease primary ankle injuries, but there seems to be a consensus on prevention of reinjury, which should include a supervised rehabilitation program with neuromuscular and proprioceptive training before return to play. Additionally, footballers with moderate to severe strains should wear an orthosis for at least 6 months following injury [57]. A prospective study by Ekstrand et al. showed that close supervision of male senior football players by a physiotherapist and doctor combined with prophylactic ankle taping and technique correction reduced all injuries by 75 % [91]. In 2003, FIFA introduced a comprehensive warm-up program (FIFA 11+) which has since been shown in several studies to decrease the risk of injury in amateur football players [92–94], although one study has shown that the injury rate is related to skill level and not adherence to the FIFA 11+ program at the amateur level of football [95].

#### 3.12 Chronic Ankle Instability

Chronic ankle instability develops over the course of a player's career through repetitive injuries and includes a wide spectrum of pathology. Alternatively, ankle instability may occur from a single insult. Two general forms of chronic ankle instability have been described: mechanical instability and functional instability. Mechanical instability includes laxity of the lateral or medial ligamentous structures, whereas functional instability involves intra- or periarticular changes without ligamentous laxity. Importantly, footballers often have a combination of the two conditions with overlapping pathology [48, 96].

In football, 20–40 % of acute sprains develop into chronic ankle instability [97]. Common symptoms may include persistent synovitis or tendinitis, stiffness, swelling, pain, muscle weakness, limping, intermittent locking, or frequent giving way [71, 98]. Diagnosis of chronic ankle injury is made clinically, based on history and physical exam, and through the use of stress radiographs or other imaging techniques. Although the anterior drawer and talar tilt tests are commonly used clinically to assess ankle instability, their use in radiographic stress imaging may underestimate the true magnitude of talar tilt or anterior displacement [99]. MRI can confirm ligament tears and help identify associated injuries that may be overlooked in the physical exam [100], including cartilage injury, peroneal tendon tear, or flexor hallucis longus stenosis. In those patients with a prolonged history (>6 months) of ankle pain refractory to conservative treatment, arthroscopy can be used as a diagnostic tool [98]. Functional rehabilitation, as previously described, is more likely to successfully treat functional rather than mechanical ankle instability [42].

#### 3.13 Mechanical Ankle Instability

#### 3.13.1 Lateral Ligament Instability

The most common mechanism of lateral ankle ligament injury is the result of combined plantar flexion and inversion motion. This position causes vulnerability of the ATFL, which is the most frequently torn ligament in ankle sprains [51, 70]. Combined rupture of the ATFL and CFL is also commonly seen in ankle sprains with more severe inversion. Lateral ligament rupture may also cause talar subluxation, resulting in collision between the medial malleolus and medial talar facet with subsequent articular cartilage damage [101]. Other injuries associated with ankle inversion injuries include loose bodies, peroneal tendon tears, flexor hallucis longus stenosis, or damage to the superficial and deep peroneal nerves [51, 98].

Lateral ligament instability is the most common diagnosis of chronic ankle pathology [98], with associated subtalar ligamentous injury and instability only present in about 10 % of patients with lateral ankle instability [51]. The PTFL is usually not involved [42]. Arthroscopy can confirm ligamentous instability and reveal the state of the articular surface, while also removing chondral fractures and loose bodies; however, the definitive treatment of lateral instability requires surgical repair of the lateral ligaments [98]. The indications for lateral ligamentous reconstruction include persistent symptomatic mechanical instability and failed functional rehabilitation. Operative treatments for lateral ankle instability can be divided into anatomic repair and anatomic or nonanatomic reconstruction [42].

Anatomic repair of the ATFL using the Bröstrom-Gould modified technique in the presence of CFL insufficiency has been shown to be highly successful with reports of 93 % excellent results at an average of 64 months follow-up [102], 80 % excellent or good results at 6 years follow-up [103], and 91 % good-to-excellent results at 26 years follow-up [104]. Major advantages of this method are its simplicity and small incision size [105]. The Bröstrom procedure also avoids use of tendon grafts, maintains normal ligamentous anatomy, and preserves physiologic tibiotalar and subtalar motion. The disadvantage lies in the fact that strong repair relies on innate tissue quality [70].

Common nonanatomic reconstruction techniques include the Watson-Jones, Evans, and Chrisman-Snook procedures. Nonanatomic reconstruction permanently changes normal ankle kinematics, resulting in impaired subtalar motion and residual instability. As such, many studies have reported poor long-term results for these procedures [42], and nonanatomic reconstructions should be avoided in the active footballer.

Use of an anatomic reconstruction has replaced historic nonanatomic reconstruction techniques [105]. Anatomic tenodesis reconstruction using an autograft or allograft augments anatomic repair without compromising lateral ankle anatomy or kinematics. This technique is indicated when poor tissue quality is present or for revision surgery and may be an excellent option for high-demand ankles with chronic instability [42]. Colville et al. found that 12 of 15 patients available at 3.5 years follow-up showed radiographically confirmed restoration of mechanical stability [106]. Sammarco et al. reported good-to-excellent results in 29 of 31 ankles (94 %) at a mean follow-up of 44 months using a split peroneus brevis tendon graft for reconstruction [107].

#### 3.13.2 Medial Ligament Instability

Football places a high demand on the medial foot and ankle structures since striking the ball requires a player to abduct and externally rotate the foot, preloading the medial structures [18]. In a prospective study of 54 cases of medial ankle instability, Hintermann et al. found that injury to the medial ankle ligaments commonly occurred during landing on an uneven surface [53]. This may apply to the footballer in scenarios such as landing after heading the ball or jumping over a tackle. Pronation with eversion and extreme rotational injuries are well known to cause deltoid ligament injury, yet most patients will report multiple ankle sprains and be unable to identify a single causal event [52]. Complete deltoid ligament rupture is rare and is more often associated with ankle fractures [108]. Due to its close proximity and similarly shared function in medial plantar arch stabilization with the tibiospring and spring ligaments, posterior tibialis (PT) tendon dysfunction is also frequently seen in medial ankle instability [52].

Patients with medial ankle instability often describe the ankle as "giving away," especially when walking on uneven surfaces, downhill, or downstairs. A history of eversion trauma to the ankle is common. After acute injury, patients can present with a medial ankle hematoma and pain along the deltoid ligament [52]. Chronic deltoid insufficiency is diagnosed based on this feeling of "giving away," pain in the medial gutter of the ankle, and a valgus and pronation deformity of the foot that can be corrected by activating the PT muscle [52]. Tenderness can also be present along the PT tendon and anterior border of the lateral malleolus. The tiptoe test is used to identify PT dysfunction [52]. Arthroscopy is the most specific way to confirm clinically suspected instability of the medial ankle [53]; however, MRI can demonstrate loss of organized medial fibers.

In the setting of symptomatic ankle instability with clinically and arthroscopically confirmed medial ankle instability, surgical exploration of the medial ankle ligaments should be performed [52]. Primary surgical repair of deltoid ligament tears yields good-to-excellent results and should be considered in the footballer to prevent the associated problems with chronic non-repaired tears such as instability, osteoarthritis, and impingement syndromes [52, 53]. Additional procedures may be necessary such as posterior tibial tendon debridement or repair, calcaneal lengthening osteotomy, or reconstruction of lateral ankle ligaments [53].

#### 3.13.3 Syndesmosis Instability

Footballers may have increased risk for syndesmotic sprains due to foot planting and cutting action [108]. Acute syndesmotic injury occurs when the fibula is pushed

away from its distal articulation with tibia. This has been described to occur through several possible mechanisms, including external rotation of foot, eversion of the talus, or ankle dorsiflexion [109]. Chronic injury can occur through instability that causes shear stress on the ankle joint with eventual ankle arthritis [108].

Syndesmotic injury is diagnosed though several physical exam findings, including pain with palpation of the AITFL, external rotation of the foot, or dorsiflexion of the foot. Proximal extension of pain indicates involvement of the underlying interosseous membrane with potential for more serious injury. Patients often walk with a heel-raise gait, thereby maintaining the foot in plantar flexion to avoid pain during push-off. An antalgic gait may also be observed in which the patient displays a shortened stance phase relative to swing phase to minimize the pain felt when weight bearing. The injury is associated with minimal swelling, which may lead to misdiagnosis of a common ankle sprain [108, 109]. Importantly, the deltoid ligament can be associated with syndesmotic sprain as they share similar external rotation and eversion mechanisms of injury [108]. Increased swelling, pain with palpation, ecchymosis over the medial ankle, and greater than 4 mm widening of the syndesmosis on plain films may be indicative of deltoid ligament involvement [110]. Special clinical tests used for diagnosis of syndesmotic injury include the external rotation test, the squeeze test, the crossedleg test, the cotton test, the point test, the dorsiflexion maneuver, and one-legged hop test [108].

Imaging for syndesmotic injury begins with anteroposterior (AP), lateral, and mortise ankle radiographs to rule out ankle fracture. Two measurements made 1 cm above the tibia plafond are used to evaluate the integrity of the syndesmosis, tibio-fibular clear space, and tibiofibular overlap [108]. Tibiofibular clear space greater than 6 mm and tibiofibular overlap less than 1 mm indicate a disrupted syndesmosis [111]. MRI is indicated for syndesmotic injuries and has high sensitivity and specificity [112]. It can reveal secondary findings such as bone bruises, ATFL injury, osteochondral lesions, and tibiofibular joint incongruity [113]. Finally, arthroscopy has been shown to be an invaluable diagnostic tool for syndesmotic injury with the characteristic triad finding of PITF scarring, disrupted interosseous ligament, and posterolateral tibial plafond chondral damage [114].

There is currently no consensus for optimal management of a syndesmotic injury [115]; however, classification of the ligaments involved can aid in selection of appropriate treatment. Grade I involves AITFL tears; grade IIa involves AITFL and interosseous ligament tears; grade IIb includes AITFL, PITFL, and interosseous ligament injury; and grade III involves injury to all three syndesmotic ligaments, as well as fibula fracture. Conservative treatment is recommended for grades I and IIa, whereas surgical intervention is necessary in grades IIb and III syndesmotic injuries. These injures take twice as long to heal as other ankle sprains [109]. Hopkinson et al. reported a mean recovery time of 55 days following syndesmosis injury in cadets at the United States Military Academy at West Point [116]. Rehabilitation should be gradual and pain-free, starting with low-level balance training, then progressing through to double leg heel raises, single leg heel raises, walking, fast walking, jogging, cutting, and finally sport-specific training [108]. Players are able to return to sport after an average of 4 months following initial syndesmotic injury.



**Fig. 3.4** Syndesmosis injury in a 21-year-old footballer. (a) Axial MRI of syndesmotic injury demonstrating a ruptured AITFL (*arrow*). (b) Arthroscopic image of open syndesmosis prior to repair. (c) Syndesmosis injury after fixation with combination of screw and TightRope (Arthrex, Inc., Naples, FL, USA). (d) Arthroscopic view of syndesmosis after repair

Historically, the most commonly used surgical technique is syndesmotic screw fixation. It is indicated if there is a laterally displaced fibula or significant mortise widening with positive lateral radiography and external rotation stress test [117]. Lateral talus subluxation greater than 1 mm is also an indication for screw fixation, which is suggested to assure reduction and stability of the ankle mortise [118]. Unfortunately, there is no consensus on optimal screw size, level of placement, or timing of removal [119, 120]. TightRope (Arthrex, Naples, FL) is a fixation technique consisting of a fiberwire secured between two endobuttons (Fig. 3.4). It has been shown to provide more accurate stabilization of the syndesmosis as compared to screw fixation [121]. Since malreduction is the most important indicator of poor long-term functional outcome, TightRope fixation should be considered in treatment of the footballer [122]. To date, there is no evidence of improvement in short-term functional outcomes with the TightRope, although there may be a decreased need for implant removal [123]. Surgical intervention is also recommended in the



Fig. 3.5 Peroneal tendon injury. (a) Axial MRI showing peroneal tendon subluxation (*arrow*). (b) Lateral incision reveals an anteriorly translocated peroneal longus tendon superficial to the fibrocartilaginous ridge (probe)

presence of persistent pain following syndesmotic injury due to the development of anterior impingement syndrome and heterotopic ossification [108].

#### 3.13.4 Superior Peroneal Reticulum Injury

The superior peroneal retinaculum (SPR) forms the roof of the superior peroneal tunnel. The tunnel contains the peroneus brevis and longus tendons and is bordered by the retromalleolar groove of the fibula and the lower aspect of the posterior intramuscular septum of the leg [124, 125]. Injury to the retinaculum results from ankle dorsiflexion and inversion and forceful reflex contraction of the peroneal muscles, which causes subluxation or dislocation of the contained tendons [124]. Since these patients present with similar injury mechanisms as simple lateral ankle sprains, the clinician must maintain high suspicion for peroneal tendon injury, especially in the presence of retrofibular pain, snapping or popping sensations about the lateral malleolus, or chronic ankle instability that worsens on uneven surfaces [124].

SPR injury can be divided into three grades [126]. A grade I injury involves SPR separation from its periosteum origin at the lateral malleolus, leading to anterior displacement of the peroneal tendons about the fibrocartilaginous ridge. In grade II injury, the fibrocartilaginous ridge is stripped with detached SPR. Grade III injury involves both SPR detachment and fibrocartilaginous ridge stripping, as well as osseous fibular fragmentation [124]. MRI is the best imaging modality to assess peroneal tendon and SPR pathologies, although ultrasound may be more useful in revealing an episodic subluxation [124]. Conservative management is often associated with poor outcomes, and surgery is indicated for all acute and chronic dislocations in footballers [127]. Anatomic reconstruction of the SPR (Fig. 3.5) is the

preferred surgical method [128]. Peroneus brevis debulking and fibular groove deepening may also augment retinaculum repair [129].

#### 3.14 Functional Ankle Instability

Painful mechanical limitation of ankle motion defines ankle impingement syndrome [52] and can be classified as either soft tissue or osseous [130]. Soft tissue impingements occur most commonly in the anterolateral gutter, medial ankle, or syndesmotic area as a result of scarring and fibrosis associated with synovial, capsular, or ligamentous injury [130]. Osseous impingements are a consequence of osteophyte formation, most commonly along the anterior distal tibia and talus or posteriorly as the os trigonum. These conditions are important to recognize as they may result in chronic ankle pain, especially in footballers [131].

# 3.14.1 Footballer's Ankle (Anterior Ankle Impingement Syndrome)

Anterior talotibial osteophytes are found in up to 60 % of footballers [16]. These lesions were first described as "athlete's ankle" by Morris in 1943 [132]. Later, McMurray speculated that the etiology of these osteophytes was repeated ligamentous strain from kicking a football with the foot in equinus, suggesting the term "footballer's ankle" [133]. It is now known that these lesions are not specific to the footballer but may occur in any sport involving running or jumping [134]. Although asymptomatic ankle osteophytes are reported in 45 % of football players [135], chronic pain may be experienced in what is now termed anterior ankle impingement syndrome (AAIS). This condition results from entrapment of hypertrophied synovial tissue or posttraumatic scar tissue between the distal tibia and malleoli and talus, exacerbated by the presence of anterior osteophytes (Fig. 3.6) [130, 135]. Early stages of anterior impingement can be treated successfully with injections and heel lifts, but surgery may be necessary for osteophyte and soft tissue debridement in more chronic cases [130].

On physical examination of a patient with anterior impingement syndrome, palpation along the anterior joint line results in point tenderness and pain can be elicited by placing the foot in maximum plantar flexion or dorsiflexion. Patients will complain of limited painful dorsiflexion exacerbated with squatting or stair climbing. Radiographic imaging is important in preoperative planning because osteophytes can be difficult to detect during arthroscopy, especially those located in the anteromedial compartment. MRI aids in preoperative planning through identification of reactive synovitis and fibrosis, subchondral marrow edema, collateral ligament complex injury, osteochondral lesions, intra-articular bodies, or osteoarthritis. Most patients with no preexisting osteoarthritis treated arthroscopically will Fig. 3.6 Radiographic image of anterior impingement in a 22-year-old footballer. An osteophyte is seen on the superior aspect of the talar neck (*arrow*)



experience pain relief and return to full activity, though recurrent osteophyte formation has been noted at long-term follow-up [130].

#### 3.14.2 Anterolateral Ankle Impingement Syndrome

Anterolateral ankle impingement syndrome is most often caused by an acute ankle inversion injury [130]. The subsequent reactive inflammation can cause fibrosis leading to distal fascicle enlargement of the AITF. Impingement in the anterolateral gutter of this enlarged fascicle can also cause chronic reactive synovitis and chondromalacia of the lateral talar dome [108]. The ATFL may also be involved with irregular thickening or nodularity [130]. Arthroscopy can reveal the common meniscoid-like lesion (Fig. 3.7) [130]. Bone scans and radiographs are not useful in diagnosis. MRI can identify abnormal areas of pathology, although 50 % of cases are diagnosed based on clinical presentation alone [98]. Common presentation is a history of anterolateral ankle pain and swelling with a popping or snapping sensation. The patient will have tenderness to palpation along the anterolateral aspect of the ankle, with pain elicited at extreme passive dorsiflexion [130]. Initial injection with local anesthetic and corticosteroid can be both diagnostic and curative. After 4 weeks, the patient should return for follow-up and further discussion of surgical treatment if symptoms have not resolved. Arthroscopy is used to confirm clinical findings prior to resecting the involved scar tissue, debriding the AITFL and ATFL, or removing any chondral lesions of the talar lateral dome [98]. The best



**Fig. 3.7** Meniscoid-like lesion of anterolateral ankle impingement syndrome

arthroscopic results occur in patients with no concurrent intra-articular lesions [130] or ankle osteoarthritis.

### 3.14.3 Anteromedial Ankle Impingement Syndrome

Anteromedial impingement syndrome is the most rare ankle impingement syndrome and likely occurs as a rare complication resulting from deltoid ligament complex disruption following an inversion event [131, 136]. Alternatively, anteromedial impingement lesions are also associated with eversion injuries or following medial malleolar or talar fractures [132]. It is thought that previous injury to the anterior tibiotalar fascicle of the deltoid complex leads to ligament thickening and subsequent impingement on the anteromedial corner of the talus. Adjacent fibrosis and synovitis are common consequences of impingement, but osteophyte formation and chondral stripping along the anteromedial talus can also be present. Typical complaints include pain along the anteromedial joint line that increases with walking, clicking sensations, and painful, limited dorsiflexion. Anteromedial impingement can be identified clinically through palpation over the anterior tibiotalar fascicle of the deltoid ligament and eversion or extreme passive dorsiflexion of the foot, all of which will elicit medial ankle tenderness [52, 130]. Initial conservative management consists of rest, physical therapy, and NSAIDs. Refractory cases may undergo arthroscopy or open resection of the anterior tibiotalar fascicle with debridement of any adjacent synovitis or scar tissue [130].

# 3.14.4 Posterior Ankle Impingement Syndrome

Posterior ankle impingement syndrome (PAIS) (Fig. 3.8) is also referred to as "os trigonum syndrome" since the posterior impingement is often associated with a



**Fig. 3.8** X-ray showing a prominent os trigonum (*arrow*)

prominent os trigonum (unfused posterolateral talar process). Pain results from impaction between the posterior tibial plafond and the os trigonum or posterior calcaneal process or as a result of soft tissue compression between the two opposing osseous structures. Because football is a sport requiring repetitive and extreme plantar flexion, players often experience posterior ankle impingement [130, 137]. In addition, slide tackles from behind resulting in acute trauma to the posterolateral talar process predispose the footballer to PAIS.

Clinically, posterior impingement can be much more difficult to detect and diagnose when compared to other types of ankle impingement because the affected structures lie much deeper and can be mimicked by or coexist with other disease processes such as peroneal tendinopathy, retrocalcaneal bursitis, osteochondral lesions of the posterior talar dome, Achilles tendinopathy, flexor hallucis longus tendinopathy or tenosynovitis, posterior tibial osteochondral injuries, tarsal tunnel compression, tarsal coalition, and Haglund's deformity [130, 137]. Patients will complain of chronic deep posterior ankle pain that is worsened with push-off activities such as jumping. Physical examination includes palpation over the posterolateral process and the crunch test. Diagnosis can be confirmed with abatement of pain following injection of an anesthetic into the posterolateral capsule of the tibiotalar joint. MRI is useful for more accurately identifying the anatomic site of abnormality, as well as revealing coexisting pathologies. Fortunately, rest is often an adequate therapy regardless of whether the symptoms are acute or chronic [130, 137]. When nonoperative measures have failed, open or arthroscopic removal of the os can quickly return the footballer to play. Calder et al. demonstrated the effectiveness of posterior ankle arthroscopy in the treatment of PAIS in the elite footballer, with return to training expected at an average of 5 weeks [138].

## 3.15 Conclusion

Ankle injuries are very common in football and can result in decreased performance or significant loss of playing time. Treatment of acute injury should be conservative, with surgical intervention reserved for severe ruptures or osteochondral fracture of the ankle joint. Chronic ankle pathology resulting in mechanical or functional instability generally requires surgery and/or arthroscopy to repair ligamentous damage and restore normal ankle kinematics. It is critical for the footballer to receive appropriate rehabilitation prior to returning to play in order to reduce the risk of reinjury and further chronic instability. Since former elite footballers are at a higher risk of ankle osteoarthritis than the general population [139–141], and lateral ligament instability is a major cause of posttraumatic ankle arthritis [142], prevention and early intervention of ankle injuries could potentially prevent the long-term incidence of arthritis in former footballers.

### References

- 1. Andersen TE, Floerenes TW, Arnason A, Bahr R (2004) Video analysis of the mechanisms for ankle injuries in football. Am J Sports Med 32(1 Suppl):69S–79S
- Chomiak J, Junge A, Peterson L, Dvorak J (2000) Severe injuries in football players. Am J Sports Med 28(5 Suppl):S58–S68
- Cloke DJ, Ansell P, Avery P, Deehan D (2011) Ankle injuries in football academies: a threecentre prospective study. Br J Sports Med 45(9):702–708
- Cloke DJ, Spencer S, Hodson A, Deehan D (2009) The epidemiology of ankle injuries occurring in English Football Association academies. Br J Sports Med 43(14):1119–1125
- Cox JS (1985) Surgical and nonsurgical treatment of acute ankle sprains. Clin Orthop Relat Res 198:118–126
- Dvorak J, Junge A, Derman W, Schwellnus M (2011) Injuries and illnesses of football players during the 2010 FIFA World Cup. Br J Sports Med 45(8):626–630
- Ekstrand J, Gillquist J (1983) Soccer injuries and their mechanisms: a prospective study. Med Sci Sports Exerc 15(3):267–270
- Fong DT, Hong Y, Chan LK, Yung PS, Chan KM (2007) A systematic review on ankle injury and ankle sprain in sports. Sports Med 37(1):73–94
- Fousekis K, Tsepis E, Vagenas G (2012) Intrinsic risk factors of noncontact ankle sprains in soccer: a prospective study on 100 professional players. Am J Sports Med 40(8):1842–1850
- Giza E, Fuller C, Junge A, Dvorak J (2003) Mechanisms of foot and ankle injuries in soccer. Am J Sports Med 31(4):550–554
- Gaulrapp H, Becker A, Walther M, Hess H (2010) Injuries in women's soccer: a 1-year all players prospective field study of the women's Bundesliga (German premiere league). Clin J Sports Med 20(4):264–271
- Hawkins RD, Fuller CW (1999) A prospective epidemiological study of injuries in four English professional football clubs. Br J Sports Med 33(3):196–203
- Heidt RS Jr, Sweeterman LM, Carlonas RL, Traub JA, Tekulve FX (2000) Avoidance of soccer injuries with preseason conditioning. Am J Sports Med 28(5):659–662
- 14. Kannus P, Renström P (1991) Treatment for acute tears of the lateral ligaments of the ankle. Operation, cast, or early controlled mobilization. J Bone Joint Surg Am 73(2):305–312
- 15. MacAuley D (1999) Ankle injuries: same joint, different sports. Med Sci Sports Exerc 31(7 Suppl):S409–S411

- 3 Epidemiology and Mechanisms of Ankle Pathology in Football
- Massada JL (1991) Ankle overuse injuries in soccer players. Morphological adaptation of the talus in the anterior impingement. J Sports Med Phys Fitness 31(3):447–451
- McCarroll JR, Schrader JW, Shelbourne KD, Rettig AC, Bisesi MA (1987) Meniscoid lesions of the ankle in soccer players. Am J Sports Med 15(3):255–257
- Morgan BE, Oberlander MA (2001) An examination of injuries in major league soccer. The inaugural season. Am J Sports Med 29(4):426–430
- Nielsen AB, Yde J (1989) Epidemiology and traumatology of injuries in soccer. Am J Sports Med 17(6):803–807
- Oztekin HH, Boya H, Ozcan O, Zeren B, Pinar P (2009) Foot and ankle injuries and time lost from play in professional soccer players. Foot (Edinb) 19(1):22–28
- Peterson L, Junge A, Chomiak J, Graf-Baumann T, Dvorak J (2000) Incidence of football injuries and complaints in different age groups and skill-level groups. Am J Sports Med 28(5 Suppl):S51–S57
- 22. Renström PA, Konradsen L (1997) Ankle ligament injuries. Br J Sports Med 31(1):11-20
- 23. Shapiro MS, Kabo JM, Mitchell PW, Loren G, Tsenter M (1994) Ankle sprain prophylaxis: an analysis of the stabilizing effects of braces and tape. Am J Sports Med 22(1):78–82
- 24. Tol JL, Slim E, van Soest AJ, van Dijk CN (2002) The relationship of the kicking action in soccer and anterior ankle impingement syndrome. A biomechanical analysis. Am J Sports Med 30(1):45–50
- Yard EE, Schroeder MJ, Fields SK, Collins CL, Comstock RD (2008) The epidemiology of United States high school soccer injuries, 2005–2007. Am J Sports Med 36(10):1930–1937
- 26. Fuller CW, Junge A, Dvorak J (2012) Risk management: FIFA's approach for protecting the health of football players. Br J Sports Med 46(1):11–17
- 27. KantarSport (2010) 2010 FIFA World Cup South Africa Television Audience Report London
- 28. Kunz M (2007) Big count: 265 million playing football. In: FIFA Magazine, pp 10–15
- 29. Arnason A et al (2004) Risk factors for injuries in football. Am J Sports Med 32(1 Suppl):5S-16S
- Hoy K et al (1992) European soccer injuries. A prospective epidemiologic and socioeconomic study. Am J Sports Med 20(3):318–322
- Giza E et al (2005) Injuries in women's professional soccer. Br J Sports Med 39(4):212–216; discussion 212–6
- 32. Wong P, Hong Y (2005) Soccer injury in the lower extremities. Br J Sports Med 39(8):473-482
- 33. Inklaar H (1994) Soccer injuries. I: incidence and severity. Sports Med 18(1):55-73
- Walden M, Hagglund M, Ekstrand J (2005) UEFA Champions League study: a prospective study of injuries in professional football during the 2001–2002 season. Br J Sports Med 39(8):542–546
- 35. Arnason A et al (1996) Soccer injuries in Iceland. Scand J Med Sci Sports 6(1):40-45
- 36. Giza E, Micheli LJ (2005) Soccer injuries. Med Sport Sci 49:140-169
- 37. Aoki H et al (2012) A 15-year prospective epidemiological account of acute traumatic injuries during official professional soccer league matches in Japan. Am J Sports Med 40(5):1006–1014
- Luthje P et al (1996) Epidemiology and traumatology of injuries in elite soccer: a prospective study in Finland. Scand J Med Sci Sports 6(3):180–185
- 39. Ekstrand J, Tropp H (1990) The incidence of ankle sprains in soccer. Foot Ankle 11(1):41-44
- 40. Woods C et al (2003) The Football Association Medical Research Programme: an audit of injuries in professional football: an analysis of ankle sprains. Br J Sports Med 37(3):233–238
- 41. Engebretsen AH et al (2010) Intrinsic risk factors for acute ankle injuries among male soccer players: a prospective cohort study. Scand J Med Sci Sports 20(3):403–410
- 42. Chan KW, Ding BC, Mroczek KJ (2011) Acute and chronic lateral ankle instability in the athlete. Bull NYU Hosp Jt Dis 69(1):17–26
- Junge A, Dvorak J (2013) Injury surveillance in the World Football Tournaments 1998–2012. Br J Sports Med 47:782–788
- 44. Carling C, Orhant E, LeGall F (2010) Match injuries in professional soccer: inter-seasonal variation and effects of competition type, match congestion and positional role. Int J Sports Med 31(4):271–276
- 45. Dvorak J, Junge A (2000) Football injuries and physical symptoms. A review of the literature. Am J Sports Med 28(5 Suppl):S3–S9

- 46. Moore KL, Dalley AF, Agur AMR (2010) Clinically oriented anatomy, 6th edn. Wolters Kluwer/Lippincott Williams & Wilkins, Philadelphia, xxix, 1134 p
- 47. Thompson JC, Netter FH (2010) Netter's concise orthopaedic anatomy, 2nd edn. Saunders Elsevier, Philadelphia, x, 404 p
- Eric Giza BM (2006) Chronic footballer's ankle. In: Football traumatology, Springer Milan, pp 333–351
- Garrick JG (1977) The frequency of injury, mechanism of injury, and epidemiology of ankle sprains. Am J Sports Med 5(6):241–242
- Agur AMR, Grant JCB (2011) Grant's atlas of anatomy, 13th edn. Wolters Kluwer Health/ Lippincott Williams & Wilkins, Philadelphia
- 51. Renstrom PA, Konradsen L (1997) Ankle ligament injuries. Br J Sports Med 31(1):11-20
- Chhabra A, Subhawong TK, Carrino JA (2010) MR imaging of deltoid ligament pathologic findings and associated impingement syndromes. Radiographics 30(3):751–761
- 53. Hintermann B (2003) Medial ankle instability. Foot Ankle Clin 8(4):723-738
- 54. Cedell CA (1975) Ankle lesions. Acta Orthop Scand 46(3):425-445
- 55. Gerber JP et al (1998) Persistent disability associated with ankle sprains: a prospective examination of an athletic population. Foot Ankle Int 19(10):653–660
- Williams GN, Jones MH, Amendola A (2007) Syndesmotic ankle sprains in athletes. Am J Sports Med 35(7):1197–1207
- Thacker SB et al (1999) The prevention of ankle sprains in sports. A systematic review of the literature. Am J Sports Med 27(6):753–760
- Tucker AM (1997) Common soccer injuries. Diagnosis, treatment and rehabilitation. Sports Med 23(1):21–32
- 59. Ivins D (2006) Acute ankle sprain: an update. Am Fam Physician 74(10):1714-1720
- 60. Lynch SA, Renstrom PA (1999) Treatment of acute lateral ankle ligament rupture in the athlete. Conservative versus surgical treatment. Sports Med 27(1):61–71
- Stiell IG et al (1992) A study to develop clinical decision rules for the use of radiography in acute ankle injuries. Ann Emerg Med 21(4):384–390
- Bachmann LM et al (2003) Accuracy of Ottawa ankle rules to exclude fractures of the ankle and mid-foot: systematic review. BMJ 326(7386):417
- 63. Stiell IG et al (1993) Decision rules for the use of radiography in acute ankle injuries. Refinement and prospective validation. JAMA 269(9):1127–1132
- Balduini FC et al (1987) Management and rehabilitation of ligamentous injuries to the ankle. Sports Med 4(5):364–380
- 65. Kerkhoffs GM et al (2001) Immobilisation for acute ankle sprain. A systematic review. Arch Orthop Trauma Surg 121(8):462–471
- Konradsen L, Holmer P, Sondergaard L (1991) Early mobilizing treatment for grade III ankle ligament injuries. Foot Ankle 12(2):69–73
- Eiff MP, Smith AT, Smith GE (1994) Early mobilization versus immobilization in the treatment of lateral ankle sprains. Am J Sports Med 22(1):83–88
- Shrier I (1995) Treatment of lateral collateral ligament sprains of the ankle: a critical appraisal of the literature. Clin J Sport Med 5(3):187–195
- Kaikkonen A, Kannus P, Jarvinen M (1996) Surgery versus functional treatment in ankle ligament tears. A prospective study. Clin Orthop Relat Res 326:194–202
- 70. DiGiovanni BF, Partal G, Baumhauer JF (2004) Acute ankle injury and chronic lateral instability in the athlete. Clin Sports Med 23(1):1–19, v
- Petersen W et al (2013) Treatment of acute ankle ligament injuries: a systematic review. Arch Orthop Trauma Surg 133:1129–1141
- 72. Pijnenburg AC et al (2003) Operative and functional treatment of rupture of the lateral ligament of the ankle. A randomised, prospective trial. J Bone Joint Surg Br 85(4):525–530
- 73. Slatyer MA, Hensley MJ, Lopert R (1997) A randomized controlled trial of piroxicam in the management of acute ankle sprain in Australian Regular Army recruits. The Kapooka Ankle Sprain Study. Am J Sports Med 25(4):544–553
- 3 Epidemiology and Mechanisms of Ankle Pathology in Football
- 74. Petrella R et al (2004) Efficacy of celecoxib, a COX-2-specific inhibitor, and naproxen in the management of acute ankle sprain: results of a double-blind, randomized controlled trial. Clin J Sport Med 14(4):225–231
- 75. McCulloch PG et al (1985) The value of mobilisation and non-steroidal anti-inflammatory analgesia in the management of inversion injuries of the ankle. Br J Clin Pract 39(2): 69–72
- Viljakka T, Rokkanen P (1983) The treatment of ankle sprain by bandaging and antiphlogistic drugs. Ann Chir Gynaecol 72(2):66–70
- 77. Dupont M, Beliveau P, Theriault G (1987) The efficacy of antiinflammatory medication in the treatment of the acutely sprained ankle. Am J Sports Med 15(1):41–45
- Dahners LE et al (1988) The effect of a nonsteroidal antiinflammatory drug on the healing of ligaments. Am J Sports Med 16(6):641–646
- Hanson CA et al (2005) The effect of analgesic agents on the healing rat medial collateral ligament. Am J Sports Med 33(5):674–679
- Elder CL, Dahners LE, Weinhold PS (2001) A cyclooxygenase-2 inhibitor impairs ligament healing in the rat. Am J Sports Med 29(6):801–805
- Mehallo CJ, Drezner JA, Bytomski JR (2006) Practical management: nonsteroidal antiinflammatory drug (NSAID) use in athletic injuries. Clin J Sport Med 16(2):170–174
- Holmes A, Delahunt E (2009) Treatment of common deficits associated with chronic ankle instability. Sports Med 39(3):207–224
- Schmikli SL et al (2011) Injury prevention target groups in soccer: injury characteristics and incidence rates in male junior and senior players. J Sci Med Sport 14(3):199–203
- Alt W, Lohrer H, Gollhofer A (1999) Functional properties of adhesive ankle taping: neuromuscular and mechanical effects before and after exercise. Foot Ankle Int 20(4):238–245
- Garrick JG, Requa RK (1973) Role of external support in the prevention of ankle sprains. Med Sci Sports 5(3):200–203
- Rovere GD et al (1988) Retrospective comparison of taping and ankle stabilizers in preventing ankle injuries. Am J Sports Med 16(3):228–233
- Sharpe SR, Knapik J, Jones B (1997) Ankle braces effectively reduce recurrence of ankle sprains in female soccer players. J Athl Train 32(1):21–24
- Surve I et al (1994) A fivefold reduction in the incidence of recurrent ankle sprains in soccer players using the Sport-Stirrup orthosis. Am J Sports Med 22(5):601–606
- Konradsen L, Olesen S, Hansen HM (1998) Ankle sensorimotor control and eversion strength after acute ankle inversion injuries. Am J Sports Med 26(1):72–77
- 90. Mohammadi F (2007) Comparison of 3 preventive methods to reduce the recurrence of ankle inversion sprains in male soccer players. Am J Sports Med 35(6):922–926
- Ekstrand J, Gillquist J, Liljedahl SO (1983) Prevention of soccer injuries. Supervision by doctor and physiotherapist. Am J Sports Med 11(3):116–120
- 92. Steffen K et al (2013) Evaluation of how different implementation strategies of an injury prevention programme (FIFA 11+) impact team adherence and injury risk in Canadian female youth football players: a cluster-randomised trial. Br J Sports Med 47(8):480–487
- 93. Steffen K et al (2013) High adherence to a neuromuscular injury prevention programme (FIFA 11+) improves functional balance and reduces injury risk in Canadian youth female football players: a cluster randomised trial. Br J Sports Med 47(12):794–802
- Junge A et al (2011) Countrywide campaign to prevent soccer injuries in Swiss amateur players. Am J Sports Med 39(1):57–63
- 95. Gatterer H et al (2012) Effects of the performance level and the FIFA "11" injury prevention program on the injury rate in Italian male amateur soccer players. J Sports Med Phys Fitness 52(1):80–84
- Silvers EGHJ (2009) Ankle instability prevention. In: DeLee J, Drez's D (ed) Orthopaedic sport medicine: principles and practice, J.C.D.D.D.M.D. Miller, (ed) Saunders, Philadelphia
- 97. Valderrabano V et al (2006) Chronic ankle instability in sports a review for sports physicians. Sportverletz Sportschaden 20(4):177–183

- Ogilvie-Harris DJ, Gilbart MK, Chorney K (1997) Chronic pain following ankle sprains in athletes: the role of arthroscopic surgery. Arthroscopy 13(5):564–574
- Hoffman E et al (2011) Accuracy of plain radiographs versus 3D analysis of ankle stress test. Foot Ankle Int 32(10):994–999
- 100. Frey C et al (1996) A comparison of MRI and clinical examination of acute lateral ankle sprains. Foot Ankle Int 17(9):533–537
- 101. van Dijk CN, Bossuyt PM, Marti RK (1996) Medial ankle pain after lateral ligament rupture. J Bone Joint Surg Br 78(4):562–567
- 102. Hamilton WG, Thompson FM, Snow SW (1993) The modified Brostrom procedure for lateral ankle instability. Foot Ankle 14(1):1–7
- Karlsson J et al (1988) Reconstruction of the lateral ligaments of the ankle for chronic lateral instability. J Bone Joint Surg Am 70(4):581–588
- Bell SJ et al (2006) Twenty-six-year results after Brostrom procedure for chronic lateral ankle instability. Am J Sports Med 34(6):975–978
- 105. Schenck RC Jr, Coughlin MJ (2009) Lateral ankle instability and revision surgery alternatives in the athlete. Foot Ankle Clin 14(2):205–214
- 106. Colville MR, Grondel RJ (1995) Anatomic reconstruction of the lateral ankle ligaments using a split peroneus brevis tendon graft. Am J Sports Med 23(2):210–213
- 107. Sammarco GJ, Idusuyi OB (1999) Reconstruction of the lateral ankle ligaments using a split peroneus brevis tendon graft. Foot Ankle Int 20(2):97–103
- 108. Lin CF, Gross ML, Weinhold P (2006) Ankle syndesmosis injuries: anatomy, biomechanics, mechanism of injury, and clinical guidelines for diagnosis and intervention. J Orthop Sports Phys Ther 36(6):372–384
- Boytim MJ, Fischer DA, Neumann L (1991) Syndesmotic ankle sprains. Am J Sports Med 19(3):294–298
- Ebraheim NA et al (1997) Radiographic and CT evaluation of tibiofibular syndesmotic diastasis: a cadaver study. Foot Ankle Int 18(11):693–698
- 111. Harper MC, Keller TS (1989) A radiographic evaluation of the tibiofibular syndesmosis. Foot Ankle 10(3):156–160
- 112. Vogl TJ et al (1997) Magnetic resonance imaging in the diagnosis of acute injured distal tibiofibular syndesmosis. Invest Radiol 32(7):401–409
- 113. Brown KW et al (2004) MRI findings associated with distal tibiofibular syndesmosis injury. AJR Am J Roentgenol 182(1):131–136
- Ogilvie-Harris DJ, Reed SC, Hedman TP (1994) Disruption of the ankle syndesmosis: biomechanical study of the ligamentous restraints. Arthroscopy 10(5):558–560
- 115. Jones MH, Amendola A (2007) Syndesmosis sprains of the ankle: a systematic review. Clin Orthop Relat Res 455:173–175
- 116. Hopkinson WJ et al (1990) Syndesmosis sprains of the ankle. Foot Ankle 10(6):325-330
- 117. Wuest TK (1997) Injuries to the distal lower extremity syndesmosis. J Am Acad Orthop Surg 5(3):172–181
- Miller CD et al (1995) Deltoid and syndesmosis ligament injury of the ankle without fracture. Am J Sports Med 23(6):746–750
- 119. Dattani R et al (2008) Injuries to the tibiofibular syndesmosis. J Bone Joint Surg Br  $90(4){:}405{-}410$
- Schepers T (2011) To retain or remove the syndesmotic screw: a review of literature. Arch Orthop Trauma Surg 131(7):879–883
- 121. Naqvi GA et al (2012) Fixation of ankle syndesmotic injuries: comparison of tightrope fixation and syndesmotic screw fixation for accuracy of syndesmotic reduction. Am J Sports Med 40(12):2828–2835
- 122. Weening B, Bhandari M (2005) Predictors of functional outcome following transsyndesmotic screw fixation of ankle fractures. J Orthop Trauma 19(2):102–108
- 123. Schepers T (2012) Acute distal tibiofibular syndesmosis injury: a systematic review of suture-button versus syndesmotic screw repair. Int Orthop 36(6):1199–1206

- 124. Roth JA, Taylor WC, Whalen J (2010) Peroneal tendon subluxation: the other lateral ankle injury. Br J Sports Med 44(14):1047–1053
- Athavale SA, Swathi, Vangara SV (2011) Anatomy of the superior peroneal tunnel. J Bone Joint Surg Am 93(6):564–571
- Eckert WR, Davis EA Jr (1976) Acute rupture of the peroneal retinaculum. J Bone Joint Surg Am 58(5):670–672
- 127. Porter D et al (2005) Peroneal tendon subluxation in athletes: fibular groove deepening and retinacular reconstruction. Foot Ankle Int 26(6):436–441
- 128. Ferran NA, Oliva F, Maffulli N (2006) Recurrent subluxation of the peroneal tendons. Sports Med 36(10):839–846
- 129. Saxena A, Ewen B (2010) Peroneal subluxation: surgical results in 31 athletic patients. J Foot Ankle Surg 49(3):238–241
- Sanders TG, Rathur SK (2008) Impingement syndromes of the ankle. Magn Reson Imaging Clin N Am 16(1):29–38
- 131. Robinson P et al (2002) Anteromedial impingement of the ankle: using MR arthrography to assess the anteromedial recess. AJR Am J Roentgenol 178(3):601–604
- 132. Morris L (1943) Report of cases of athlete's ankle. J Bone Joint Surg 25:220
- 133. McMurray T (1950) Footballer's Ankle. J Bone Joint Surg Br 32:68-69
- 134. Parkes JC 2nd., Hamilton WG, Patterson AH, Rawles JG Jr (1980) The anterior impingement syndrome of the ankle. J Trauma 20(10):895–898
- Tol JL, van Dijk CN (2004) Etiology of the anterior ankle impingement syndrome: a descriptive anatomical study. Foot Ankle Int 25(6):382–386
- 136. Mosier-La Clair SM, Monroe MT, Manoli A (2000) Medial impingement syndrome of the anterior tibiotalar fascicle of the deltoid ligament on the talus. Foot Ankle Int 21(5):385–391
- 137. Maquirriain J (2005) Posterior ankle impingement syndrome. J Am Acad Orthop Surg 13(6):365–371
- Calder JD, Sexton SA, Pearce CJ (2010) Return to training and playing after posterior ankle arthroscopy for posterior impingement in elite professional soccer. Am J Sports Med 38(1):120–124
- 139. Kuijt MT et al (2012) Knee and ankle osteoarthritis in former elite soccer players: a systematic review of the recent literature. J Sci Med Sport 15(6):480–487
- 140. Armenis E et al (2011) Osteoarthritis of the ankle and foot complex in former Greek soccer players. Foot Ankle Spec 4(6):338–343
- 141. Drawer S, Fuller CW (2001) Propensity for osteoarthritis and lower limb joint pain in retired professional soccer players. Br J Sports Med 35(6):402–408
- 142. Valderrabano V et al (2006) Ligamentous posttraumatic ankle osteoarthritis. Am J Sports Med 34(4):612–620
- 143. Mattacola CG (2002) Rehabilitation of the ankle after acute sprain or chronic instability. J Athl Train 37(4):413–429
- 144. Penner MJ (2007) Instability of the ankle. In: Practical orthopaedic sports medicine & arthroscopy, 1st edn. Lippincott Williams & Wilkins, Philadelphia

# Chapter 4 Interview – Ricardo Pruna

Pieter P.R.N. d'Hooghe and Gino M.M.J. Kerkhoffs

**Abstract** Dr. Ricardo Pruna is a world-renowned expert in sport medicine and sports traumatology. Since 1997 he has been active in elite professional football, and he is the first team doctor of FC Barcelona. Last season (2013–2014), many of his athletes on the team suffered significant time loss due to ankle injuries.

He is also the author of numerous well-recognized publications within the field of football medicine and an active member in the world of sports medicine.

P.P.R.N. d'Hooghe (🖂)

G.M.M.J. Kerkhoffs Department of Orthopedic Surgery, Academic Medical Centre Amsterdam, University of Amsterdam, Meibergdreef 9, Amsterdam, AZ 1105, The Netherlands e-mail: g.m.kerkhoffs@amc.uva.nl

Department of Orthopaedic Surgery, Aspetar Hospital, Doha, Aspire Zone, Qatar e-mail: pieter.orthopedie@gmail.com



- Did your players experience many ankle problems last season? Really, lesional ankle incidents have not increased in recent seasons. The prevention programs in the team are demanding, and in recent years we have reduced the incidents specifically in the ankle. In any case, the most frequently affected pathologies are capsular impingements (chronic pathology) and syndesmosis (acute).
- Did you see an increase in ankle pathology related to the high number of competitions your players perform in? *No. we didn't observe more pathology specifically in the ankle..*
- Is it important for you to talk a lot with an injured player? Is your approach different for a player who has just come back from injury? *Obviously, empathy, knowing how to explain the injury, and paying attention from different angles without forgetting the emotion are crucial in dealing with an injured player. It is extremely important to be in the day-to-day beside the injured player; otherwise he might feel abandoned even by the medical team.*
- How do you manage medical secrets with TV and journalists watching your neck?
   The most important thing is to communicate well with the coach and the player.
   The press should be informed, but always under the umbrella of professional secrecy.
- How do you make the call on the pitch on the severity of a player's lesion? The physician should always impose his authority, otherwise there are daily arguments with players, and be very expeditious and clear in the orders given.
- Is the stress higher when treating million-dollar players medically? Of course, these players are a special group of people whose management takes great care. A single mistake can cost you the confidence of all the players.

#### 4 Interview - Ricardo Pruna

• What's your philosophy on injury prevention? Are club directors in general supportive on this topic?

Injury prevention should be daily and strictly attended to. My experience has led me to verify that a single physiotherapist should serve players for a particular type of pathology. Therefore, our philosophy is to have professionals specializing in each pathology in our medical services to prevent injuries. We have different areas of work: knee, ankle, hip, hamstrings ....

• How do you decide to send a player for further treatment and to who/where? Do you have a network?

Yes, it is very important to have a network of several professionals, depending on the pathology affected, to get complementary opinions. In my experience, it is not important where the best specialist is located. We have the philosophy to look for the best in the world in cases of severe and complicated injury, especially if surgery will be required.

- How do you treat a player when traveling abroad? We contact the medical network in the country before we travel in order to have everything prepared in case injuries occur. I had an intense experience with David Villa, who was, injured in Japan during the world team championship. We came with the player on the 14-h flight to Barcelona on a highly medicalized aircraft to serve the maximum tibia fracture he suffered during the championship tournament.
- Are the new generation of artificial grass, newer football shoe designs, and new training strategies beneficial to avoid ankle injuries in your opinion? I believe only the shoe companies are able to collaborate with this specific site (ankle). We have seen that five metatarsal stress fractures have decreased due to protection of this area provided by new shoe technology. Regarding the new generation of artificial turf, no differences are shown. Otherwise, our training and match procedures are always on natural grass.
- How do you form your opinion of new medical treatment strategies, and how do you communicate this with players who ask about it (e.g. PRP)? We are aware that new treatments have to be investigated to find out if they are effective for elite football players' injuries. Every time that a new technology appears in the medical world, we plan a meeting with players to explain clearly this option and possibilities. Growth factors are used in severe muscular injuries and in several injured ligaments in our club (like MCL, collateral ligament—ankle).

• Has the medical setup in your club changed in the last 10 years, and what do you think about the evolution seen? *Immensely. We are the first football club to achieve accreditation of FIFA. The* 

structure, hierarchies, working groups, and technological possibilities provided as well as the level of research and scientific publications have grown tremendously in the last 7 years.

- What constitutes a good football club doctor in your opinion? This is a complete definition: The team doctor has to meet various qualities, have empathy, be a great communicator, be able to make decisions quickly; they must be aware that their main function is to manage the healthcare structure while working in group network. They must have deep knowledge of soft tissue pathology and physiology of performance.
- Who will win the World Cup in Brazil 2014? *For me, the favorite national team is Brazil.*

# Chapter 5 Prevention of Ankle Injuries

Miriam van Reijen and Evert Verhagen

Some people say that Football is a matter of life and death. They are wrong since Football is far more important than that.

> Bill Shankly Ex-manager Liverpool F.C

**Abstract** Prevention of injuries is important for the health of an individual player, as well as for the overall performance of the team. Injured players do not play at their full physical capacity or may not be able to play at all. The majority of ankle injuries that occur in football result in complete inability to participate in training and/or competition. This chapter will discuss strategies for the prevention of ankle injuries in football. First, we will introduce a pragmatic approach towards injury prevention in general, after which we will discuss a number of general preventive measures to be employed in football. Thereafter, measures to prevent acute lateral ligament injury will be described in depth. These injuries are the most commonly encountered by football players and are the underlying cause of many of the other injuries described in this book. We will finish this chapter with general considerations regarding the implementation of prevention measures within a team sport as football.

Keywords Ankle • Prevention • Prophylaxis

# 5.1 Introduction

Prevention of injuries is important for the health of an individual player, as well as for the overall performance of the team. Injured players do not play at their full physical capacity or may not be able to play at all. The majority of ankle injuries that

M. van Reijen (🖂) • E. Verhagen

Department of Public and Occupational Health, EMGO+ Institute for Health and Care Research, VU University Medical Center, Amsterdam, The Netherlands occur in football result in complete inability to participate in training and/or competition. This chapter will discuss strategies for the prevention of ankle injuries in football. First, we will introduce a pragmatic approach towards injury prevention in general, after which we will discuss a number of general preventive measures to be employed in football. Thereafter, measures to prevent acute lateral ligament injury will be described in depth. These injuries are the most commonly encountered by football players and are the underlying cause of many of the other injuries described in this book. We will finish this chapter with general considerations regarding the implementation of prevention measures within a team sport as football.

# 5.2 A Pragmatic Approach Towards Injury Prevention

Effective prevention of ankle injuries does not stand by itself and requires a systematic approach [12]. The underlying factors that lead to injury to the ankle are multifactorial and may differ between players [10]. As such, optimal prevention would imply an individualised approach to tackle ruling risks and risk factors. However, from a practical approach – especially where it concerns a team sport as football – an individualised effort to prevent injury proves to be a challenge. At the elite level, an individual programme with detailed analysis of individual risk factors should be the preferred approach. For recreational footballers, a team-based approach might be more realistic. Yet, for both, the objective of prevention is identical: to take away the cause of injury and hence to prevent injury. Before we go into more detail of prevention programmes for ankle injury, we first introduce a theoretical framework for successful prevention.

Defining preventive measures and its potential consequences on a timeline allows for a step-to-step approach to prevention. The levels on which prevention can interfere in the presentation of an injury are as follows:

- (a) Primary prevention. Prevent the injury from occurring or reduce its impact. Primary prevention can include prevention or preventive measures without the existence of a prior injury.
- (b) Secondary prevention. Rapidly diagnose and treat the injury in order to reduce the clinical symptoms and severity of the injury. Secondary prevention is important to reduce the risk for reinjury. Accurate and rapid diagnosis and correct treatment are crucial for success in this phase.
- (c) *Tertiary prevention*. Limit the long-term consequences of the injury by promoting recovery. Tertiary prevention can be described as (long-term) rehabilitation.

These three prevention levels can also be placed in a so-called Haddon matrix. This matrix was initially modelled on motor-vehicle collisions but has been adapted to be applicable to sports-related injuries [2, 7]. This matrix summarises potential preventive hooks by presenting injury timing phases (pre-injury, crash and post-injury) in relation to levels of prevention (athlete, equipment and environment). The pre-injury phase consists of primary preventive efforts that ensure that the player

	Pre-event	Crash	Post-event
Athlete	Technique training Fair play		Rehabilitation
Environment	Playing surface		First aid given by medical staff
Equipment	Proper shoes	Tape/brace	

Table 5.1 Haddon's matrix applied to ankle sprains in football

will abstain from a harmful situation. The crash phase constitutes measures that will prevent the athlete from suffering an injury in case a harmful situation occurs. The post-event phase is made up of secondary and tertiary preventive measures that reduce the immediate- and long-term consequences of injury.

An example of a completed Haddon matrix for acute lateral ankle ligament injuries (ankle sprains) in football is presented in Table 5.1. As one can see in the table, it is not always possible or required to apply all the cells. Such a matrix can be applied for various specific injuries by coaches and medical staff of football teams. This aids in the establishment of multifaceted preventive approaches applicable to a team's or club's situation.

Ideally, one wants to fill the different cells in the Haddon matrix with evidencebased preventive measures, i.e. measures for which the preventive effects have been proven in scientific studies. However, this is not fully possible. The reason for this is simply being that measures to prevent sports injuries do not stand by themselves. They are part of what might be called the sequence of prevention [12] which has not been completed for the majority of preventive measures. First, the problem must be identified and described in terms of incidence and severity of injuries. Then, the factors and mechanisms that play a part in the occurrence of injuries have to be identified. The third step is to introduce measures that are likely to reduce the future risk and/or severity of injuries. Such measures should be based on the aetiologic factors and the mechanisms as identified in the second step. Finally, the effect of the measures must be evaluated by repeating the first step, which will lead to so-called time-trend analysis of injury patterns. However, from an epidemiological standpoint, it is preferable to evaluate the effect of preventive measures by means of a randomised controlled trial (RCT). Unfortunately, RCTs have only very rarely been conducted in sports injury prevention studies, and most of the RCTs that have been conducted so far in this area of research were carried out in settings other than football. As such, there is diminutive scientific evidence on the value of preventive measures. This holds especially true for less common and less severe injuries that - apart from methodological issues - have not yet gathered sufficient attention of the scientific sports medicine field.

Evidence should therefore not be the holy grail in effective injury prevention programmes but function as supplement to practical and clinical experience combined with in-depth knowledge of players' health status. The pragmatic preventive approach outlined above can aid the development and introduction of preventive measures when combined with personal expertise and the information on specific injuries provided in subsequent chapters.

# 5.3 General Preventive Measures

Historically, a number of general preventive measures have been proposed. Although for some there is ongoing debate on their true effectiveness and scientific validation for their effect on ankle injuries has not been reported, good practical results have been reported. As such, the measures presented below are recommended to keep players in good health.

# 5.3.1 Warm-Up and Stretching

To progress from rest to high-intensity exercise requires a transformation of the whole body including the muscles, tendons and blood vessels. Proper warm-up ensures a more gradual process and may add to injury prevention. The likely benefit of warm-up includes increased blood flow to the muscles, increased oxygen delivery to the muscles, increased nerve conduction, decreased stiffness of connective tissue leading to decrease risk of tears, increased cellular metabolism and increased range of motion [1]. With regard to football, the FIFA 11+ programme can be used as a complete warm-up protocol (Fig. 5.1). The strength of the 11+ programme is that in addition to providing a complete football-specific warm-up programme, it also includes injury-specific preventive exercises, including prevention of ankle injuries [6]. The 11+ programme has been shown to reduce injury risk at various levels of football play [11].

# 5.3.2 Appropriate Progressive Training Load

Training load is the combination of training duration, frequency and intensity. Each individual player has its own capacity depending upon, amongst others, overall health status as well as training and competitive cycle. In football, with a clear annual cycle, training load should be adjusted in such a way that there is room for adequate recovery between training sessions and matches. Periodisation, the variation in training load according to the season, should be aiming at peak performance during the end of the competitive season with a championship or final. There is general consensus that in order to improve performance, training load should gradually increase in either duration, frequency and intensity or a combination of these. However, when athletes are asked to perform at a greater intensity or to perform a greater volume of work, there is the risk of overload and consequently an increased risk for injuries. To avoid excessive overload, a number of principles should be considered:

- 1. Increase volume *or* intensity *or* frequency. Increases in frequency should precede increased volume which should precede increasing intensity.
- 2. Allow for adequate recovery in order to benefit fully from supercompensation the additional improvement in performance as a result of temporary overload.

	RUNNING     STRAIGHT AHEAD     Straight and a	No.	RUMAING     HIP OUT     The second control of a figure at the second control of a figure at the second control of the second co	+:+	RUNNING HIP IN Reactive interaction of the second second
M	CAUNING CRICLING PARTNER Another and the second se		BURNING     SHOULDER CONTACT      MOULDER CONTACT      Mould and a service of the service o	1	RUNNING QUICK FORWARDS & BACKWARI Network of the transmission of the second sec
	STRENGTH · PLYOMETRICS	BALAN	CE - 10 MINUTES		
-	TANKS 1 2 3 3 4 4 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5		THE BEINCH ALTERNATE LEGS ALTERNATE LEGS MARKENS IN AND AND AND AND AND AND AND AND AND AN	-	THE BENCH THE BENCH ONE LEG LIFT AND HOLD When the state is the state participation of the state of the st
	SDEWAYS BENCH STATE		SOCIAMENTS EENCH RAISE & LOWER HIP The second secon		BIDE WAY'S BENCH WITH LEG UPT The second sec
ų	HANSTRINGS EGUNER BARSTRINGS Description of the second sec	1	HAMSTRINGS INTERMEDIATE REPORT AND A Second	10	HAMSTRINGS ADVANCED The advance of the second secon
	SINGLE-LEG STANCE     HOLD THE BALL	#*	SINGLE-LEG STANKE THROWING BALL WITH PARTNER Description of the standard st		SINGLE-LEG STANCE TEST YOUR PARTNER THE YOUR PARTNER THE TOTOL AND
	11 SQUATS WITH TOE RAISE Descent of the second and	*	SQUATS WALKONG LUNGES Hanspace Into Art on our for a work of a year work of a year of the second sec	**	SQUATS ORE-LEG SQUATS COMPLETE SQUATS The second se
*	ADAPTING VERTICAL JUMPS VERTICAL JUMPS Description of the second second second second second means that the second second second second second second that is also be second seco	*	22 RUMPING EATERAL JUMPS Internet and the second se	*	12 AUMPING 10 X JUMPS The perfect of the set of the s
ART 3	RUNNING EXERCISES • 2 MI	NUTES			
1	BLANNING ACROSS THE PITCH	Å	RUNNING BOUNDING An advised to the same and state and to the the same and of an advised to the advised to the same and the same and the same and the same advised to the same advised to the same advised to the same same advised to the same advised to the same advised to the same same advised to the same advised to the same advised to the same same advised to the same advised to the sa	A	RUNNING PLANT & CUT

Fig. 5.1 FIFA F-MARC 11+ programme (Accessible through www.f-marc.com/11plus/home/. Accessed 20 July 2013)

Coaches/trainers should be willing to adjust training load according to the individual players' capacity.

3. Focus on accurate execution instead of fast execution when introducing new training activities.

- 4. Monitor the athlete closely for signs of overtraining such as decreased performance, fatigue and mood disturbances. This can be done by retrospective questionnaires, training diaries, physiological screening and direct observation.
- 5. Avoid excessive monotony of training.
- 6. Encourage and reinforce optimal nutrition, hydration and sleep.
- 7. Schedule regular health checks performed by a multidisciplinary team consisting of at least (sports) physicians, psychologists and nutritionists.
- Allow full recovery after illness and be aware of upper respiratory tract infections or other infectious episodes during which athletes should suspend from all training.
- 9. Take notice of personal matters of each individual players such as relational changes, alteration of lodgings or other potential stress factors.
- 10. Special care must be given to the introduction of high-intensity speed training, plyometric (jump) training and eccentric exercises as their explosive nature implies a high risk of injury.

# 5.3.3 Fair Play

Data from the FIFA World Cup 1998–2010 showed that foul play during competition is the cause of a large percentage of all injuries. The incidence of match injuries due to foul play during the World Cup was 23 % (2010), 61 % (2006) and 51 % (2002). This leads to the conclusion that stricter application of the laws of the game by referees will lead to a further reduction of injures [3]. In a precedent study, it was shown that during the three men's world soccer tournaments (the 2000 Olympics, the 1999 Under-17 World Championships and the 2001 Under-21 World Championships), the vast majority (72 out of 76) of injuries were due to players contact. Noteworthy is that significantly more injuries involved a tackle from the side (52 side, 18 behind, 6 front) and a tackle force from the lateral of medical direction (37 lateral, 31 medial, 4 posterior, 3 anterior, 1 not identified). Players who stay on their feet during the tackle are more likely to sustain an injury (43 stayed on feet, 28 sliding, 14 horizon-tal jumps and 1 vertical jump). In addition, ankle sprains are more likely to occur on a player's dominant side as this leg is more exposed to force inversion [5].

Initially, the FIFA 11+ consisted of 10 exercises. The 11th point added was meant to promote fair play. The FIFA actively encourages fair play as a means to reduce the incidence of injuries.

# 5.4 Ankle Sprain Prevention

An ankle sprain involves the lateral ankle ligaments and is the result of an acute inversion trauma, damaging the lateral ankle ligament complex. This injury is not only common in football but the most commonly encountered specific sports-related

injury in general. In contrast to other ankle injuries, ankle sprains have received much attention from a preventive standpoint. Also, although generally deemed a 'minor' injury, without proper care, an ankle sprain can develop into chronic complaints such as ankle impingement or osteochondral defects. The risk of such chronic complaints is especially high due to recurrent sprains that may lead to subsequent damage to the ankle joint. As such, primary and secondary prevention of ankle sprains can also be regarded as a preventive means to counteract other, more chronic, ankle injuries in football.

Prophylactic taping, braces, shoes (specially designed) and neuromuscular training (e.g. balance board training) have been postulated as preventive measures against ankle sprains. Multiple reports have been published in which the effects of each of these preventive measures have been studied. According to the literature – which varies in quality and quantity between different measures – the prevention of ankle sprains with external measures that limit ankle range of motion (i.e. brace or tape) is equally effective as the use of neuromuscular training. Both types of measures are linked to a 50 % reduction of the risk to sustain an ankle sprain. A preventive effect has been shown only for athletes with a previous injury [13].

## 5.4.1 Shoe Type

Few studies have examined the effect of shoes on the risk to sustain an ankle sprain, and studies have been conducted in basketball alone. No difference between low- and high-top shoes has been reported [13]. It has been suggested that it is the newness of shoes – irrelevant of shoe height – which has most effect on injury prevention. Newer shoes are hypothesised to be more rigid, providing stability to the ankle when in a vulnerable position. On the other hand, it can be postulated that the sole of a new shoe generates more shoe-surface friction. From a biomechanical standpoint, this may predispose to injury as inversion forces can be increased. Shoe-surface interaction may specifically play a role in football where improper cleats may affect ankle injury. Cleats that provide too much traction will not allow the player to react properly to an inversion movement as the foot is fixed. This may happen, for instance, during a misstep or when the player is being tackled while the foot is planted on the pitch. While different shoe manufacturers provide different cleat designs for various circumstances, it is not feasible to give a general guideline on cleat usage here. As such, it recommended to have players follow the manufacturers' recommendations.

# 5.4.2 Taping

Taping of the ankle is the earliest prophylactic measure used to prevent ankle sprains and arguably the most well-known and widely used preventive measure against these injuries. Although there is limited scientific evidence, taping has shown to effectively reduce the risk of injury in athletes with a history of ankle sprains. Taping is a form of strapping by which tape is attached to the skin in order to physically maintain a certain joint position. While tape loosens during play, part of its effectiveness comes from stimulation of additional nerve receptors on the ankle surface, allowing for earlier response to ankle inversion movements.

Over time, a large variety of taping methods have been developed. Additionally, many different types of athletic tape are manufactured, which can be divided into a nonelastic and an elastic variant. For standard ankle application, the tape of choice is 3.8 cm (1.50 in.) or 5.1 cm (2.01 in.) white porous athletic nonelastic tape.

The two most commonly used and widely accepted methods are the so-called basket weave method and the figure-eight method. Other taping methods as well as different variations are also applied. This illustrates one of the greatest benefits of taping, being that one can adjust the method of taping to the needs and preferences of the individual player. On the downside, taping is the most expensive preventive measure discussed in this chapter. Taping provides single-use prophylactic measures, and in one season, the material costs to tape the ankles of an entire football team can be substantial.

# 5.4.3 Bracing

The concept of ankle bracing evolved from ankle taping. Braces are currently being used instead of traditional taping by many athletes at all levels of competition. They offer several advantages; amongst others, they are self-applied, reusable and read-justable. In the long run, they are argued to be more cost-effective than taping. Also, there is much more evidence available of a preventive effect of bracing. Recent trials have even indicated a strong primary preventive effect of bracing [9].

Although braces are considered to have important advantages over tape, many players do not feel as comfortable or as stable wearing braces. More importantly for football, braces attach a bulk to the outside of the ankle which may compromise play. Braces have never been linked to a decrease in sporting performance, but results of studies have been derived using standardised exercise protocols that measure speed, agility and jumping ability. Ball handling in football while wearing a brace has not been the subject of research and may very well be compromised due to restricted ankle motion. Thereby, the advice to use braces in soccer may result in non-compliance, and it would be better to recommend the use of ankle tape.

Braces come in different basic varieties which may go by different names. In general, one can speak of the following:

- (a) *Ankle sleeves*, which do not provide stability but provide compression and can be used for prevention due to their positive proprioceptive (balance and positioning) effect.
- (b) *Non-rigid ankle braces*, which come in two basic types: (1) with an elastic sleeve with 'wrap-around' straps and (2) nylon, canvas or neoprene lace-up

brace with elastic wrap-around straps. Both types provide minimal stabilising effect and assist with proprioception. Although many of these braces are reported to be uncomfortable, there is an abundance of various types and fits available, making it possible for each player to find a suitable model.

(c) Semi-rigid braces, which are similar to the non-rigid versions, with the added feature of medial and lateral moulded plastic struts or air cushions. These braces are effective at stabilising the ankle in inversion (rolling out) and eversion (rolling in), but less effective when the athlete is in a dorsiflexed position (the most common mechanism of ankle sprain).

# 5.4.4 Balance Training

One measure that has been studied extensively in recent years is improvement of neuromuscular function and more specifically balance board (proprioceptive) training. Trauma to mechanoreceptors of the ankle ligaments after an ankle sprain can produce a proprioceptive impairment in the ankle. This might explain the increased risk of reinjury within 1 year after an ankle sprain. Neuromuscular training is designed for the rehabilitation after an ankle sprain and is thought to improve proprioception by re-establishing and strengthening the protective reflexes of the ankle. Thereby, neuromuscular training has the potential to be very effective measure in reducing the risk of injury recurrences. This potential effect has been shown in studies in a variety of different sports, all showing an injury risk reduction for players with previous ankle sprains. A basic proprioceptive training programme that can be embedded in regular football practice is described in Table 5.2.

## 5.4.5 Strength Training

Both range of motion (ROM) and strength deficits have been established in players who suffered an ankle injury [8]. Although no research is available on the effectiveness of strength training programmes to reduce the risk of ankle injury, these deficits suggest that strength and flexibility training may have a preventive effect. Individual functional performance testing should determine the presence and severity of strength and ROM deficits. Repetition of such testing at regular intervals may chart a player's progress after exercises are prescribed. Although there is no consensus on the exact training mode and intensity for optimal recovery, it is recommended that both open and closed chain exercise in a variety of modes should be performed [8]. For range of motion, especially ankle dorsiflexion should be targeted.

No material	Ball	Ball and balance board	Balance board
One-legged stance with flexed knee. Step out on the other foot with the knee flexed. Balance for 5 s. Repeat ten times for both legs	Make pairs and stand in one-legged stance with flexed knee. Keep a distance of 5 m (16.4 ft) and throw a ball five times while keeping balance. Repeat ten times for both legs	Make pairs. One stands with both feet on the balance board. Throw a ball ten times with one hand. Repeat two times for both players on the balance board	One-legged stance on the balance board with the knee flexed. Maintain balance for 30 s and change stance leg. Repeat two times for both legs
Variations A–D	Variations A, B		Variations A-D
One-legged stance with hip and knee flexed. Step out on the other foot with the hip and knee flexed. Balance for 5 s. Repeat ten times for both legs	Make pairs and stand in one-legged stance with the hip and knee flexed. Keep a distance of 5 m (16.4 ft) and throw a ball five times while keeping balance. Repeat ten times for both legs	Make pairs. One stands in one- legged stance with flexed knee. Throw a ball ten times with one hand. Repeat two times for both players and both legs on the balance board	One-legged stance on the balance board with the hip and knee flexed. Maintain balance for half a minute and change stance leg. Repeat two times for both legs
variations A–D	variations A, D	Variations A, B	Variations A–D
Instructions Choose 1 exercise p Each category (no r balance board, bal chosen once per w Choose variations tr challenging Variation A: stretch Variation B: flex the bearing leg Variation C: stretch	ber warm-up session naterial, ball, ball and ance board) may only be eek o make exercises more the weight-bearing leg e knee of the weight- n the weight-bearing leg	Make pairs. One stands in one- legged stance with flexed hip and knee. Throw a ball ten times with one hand. Repeat two times for both players and both legs on the balance board	Move slowly over the balance board with one leg while the other foot is on the balance board. Repeat ten times for both legs
and keep eyes clos Variation D: flex th bearing leg and ke	ed e knee of the weight- ep eyes closed	Variations A, B	Stand with both feet on the balance board and perform knee flexions. Repeat ten times

 Table 5.2 Neuromuscular training progreblamme for football warm-up

Adapted from Verhagen et al. [15]

# 5.5 Prevention of Ankle Injury in Practice: Additional Considerations

In order to effectively reduce the risk of an ankle injury, the compliance to introduced measures is of utmost importance. Especially in preventive measures that require active participation, such as the neuromuscular training programme, programme adherence is the key to success or failure [14]. Such programmes are only effective when players are exposed to a critical dose of exercises. This is very much equal to the acquisition of a cardiovascular training effect: one will only achieve better cardiovascular fitness when exposed to a substantial amount of endurance training.

However, the introduction of preventive measures implies a modification of sporting behaviour which may be in conflict with the ruling behaviour. Simply put, players and coaches do not like to change their regular routine. This is not unique to football, but rather a generic issue surrounding the introduction of preventive measures in sports. As such, it is crucial to determine which preventive measures will work in each individual setting. It is important that there is effective communication between players, coaches and medical staff to get a feel of how they perceive the problem that needs to be solved and what solutions they believe in or apply, as these are usually the solutions they will comply to.

Having said so, we do recommend the following to prevent ankle injury in football. Earlier in this chapter, we already mentioned the FIFA F-MARC 11+ warmup programme. The 11+ is a multifaceted preventive programme that has been developed especially for football by the FIFA Medical Assessment and Research Centre (F-MARC, www.f-marc.com). It includes exercises to develop core stability, strength, balance and agility. A fair play component is also included. Figure 5.1 depicts the complete FIFA 11+ programme, which has been designed as full warmup programme aimed to reduce injury risk in male and female football players aged 14 years and older. Most of the recommendations made in this chapter on ankle injury prevention are also embedded in the 11+ programme, e.g. balance, coordination, strength, endurance and fair play. It is highly recommended for all football coaches to replace regular warm-up with the 11+ programme.

Also, additional preventive measures may be implemented for players who are at increased risk and thus require special measures. This goes especially for the subgroup with a history of previous injury or episodes of ankle instability [4]. In this subgroup, subsequent reinjury risk is highly dependent on the type of rehabilitation employed, whether or not the subject complied with the rehabilitation programme, and the quality of recovery. From this perspective, structured rehabilitation programmes that include restoration of normal ankle motion, strengthening and restoration of neuromuscular control and proprioception of the ankle complex should be advocated to all injured players. Until function is completely normal, players should be urged to make use of either a tape or a brace.

## References

- Bahr R (2007) Principles of injury prevention. In: Brukner P, Khan K (eds) Clinical Sports Medicine, 4th edn. McGraw-Hill, Sydney, pp 113–137
- Bahr R, Krosshaug T (2005) Understanding injury mechanism; a key component of preventing injuries in sport. Br J Sports Med 39(6):324–329
- Dvorak J, Junge A, Derman W, Schwellnus M (2011) Injuries and illnesses of football players during the 2010 FIFA World Cup. Br J Sports Med 45:626–630
- Fong DT-P, Hong Y, Chan L-K, Yung PS-H, Chan KM (2007) A systematic review on ankle injury and ankle sprain in sports. Sports Med 37(1):73–94

- Giza E, Fuller C, Junge A, Dvorak J (2003) Mechanism of foot and ankle injuries in soccer. Am J Sports Med 3(4):550–554
- Grooms DR, Palmer T, Onate JA, Myer G, Grindstaff T (2013) Comprehensive Soccer-specific warm-up and lower extremity injury in collegiate male soccer players. J Athl Train 48:782–789
- Haddon W (1972) A logical framework for categorizing highway safety phenomena and activity. J Traum 12:193–207
- Kaminski TW, Hertel J, Amendola N, Dochtery CL, Dolan MG, Hopkins JT, Nussbaum E, Poppy W, Richie D (2013) National athletic trainers' association position statement: conservative management and prevention of ankle sprains in athletes. J Athl Train 48(4):528–545
- 9. McGuine TA, Brooks A, Hetzel S (2011) The effect of lace-up ankle braces on injury rates in high school basketball players. Am J Sports Med 39(9):1840–1848
- 10. Meeuwisse WH, Tyreman H, Hagel B, Emery CA (2007) Dynamic model of etiology in sport injury: the recursive nature of risk and causation. Clin J Sports Med 17:215–291
- 11. Soligard T, Myklebust G, Steffen K, Holme I, Silvers H, Bizzini M, Junge A, Dvorak J, Bahr R, Andersen TE (2008) Comprehensive warm-up programme to prevent injuries in young female footballers: cluster randomised controlled trial. BMJ 37(dec09 2):a2469–9
- 12. Van Mechelen W, Hlobil H, Kemper HC (1992) Incidence, severity, aetiology and prevention of sports injuries. A review of concepts. Sports Med 14(2):82–99
- 13. Verhagen EALM, Bay K (2010) Optimising ankle sprain prevention: a critical review and practical appraisal of the literature. Br J Sports Med 44(15):1082–1088
- Verhagen EALM, Hupperets MDW, Finch CF, van Mechelen W (2011) The impact of adherence on sports injury prevention effect estimates in RCTs: looking beyond the CONSORT statement. J Sci Med Sports 14(4):287–292
- 15. Verhagen EALM, van der Beek AJ, Twisk J, Bouter L, Bahr R, van Mechelen W (2004) The effect of a proprioceptive balance board training program for the prevention of ankle sprains: a prospective controlled trial. Am J Sports Med 32(6):1385–1393

# Chapter 6 Interview – Frank de Bleeckere

Pieter P.R.N. d'Hooghe and Gino M.M.J. Kerkhoffs

**Abstract** Frank de Bleeckere is a Belgian international elite football referee, born in 1966.

He has been a referee since 1984 till 2012 and an international (FIFA) official since 1998 till 2012. De Bleeckere was refereeing in his first World Cup finals in Germany 2008 and had to pass a late fitness test following injury. He has been appointed to referee elite football matches in European and international competitions for three decades and is accredited to be one of the world's best referees of his generation.

P.P.R.N. d'Hooghe, MD ()

G.M.M.J. Kerkhoffs, MD, PhD Department of Orthopedic Surgery, Academic Medical Centre Amsterdam, University of Amsterdam, Meibergdreef 9, Amsterdam, 1105 AZ, The Netherlands e-mail: g.m.kerkhoffs@amc.uva.nl

P.P.R.N. d'Hooghe, G.M.M.J. Kerkhoffs (eds.), *The Ankle in Football*, Sports and Traumatology, DOI 10.1007/978-2-8178-0523-8\_6, © Springer-Verlag France 2014

Department of Orthopaedic Surgery, Aspetar Hospital, Aspire Zone, Doha, Qatar e-mail: pieter.orthopedie@gmail.com



• How did you experience medical evolution of refereeing in football over the years?

Medical evolution has progressed significantly during the last decades. Especially in refereeing, there has been a huge increase of awareness towards injury prevention. This is an important topic for referees since we are generally 15 years older than the players. During a game, we normally run backwards for about 2 km, and because this puts a high load on our Achilles tendons, injury prevention is a hot topic for referees nowadays.

• Sport-specific ankle lesions are very frequent in football refereeing due to the high amount of backward running. Is that a topic when you prepare for big tournaments with your colleagues?

Yes, sure this is a topic for me and my colleagues. Backward running overloads the tendons quickly; therefore, the choice of shoes is very important. Personally, I always used my "running shoes" during my games. It offers you more energy absorption on the heels and consequently unloads the Achilles tendon also. This is in contrast with regular football shoes where you can see less absorption and supportive areas.

• Did you suffer any significant ankle-related time-loss injuries in your career of elite refereeing (Achilles tendon)?

The last 2 years in my international career, I suffered from time-loss Achilles tendinopathy injuries. This was mainly overuse – triggered due to the high load

of games in combination with my daily workouts. Laser therapy helped me somewhat during that period, and it kept me going for a while and train without significant pain. Although, thanks to my injury-preventative program before and after training, I never suffered from severe surgery-related injuries, I was lucky to have a professional team around me consisting of performance professor Werner Helsen (Leuven University) and my loyal physiotherapist Bernard van de Velde (physiotherapist Belgian National Team).

• Do you think you are performing enough injury prevention strategies for your ankle in your daily workout?

I am personally convinced that injury prevention attention led me to continue until my 45 years of age (which is the maximum age to allow international refereeing). Treatment, rest, and expert follow-up on all levels helped me to reach the international level and stay there for all these years. A referee is nowadays expected to be a top sport athlete, and that requires balance and a lot of discipline to get to the international level.

• Do you think the newer football shoe designs are improved compared to previous designs?

More and more big companies have taken interest to create a referee football shoe. I personally took part in a Belgian project in order to develop the referee football shoe with ultimately great success. Since the demands on the feet in refereeing are significantly different to that of the football player, the "referee shoe" should be tailored to the specific needs.

• Do you fear joint problems in your ankle after your career?

I quit refereeing now for 2 years, and my Achilles tendons do well. I train three times a week still, and no more inflammations are present. Still, my training load is totally different now: I changed my interval training towards endurance (10 K) training. This change in training has shifted my workout perception from "necessary" to "fun."

*My previous ankle issues have completely resolved by training less on interval and frequency.* 

• Who will win the World Cup in Brazil 2014?

That's probably the trickiest question of all! Of course, my team is the Belgian Red Devils. They have a strong generation of players and attitude. Although I honestly believe that Brazil comes too early for them and that we will enjoy them more on the Euro 2016 stage, Belgium has potential to win in Brazil so let's stick to that.

# Chapter 7 Ankle Ligament Lesions

Gino M.M.J. Kerkhoffs, Peter A.J. de Leeuw, Joshua N. Tennant, and Annunziato Amendola

You only stop learning about Football when you quit.

Ruud Gullit

**Abstract** This chapter describes injury to the ankle ligaments, including the lateral ankle ligaments, the syndesmotic ligaments and the deltoid ligaments. All aspects on diagnosis, clinical appearances and therapeutic treatment options are highlighted, specified if possible for the footballer.

Keywords Ankle • Lateral ligaments • Deltoid ligaments • Syndesmosis

# 7.1 Introduction Including Epidemiology

The majority of ankle sprains occur in individuals under 35 years of age, most commonly in those aged 15–19 years. They account for up to 40 % of all athletic injuries, and 29 % of football injuries can be attributed to ankle injuries, and 12 % of time lost in football is due to ankle injuries. Three-quarters of ankle injuries involve the lateral ligamentous complex; there is conflicting evidence as to whether females

G.M.M.J. Kerkhoffs, MD, PhD (>) • P.A.J. de Leeuw, MD

Department of Orthopaedic Surgery, Academic Medical Center, University of Amsterdam, PO Box 22700, Amsterdam 1100 DE, The Netherlands e-mail: g.m.kerkhoffs@amc.nl; p.a.deleeuw@amc.nl

J.N. Tennant, MD, MPH Department of Orthopaedic Surgery, University of North Carolina, Chapel Hill, NC 27599-7055, USA e-mail: josh\_tennant@med.unc.edu

A. Amendola, MD Department of Orthopaedics and Rehabilitation, University of Iowa Hospitals and Clinics, 200 Hawkins Drive, Iowa City, IA 52242, USA e-mail: ned-amendola@uiowa.edu are more prone to these injuries when compared to males [3]. Without adequate diagnosis and treatment, ankle injuries may lead to chronic instability, osteoarthritis and other permanent sequelae. Football players have significantly greater load and demand on their ankle joints than the average population, and in addition sports performed at the highest level are possibly an unfavourable prognostic factor for the development of residual complaints.

Syndesmotic injuries can be associated with prolonged pain, disability and an unpredictable time away from the pitch or sports in general. The incidence ranges from 1 to 18 % of ankle ligament sprains [19]. Syndesmosis involvement following lateral ankle ligament injury was the most predictive factor of chronic ankle dysfunction at 6 months post-injury [11] and then becomes chronic by definition. Also patients take twice as long to return to sports if associated syndesmosis injury is present as compared to isolated lateral ligament sprains.

These injuries may occur in isolation or in association with other ligamentous, bony or cartilaginous injuries around the ankle. Missed and chronically unstable injuries may lead to osteoarthritis. It is likely that this is an underestimate as these injuries are frequently missed or undertreated.

## 7.1.1 Deltoid Ligaments

Isolated deltoid ligament injuries are a rare occurrence, representing 3-4 % of all ankle sprains [3, 11]. Lesions of the deltoid ligament may present as acute, chronic or concomitant injuries involving other ligamentous or bony injuries. Medial-sided ankle injuries may be associated with syndesmosis injury, but those isolated to the medial side may also involve the tibialis posterior tendon and spring ligament complex.

## 7.2 Functional Anatomy

The ligaments around the ankle can be divided in groups depending on their location and function; from lateral to medial, the first group consists of the ligaments that form the tibiofibular syndesmosis and the second group the lateral ligaments, and the third group located on the medial side is formed by the deltoid ligaments.

The ankle joint can be regarded as a fork, at one side formed by the distal tibia and at the other side the fibula. In between this fork, the talus is orientated. The tibiofibular syndesmotic ligaments ensure that the fork can resist axial, rotational and translational forces which tend to distend the distal tibia and fibula. The syndesmotic ligament complex is formed by the anterior tibiofibular ligament, the posterior tibiofibular ligament and the interosseous tibiofibular ligament. The anterior tibiofibular ligament originates in the anterior tubercle of the tibia and inserts in the anterior margin of the lateral malleolus. Following a lateral ligament injury, the most distal fibres will have an increased contact pressure with the anterolateral talus due to the increased anterior to posterior talar laxity. Subsequent fraying of the anterior tibiofibular ligament could cause anterolateral soft tissue impingement secondary to an inversion injury. The posterior tibiofibular ligament is formed by two independent bands: the superficial and deep component, also known as the transverse ligament. The superficial component originates at the posterior edge of the lateral malleolus and directs proximally and medially to insert in the posterior tibial tubercle. This component would be homologous to the anterior tibiofibular ligament. The deep component originates in the proximal area of the malleolar fossa to insert in the posterior edge of the tibia and is responsible for ankle joint stability and also to prevent posterior talar translation [12]. The interosseous tibiofibular consists of a mass of short fibres connecting the distal fibula and tibia and can be regarded as a continuation of the interosseous membrane. Its exact function remains unclear; some investigators have suggested that the interosseous ligament is mechanically insignificant, whereas others consider it the primary bond between the tibia and fibula.

The lateral ligament complex of the ankle is formed by three ligaments: the anterior talofibular ligament (ATFL), the posterotalofibular ligament (PTFL) and the calcaneofibular ligament (CFL). The ATFL originates at the distal part of the anterior fibula to insert in the talar body. This ligament is mainly responsible to prevent anterior translation of the talus. In the neutral ankle position, it is orientated horizontally with respect to the anterior ankle joint line. Ankle plantar flexion results in tensioning of this ligament. This lateral ligament is the most frequently damaged ligament in an inversion injury [4]. Just distal to the origin of the ATFL, the CFL originates. In the neutral ankle position, the ligament runs in an oblique orientation to insert in the lateral surface of the calcaneus. In the valgus ankle position, the ligament is relaxed, whereas in varus the ligament is in tension and could potentially be damaged. Isolated injuries to the CFL are very rare; nevertheless, combined injuries to both the ATFL and the CFL account for up to 20 % of the ankle sprains. The PTFL originates from the posterior and distal part of the lateral malleolus to run almost horizontally to insert in the posterolateral talar process. This ligament is tensed in ankle dorsiflexion and is relaxed in the neutral and in the plantar flexion position. Therefore, this ligament is usually not damaged in inversion injuries.

## 7.2.1 Deltoid Ligaments

The deltoid ligament has deep and superficial layers, each consisting of multiple components. Milner and Soames [27] described the superficial layer with four components, each originating from the anterior colliculus of the medial malleolus: the tibiospring ligament inserts on the superomedial band of the calcaneonavicular ligament; the tibiocalcaneal ligament inserts on the dorsomedial surface of the navicular; the tibiocalcaneal ligament inserts on the sustentaculum tali; and the tibiotalar ligament inserts at the medial talar tubercle. The deep layer of the deltoid only spans the ankle joint. A small anterior component of the deep layer originates from the anterior colliculus and inserts on the medial talus. The larger and stronger posterior deep layer originates from the intercollicular groove and the posterior colliculus and inserts on the medial talus. Not all anatomic components are constant, and other investigators have classified the distinct ligaments and layers with slight variation [2].

The deltoid ligament as a whole functions to resist talar abduction within the mortise, although the discreet functions of individual components of the deltoid complex have been variably described in the literature. The tibiocalcaneal portion of the superficial deltoid is the strongest check to talar eversion. The deep deltoid serves as a secondary restraint to lateral and anterior displacement of the talus, with the lateral ligaments and fibula acting as a primary restraint [13].

## 7.3 Aetiology and Injury Mechanism

### 7.3.1 Tibiofibular Syndesmosis

Distal tibiofibular syndesmotic injuries most commonly are caused by pronationexternal rotation, pronation-abduction and infrequently the supination-external rotation mechanism. In the patients with an ankle fracture following the pronationexternal mechanism (Lauge-Hansen), also known as the Weber C ankle fractures, damage to the syndesmotic ankle ligaments is present by definition. Isolated total ruptures of the syndesmosis are relatively rare but are reported in football as well as in American football and skiing [36].

# 7.3.2 Lateral Ankle Ligaments

Understanding the injury mechanism is important for optimising treatment, injury prevention and research goals. A twisting injury or 'going over on the ankle' usually results in an inversion of the foot and ankle. The most common trauma mechanism is supination and adduction (inversion) of the plantar-flexed foot. Sometimes, there is also an external rotation of the lower leg with respect to the ankle joint [14]. Inversion injuries of the plantar-flexed foot result in ATFL injury because the ligament is tight in this position. The majority of ankle sprains during football were sustained during player contact (59 %) except for goalkeepers in whom 79 % occurred during noncontact situations [37]. Andersen et al. analysed the ankle supination sprain injury with video and reported that there were two major mechanisms: (1) impact by opponent on the medial aspect of the leg just before or at foot strike, resulting in a laterally directed force causing the player to land with the ankle in a vulnerable inverted position, and (2) forced plantar flexion when the injured player hits the opponent's foot when attempting to shoot or clear the ball.

## 7.3.3 Deltoid Ligaments

Deltoid ligament injury is most commonly associated with other injury patterns, including the syndesmosis, lateral ligament ankle sprains and rotational ankle

fractures. In the rare cases of isolated deltoid injury, eversion, external rotation or abduction mechanism may be recalled by the patient. Common sporting activities involved in deltoid ligament injuries include soccer, American football, basketball, long and triple jump and dancing [30].

Considerable force is required to injure the deltoid ligament, which has a higher load to failure than the lateral ligaments. The posterior tibiotalar component of the deep deltoid typically ruptures near its talar insertion. The posterior inferior tibiofibular ligament of the syndesmosis is the only ligamentous structure about the ankle with a higher load to failure [1].

# 7.4 Clinical Examination

### 7.4.1 Tibiofibular Syndesmosis

In the acute setting, a patient presenting with ankle pain following a twisting injury, a fracture with possible subsequent syndesmotic ligament injury must be ruled out; see also the chapter on ankle fractures in this book. In the acute setting, a high percentage of the 'normal' ankle sprain patients also presents with pain at the area of anterior distal tibiofibular ligament; therefore, it is difficult to distinguish a lateral ankle ligament rupture from a tibiofibular ligament injury solely on (delayed) physical examination.

In patients with persistent ankle pain following a healed ankle fracture or following a severe ankle sprain, instability of the syndesmosis must be ruled out. Patients typically have a sensation of giving way and difficulty to walk on uneven grounds, and also difficulty 'pushing' off when running is a common presentation. On clinical examination, stiffness, limited dorsiflexion and sometimes swelling in the anterolateral aspect, just proximal to the ankle joint, can be observed. A recent systematic review on eight different tests, all aimed to reproduce symptoms by applying stress to the syndesmosis, shows that not a single test can accurately diagnose syndesmotic ligament injuries. However, we feel that in experienced hands, a combination of a positive squeeze test, a positive fibular translation test and local palpation pain—that is, recognisable for the patient—is highly specific for the presence of chronic syndesmotic injury. However, we advocate that additional diagnostics should be used in case of a clinical suspicion [32].

# 7.4.2 Lateral Ankle Ligaments

After a supination trauma, it is important to distinguish a simple distortion from an acute ankle ligament rupture because adequate treatment is associated with a better prognosis [35]. A player with a simple distortion can be back on the pitch in just a few days; a complete rupture however needs serious treatment to prevent chronic problems later on in the career. If a haematoma is present accompanied by local

pressure pain at palpation or a positive anterior drawer test is present or both, it is most likely that a (partial) lateral ankle ligament rupture exists. Delayed physical diagnostic examination (4–5 days) gives a better diagnostic result than the same examination done within 48 h after the initial trauma. The sensitivity to correctly diagnose an acute lateral ligament rupture during a delayed physical examination is 96 % with a specificity of 84 % [35].

## 7.4.3 Deltoid Ligaments

A patient with suspected acute deltoid ligament injury presents with tenderness, swelling and often ecchymosis at the medial ankle, although none of these are absolutely predictive [34]. A feeling of instability may be described on the medial side. Chronic injuries may have pain in the medial gutter, especially with palpation of the anterior medial malleolus, as well as a progressive hindfoot valgus deformity. Posterior tibial dysfunction contributing to the deformity can be excluded by a reconstitution of the arch of the foot when the patient is asked to go onto his or her toes.

With the patient sitting and the feet hanging free, stability testing is performed. Anterior drawer stress as well as varus and valgus tilt stress testing of the ankle is compared to the contralateral side.

## 7.5 Diagnostic Imaging

## 7.5.1 Tibiofibular Syndesmosis

A mortise or AP and lateral ankle weight-bearing radiograph should be used to evaluate the tibiofibular clear space, tibiofibular overlap and medial clear space and to rule out any ankle fracture. Also stress radiographs are recommended. With these 'conventional' additional diagnostics, still syndesmotic ligament injuries can easily be missed. For this reason, more advanced imaging such as magnetic resonance imaging (MRI) is recommended, which has been reported to have a sensitivity and specificity of respectively 95 and 90 % [3] (Fig. 7.1).

# 7.5.2 Lateral Ankle Ligaments

The Ottawa ankle rules have been developed to rule out fractures after acute ankle injuries in the emergency setting, within the first week after acute ankle injury. Weight-bearing radiographs should be made in case the Ottawa ankle rules are

#### 7 Ankle Ligament Lesions

**Fig. 7.1** Axial T1 MR image of a left ankle indicating the presence of a rupture of the anterior tibiofibular ligament (\*)



positive and there subsequently is a 'high' suspicion of an ankle fracture following a lateral ankle injury. Stress radiographs can demonstrate the degree of laxity in the ankle joint but are difficult to perform for acute ankle sprains because of patient pain, oedema and muscle spasms, and this is therefore not advised. Ultrasound and MRI can be useful in diagnosing associated injury (bone, chondral or tendon) and are routine investigations in professional athletes. Ultrasound has been demonstrated to be an accurate investigation that leads to little discomfort in patients but requires technical expertise, and the data may be difficult to interpret on retrospective review by other physicians. The sensitivity and specificity of ultrasound investigation for a ligament rupture are 92 and 64 %, respectively. The predictive value of a positive ultrasound investigation is 85 % and of a negative ultrasound investigation 77 % [35]. Ultrasound is accurate in demonstrating the presence of joint effusion. MRI is reliable in the diagnosis of acute ligamentous ankle injuries (Fig. 7.2), evaluation of tendon disorders, occult fractures and osteochondral lesions, although the last can be diagnosed more reliably with a computed tomography (CT) scan.

For chronic ankle instability, the lateral instability/laxity test (talar tilt) and the anterior instability/laxity test (anterior talar translation) are the two radiographic tests that can be used. Increased laxity can be defined as anterior talar translation of

**Fig. 7.2** Axial T1 MR image of a right ankle indicating the presence of a rupture of the anterior talofibular ligament (\*)



more than 10 mm or talar tilt of more than  $9^{\circ}$ . Another way of defining increased laxity is a difference in anterior talar translation between the functionally unstable ankle and the contralateral ankle of more than 3 mm or a talar tilt of more than  $3^{\circ}$  in patients who have unilateral instability.

# 7.5.3 Deltoid Ligaments

Similar to evaluation of other ankle ligamentous injuries, plain radiographs of the ankle are the initial choice for imaging to assess suspected injuries of the deltoid ligament. In addition to the AP, mortise and lateral weight-bearing views, a hindfoot alignment view [31] is important, particularly in the setting of chronic injury when a planovalgus deformity may be present.

In the setting of an acute injury, particularly rotational ankle fractures, stress radiographs are helpful to assess the competency of the deltoid ligament [33]. With fractures of the lateral malleolus or syndesmotic injury, medial clear space widening of the mortise may be evident. With an isolated deltoid ligament injury, no widening

#### 7 Ankle Ligament Lesions



**Fig. 7.3** (a) T2 weighted MRI coronal image showing normal deltoid ligament, and (b) same image from a patient with an acute deltoid injury interposed into the medial gutter of the ankle from an associate rotational ankle injury

at the medial mortise will occur. A radiographic stress examination using both varus and external rotation may help detect occult deep deltoid ligament injuries [10].

Ultrasound has been used to evaluate the deltoid ligament in supination-external rotation ankle fractures as a means of assessing stability of the fracture pattern, with sensitivity and specificity of 100 % in small case series [5]. CT scan is a useful modality if injury to the articular surface, avulsion fractures or abnormal osseous anatomy, such as an underlying talocalcaneal coalition, are suspected. MRI may also be able to show injury to individual components of the deltoid ligament [6], although the benefit of the additional detail should be measured against the increased expense of the study (Fig. 7.3).

# 7.6 Classification

# 7.6.1 Tibiofibular Syndesmosis

A differentiation must be made between functional and mechanical instability. Functional instability refers to the subjective patient symptoms, whereas mechanical instability refers to objective parameters based on both clinical and radiological criteria. The West Point Ankle Grading System is widely used and is based on the clinical examination of the syndesmosis and divides pathology into three grades. Grade I represents a mild sprain/tear to the anteroinferior tibiofibular ligament without instability, and grade III represents definite instability with complete rupturing of all the ligaments which form the syndesmosis. Grade II is somewhat in between these severities with some instability with a tear of the anteroinferior tibiofibular ligament and a partial tear of the interosseous ligament [11].

# 7.6.2 Lateral Ankle Ligaments

There are several grading and staging systems for lateral ankle ligament injuries based on anatomic injury, clinical symptoms, trauma mechanism, stability and 'severity' of the injury. A classification is only relevant when it has consequences for treatment of prognosis. Grade I injuries include stretching of the ligament without a macroscopic rupture. There is a minor swelling and tenderness on palpation without increased laxity. Grade II injuries include partial ruptures of the ligaments, with moderate swelling, tenderness and pain. There is a mild to moderate increase in laxity, some loss of motion and moderate functional disability. In grade III injuries, a complete rupture of the ligaments and the joint capsule are present with severe bruising, swelling and pain. There is a major loss of function and an increased laxity. The patient is most frequently unable to bear weight. In clinical practice, only the difference between a simple sprain (grade I) and real instability (grade II or III) is relevant because only grades II and III require an additional treatment.

# 7.6.3 Deltoid Ligaments

A classification of deltoid ligament injury based on anatomic location has been proposed by Hintermann [16], divided into three types. Type I is a proximal avulsion involving the tibionavicular or tibiospring ligament. A Type II lesion is an intermediate tear in which the deep deltoid remains attached distally and the superficial deltoid remains attached proximally, again involving the tibionavicular and tibiospring. Lastly, the Type III injury involves a distal tear or avulsion of the tibionavicular and spring ligaments.

## 7.7 Treatment and Rehabilitation

## 7.7.1 Tibiofibular Syndesmosis

For grade I sprains without instability and only partial disruption of the AITFL, the maximum management entails immediate rest, ice and immobilisation in a pressure

bandage, or compression-cooling system is advocated, and rehabilitation can be initiated with players' pain complaints as a guide. If the ankle is very painful, because of concomitant injuries, immobilisation in a cast or non-weight-bearing boot for 5–7 days can be considered to allow the acute inflammation and swelling to subside. Partial weight bearing commences when pain complaints have subsided or approximately 7–14 days post-injury as tolerated, and active-assisted physiotherapy concentrating on range of motion and light proprioception exercises is instituted. Progression to the next phase, also with players' complaints as the guide. Full weight bearing as tolerated, strength training and proprioception are emphasised, and thereafter sport-specific functional exercises are started—toe standing and light running are commenced initially, increasing to toe–toe running and single-leg hopping. A sign of a healing syndesmosis is the ability to repeatedly single-leg hop. Return to sporting activity is permitted when able to single-leg hop for 30 s without significant pain. The time to pain-free full recovery is variable.

Grade II injuries are varied, and decision-making can be difficult. The recreational football player without diastasis can be treated nonoperatively as discussed above with good results. Prolonged time to return to sports is expected, and patients must be told this. For the professional player with a grade II injury and clinical suspicion of dynamic instability, we recommend an examination under anaesthesia (EUA) and arthroscopy with assessment of the syndesmosis. Dynamic diastasis of 2 mm or more warrants fixation. Post-operative CT scanning is indicated in some cases where an accurate reduction is a concern.

Grade III injuries are uncommon in the professional football player and are often associated with other injuries around the ankle. An arthroscopy prior to fixation can identify intra-articular pathology and address this pathology if present (Fig. 7.4). If the syndesmosis is grossly unstable, two screw fixation or two tightropes will stabilise the ankle. Post-operatively, the ankle is treated in a non-weight-bearing splint for 10 days to 2 weeks allowing wound healing and resolution of inflammation. Range of motion is regained first with early proprioception training and partial weight bearing at 3–4 weeks post-op. Full weight bearing is commenced at 4 weeks as tolerated and strength training continued. Return to running and high-impact activity is commenced after 8 weeks as dictated by rehabilitation progress and resolution of pain [26]. Screw removal is optional and depends grossly on the surgeons' preference.

Chronic syndesmosis injuries usually do not respond to a conservative treatment method and require a surgical intervention. These surgical interventions include arthroscopic debridement, screw fixation, anatomic reconstruction of the syndesmotic ligaments or an arthrodesis. A recent meta-analysis found that none of these surgical treatment modalities is superior, with a success rate for screw fixation, arthrodesis and arthroscopic debridement of 88, 80 and 78 %, respectively [28]. Most studies published used screw fixation as surgical intervention. Noteworthy is that the rates of success of this procedure are not significantly affected by gender, age, average follow-up, mean duration of symptoms or the method in which the diagnosis was initially set [28]. We feel that in cases where syndesmotic pain rather than functional syndesmotic instability is the key complaint (syndesmotic impingement), an arthroscopic debridement can be successful as sole treatment. In all other



Fig. 7.4 (a) Anterior arthroscopic image of a right ankle indicating this might be an intact syndesmosis. (b) On ankle dorsiflexion in the same patient the syndesmosis clearly widens, indicating its instability

cases, an arthroscopy is useful to inspect the ankle joint and to treat concomitant intra-articular pathology, but surgery should really consist of a ligament reconstruction technique or an arthrodesis of the distal tibiofibular joint.

## 7.7.2 Lateral Ankle Ligaments

For over many years, there is a wide spread debate concerning the optimal treatment of acute lateral ligament injuries. Recently, the current best evidence was formulated in a guideline [23], consisting of recommendations based on formerly published research. The quality of included articles was assessed by epidemiologists on the basis of 'evidence-based guideline development' assessment forms (EBRO) and classified in order of probative and scientific value [23]. This guideline was then used by experts in the field of sports for a debate on the best treatment for acute lateral ligament injuries in athletes.

A patient suffering from an acute lateral ankle ligament injury benefits from the use of the so-called RICE (rest, ice [cryotherapy], compression and elevation) [23]; also, the use of nonsteroidal anti-inflammatory drugs is recommended in the acute phase of recovery. Manual mobilisations of the ankle must be discouraged. More than 2 weeks of immobilisation in a lower-leg cast is not an effective treatment strategy. However, a short period of plaster immobilisation or similar rigid support could well facilitate a rapid decrease of pain and swelling and can thus be helpful in this phase of the treatment.

Thereafter, a functional treatment is recommended, and, as part of this functional approach, the use of an ankle support is advocated. An elastic bandage gives fewer complications than tape but is associated with a delayed return to work and sports. It seems that overall, a lace-up brace or semirigid brace is preferable. In professional

players, the use of tape can be considered, although this requires careful application because the risk of complications such as skin problems is greater than when a brace or elastic bandage is used [21]. We know from years on the pitch that most professional football players do not like brace because they do not seem to fit in the typical football shoes.

Exercise therapy should also be recognised as an essential element of the functional treatment of acute lateral ankle ligament injury, and this form of therapy can also be effectively performed at home [23]. In the general population, functional treatment is preferred over surgical therapy [21]. However, in professional athletes, surgical treatment may be considered on an individual basis [23]. A personal treatment approach for the athlete with an acute lateral ankle ligament injury is advocated in which a direct anatomic repair of the ruptured ligaments by an expert foot/ ankle or sports surgeon may be considered. This might result in a more stable ankle joint at follow-up without compromising or delaying return to participation in sports [24, 26]. The rehabilitation regime after direct anatomic repair of the ruptured ligaments is a lower-leg cast for 1 or 2 weeks followed by 2–4 weeks in a walking boot. Then an active exercise protocol with the use of an ankle support is advocated.

In case of chronic ankle instability, the initial treatment consists of neuromuscular training, thereby optimising lower limb postural control and restoring active stability by training, which provides good results in a short time [8]. If however symptoms persist and increased ankle laxity remains present, surgical treatment can be considered [20].

Surgical procedures fall into two main categories. In 'anatomic' reconstructions, the previously ruptured ligaments are tightened by overlapping (imbrication) or by reattaching one end of the ligament into the bone (reinsertion) [4]. In 'nonanatomic' reconstructions, the structural laxity is corrected using other tissues, normally tendon (tenodesis). A Cochrane review by de Vries et al. showed that there is not enough evidence to support one specific superior surgical intervention in the treatment of chronic ankle instability. However, based on Krips' early work on sports after surgical treatment of chronic ankle instability and our personal experience, we advocate anatomic reconstruction for chronic lateral ankle ligament laxity in football players [25]. The rehabilitation regime after anatomic reconstruction for chronic lateral ankle instability is the same as in the acute phase with a lower-leg cast for 1 or 2 weeks followed by 2–4 weeks in a walking boot. Then an active exercise protocol with the use of an ankle support is advocated. Furthermore, it is recommended that the rehabilitation protocol after a surgical intervention for chronic ankle laxity is functional with early mobilisation of the ankle joint, rather than 6 weeks of immobilisation. This will reduce the time to return to work and the football field [8].

### 7.7.3 Deltoid Ligaments

Isolated deltoid ligament injuries typically recover well with a functional rehabilitation programme without surgical intervention. Initial rest and bracing should be used until acute inflammation and tenderness resolve. The decision to surgically



Fig. 7.5 Arthroscopic visualization of an acute deltoid injury associated with a rotational ankle injury (\* medial malleolus, \*\* talus)

versus conservatively treat the deltoid injury in a combination injury will depend on the severity of the lateral ligament, syndesmotic and osseous injuries and resultant instability. In the setting of chronic injuries to the deltoid ligament, nonoperative management may be attempted initially. Surgical options are discussed when physical therapy, shoe modifications and other nonoperative modalities fail.

Arthroscopy may be an initial tool in the surgical approach to deltoid ligament injury (Fig. 7.5). Direct intra-articular visualisation with the arthroscope may elucidate or confirm the preoperative diagnosis. The usual open approach to the medial deltoid ligament employs a longitudinal curving incision along the course of the posterior tibial tendon, first incising the laciniate ligament. The superficial deltoid ligament may be inspected anteriorly. The posterior tibial tendon is directly visualised and inspected and then retracted to allow exposure of the underlying deltoid and spring ligament complex.

Repair or reattachment of the ligament is frequently possible with the use of proper suture materials with or without anchors, even in chronic cases [18]. When direct repair is not possible, the use of autograft or allograft tendon for reconstruction has been described [9, 16, 18]. When appropriate, midfoot and hindfoot realignment osteotomies should be considered to place the repaired or reconstructed deltoid ligament in an advantageous mechanical position.

Medial ankle impingement is less common than its anterior and posterior counterparts. The location may be anteromedial or posteromedial, caused by injury or irritation to the superficial or deep components of the ligament, respectively [29]. For cases refractory to conservative management, surgical treatment with arthroscopy or small arthrotomy may be indicated.
#### 7 Ankle Ligament Lesions

Degenerative changes and ossicles at the medial malleolar tip are a relatively common finding, as high as 4.6 % in the series reported by Coral [7]. Early arthritic changes involve a sharpening of the tip of the medial malleolus from its rounder, native shape. Avulsion injuries of the superficial deltoid from the anterior colliculus may contribute to inframalleolar ossicle formation. An accessory ossicle called an os subtibiale is a potential source of misdiagnosis for acute or chronic avulsion injuries. Further imaging investigation such as plain CT, MRI or single-photon emission CT may help to determine the possible donor site for an avulsion injury and the contribution of the ossicle to the patient's symptoms.

#### References

- 1. Beumer A, van Hemert WL, Swierstra BA et al (2003) A biomechanical evaluation of the tibiofibular and tibiotalar ligaments of the ankle. Foot Ankle Int 24:426–429
- Boss AP, Hintermann B (2002) Anatomical study of the medial ankle ligament complex. Foot Ankle Int 23:547–553
- Brostrom L (1964) Sprained ankles. I. Anatomic lesions in recent sprains. Acta Chir Scand 128:483–495
- Brostrom L (1966) Sprained ankles. V. Treatment and prognosis in recent ligament ruptures. Acta Chir Scand 132:537–550
- Chen PY, Wang TG, Wang CL (2008) Ultrasonographic examination of the deltoid ligament in bimalleolar equivalent fractures. Foot Ankle Int 29:883–886
- Chhabra A, Subhawong TK, Carrino JA (2010) MR imaging of deltoid ligament pathologic findings and associated impingement syndromes. Radiographics 30:751–761
- Coral A (1987) The radiology of skeletal elements in the subtibial region: incidence and significance. Skeletal Radiol 16:298–303
- de Vries JS, Krips R, Sierevelt IN et al (2011) Interventions for treating chronic ankle instability Cochrane Database Syst Rev (10):CD004124
- Ellis SJ, Williams BR, Wagshul AD et al (2010) Deltoid ligament reconstruction with peroneus longus autograft in flatfoot deformity. Foot Ankle Int 31:781–789
- Femino JE, Vaseenon T, Phisitkul P et al (2013) Varus external rotation stress test for radiographic detection of deep deltoid ligament disruption with and without syndesmotic disruption: a cadaveric study. Foot Ankle Int 34:251–260
- 11. Gerber JP, Williams GN, Scoville CR et al (1998) Persistent disability associated with ankle sprains: a prospective examination of an athletic population. Foot Ankle Int 19:653–666
- Golano P, Vega J, de Leeuw PA et al (2010) Anatomy of the ankle ligaments: a pictorial essay. Knee Surg Sports Traumatol Arthrosc 18:557–569
- 13. Harper MC (1987) Deltoid ligament: an anatomical evaluation of function. Foot Ankle 8:19-22
- Hertel J (2002) Functional anatomy, pathomechanics, and pathophysiology of lateral ankle instability. J Athl Train 37:364–375
- 15. Hintermann B (2003) Medial ankle instability. Foot Ankle Clin 8:723-738
- Hintermann B, Knupp M, Pagenstert GI (2006) Deltoid ligament injuries: diagnosis and management. Foot Ankle Clin 11:625–637
- Hintermann B, Valderrabano V, Boss A et al (2004) Medial ankle instability: an exploratory, prospective study of fifty-two cases. Am J Sports Med 32:183–190
- Hintermann B, Valderrabano V, Kundert HP (1999) Lengthening of the lateral column and reconstruction of the medial soft tissue for treatment of acquired flatfoot deformity associated with insufficiency of the posterior tibial tendon. Foot Ankle Int 20:622–629

- Jones MH, Amendola A (2007) Syndesmosis sprains of the ankle: a systematic review. Clin Orthop Relat Res 455:173–175
- Karlsson J, Eriksson BI, Sward L (1996) Early functional treatment for acute ligament injuries of the ankle joint. Scand J Med Sci Sports 6:341–345
- 21. Kerkhoffs GM, Struijs PA, Marti RK et al (2003) Functional treatments for acute ruptures of the lateral ankle ligament: a systematic review. Acta Orthop Scand 74:69–77
- 22. Kerkhoffs GM, Tol JL (2012) A twist on the athlete's ankle twist: some ankles are more equal than others. Br J Sports Med 46:835–836
- 23. Kerkhoffs GM, van den Bekerom M, Elders LA et al (2012) Diagnosis, treatment and prevention of ankle sprains: an evidence-based clinical guideline. Br J Sports Med 46:854–860
- 24. Kerkhoffs GM, van Dijk CN (2013) Acute lateral ankle ligament ruptures in the athlete: the role of surgery. Foot Ankle Clin 18:215–218
- Krips R, van Dijk CN, Lehtonen H et al (2002) Sports activity level after surgical treatment for chronic anterolateral ankle instability. A multicenter study. Am J Sports Med 30:13–19
- 26. McCollum GA, van den Bekerom MP, Kerkhoffs GM et al (2013) Syndesmosis and deltoid ligament injuries in the athlete. Knee Surg Sports Traumatol Arthrosc 21:1328–1337
- 27. Milner CE, Soames RW (1998) The medial collateral ligaments of the human ankle joint: anatomical variations. Foot Ankle Int 19:289–292
- Parlamas G, Hannon CP, Murawski CD et al (2013) Treatment of chronic syndesmotic injury: a systematic review and meta-analysis. Knee Surg Sports Traumatol Arthrosc 21:1931–1939
- Paterson RS, Brown JN (2001) The posteromedial impingement lesion of the ankle. A series of six cases. Am J Sports Med 29:550–557
- Ribbans WJ, Garde A (2013) Tibialis posterior tendon and deltoid and spring ligament injuries in the elite athlete. Foot Ankle Clin 18:255–291
- Saltzman CL, el-Khoury GY (1995) The hindfoot alignment view. Foot Ankle Int 16:572–576
- Sman AD, Hiller CE, Refshauge KM (2013) Diagnostic accuracy of clinical tests for diagnosis of ankle syndesmosis injury: a systematic review. Br J Sports Med 47:620–628
- Tornetta P 3rd (2000) Competence of the deltoid ligament in bimalleolar ankle fractures after medial malleolar fixation. J Bone Joint Surg Am 82:843–848
- 34. van den Bekerom MP, Mutsaerts EL, van Dijk CN (2009) Evaluation of the integrity of the deltoid ligament in supination external rotation ankle fractures: a systematic review of the literature. Arch Orthop Trauma Surg 129:227–235
- 35. van Dijk CN, Mol BW, Lim LS et al (1996) Diagnosis of ligament rupture of the ankle joint. Physical examination, arthrography, stress radiography and sonography compared in 160 patients after inversion trauma. Acta Orthop Scand 67:566–570
- 36. Veltri DM, Pagnani MJ, O'Brien SJ et al (1995) Symptomatic ossification of the tibiofibular syndesmosis in professional football players: a sequela of the syndesmotic ankle sprain. Foot Ankle Int 16:285–290
- 37. Woods C, Hawkins R, Hulse M et al (2003) The Football Association Medical Research Programme: an audit of injuries in professional football: an analysis of ankle sprains. Br J Sports Med 37:233–238

# Chapter 8 Interview – Jan Wouters

Pieter P.R.N. d'Hooghe and Gino M.M.J. Kerkhoffs

**Abstract** Wouters played for several clubs including PSV, FC Utrecht, Bayern Munich, and Ajax Amsterdam. He was also a Dutch international (70 caps, 4 goals) and was hugely influential in 1988 when the Netherlands won the European Football Championship. As a coach, Jan Wouters worked for AJAX Amsterdam, PSV Eindhoven, and Glasgow Rangers and from 2011 onward as coach of his club FC Utrecht. He represents a new generation of international top coaches with a successful history as a player in football at the highest level.

G.M.M.J. Kerkhoffs, MD, PhD

P.P.R.N. d'Hooghe, MD ()

Department of Orthopaedic Surgery, Aspetar Hospital, Aspire Zone, Doha, Qatar e-mail: pieter.orthopedie@gmail.com

Department of Orthopedic Surgery, Academic Medical Centre Amsterdam, University of Amsterdam, Meibergdreef 9, Amsterdam 1105 AZ, The Netherlands e-mail: g.m.kerkhoffs@amc.uva.nl



• Jan, what do you feel you can add as a coach to a player's ankle injury rehabilitation?

The most important for me is that I have confidence in the medical staff so I feel comfortable listening to their strategy. In good harmony with the players' personal experience and the opinion of the medical professionals, my role will be to allow a smooth return of the player into the training with the group.

• Do you address a player differently when just coming out of an ankle injury? Yes of course. Again after consulting the medical staff, we will adjust the program of the player according to the functional phase of his rehabilitation program. In addition, it is important to anticipate the play time of the player for the upcoming games; ideally, there is a logical buildup in the number of minutes that the player will be in the team; however, sometimes necessity knows no law, again in close collaboration with the medical staff. This whole process goes for the injured player as much as for the other players as well. Medical staff, technical staff, physical trainer, and players work together to make this a smooth process. • Do you feel that an ankle-injured player should always do his rehabilitation near the team?

Usually yes; however, there can be exceptions to the rule. For example, the foreign players that are injured for a long time, they can do part of the rehabilitation program in their home country in familiar circumstances. Long-term rehabilitation programs are also mentally tough so a part of the total program in domestic surroundings can help with this aspect too. External rehabilitation programs are always discussed in detail with the medical staff with periodical feedback moments.

- How do you manage the team's confidence if one of the best players is injured? Actually by focusing on the rest of the group. I try to prevent the other players to take notice that I am disappointed. It is an important part of the job to coach positive and not to emphasize on negative issues as, for example, the absence of injured players.
- What do you expect exactly from a medical staff? A medical staff should facilitate a quick diagnosis and thereafter a fast return to the pitch! It needs no saying that the return should be within good reason in order to prevent re-injuries. The medical staff should take a clear decision and communicate this within the team; these are decisions that I cannot make, and for me it is crucial that there is a good and early communication on diagnosis and prognosis. I will then always follow the advice of the medical staff.
- How did you experience medical staff evolution in football over the years? There has changed a lot since my time as an active player. The medical staff has grown and involves more people. An injured player gets a quick analysis of his injury; the medical staff acts as a case manager for the injured player, taking care of getting him to the right people in time and providing the best treatment and rehabilitation protocol. The rehabilitation possibilities are much better than in earlier years. In my time as a player, we had to run blocks around the pitch; nowadays, the rehabilitation is much more specific and better thought of. *Certainly, the increased knowledge and experience help us a lot; for each player* and for each injury, there is an individual rehabilitation plan. At the start of the season, there are numerous tests in order to facilitate injury prevention programs to be developed per player. In my days, a player went out on the pitch even with a fever; nowadays, we are much more careful with the players, sometimes maybe too careful. Although the pressure is high for the players, I feel that there is a danger that players are mentally pampered too much, and mental toughness is an important feature in top football. This aspect needs special attention.
- Do you think that the frequency of ankle injuries has increased compared to your time as a player?

The margins for the play on the pitch are smaller, the game has become more physical, there are more duels, and thus the physical demands for the players

have become much bigger. The whole body needs to be in optimum state, and the training has to take care of this aspect too.

- Do you discuss a medical record of a player that you want in your team extensively before getting the player to the club? *Yes, certainly I want to know the injury pattern of a player. If the risk of being injured for a certain player is very high, then I would rather go for a player with less chance of being injured. You can spend your money only once.*
- How important is it for you as a coach that a player is 100 % fit to play? Or how fit should a player be in order to put him on the team? If players are injured, they will only be at my disposal once they are medically fit. Thereafter, we have to get them to be game fit. This really is a gray zone where sometimes I will bring a player on the pitch that is not 100 % game fit, although always 100 % medically fit. The arguments to bring a player that is not yet 100 % game fit depend on the experience of the player, position on the pitch, or the sake of the upcoming game. In principle, a player will first make minutes in the development team before returning to team; experienced players can make their first minutes after injury in the team.
- What's your philosophy on ankle injury prevention, and is ankle injury prevention a topic in coach courses?

Sure there is a lot of attention for injury prevention. Injury prevention is a permanent topic in the courses coach in professional football of the Royal Dutch Football Association (KNVB).

Physical tests at the start of the season provide solid information for an individual training schedule for core stability and strength exercises as well as specific injury prevention strategies. Medical staff and physical fitness trainer work closely together to get the best results.

• Which nation will win the World Cup 2014? *Brazil* 

# Chapter 9 Osteochondral Defects in the Ankle Joint

Rogier Gerards, Maartje Zengerink, and C. Niek Van Dijk

Football is a simple game, but the hardest in Football is to play simple.

Johan Cruijff

**Abstract** Osteochondral defects (OCDs), also known as osteochondritis dissecans, can cause pain and decreased function in patients and offer a significant challenge to the foot and ankle surgeons. An OCD is a lesion involving articular hyaline cartilage, the subchondral bone plate and the subarticular spongiosa. An OCD is mostly caused by a single or multiple traumatic events leading to partial or complete detachment of the osteochondral fragment with or without osteonecrosis. Osteochondral ankle defects can be seen in the tibial plafond but occur predominantly on the talar dome.

#### 9.1 Introduction

Osteochondral defects (OCDs), also known as osteochondritis dissecans, can cause pain and decreased function in patients and offer a significant challenge to the foot and ankle surgeons. An OCD is a lesion involving articular hyaline cartilage, the subchondral bone plate and the subarticular spongiosa. An OCD is mostly caused by a single or multiple traumatic events leading to partial or complete detachment of the osteochondral fragment with or without osteonecrosis. Osteochondral ankle defects can be seen in the tibial plafond but occur predominantly on the talar dome.

Osteochondral defects can occur in any joint; however, the most common location is the knee, followed by the elbow. Of the total number of OCDs, the ankle comprises approximately 4 %, and they occur most frequently in 20- to 30-year-old males [1, 2]. Little is known about the incidence of osteochondral defect in the

The Ankle in Football, Sports and Traumatology,

R. Gerards (🖂) • M. Zengerink • C.N. Van Dijk

Department of Orthopedic Surgery, Academic Medical Center, Amsterdam, The Netherlands e-mail: r.m.gerards@amc.uva.nl

P.P.R.N. d'Hooghe, G.M.M.J. Kerkhoffs (eds.),

DOI 10.1007/978-2-8178-0523-8\_9, © Springer-Verlag France 2014

general population. A study following US military personnel (1999–2008) described an average incidence of 27 per 100,000 people. The incidence rate was 16 per 100,000 in 2002, with steady annual increases resulting in an incidence rate of 56 per 100,000 in 2008, corresponding to the years of active involvement in combat operations and thus increased physical activities [3].

The ankle is one of the most frequently injured joints in sporting activities and in the general population [4, 5]. Ankle sprain injuries account for 14.8 % of all reported injuries in the emergency department. These injuries are more common among athletes who participate in sports that involve running on changing terrains, repetitive jumping, or frequent changes in direction, such as in basketball, volleyball, football, American football and cross-country [6–8]. Ankle injuries comprise of 45 % of all basketball injuries, 31 % of all football injuries and 25 % of all volleyball injuries [9, 10].

Ankle sprains are accepted as being the most common cause of osteochondral ankle defects. The incidence of osteochondral lesion after chronic ankle sprains is reported in the literature as 7 % [11, 12]. The results of acute ankle arthroscopy in a series of acute ankle sprains revealed a medial osteochondral talar lesion in 66 % of cases (20 lesions in 30 patients) [13]. In a retrospective study on 108 ankle sprains, the MRI findings showed bone bruises in 39 % [14].

Treatment of an ankle sprain is directed at returning the athlete to the previous level of competition in the shortest possible time. However, after standard treatment for acute ankle sprains, residual symptoms are reported in up to 40 % of patients [11, 15]. In case of persisting symptoms, 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.

#### 9.2 History

Osteonecrosis of subchondral bone was first described by Ambroise Pare in 1558 after finding loose bodies in a patient's knee. Monro was, in 1856, the first to report the presence of cartilaginous bodies in the ankle joint [16]. Paget further described the pathology and named the process "quiet necrosis" when describing two patients with knee pain in 1870 [17]. In 1887–1888, König was given credit for his original description. He suggested that the loose bodies, found in the knee joint, resulted from a 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 [18]. However, throughout the years, inflammation was never revealed as a contributing factor, making the name osteochondritis a misnomer. In 1922, Kappis [19] was the first to describe a similar lesion in the talar dome, but it was not until 1959, when Berndt and Harty were the first to mention trauma as the main aetiological 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. 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 [20].

### 9.3 Aetiology

Although trauma is mentioned as the main aetiological factor of OCDs, not all patients report a history of ankle injury. A subdivision can be made between traumatic and non-traumatic defects. In the aetiology of traumatic OCDs, ankle sprains play a large role. A severe ankle sprain can cause a small fracture and subsequent impaired vascularity, leading to the formation of an OCD. Microtraumas, caused by repetitive articular cartilage surface loading or excessive stress, can lead to cellular degeneration or death by the disruption of the collagen fibril ultrastructure and thickening of the subarticular spongiosa [21]. In lateral OCD lesions, trauma is described in 98 % of cases; in medial lesions, this is 70 % [22]. In non-traumatic OCDs, ischemia, subsequent necrosis and genetics are possible aetiological factors. OCDs have been described in identical twins and in siblings [23–25]. And in 10–25 % of patients, the occurrence of the defect is bilateral [20, 26, 27].

#### 9.4 Mechanism of Injury

When an inversion trauma occurs, the talus twists inside its boxlike housing, and the cartilage lining and the underlying bone can be damaged. Trauma may lead to a bone bruise and softening of the cartilage or even a crack in the cartilage with subsequent delamination. Due to the shearing forces separation may also occur in the subchondral bone, giving rise to a subchondral lesion. These fragments can detach completely and become a loose body in the ankle joint or remain partially attached. When trauma has caused microfractures in the subchondral plate and subarticular spongiosa, it creates a situation in which liquid from the damaged cartilage can be forced into the subchondral plate, the higher the fluid pressure. This intermittent local rise in high fluid pressure will cause osteolysis and the eventual formation of a subchondral cyst. The ongoing intermittent flow of fluid from the joint through the damaged subchondral bone plate into the spongiosa can prevent healing of the lesion in the subchondral bone plate [28].

In cadaver ankles, Berndt and Harty could reproduce lateral defects by strong inversion of a dorsiflexed ankle [20]. When the foot is inverted, the lateral part of the talar dome is compressed against the fibula (Fig. 9.1). Due to the forces released when the lateral ligament ruptures, an avulsion of the lateral talar border can occur. They were also able to reproduce a medial lesion in a plantar-flexed ankle with a slight anterior displacement of the talus accompanied by an inversion and internal rotation of the talus. Until recently, it was estimated that lateral osteochondral lesions were usually located in the anterior third of the talar dome and medial lesions were also seen but less frequent. A study to evaluate the location and morphologic characteristics of osteochondral lesions. Lateral lesions are typically shallow



**Fig. 9.1** Inversion of the talus; a shear mechanism injury resulting in a lateral osteochondral defect

and wafer shaped, indicating a shear mechanism of injury. In contrast, medial lesions are generally deep and cup-shaped, indicating a mechanism of torsional impaction. Because of their shape, location and trauma mechanism, lateral lesions are more often displaced than medial lesions (Fig. 9.2) [29].

### 9.5 Clinical Presentation

In the acute situation, after a traumatic incident, an OCD of the talus often remains unrecognised. This is because of swelling and pain from soft tissue injury like lateral ligament lesion. The radiographs taken at the emergency unit may not reveal any pathology. In case of a large OCD, the initial radiographs may be positive. When the symptoms of the ligament injury have resolved after some weeks, symptoms like persistent swelling, limited range of motion and pain on weight bearing



Fig. 9.2 Main shape and locations of OCDs of the right talar bone [29]

may persist. 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 4–6 weeks, an OCD of the talus should be suspected. Locking and catching are symptoms of a displaced fragment.

A differentiation has to be made between the acute and chronic situation. Chronic lesions classically present as deep lateral or medial ankle pain associated with weight bearing. Reactive swelling and stiffness can be present, but the absence of swelling, locking or catching does not rule out an OCD. Recognisable tenderness on palpation is typically not present in these patients, but can be present in case of synovitis. Some patients have a diminished range of motion.

Differential diagnoses are:

- Posttraumatic synovitis
- OCD of tibial plafond
- Sinus tarsi syndrome
- Ligament laxity
- Osteoarthritis
- Subtalar joint pathology

Cartilage has a liquid and a solid component (i.e. collagen and proteoglycans) that enables it to withstand compressive stress. Fluid from the damaged cartilage can be forced into the microfractured subchondral bone plate underneath during loading. The smaller the diameter of the defect in the subchondral plate, the higher the fluid pressure. This intermittent local rise in high fluid pressure will cause osteolysis and the eventual formation of a subchondral cyst. Malalignment of the ankle joint may aggravate this process by increasing the local pressure in specific locations of the ankle. The pain in osteochondral defects is most probably caused by the repetitive high fluid pressure, sensitising the highly innervated subchondral bone [28].

#### 9.6 Diagnosis

After medical history and physical examination of the ankle, routine radiographs are made consisting of weight-bearing anteroposterior (mortise) and lateral views of both ankles. OCDs may be visible on the plain AP radiograph shown as an area of radiolucency (Fig. 9.3), although the findings may be subtle and require very careful attention. Displaced fragments are more likely to be detected on the plain radiograph than those which are undisplaced. However, it is not unusual for the initial radiograph to be normal. Small fragments are rarely visible. A heel-rise view with the ankle in a plantar-flexed position may reveal a posteromedial or posterolateral defect [30].



**Fig. 9.3** Radiolucency of the medial talar dome indicating an osteochondral defect (x-ray)



Fig. 9.4 Computed tomography (CT) scan of a medial osteochondral defect before and after excision, debridement of the sclerotic bone and bone marrow stimulation

Table 9.1 Classification and staging of lesions

Stage	Description	
I	Small compression fracture	
II	Incomplete avulsion of a fragment	
III	Complete avulsion of a fragment without displacement	
IV	Displaced fragment	
According to Berndt and Harty [20]		

The sensitivity of routine radiography is 50-75 %, whereas pickup on bone scan is 99 % sensitive and can differentiate between a symptomatic and asymptomatic lesion. CT scan may be useful for bony anatomy and location of the lesion and is therefore more valuable for preoperative planning (Fig. 9.4) [30].

MRI is indicated if standard radiographic results are normal; it may give information regarding vascularity, healing and cartilage integrity. However, the true extent of the OCDs may be obscured by concomitant bone marrow oedema [31].

#### 9.7 **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 [20].

In stage I, there is local compression of the cartilage and subchondral bone, and usually there are no radiographic findings. In stage II, there is avulsion or partial detachment of the osteochondral fragment, but the main part is still attached to the talus. In stage III, there is complete avulsion of an osteochondral fragment without any displacement. In stage IV, the osteochondral fragment is completely detached and displaced inside the ankle joint (Table 9.1 and Fig. 9.5).

Loomer et al. later modified the staging system to include stage 5, subchondral cysts [32]. Ferkel and Sgaglione developed a classification system based on CT:



Fig. 9.5 Classification of osteochondral ankle defects [20]

stage I, intact roof/cartilage with cystic lesion beneath; stage IIA, cystic lesion with communication to the surface; stage IIB, open surface lesion with overlying fragment; stage III, non-displaced fragment with lucency underneath; and stage IV, displaced fragment [33]. Hepple et al. revised the MRI classification in 1999 to resemble Berndt and Harty's original classification. Stage 1 represents articular cartilage damage only. Stage 2a represents articular cartilage damage with underlying fracture and bony oedema. Stage 2b is similar to 2a without bony oedema. Stage 3 represents a detached but undisplaced osteochondral fragment. The fragment is displaced in stage 4, and in stage 5, subchondral cyst formation occurs. Pritsch et al. were one of the first to stage talar osteochondral lesions with arthroscopic findings according to cartilage quality [34]. Cheng et al. further developed arthroscopic staging of the lesions. In stage A, the articular cartilage is smooth and intact, but soft. In stage B, the articular cartilage surface is rough. In stage C, fibrillation or fissuring of the cartilage is present. In stage D, an osteochondral flap is present or bone is exposed. In stage E, the osteochondral fragment is detached but undisplaced. In stage F, the osteochondral fragment is detached and displaced. Arthroscopy is useful in staging talar osteochondral lesions, but it is unable to completely assess underlying bony lesions [35].

#### 9.8 Current Treatment Options

There are widely published nonsurgical and surgical techniques for treatments of symptomatic osteochondral lesions.

#### 9.8.1 Nonoperative Treatment

Conservative treatment consists of rest and/or restriction of (sporting) activities, with or without treatment with nonsteroidal anti-inflammatory drugs (NSAIDs) or a cast immobilisation for at least 3 weeks and up to 4 months. A systematic review of treatment strategies for OCDs demonstrated only a 45 % success rate for nonoperative treatment [38]. The treatment aim is to unload the damaged cartilage, so oedema can resolve and necrosis is prevented.

#### 9.8.2 Bone Marrow Stimulation (BMS)

Surgical treatment can include excision, in which the partially detached fragment is excised and the defect itself is left untreated; excision and debridement, in which, after excision of the loose body, the surrounding necrotic subchondral bone is curetted using either an open or arthroscopic technique; and finally excision, debridement and bone marrow stimulation (BMS) (Fig. 9.4). BMS is the current treatment of choice regarding OCDs. Following excision and curettage (debridement), multiple connections with the subarticular spongiosa are created. This can be accomplished by drilling or microfracturing. The objective is to partially destroy the calcified zone that is most present and to create multiple openings into the subchondral spongiosa. Intraosseous 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, bone marrow cells are introduced in the osteochondral defect, and fibrocartilaginous tissue is formed. Diameter of the lesions usually did not exceed 1.5 cm. Treatment was reported to be successful in 85 % of described cases [36]. In the case of large defects, a cancellous bone graft can be placed.

#### 9.8.3 Antegrade (Transmalleolar) Drilling

Transmalleolar antegrade drilling is considered in cases of OCDs that are difficult to approach because of their location on the talar dome. In this technique, a K-wire is inserted about 3 cm proximal to the tip of the medial malleolus and directed across the medial malleolus into the lesion through the intact cartilage. In case an osteochondral lesion is hard to reach because of its location on the talar dome, the defect can be drilled through the malleolus. A K-wire is inserted about 3 cm proximal to the tip of the medial malleolus and directed across the medial malleolus into the lesion through the intact cartilage [36]. This method is not recommended in football players since it damages the intact cartilage of the distal tibia.

### 9.8.4 Retrograde Drilling

Retrograde drilling is done for primary OCDs when there is more or less intact cartilage with a large subchondral cyst or when the defect is hard to reach via the usual anterolateral and anteromedial portals. For medial lesions, arthroscopic drilling can take place through the sinus tarsi. For lateral lesions, the cyst is approached from anteromedial. A posterior arthroscopic approach is possible by drilling through the posterior talar process. The aim is to induce subchondral bone revascularisation and subsequently to stimulate the formation of new bone. A cancellous graft may be placed to fill the gap [36, 37]. It is the treatment of choice when there is a large subchondral cyst with overlying healthy cartilage.

#### 9.8.5 Osteochondral Autograft Transplantation

Osteochondral autografts have been introduced 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 which has become damaged. It is carried out either by an open approach or by an arthroscopic procedure. Indications involve large, often medial lesions, sometimes with a cyst underneath [36, 37]. Osteochondral grafting of defects yielded 90-94 % good to excellent results at intermediate followup; recent studies suggested significant midterm donor-site morbidity at the previously uninjured knee joint [39–41]. On the search for an alternative method that addresses both the osseous and chondral levels and provides intrinsic osteochondral stability without harming another joint, a modified mosaicplasty procedure was developed for severe and recurrent talus OCL: bony periosteum-covered iliac crest plug transplantation. But more research is needed to prove its effect [42]. OATS is not recommended as initial treatment in football players because of the high donor-site morbidity.

#### 9.8.6 Autologous Chondrocyte Implantation (ACI)

Autologous chondrocyte implantation attempts to regenerate tissue with a high percentage of hyaline-like cartilage. The ACI technique involves placing cultured chondrocytes under a periosteal patch that covers the lesion. It is done for lesions larger than 1 cm<sup>2</sup> in the absence of generalised osteoarthritic changes. Harvesting is first accomplished from either the knee or ankle from the region on the perimeter of the talus lesion. A second procedure is performed after the cells have been cultured for 6–8 weeks. An osteotomy of the medial malleolus can be performed for medial defects. The damaged articular surface is curetted to a stable border, and a periosteal patch is harvested from the tibia. The patch is sutured to the defect and sealed with fibrin glue. Finally, cultured chondrocytes are injected under the patch. Matrix-based chondrocyte implantations (MACI) are also available [43]. It differs from traditional ACI in that chondrocytes are not placed under the periosteal patch but embedded in a type I/III collagen membrane bilayer. As with ACI, the membrane is placed in the defect, but sutures are not required. The membrane bilayer is secured using fibrin sealant. MACI is technically easier than ACI and does not require an osteotomy [36, 37].

#### 9.8.7 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. The advantage is that the graft fits anatomically. Fixation is recommended for lesions of more than 15 mm [36].

#### 9.9 Treatment in Primary Lesions

The surgical treatment of osteochondral lesions of the talus remains controversial among orthopaedic surgeons worldwide. 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 [30]. Pure cartilage lesions and asymptomatic and low symptomatic lesions are treated conservatively with rest, ice, temporarily reduced weight bearing or non-weight bearing using a cast and, in case of giving way, an orthosis. Consideration for surgical treatment is failure of nonoperative treatment or continuing or exacerbation of symptoms after 6 months or residual symptoms after previous surgical treatment.

Arthroscopic bone marrow stimulation is the primary treatment in primary OCDs smaller than 15 mm, with good success proven by Level II or III studies with consistent findings (Table 9.2) [36, 44].

The treatment of symptomatic OCDs has difficulties and limitations because of the poor regeneration of articular cartilage and the limited access to the ankle joint. It is important that the surgeon understand the causes of failure as well as the factors influencing the results of BMS and other treatments of OCDs (Table 9.3).

Table 9.2   Primary OCDs in	Lesion type	Best treatment
tootball players, best	Asymptomatic lesions	Conservative
ireatment options	Symptomatic lesions <15 mm	BMS
	Symptomatic lesions >15 mm	Fixation
	Talar cystic lesions	Retrograde drilling

 Table 9.3 Conceptions and misconceptions regarding results after BMS

Factors influencing the results of BMS of OCDs

Increasing age is not an independent risk factor for poor clinical outcome after the arthroscopic treatment of OCDs

In patients with a large area of more than 15 mm, the clinical failure rate is significantly higher. The existence of a cyst in osteochondral defects has not demonstrated to affect the postoperative prognosis

Patients with an uncontained lesion experienced inferior clinical outcomes as compared with patients with a contained lesion after arthroscopic treatment

Osteochondral transplantation is a viable alternative secondary procedure for treating unstable OCDs that are refractive to arthroscopic treatment

#### 9.9.1 BMS Surgical Technique

Preoperatively, the best approach to the defect is decided. Upon the preference of the surgeon and the location of the lesion, the approach can be either from the anterior, from the posterior or by means of a medial malleolar osteotomy. On the lateral side, a detachment of the anterior talofibular ligament (ATFL) and the calcaneofibular ligament (CFL) can extend the approach to a posterolateral lesion. In case of arthroscopic treatment, we recommend to place the ankle in full plantar flexion. We use a 4.0-mm arthroscope or a 2.7-mm scoop of 11-cm length with a high volume shaft of 4.6 mm. 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. The standard anteromedial and anterolateral approaches are created as described [45]. When introducing instruments for an anterolateral defect, the ankle is in fully dorsiflexed position, and the scoop is introduced through the anteromedial portal and a 4.5- or 5.5-mm shaver through the anterolateral portal. If the OCD is located anteromedially, the arthroscope is moved over to the anterolateral portal, and the instruments are introduced through the anteromedial portal.

If osteophytes are present, they can be removed by chisel, burr or aggressive fullradius resector (bone cutter). Synovitis located anterolaterally (in case of an anterolateral defect) or anteromedially (in case of an anteromedial defect) is removed by a 4.5- 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 plantar flexion. It should be possible to palpate and visualise the OCD



Table 9.4 Timetable recovery after BMS

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 is possible to identify the lesion with the ankle in the forced plantar-flexed position by palpating the cartilage with a probe. In case of a posteriorly located osteochondral lesion, a full forced plantar flexion is needed for adequate visualisation. A little joint laxity helps to open up the joint. During this part of the procedure, we apply a soft tissue distractor [46]. Debridement 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 debridement procedure is checked by regularly switching portals. A precise and complete debridement with removal of all loose fragments can be performed.

After full debridement, the sclerotic zone is drilled by multiple drill holes using a 2-mm burr or a 1.4-mm Kirschner 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. Microfracturing by means of a microfracture probe offers the possibility to work "around the corner". Make sure that the calcified area is penetrated.

#### 9.9.2 Aftercare

Aftercare depends on the type of surgical treatment. After arthroscopic treatment of OCDs, a 4-level activity scheme, derived from rehabilitation after Achilles tendon rupture, has been described (Table 9.4) [46, 50].

Level 1: The first level of activity phase is a return to normal walking that commences on the day of the operation with partial weight bearing. Training for ROM is important in this phase. Patients are encouraged to make active plantar-flexed and dorsiflexed ankle movements. The most important factor is the quality and strength of the tissue repair. The formation of granulation and thereafter fibrocartilaginous tissue starts on the day of the operation. Partial weight bearing provides synovial fluid to nourish chondrocytes. Allowing full weight bearing depends on size and location of the lesion. A lesion of up to 1 cm is allowed to progress to full weight bearing within 2–4 weeks. Larger lesions and anteriorly located lesions require partial weight bearing of up to 6 weeks. After 6–8 weeks, fibrocartilaginous tissue is formed, and full weight bearing is allowed to further stimulate osteoblasts in the formation of bone underneath the cartilage. At the end of this phase, training of proprioception is commenced to regain normal active stability.

Level 2: The next level of activity phase is to resume running on even ground. Progression from walking to running on even ground is permitted between 12 and 16 weeks. Further training of proprioception might be needed, in case active stability has not yet been achieved. The ROM should be normal. By training for force, endurance and technical skills, the aim is to achieve controlled sideways movement, with the lower-leg force increasing to a left/right difference of less than 12 %. After increased activity, pain and swelling should have ceased after 24 h.

Level 3: The third level of activity phase is a return to noncontact activities. Depending on the size and location, full return to noncontact sporting activities is usually possible 20–24 weeks postoperatively. By means of further training for speed and endurance, running on even ground and sprinting should become possible. At the end of this phase, rope jumping, turning and twisting should also be possible. Some pain may occur after increased activity but should be absent after 24 h.

Level 4: This, the highest level of activity phase, is defined as a return to contact sports. Contact sports are permitted from 24 weeks and up. Final training for speed, muscle strength and endurance should enable running on uneven ground, generating explosive force, changing direction and other sports-specific movements.

The course of rehabilitation after other treatment options, like fixation or OATS, is slightly different. Large fragments are treated surgically with reduction and fixation of the osteochondral fragment. If a fragment is fixed, the period of non-weight bearing is 6 weeks followed by another 4–6 weeks of controlled weight bearing to ensure proper fixation.

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. Furthermore, the literature describes several factors, like growth factors, PRP, bisphosphonates, hyaluronic acid and PEMF, that can influence the natural recovery of an OCD and, thereby, the speed of rehabilitation and return to sports. Since most factors are investigated in vitro and in animal studies, more research on potentially influencing factors is needed for talar OCDs in humans [46].

#### 9.10 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 2011 *Cochrane Systematic Database Review* provides good evidence for the beneficial effect of

ankle supports in the form of semi-rigid orthosis or air-cast braces to prevent ankle sprains during high-risk sporting activities. Football players 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 device and – for some – the possible or perceived loss of performance [47]. Proprioceptive training has also been shown to be effective for prevention of ankle sprain recurrences [48]. 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, malunion, non-union and degenerative changes due to the osteotomy over the long term [49]. We do not recommend the use in professional football players.

Morbidity at the donor site in OATS is seen in up to 36 % of cases [36]. For a football player, this is a concern since knee pain may prevent the patient from returning to competitive play. For secondary lesions, BMS is still a good option, with a success rate of 75 %. Other options are OATS and ACI.

Rehabilitation in athletes is directed not only at progressing the patient from protected mobilisation to partial and full weight bearing but also at strengthening and proprioceptive activity.

#### References

- DeBerardino TM, Arciero RA, Taylor DC (1997) Arthroscopic treatment of soft tissue impingement of the ankle in athletes. Arthroscopy 13:492–498
- McCullough CJ, Venugopal V (1979) Osteochondritis dissecans of the talus: the natural history. Clin Orthop Relat Res 144:264–268
- Orr JD, Dawson LK, Garcia EJ, Kirk KL (2011) Incidence of osteochondral lesions of the talus in the United States military. Foot Ankle Int 32:948
- 4. van Dijk CN, Molenaar AH, Cohen RH et al (1998) Value of arthrography after supination trauma of the ankle. Skeletal Radiol 27:256–261
- van Dijk CN, Lim LS, Bossuyt PM et al (1996) Physical examination is sufficient for the diagnosis of sprained ankles. J Bone Joint Surg Br 78:958–962
- Purcell SB, Schuckman BE, Docherty CL, Schrader J, Poppy W (2009) Difference in ankle range of motion before and after exercise in 2 tape conditions. Am J Sports Med 37(2):383–389
- Borowski LA, Yard EE, Fields SK, Comstock RD (2008) The epidemiology of US high school basketball injuries, 2005–2007. Am J Sports Med 36(12):2328–2335
- Halasi T, Kynsburg A, Tallay A, Berkes I (2004) Development of a new activity score for the evaluation of ankle instability. Am J Sports Med 32(4):899–908
- 9. Garrick JG (1977) The frequency of injury, mechanism of injury, and epidemiology of ankle sprains. Am J Sports Med 5:241–242
- Hootman JM, Dick R, Agel J (2007) Epidemiology of collegiate injuries for 15 sports: summary and recommendations for injury prevention initiatives. J Athl Train 42(2):311–319

- Bosien WR, Staples OS, Russell SW (1955) Residual disability following acute ankle sprains. J Bone Joint Surg Am 37:1237–1243
- 12. Lippert MJ, Hawe W, Bernett P (1989) Surgical therapy of fibular capsule-ligament rupture [Article in German]. Sportverletz Sportschaden 3:6–13
- 13. Van Dijk CN (1994) On diagnostic strategies in patients with severe ankle sprain. Thesis, University of Amsterdam, Amsterdam
- Labovitz JM, Schweitzer ME (1998) Occult osseous injuries after ankle sprains: incidence, location, pattern, and age. Foot Ankle Int 19:661–667
- van Rijn RM, van Os AG, Bernsen RMD, Luijsterburg PA, Koes BW, Bierma-Zeinstra SMA (2008) What is the clinical course of acute ankle sprains? A systematic literature review. Am J Med 121:324–331
- 16. Monro A (1856) Microgeologie. Th Billroth, Berlin, p 236
- 17. Paget J (1870) On the production of the loose bodies in joints. St Bartholomews Hosp Rep 6:1
- 18. König F (1888) Über freie Körper in den Gelenken. Deutsch Zeit Chirurg 27:90–109
- Kappis M (1922) Weitere beiträge zur traumatisch-mechanischen entstenhung der "spontanen" knorpelabiösungen. Dtsch Z Chir 171:13–29
- Berndt AL, Harty M (1959) Transchondral fractures (osteochondritis dissecans) of the talus. J Bone Joint Surg Am 41:988–1020
- Frenkel SR, Di Cesare PE (1999) Degradation and repair of articular cartilage. Front Biosci 15:671–685
- 22. Flick AB, Gould N (1985) Osteochondritis dissecans of the talus (transchondral fractures of the talus): review of the literature and new surgical approach for medial dome lesions. Foot Ankle 5:165–185
- 23. Woods K, Harris I (1995) Osteochondritis dissecans of the talus in identical twins. J Bone Joint Surg Br 77:331
- Anderson DV, Lyne ED (1984) Osteochondritis dissecans of the talus: case report on two family members. J Pediatr Orthop 4:356–357
- Erban WK, Kolberg K (1981) Simultaneous mirror image osteochondrosis dissecans in identical twins [Article in German]. Rofo 135:357
- 26. Canale ST, Belding RH (1980) Osteochondral lesions of the talus. J Bone Joint Surg Am 62:97–102
- Hermanson E, Ferkel RD (2009) Bilateral osteochondral lesions of the talus. Foot Ankle Int 30:723–727
- Dijk CN, Reilingh ML, Zengerink M, van Bergen CJA (2010) Osteochondral defects in the ankle: why painful? Knee Surg Sports Traumatol Arthrosc 18:570–580
- Raikin SM, Elias I, Zoga AC, Morrison WB, Besser MP, Schweitzer ME (2007) Osteochondral lesions of the talus: localization and morphologic data from 424 patients using a novel anatomical grid scheme. Foot Ankle Int 28:154
- Verhagen RA, Maas M, Dijkgraaf MG et al (2005) Prospective study on diagnostic strategies in osteochondral lesions of the talus. Is MRI superior to helical CT? J Bone Joint Surg Br 87: 41–46
- Lahm A, Erggelet C, Steinwachs M, Reichelt A (2000) Arthroscopic management of osteochondral lesions of the talus: results of drilling and usefulness of magnetic resonance imaging before and after treatment. Arthroscopy 16:299–304
- 32. Loomer R, Fischer C, Lloyd-Schmidt R, Sisler J, Cooney T (1993) Osteochondral lesions of the talus. Am J Sports Med 21:13–19
- Ferkel RD, Sgaglione NA, DelPizzo W et al (1990) Arthroscopic treatment of osteochondral lesions of the talus: long-term results. Orthop Trans 14:172–173
- Pritsch M, Horoshovski H, Farine I (1986) Arthroscopic treatment of osteochondral lesions of the talus. J Bone Joint Surg Am 68:862–865
- 35. Cheng MS, Ferkel RD, Applegate GR (1995) Osteochondral lesions of the talus: a radiologic and surgical comparison. In: Presented at the annual meeting of the American Academy of Orthopedic Surgeons, New Orleans, 16–21 Feb 1995. American Academy of Orthopedic Surgeons, New Orleans

- Zengerink M, Struijs PAA, Tol JL, van Dijk CN (2010) Treatment of osteochondral lesions of the talus: a systematic review. Knee Surg Sports Traumatol Arthrosc 18:238–246
- Badekas T, Takvorian M, Souras N (2013) Treatment principles for osteochondral lesions in foot and ankle. Int Orthop (SICOT) 37:1697–1706
- Verhagen RA, Struijs PA, Bossuyt PM et al (2003) Systematic review of treatment strategies for osteochondral defects of the talar dome. Foot Ankle Clin 8:233–242
- Scranton PE Jr, Frey CC, Feder K (2006) Outcome of osteochondral autograft transplantation for type-V cystic osteochondral lesions of the talus. J Bone Joint Surg Br 88:614–619
- Hangody L, Fules P (2003) Autologous osteochondral mosaicplasty for the treatment of fullthickness defects of weight-bearing joints: ten years of experimental and clinical experience. J Bone Joint Surg Am 2:25–32
- Paul J, Sagstetter A, Kriner M, Imhoff AB, Spang J, Hinterwimmer S (2009) Donor-site morbidity after osteochondral autologous transplantation for lesions of the talus. J Bone Joint Surg Am 91:1683–1688
- 42. Leumann A, Valderrabano V, Wiewiorski M, Barg A, Hintermann B, Pagenstert G (2013) Bony periosteum-covered iliac crest plug transplantation for severe osteochondral lesions of the talus: a modified mosaicplasty procedure. Knee Surg Sports Traumatol Arthrosc
- 43. Anders S, Goetz J, Schubert T, Grifka J, Schaumburger J (2012) Treatment of deep articular talus lesions by matrix associated autologous chondrocyte implantation–results at five years. Int Orthop 36(11):2279–2285. doi:10.1007/s00264-012-1635-1, Epub 2012 Aug 12 PMID: 22885840
- Murawski CD, Kennedy JG, Operative treatment of osteochondral lesions of the talus. J Bone Joint Surg Am. 2013 Jun 5;95(11):1045–1054. doi: 10.2106/JBJS.L.00773. Review
- 45. Van Dijk CN, Scholte D (1997) Arthroscopy of the ankle joint. Arthroscopy 13:90-96
- 46. van Eekeren IC, Reilingh ML, van Dijk CN (2012) Rehabilitation and return-to-sports activity after debridement and bone marrow stimulation of osteochondral talar defects. Sports Med 42(10):857–870
- Tol JL, Struijs PA, Bossuyt PM et al (2000) Treatment strategies in osteochondral defects of the talar dome: a systematic review. Foot Ankle Int 21:119–126
- 48. Verhagen E, van der Beek A, Twisk J et al (2004) The effect of a proprioceptive balance board training program for the prevention of ankle sprains: a prospective controlled trial. Am J Sports Med 32:1385–1393
- Anderson IF, Crichton KJ, Grattan-Smith T et al (1989) Osteochondral fractures of the dome of the talus. J Bone Joint Surg Am 71:1143–1152
- van Sterkenburg MN, Donley BG, van Dijk CN (2008) Guidelines for sport resumption. In: van Dijk CN, Karlsson J, Maffuli N et al (eds) Achilles tendon rupture. DJO Publications, Surrey, pp 107–116

### Chapter 10 Interview – Velibor "Bora" Milutinović

Pieter P.R.N. d'Hooghe and Gino M.M.J. Kerkhoffs

**Abstract** Velibor "Bora" Milutinović (born 7 September 1944) is a Mexican-Serbian football coach and former player. He and Carlos Alberto Parreira are the only two persons to have coached five different teams at the World Cup: Mexico (1986), Costa Rica (1990), the United States (1994), Nigeria (1998), and China (2002). He is also the first coach to take four different teams beyond the first round – Mexico (1986), Costa Rica (1990), the United States (1994), and Nigeria (1998) – earning the nickname of *Miracle Worker*, first given to him by Alan Rothenberg, then president of the United States Soccer Federation. In total, Milutinović has coached eight different national football teams.

He has been in professional elite football for over 35 years and therefore has extensive experience in the evolution of football throughout the last three decennia.

P.P.R.N. d'Hooghe, MD (🖂)

G.M.M.J. Kerkhoffs, MD, PhD

Department of Orthopedic Surgery, Academic Medical Centre Amsterdam, University of Amsterdam, Meibergdreef 9, Amsterdam, 1105 AZ, The Netherlands e-mail: g.m.kerkhoffs@amc.uva.nl

P.P.R.N. d'Hooghe, G.M.M.J. Kerkhoffs (eds.), *The Ankle in Football*, Sports and Traumatology, DOI 10.1007/978-2-8178-0523-8\_10, © Springer-Verlag France 2014

Department of Orthopaedic Surgery, Aspetar Hospital, Aspire Zone, Doha, Qatar e-mail: pieter.orthopedie@gmail.com



• What do you expect exactly from a medical staff?

A good team doctor is extremely important to a coach; it is even of importance nr.1. The doctor needs to predict and prevent problems and deal with it in sometimes very intense situations. To do that, the doctor should be competent, understand football, and also be a great psychologist. The confidence with the player is created through adequate medical management and close availability, and that's why the doctor should always be operating close to the team. A coach wants his player back as quick as possible and a good team doctor prioritizes this without compromising the player's health.

 How did you experience medical staff evolution in football over the years? Impossible to still compare! When I started as a coach in 1976, a severe ankle injury was a career-ending lesion for a player. Now they complain if they can't play yet after 6 weeks.... Also in that era, we didn't know what a cartilage lesion of the ankle was. What an evolution we have gone through with football-related medical knowledge since then but don't underestimate the experience-based remedies from the past either (starts to talk with melancholy): I remember in 1977 in Partizan Belgrade, a player sustained a severe lesion over the ankle. Surgeons advised him ligament reconstruction but couldn't give him any guarantee. The player decided to stay conservative and was treated by an old village herb lady with salt, pili pili, and special olive oil. He sweated for 6 nights like a mad man but played afterwards all games until the end of his professional career without any problem.

Nowadays players are even called injured if they have inflammatory maceration between the fourth and fifth toe of their feet (opens brown sugar package next to his coffee, removes his shoe and socks, and puts the sugar in the webspace). In the past we treated it with brown sugar, and I assure you that it heals much quicker than with all the other fancy treatments of today!

- Do you address a player differently when just coming out of an injury? All players are different and should be coached individually. A coach needs to know exactly what happened to the player in order to give confidence. A bad injury period for the player can result in a major mental advantage afterwards, if managed well by the coach and doctor together.
- How do you manage the team's confidence if one of the best players is injured? Every player in the team has to give its maximum and that's what makes a team. Therefore, dialogue is the key and the team needs to take that to the next step. Due to the high load of games nowadays, we adapt and individualize training sessions much more now. Still, especially for the young players, even if everything has changed, there's no need to forget or change the fundamentals of the game.
- Is injury prevention (especially in ankles) a topic in coach courses? *This has become imperative because of the effect it can have on a team's performance. This is for sure not underestimated in modern coaching courses.*

The problem nowadays is that many have forgotten the fundamentals of the game: how to run, turn, play, and feel the game. Electronic tools are now "hot" but football is TALENT and GAME; you have to learn the game itself, how to stay safe and how to enjoy what you make out of it.

This is also a responsibility of the player. Due to economic reasons, players sometimes decide to play with different shoe types, etc.... This can have detrimental effects on his/her performance and making him/her more prone to specific injuries. It's all about looking for quality and depending on experience. There is nowadays a business danger that can make players more prone to injuries, and the players also take a big responsibility in that regard.

- How do you manage the medical secret with all the media around? The most important is that you protect the club and the player. Everybody has to be on the same page, work in the same direction. In that way, you can avoid many problems regarding that sensitive issue.
- What is the secret of your coaching? Talent detection – football strategy – psychology
- Who will win the World Cup 2014? The one who scores the most goals in the final...

# Chapter 11 Anterior Ankle Impingement

Johannes L. Tol, Pieter P.R.N. d'Hooghe, Peter A.J. de Leeuw, Mario Maas, and Gino M.M.J. Kerkhoffs

Football is like life: pure art with little pure artists.

Herman Brusselmans Writer

**Abstract** The anterior ankle impingement syndrome is the most common cause of chronic ankle pain in football players. It is characterized by anterior ankle pain on forced dorsiflexion movements. Clinical investigation reveals pain on palpation along the anterolateral and/or anteromedial ankle joint line. There is recognizable pain on forced dorsiflexion which is limited as compared to the contralateral side. In patients with anteromedial impingement, the plain X-rays are often negative and are therefore insufficient. An additional oblique view is required for visualization of the anteromedial osteophytes. The effectiveness of conservative treatment has not yet

J.L. Tol, MD, PhD (🖂)

Department of Sports Medicine, Aspetar, Qatar Orthopaedic and Sports Medicine Hospital, Aspire Zone, PO Box 29222, Doha, Qatar e-mail: johannes.tol@aspetar.com

P.P.R.N. d'Hooghe, MD Department of Orthopaedic Surgery, Aspetar, Qatar Orthopaedic and Sports Medicine Hospital, Aspire Zone, PO Box 29222, Doha, Qatar e-mail: pieter.dhooghe@aspetar.com

P.A.J. de Leeuw, MD Department of Orthopaedic Surgery, Academic Medical Center, Meibergdreef 9, Amsterdam 1105 AZ, The Netherlands e-mail: p.a.deleeuw@amc.nl

M. Maas, MD, PhD Department of Radiology, Academic Medical Center, Meibergdreef 9, Amsterdam 1105 AZ, The Netherlands e-mail: m.maas@amc.nl

G.M.M.J. Kerkhoffs, MD, PhD Department of Orthopaedic Surgery, Orthopaedic Research Center Amsterdam, Academic Medical Center, Meibergdreef 9, Amsterdam 1105 AZ, The Netherlands e-mail: g.m.kerkhoffs@amc.nl

P.P.R.N. d'Hooghe, G.M.M.J. Kerkhoffs (eds.), *The Ankle in Football*, Sports and Traumatology, DOI 10.1007/978-2-8178-0523-8\_11, © Springer-Verlag France 2014 been well documented. After conservative treatment fails, an arthroscopic intervention is the treatment of choice for professional football players. Arthroscopic excision of soft tissue and/or the bony impediment is known to be successful in the players without signs of joint space narrowing. The majority of professional football players will return to their former level within 2 months after surgery.

Keywords Anterior impingement • Ankle • Football

#### 11.1 Introduction Including Epidemiology

Anterior ankle impingement syndrome is the most common cause of chronic ankle pain in football players. The overall percentage of ankle injuries in elite football is 19 % [10], but the exact prevalence and incidence for anterior ankle impingement has not been documented. The typical clinical features are anteriorly located ankle pain, which increases with forced hyper dorsiflexion. The cause can either be soft tissue or bony impingement. In literature, the first authors describing bony impingement lesions in the ankle were Morris [13] and McMurray and classified this pathology as "athlete's" ankle or "footballer's" ankle. In recent literature, these terms have been replaced by the anterior ankle impingement syndrome [18, 22, 25].

#### 11.2 Functional Anatomy

As for any pathology, anatomical knowledge is essential in the understanding and treatment of this pathology. Some believe that the traction spurs (osteophytes) originate due to recurrent capsular traction at the attachment sites at the distal tibia and talus.

Probably the anterior ankle pain is caused by the soft tissues being compressed in between the distal tibia and talus during forced dorsiflexion movements.

In specimens, the anterior joint capsule inserts in the distal tibia on an average of 6 mm proximal to the anterior tibial cartilage rim. On the talar site, the capsule inserts approximately 3 mm from the distal talar cartilage border [23]. Based on these anatomic observations, the hypothesis of formation of talotibial spurs due to recurrent traction to the joint capsule (traction spurs) is not likely to be true. In patients with bony impingement, the location of tibial spurs is reported to be at the joint level and within the confines of the joint capsule [26]. Along the distal tibia, the width of the non-weightbearing cartilage rim extends up to 3 mm proximal to the joint line. It is this non-weightbearing anterior cartilage rim that undergoes the osteophytic transformation. On the talar side, the typical osteophytes are found proximal to the talar neck notch [22].

#### 11.3 Etiology and Injury Mechanism

In the ankle, osteophytes typically originate at the site of the non-weight-bearing cartilage, without pathologic changes of the weight-bearing ankle articular cartilage. It should therefore be differentiated from osteoarthritis.

In football players, the ankle osteophytes are a manifestation of repetitive trauma in combination with recurrent kicking of the ball [21]. The cause of the pain is most probably the inflamed and increased amount of soft tissue in the anterior ankle compartment which is squeezed in between the osteophytes during dorsiflexion, causing an impingement [26]. Typically in football players, pain is experienced on kicking, when the anterior capsule is stretched over the tibial and talar osteophytes. Recurrent trauma to this soft tissue component may lead to hypertrophy of the synovial layer, subsynovial fibrotic tissue formation, and infiltration of inflammatory cells.

#### **11.4 Clinical Features**

The typical symptomatic player will present with a history of previous ankle injuries [22]. On history taking, the main symptoms are persistent anteriorly located ankle pain during dorsiflexion movements or while kicking the ball, post-exercise ankle swelling, and restricted dorsiflexion. With an adapted training program, most players can continue playing.

The physical examination reveals recognizable pain on palpation along the anterolateral and/or anteromedial joint line. In plantar flexion the joint capsule stretches over the osteophytes, inducing pain and difficulties palpating the osteophytes. The optimal palpation position is at slight ankle dorsiflexion. Depending on the recognizable pain on palpation, a differentiation can be made being either an anteromedial or anterolateral ankle impingement. Forced hyper dorsiflexion can provoke the pain, but this maneuver might be negative in the prone position [26].

#### 11.5 Diagnostic Imaging

Standard weight-bearing lateral and anteroposterior radiographs can detect the anteriorly located osteophytes but might be false negative. Due to the anteromedial notch, anteromedial osteophytes up to 7 mm are undetected on these "standard" radiographs [23]. Medially located talar osteophytes remain undetected due to overprojection of the lateral part of the talar neck and body.

The oblique anteromedial impingement (AMI) view is recommended to detect the anteromedial osteophytes (Fig. 11.1). As compared to the standard lateral projection, the beam is tilted into a 45° craniocaudal direction with the leg in 30° external rotation and the foot in plantar flexion (Fig. 11.2). Routine computed tomography (CT) and/or magnetic resonance imaging (MRI) for the ankle impingement syndrome is not advised, although MRI has a high sensitivity to detect the (anterolateral) soft tissue impediments.

Ultrasound and conventional MRI have a debatable role on the footballers' population. Literature demonstrated that osteophytes are not strictly solely related to the clinical entity of impingement, suggesting that soft tissue pathology may also play a significant role [8]. As mentioned previously, the associated synovial abnormality



**Fig. 11.1** Professional footbal player with an anteromedial impingement syndrome (*red circle*). (**a**) Lateral view shows a normal joint space and no osteophytes. (**b**) Anteroposterior view shows no significant abnormalities. (**c**) Anteromedial impingement (AMI) view shows a tibial osteophyte (*red circle*)

secondary to the osseous spurs is critical for inducing the clinical syndrome rather than the osseous spurs alone [17]. Gray-scale ultrasonography is performed with a linear probe (12–17 MHz) in an axial and coronal plain with the foot in slight dorsiflexion for better visualization of the etiologic causes. Sensitivity and specificity of the ultrasound examination is conflicting. Thickening of the synovium over 10 mm with nodular appearance of the synovial capsule, especially within the anterolateral recess (meniscoid lesion or synovitic lesion), is strongly associated with impingement [11, 27]. Cochet et al. elucidated in a more recent study that detection of smaller lesions can have a positive predictive value for diagnosing impingement [1]. Fluid effusion at the anterolateral side of the ankle can most efficiently be visualized in the sagittal plain. Even spurs in an early phase of formation can hereby be detected. The detection of these findings is not hampered by the presence of fluid at the ankle recess, which is a potential limitation in the conventional MRI. Color Doppler ultrasound might be helpful, since hypervascularity of the synovial or meniscoid mass seems to depend on the repetitive injuries and the amount of the fibrosis in it [1, 11]. Another advantage of sonography over MRI is the possibility to directly infiltrate the inflamed tissue with steroids in a controlled manner, most suitable anterolaterally.



Fig. 11.2 Following patient's history and the physical examination, in case an anteromedial impingement is suspected, an anteromedial impingement view (AMI) can be made to confirm or reject an anteromedial bony impingement on the tibia and/or talus. The AMI view showing the medial osteofyt (*red circle*) is made with the ankle in plantar flexion and the leg in  $30^{\circ}$  external rotation, the beam tilted in a  $45^{\circ}$  craniocaudal direction

Conventional MRI is nowadays widely used as an additional diagnostic tool in case a clinical ankle impingement is suspected; however, results are conflicting. Sensitivity and specificity for the detection of abnormality varies widely, and the accuracy depends on the presence of a significant joint effusion [17]. Although the spatial resolution of the conventional MRI is nowadays increased and special sequences are used, anatomical variants are still difficult to recognize. An example in this case is the Bassett ligament; this anatomical variant, being an accessory anteroinferior tibiofibular ligament, can easily be pointed out as an increased soft tissue mass responsible for a clinically suspected anterolateral impingement with a negative conventional radiograph. In these cases, a static and dynamic ankle ultrasonography and/or anterior ankle arthroscopy is superior [8, 16, 27].

Although some authors have proposed MR arthrography enabling a high sensitivity and specificity as an additional diagnostic tool to diagnose ankle impingement [1, 8, 17], no large radiographic series have been published in literature up to present [8]. On an MR arthrography, an irregular or nodular contour of the anterolateral

Туре	Characteristics		
Туре І	Synovial impingement. X-rays show an inflammatory reaction, up to 3 mm spur formation		
Type II	Osteochondral reaction exostosis. X-rays manifest osseous spur formation greater than 3 mm in size. No talar spur is present		
Type III	Significant exostosis with or without fragmentation, with secondary spur formation on the dorsum of the talus seen, often with fragmentation of osteophytes		
Type IV	Pantalocrural arthritic destruction. X-rays suggest medial, lateral, or posterior, degenerative, arthritic changes		
Table 11	.2 Classification Grade Characteristics		

soft tissues is considered to be pathological and would be highly correlated to ankle

 Table 11.1
 Classification of anterior ankle impingement [19]

Table 11.2         Classification	Grade	Characteristics
for osteoarthritic changes	Grade 0	Normal joint or subchondral sclerosis
of the alikie joint [20]	Grade I	Osteophytes without joint space narrowing
	Grade II	Joint space narrowing with or without osteophytes
	Grade III	(Sub) total disappearance/deformation of the joint
		space

scar tissue and synovitis at arthroscopy. An indirect positive sign for anterolateral impingement is the extent of the anterior recess [17]. CT arthrography has also been used for the evaluation of anterolateral impingement with a lower sensitivity and specificity as compared to MR arthrography [1, 17].

#### 11.6 Classification

Up to present, there is not a uniform classification system for anterior ankle impingement. Scranton and McDermott [19] published on the size of the osteophytes and their location, based on the lateral radiographs (Table 11.1). To predict the surgical outcome following the removal of the osteophytes, van Dijk et al. published an osteoarthritic classification [26] (Table 11.2).

#### 11.7 Treatment

#### 11.7.1 Conservative Treatment

Conservative treatment, consisting of intra-articular injections and/or heel lifts, is recommended in the early stages but has never been systematically studied and might frequently be unsuccessful [22].

#### 11.7.2 Invasive Treatment

McMurray reported on the first surgically treated patients [12]. After removal of anterior located osteophytes by open arthrotomy, the patients successfully returned to professional soccer. In subsequent studies numerous authors have reported good results with an open ankle arthrotomy [7, 14, 15]. Open ankle arthrotomy can be complicated by cutaneous nerve entrapment, damage of the long extensor tendons, wound dehiscence, and formation of hypertrophic scar tissue [3]. Arthroscopic intervention with an earlier return to sports is generally accepted as the preferred procedure for professional football players [27].

Anterior ankle arthroscopy is carried out as an outpatient procedure under general or spinal anesthesia. The patient is positioned in the supine position with slight elevation of the ipsilateral buttock. The heel of the affected ankle is placed at the very end of the operating table (Fig. 11.3). In this way, the surgeon can fully dorsiflex the ankle by leaning against the foot sole. Routine anterior portals used are the anteromedial and anterolateral portal. A soft tissue distraction device can be used when indicated (Fig. 11.4). Accessory portals are located just in front of the tip of the medial or lateral malleolus.

The anteromedial portal is made just medially to the anterior tibial tendon through the skin only (Fig. 11.5); subsequently the subcutaneous tissue is spread with a mosquito clamp in ankle dorsiflexion, thereby preventing iatrogenic damage



**Fig. 11.3** Patient positioning in anterior ankle arthroscopy: the patient is in the supine position, the hip on the contralateral side is supported, the ipsilateral buttock is elevated, and a tourniquet applied around the upper leg, with the affected ankle at the end of the operation table. Hereby, ankle dorsiflexion can be achieved by leaning against the foot during surgery



Fig. 11.4 In some cases, a noninvasive soft tissue distractor can be helpful. This device is positioned around the ankle and is connected to a belt around the surgeon's hips. Hereby, the surgeon can distract the ankle by leaning backwards

Fig. 11.5 Anteromedial portal location in anterior ankle arthroscopy: while palpating the joint line with the thumb (notch of Harty), the location of the anterior tibial tendon is observed, and subsequently with the ankle in dorsiflexion, the portal is made by a vertical incision through the skin only just medial to the anterior tibial tendon



to the tibial and talar cartilage. Routinely a 4.0 mm 30° angled arthroscope is introduced, again in maximal ankle dorsiflexion (Fig. 11.6). Iatrogenic cartilage lesions by instrument introduction can thereby be prevented. For irrigation, normal saline by an arthroscopic pump (50 mmHg, FMS DUO®+, DePuy Synthes-Mitek) is nowadays routinely used. By arthroscopic visualization laterally, the location of the anterolateral portal can be controlled directly. A spinal needle is introduced just



**Fig. 11.6** A 4.0 mm 30° angled arthroscope is introduced while the ankle is in full dorsiflexion, thereby preventing iatrogenic damage to the talar cartilage (\* ankle capsule, \*\* talus)

lateral to the peroneus tertius tendon. A vertical skin incision is made with respect for the local anatomy, being the superficial peroneal nerve. Although this nerve is unique for the human body with respect to its possibility being visualized by combined ankle plantar flexion and inversion, it is the most frequently reported complication with the creation of the anterolateral portal [5]. Another possibility to visualize this nerve is by means of fourth toe flexion [20]. Marking its course prior to the creation of the anterolateral portal is advised; nevertheless, one should realize that the course of this nerve changes with the *ankle* position. The anterolateral portal should be made medially to the position of the nerve in ankle plantar flexion and inversion [4]. Other important tricks to prevent superficial peroneal nerve injury include vertical skin incisions, through the skin only, followed by blunt dissection up to the joint and the use of transillumination [5, 20]. The contour of the anterior tibia is identified, and in case of an osteophyte, soft tissue superior from this osteophyte is removed with a shaver. The extent of the osteophyte is determined, and the osteophyte is subsequently removed using a 4 mm chisel and/or shaver. When an osteophyte is located on the medial distal tibial rim or the front of the medial malleolus, the arthroscope is moved to the anterolateral portal and the instruments are introduced through the anteromedial portal. Osteophytes at the tip of the medial malleolus and ossicles or avulsion fragments in this area can be removed in a similar manner. It can be helpful to create an accessory portal in front of the tip of the medial malleolus. In case of osteophytes at the tip of the medial malleolus, usually overcorrection of the tip is feasible using a bonecutter shaver. Also in case of a soft tissue impingement, a shaver is helpful for the debridement (Fig. 11.7).

In case besides the impingement also, an osteochondral defect needs to be addressed; the talus can be inspected by ankle plantar flexion. In some cases a soft tissue distractor



Fig. 11.7 Arthroscopic treatment anterolateral soft tissue impingement in a right ankle. (a) The  $4.0 \text{ mm } 30^{\circ}$  arthroscope is introduced through the anteromedial portal, and the soft tissue impingement anterolaterally can be identified. (b) Under arthroscopic control a spinal needle is introduced to determine the optimal anterolateral portal position. (c) After skin incision only, a hemostat is used to spread the subcutaneous tissue to subsequently open the anterolateral ankle capsule. (d) A shaver is introduced to debride the soft tissue responsible for the impingement. (e) Postoperative arthroscopic image

can be helpful, mainly in case these lesions are situated in the tibial plafond. Most anteriorly located osteochondral lesions can subsequently be treated arthroscopically by debridement and bone marrow stimulation. In case history taking, physical examination, and the additional diagnostics indicate an anterior ankle impingement and an additional syndesmotic instability, the syndesmosis can be tested arthroscopically.

To prevent sinus formation, at the end of the procedure, the skin incisions are sutured with 3.0 Ethilon. A sterile compressive dressing is applied (Klinigrip, Medeco BV, Oud Beijerland, the Netherlands). Prophylactic antibiotics are not routinely given.

A recent review of the literature showed sufficient evidence for arthroscopic treatment of anterior ankle impingement [6]. The number of prospective cohort studies is still limited but shows an overall success rate of grade I (bony impingement without joint space narrowing) in over 85 %.

Osteophytes reoccur in 67 % at 5–8 years follow-up [24]. Coull et al. [2] reported even a recurrence of osteophytes in all their 27 patients who underwent open debridement. There was, however, no statistical correlation between the recurrence of osteophytes and the return of symptoms [24]. As mentioned previously it is probably not the osteophyte itself which causes the pain, but the compression of the synovial fold or fibrotic (scar) tissue. In theory, arthroscopic excision of the soft tissue can relieve pain. Talar and tibial osteophytes, however, reduce the anterior joint space. After arthroscopy, a postoperative hematoma may develop and again form an anterior soft tissue impediment. It is therefore important to restore the anterior space and reduce the chance of symptoms to reoccur.
## 11.8 Rehabilitation

Postoperative rehabilitation treatment consists of a compression bandage and partial weight bearing for 3–5 days. The athlete is instructed to actively dorsiflex his or her ankle and foot upon awakening and to repeat this exercise a few times every hour for the first 2–3 days after surgery [22, 24]. The added value of physical therapy has insufficiently been documented. A small retrospective series showed that patients receiving more than 1 month physical therapy scored better on a 7-point ankle scale as compared to the ones who were not treated with physical therapy [9]. In the absence of high-level evidence, in professional football players, physical therapy with a focus on restoration of dorsiflexion, reduction of swelling, functional training, and supervised return to sport specific training after 6–8 weeks is advised.

## References

- Cochet H, Pele E, Amoretti N et al (2010) Anterolateral ankle impingement: diagnostic performance of MDCT arthrography and sonography. AJR Am J Roentgenol 194:1575–1580
- 2. Coull R, Raffiq T, James LE et al (2003) Open treatment of anterior impingement of the ankle. J Bone Joint Surg Br 85:550–553
- Cutsuries AM, Saltrick KR, Wagner J et al (1994) Arthroscopic arthroplasty of the ankle joint. Clin Podiatr Med Surg 11:449–467
- de Leeuw PA, Golano P, Sierevelt IN et al (2010) The course of the superficial peroneal nerve in relation to the ankle position: anatomical study with ankle arthroscopic implications. Knee Surg Sports Traumatol Arthrosc 18:612–617
- 5. Ferkel RD, Heath DD, Guhl JF (1996) Neurological complications of ankle arthroscopy. Arthroscopy 12:200–208
- Glazebrook MA, Ganapathy V, Bridge MA et al (2009) Evidence-based indications for ankle arthroscopy. Arthroscopy 25:1478–1490
- Hensley JP, Saltrick K, Le T (1990) Anterior ankle arthroplasty: a retrospective study. J Foot Surg 29:169–172
- Hopper MA, Robinson P (2008) Ankle impingement syndromes. Radiol Clin North Am 46:957–971
- Japour C, Vohra P, Giorgini R et al (1996) Ankle arthroscopy: follow-up study of 33 ankles effect of physical therapy and obesity. J Foot Ankle Surg 35:199–209
- Junge A, Dvorak J (2013) Injury surveillance in the World Football Tournaments 1998–2012. Br J Sports Med 47:782–788
- McCarthy CL, Wilson DJ, Coltman TP (2008) Anterolateral ankle impingement: findings and diagnostic accuracy with ultrasound imaging. Skeletal Radiol 37:209–216
- 12. McMurray T (1950) Footballer's ankle. J Bone Joint Surg 32:68-69
- 13. Morris LH (1943) Report of cases of athlete's ankle. J Bone Joint Surg 25:220
- O'Donoghue DH (1957) Impingement exostoses of the talus and the tibia. J Bone Joint Surg 39:835–852
- Parkes JC II, Hamilton WG, Patterson AH et al (1980) The anterior impingement syndrome of the ankle. J Trauma 20:895–898
- Philbin TM, Lee TH, Berlet GC (2004) Arthroscopy for athletic foot and ankle injuries. Clin Sports Med 23:35–53
- 17. Robinson P (2007) Impingement syndromes of the ankle. Eur Radiol 17:3056-3065

- Russo A, Zappia M, Reginelli A et al (2013) Ankle impingement: a review of multimodality imaging approach. Musculoskelet Surg Suppl 2:S161–S168
- Scranton PE Jr, McDermott JE (1992) Anterior tibiotalar spurs: a comparison of open versus arthroscopic debridement. Foot Ankle 13:125–129
- Stephens MM, Kelly PM (2000) Fourth toe flexion sign: a new clinical sign for identification of the superficial peroneal nerve. Foot Ankle Int 21:860–863
- Tol JL, Slim E, van Soest AJ et al (2002) The relationship of the kicking action in soccer and anterior ankle impingement syndrome. A biomechanical analysis. Am J Sports Med 30:45–50
- 22. Tol JL, van Dijk CN (2006) Anterior ankle impingement. Foot Ankle Clin 11:297-310
- Tol JL, Verhagen RA, Krips R et al (2004) The anterior ankle impingement syndrome: diagnostic value of oblique radiographs. Foot Ankle Int 25:63–68
- 24. Tol JL, Verheyen CP, van Dijk CN (2001) Arthroscopic treatment of anterior impingement in the ankle. J Bone Joint Surg Br 83:9–13
- 25. Valkering KP, Golano P, van Dijk CN et al (2013) "Web impingement" of the ankle: a case report. Knee Surg Sports Traumatol Arthrosc 21:1289–1292
- 26. van Dijk CN, Tol JL, Verheyen CC (1997) A prospective study of prognostic factors concerning the outcome of arthroscopic surgery for anterior ankle impingement. Am J Sports Med 25:737–745
- Vaseenon T, Amendola A (2012) Update on anterior ankle impingement. Curr Rev Musculoskelet Med 5:145–150

# Chapter 12 Interview – Eva Blewanus

Pieter P.R.N. d'Hooghe and Gino M.M.J. Kerkhoffs

**Abstract** Eva is a sport physiotherapist and manual therapist who was trained at the Hogeschool van Amsterdam in the Netherlands. She has a broad experience as a physio- and manual therapist in professional football. She worked with the Royal Dutch Football Association (KNVB) from 2005 to 2011 and worked for Dutch premier league AZ from 2007 to 2012. AZ won the Dutch championship in 2008, 2009, and 2010 and the National Cup in 2011. She switched to AJAX Amsterdam in 2012 and is currently working with the professional AJAX female football team that plays in the BENE League.

P.P.R.N. d'Hooghe, MD (🖂)

G.M.M.J. Kerkhoffs, MD, PhD Department of Orthopedic Surgery, Academic Medical Centre Amsterdam,

Department of Orthopaedic Surgery, Aspetar Hospital, Aspire Zone, Doha, Qatar e-mail: pieter.orthopedie@gmail.com

University of Amsterdam, Meibergdreef 9, Amsterdam 1105 AZ, The Netherlands e-mail: g.m.kerkhoffs@amc.uva.nl



- What do you feel you add as a physical therapist to a players' injury rehabilitation? When a player is injured the physiotherapist, team physician, and, if necessary, a medical specialist together develop the rehabilitation program. The physiotherapist takes care of the patient on a daily basis. You correspond with the team physician regularly to monitor the player's progress and to adjust the rehabilitation program if necessary. As a team physiotherapist, you work with a player one-on-one during a longer period; hereby, you develop a relationship of trust with the player, which is beneficial for the rehabilitation.
- Do you feel that an injured player should always do his rehabilitation near the team?

The answer is not solely "yes" or "no;" it depends on the type of injury and the player's preference. Advantages for a rehabilitation program near the team are, for example, that a player will feel more connected with the team and can feel the support of the rest of the team during the rehabilitation. The last phase of sportspecific rehabilitation can be easier if the rehabilitation takes place near the team. Besides, as a team physiotherapist, it is easier to control the rehabilitation and to monitor the player's progress.

Rehabilitation near the team has also disadvantages such as that the injured player can interfere with the rest of team and it can be difficult for the player to be close to a team where he/she can not be part of because of his/her injury. My preference is a rehabilitation program near the team, but in case of a longer rehabilitation period, it should be possible for a player to rehabilitate somewhere else.

#### • Can you describe your role in the medical staff?

The medical staff of the team where I am part of consists of two physiotherapists and one sports medicine physician. Sometimes, also exercise physiologists and human movement scientists are involved to monitor the physical condition and recovery of the players. We are a "performance team" within a larger team of medical specialists and paramedics who work for the club. Our common goal is to improve the performance of the players to keep them fit (prevention), to get them fit after an injury (cure), and to improve their health by offering them support with sport-related issues, such as nutrition, recovery, and individual performance programs.

Together with my colleague, we take care of the necessary preparations and posttreatments for the practices and matches (e.g., mobilization, strapping, detonizing). During a game, I am responsible for the treatment of injuries sustained during the game and to make the decision if a player can play with an injury or not. Besides, I am responsible for an individual performance program for each player based on his or her injury risk profile. This performance program also consists of injury prevention. Furthermore, I am strongly involved with the rehabilitation of the injured players. I am in service of the team and contribute to an optimal environment for the elite player.

What do you expect from the team coach?

Trust and mutual respect for each other and each other's work. I think that those are the most important aspects for a good working relationship with a coach. In which clear communication has a key role. I expect from the coach that he will give me insight in his training schedule, so I can take this into consideration for the rehabilitation of the injured players, for example, to effortlessly make the transition to the team practices. Furthermore, I appreciate it when a coach gives me insight in the content of the practices so that I can advise the coach and the players who are not completely fit which exercises they can or cannot do and whether a player can make the transition to group practices. Moreover, I expect a coach to pay attention to injury prevention, which sometimes requires exercises outside the soccer field.

On the contrary, it is necessary that the medical staff informs the coach on the ability of the players, presence of injuries, and the expected return to playtime; hereby the coach can adjust the training exercises and make the line up on time. I expect from the coach that he will take my advice into consideration. Yet, a coach will only take advice into consideration if he is confident about the quality of the medical staff.

- How important is it for you as a physical therapist that a player is 100 % fit to play? Or how fit should a player be in order to advise him to return on the team? *I think it is important that a player is completely recovered after an injury before he/she returns to play matches. When a player returns to sports too soon, it will increase the risk on reinjury or delayed recovery. In case of a more serious injury, we will conduct "return-to-play" tests to measure the player's physical capacity to resume full training load. If a player practices again with the team for some time and the physical condition is also on the desired level, the player can gradually increase the minutes he can play during a match. In case of minor injuries or early signs of overtraining, the medical staff can decide to adjust the player's training schedule in frequency or content. It is possible that we advise the coach not to let a player play a complete match. These decisions are always in agreement with the player; the history of previous injuries and the type of injury are taken into consideration.*
- You are working with male as well as with female football players; is there a difference in physical fitness, training schedule, and injury prevention program? There is definitely a huge difference between men's and women's soccer. Despite women's soccer being the fastest growing sport for girls and women it is less professional compared with men's soccer. This is understandable but the fact remains that you have to recognize the differences and have to take these differences into consideration as medical or technical staff. The female players of my team are offered many facilities and a small financial compensation, yet this is not enough for daily living expenses. Besides playing soccer they have to work or study. On average there are six practices and one match in a week; this is less when compared with the men's soccer on the elite level. Because the players have to work or study besides playing soccer, they have less time to recover and rest, and therefore, they are at increased risks, for example, to over train. Furthermore, there is a difference between physical condition between men and women such as physical stature, muscle strength, and hormonal differences. These physical differences account for the difference in injury prevention programs for men and women.
- What's your philosophy on injury prevention, and is injury prevention a topic in physical therapy courses?

Injury prevention is one of the most important tasks of the medical staff. Moreover, injury prevention has gained a lot of attention within the past years. At the start of the season, I develop an individual injury prevention program for each player based on the player's risk profile; this program consists of strengthening and stability exercises. You can clearly see the improvement in strength and physical capacity of the players during the season.

The subject "prevention" is also part of the postgraduate training sports physiotherapy. Injury prevention was a neglected subject in the undergraduate study physiotherapy when I completed it 8 years ago. I am unaware if injury prevention is implemented in the study at the moment. • Do you think injury prevention is also the responsibility of coaches and players? *Yes, absolutely! Injury prevention is the responsibility of the whole team including the coach. As I said before, I expect a coach to reserve time in his trainings schedule for injury prevention, as it is an important part of elite sports.* 

We expect a certain degree of self-responsibility from the players as they have to do part of the prevention programs by themselves and sometimes also away from the club. If they fail to do this or less serious than we expect, it is clear that the injury prevention programs will be less effective. Overall physical ability is also part of injury prevention; this is for the most part the responsibility of the player. It can be difficult for a player to truthfully answer questions about overtraining and fatigue, because a player wants to play as many matches as possible. Yet, we expect the players to pay attention to their work and rest schedule as well as their nutrition and to report physical complaints. Advice and guidance is offered, but in the end the player has his or her own responsibility.

- Who will win World Cup 2014? Brazil
- Who will win Women's World Cup 2015? USA

# Chapter 13 Posterior Ankle Impingement

Gino M.M.J. Kerkhoffs, Peter A.J. de Leeuw, and Pieter P.R.N. d'Hooghe

Behind every kick of the ball there has to be a thought.

Dennis Bergkamp

**Abstract** The posterior ankle impingement syndrome is a common cause of ankle pain in football players. It is characterised by posterior ankle pain on forced plantar flexion movements. Clinical investigation reveals pain on palpation along the posterolateral and/or posteromedial ankle joint line. There is recognisable pain on forced plantar flexion which can be limited as compared to the contralateral side. An infiltration with 1 cc anaesthetic posterolateral over the pain spot that temporarily reduces the pain is an additional proof for the diagnosis posterior impingement pain. In patients with posterior impingement pain, the plain X-rays can be negative and are therefore insufficient. An additional straight lateral (posterior impingement) view is required for visualisation of posterior bone structures, i.e. an os trigonum or an elongated posterior talar process that can cause the posterior impingement pain. The effectiveness of conservative treatment has not yet been well documented. After conservative treatment fails, an arthroscopic intervention is the treatment of choice for professional football players. Posterior ankle arthroscopic release of the retinaculum of the flexor hallucis longus with reduction of the elongated posterior

G.M.M.J. Kerkhoffs, MD, PhD ()

Department of Orthopaedic Surgery, Orthopaedic Research Center Amsterdam, Academic Medical Center, Meibergdreef 9, Amsterdam 1105 AZ, The Netherlands e-mail: g.m.kerkhoffs@amc.nl

P.A.J. de Leeuw, MD Department of Orthopaedic Surgery, Academic Medical Center, Meibergdreef 9, Amsterdam 1105 AZ, The Netherlands e-mail: p.a.deleeuw@amc.nl

P.P.R.N. d'Hooghe, MD Department of Orthopaedic Surgery, Aspetar Orthopaedic and Sportsmedicine Hospital, Aspire Zone, 29222, Doha, Qatar e-mail: pieter.dhooghe@aspetar.com talar process or removal of the os trigonum or excision of loose bodies is known to be successful in football players, without signs of joint space narrowing. The majority of professional football players will return to their former level within 2 months after surgery.

Keywords Ankle • Impingement • Flexor hallucis longus • Arthroscopy • Football

#### 13.1 Introduction Including Epidemiology

Posterior ankle impingement syndrome is a common cause of chronic ankle pain in football players. The overall percentage of ankle injuries in elite football is 19 % [3], but the exact prevalence and incidence for posterior ankle impingement has not been documented. The typical clinical features are posteriorly located ankle pain, which increases with forced hyper plantar flexion. The cause can either be soft tissue or bony impingement.

In the early 1930s – and mainly because of its anatomical features – the ankle joint was found unsuitable for arthroscopy. Forty years later, in 1970 Tagaki and later Watanabe made considerable contributions to the arthroscopic surgery of the ankle, and the latter published a series of 28 ankle arthroscopies in 1972.

Over the last three decades, arthroscopy of the ankle joint has become a standardised and important procedure, with numerous indications for both anterior and posterior intra-articular pathology, as well as for tendinous problems around the ankle.

The advantages of ankle arthroscopy are the direct visualisation of the structures, improved assessment of the articular cartilage, faster rehabilitation and earlier resumption towards sports.

There is nowadays enough evidence that there is a limited value in performing a diagnostic arthroscopy because of the increased imaging modalities in ankle pathology.

However, because of the lack of direct access and the nature and deep location of its hindfoot structures, posterior ankle problems still pose a diagnostic and therapeutic challenge nowadays.

Historically, the hindfoot was approached by a three-portal technique, i.e. the antero-medial, anterolateral and posterolateral portals, with the patient in the supine position. It is known that the traditional posteromedial portal is associated with potential damage to the tibial nerve, the posterior tibial artery and its surrounding tendons locally.

Therefore, a two-portal endoscopic technique was introduced in 2000 by Van Dijk et al. [9], and since then, this technique has shown to give a safe [4] and excellent access to the posterior ankle compartment, the subtalar joint and the surrounding extra-articular posterior ankle structures [6, 9].

Posterior ankle arthroscopy has shown the need for specific anatomical knowledge, has modified classic arthroscopic tools and skills and has introduced a broad spectrum of new indications in posterior ankle pathology.

## **13.2 Functional Anatomy**

As for any pathology, anatomical knowledge is essential in the understanding and treatment of this pathology. Anatomical knowledge is particularly important in ankle arthroscopy since the significant risk of associated complications can be prevented or decreased only by profound familiarity with the anatomy of the region.

The main anatomical structure for the orientation and to determine the safe working area is the flexor hallucis longus tendon (FHL). Just medial to this tendon runs the posterior neurovascular bundle (tibial nerve and posterior tibial artery and veins).

The posterior ankle arthroscopy should therefore routinely be performed lateral to the FHL tendon. Proper positioning of the ankle and the hallux results in better visualisation of the tendinous portion of the FHL muscle and avoids unnecessary resection of some of the muscle fibres that reach the lateral tendinous border in a semipeniform morphology.

Plantar flexion of the ankle or hallux flexion facilitates visualisation of the FHL tendon proximal to the lateral talar process.

Also obligatory for the orientation during posterior ankle arthroscopy are the posterior ankle ligaments. The most distal and vertically orientated ligament is the talocalcaneal ligament, originating from the posterior talar process to the calcaneus. Superior to this ligament, also originating from this process, is the posterior talofibular ligament (PTFL), running in a slight oblique orientation to insert in the medial border of the posterior distal fibula. Both the posterior talocalcaneal ligament and the PTFL need to be (partly) released in case of removal of a symptomatic os trigonum/hypertrophic posterior talar process.

Superiorly to the PTFL, the posterior intermalleolar ligament, also called the tibial slip in arthroscopic literature, is recognised (Fig. 13.1) and proximal to this ligament is the posterior tibiofibular ligament which is composed of a superficial and deep component or transverse ligament. A distinction between the transverse and intermalleolar ligament can easily be made by dorsiflexion of the ankle [8].

In order to gain arthroscopic access to the talocrural joint, the posterior intermalleolar ligament and the transverse ligament need to be lifted and/or partly excised. Until now no literature is present on the function of these ligaments, but most probably these can be at least partly removed without significant consequences.

## **13.3** Aetiology and Injury Mechanism

In the ankle, the posterior area is mostly well protected from trauma, and posterior impingement complaints therefore mostly originate from repetitive strains or overuse as is seen in professional (ballet) dancers. In contrast, in football players, the



Fig. 13.1 Arthroscopic images indicating the important anatomical landmarks during posterior ankle arthroscopy in a right ankle. Superior to the subtalar joint (\*), the posterior talofibular ligament is orientated and proximal to this ligament is the tibial slip (TS). By elevation of the TS, the ankle joint (\*\*) can be entered. Prior to treatment of any pathology, the flexor hallucis longus (*FHL*) must be identified

main cause is a trauma to the posterior part of the ankle joint; this can be an inversion injury with an additional injury to the posterior ligaments or a direct trauma from a football shoe into the posterior area causing a fracture of the posterior talar process, an avulsion fracture of the posterior ligament complex, or an instability in the pseudo-joint of the os trigonum. The latter will then remain symptomatic in most cases because there is now instability in this joint allowing (painful) motion of the os trigonum in relation to the posterior talar process (Fig. 13.2). The pain in posterior impingement syndrome can originate from a number of causes, as mentioned above already: a traumatised os trigonum, a fracture of posterior talar process, an avulsion fracture of the posterior ligament process, a tear in the posterior ligament process, a flexor hallucis longus tendinitis and an impingement of a bulky distal flexor hallucis longus tendon in the flexor retinaculum in dorsal flexion of the hallux. Typically in football players, pain is experienced on kicking, when the posterior structures are crushed in the little posterior space between the Achilles tendon, talus and distal tibia (Fig. 13.3). Recurrent trauma to the soft tissue component can lead to hypertrophy of the synovial layer, subsynovial fibrotic tissue formation and infiltration of inflammatory cells and can thus cause a posterior soft tissue impingement.



Fig. 13.2 Schematic drawings of the talus. (a) At the posterolateral aspect of the talus, the posterolateral talar process can be identified. This bony prominence, forming the groove for the flexor hallucis longus (\*) together with the posteromedial talar process, can be enlarged (hypertrophic) and can subsequently cause a posterior ankle impingement. (b) In case the posterolateral talar process is not fused after the ossification is finished, it is called an os trigonum and could also become symptomatic



Fig. 13.3 With repetitive trauma in the posterior ankle compartment, specifically kicking the ball in football, the anatomical structures become compressed in between the Achilles tendon, the talus and the distal tibia which can result in posterior ankle impingement

## 13.4 Clinical Features

The typical symptomatic football player will present with a history of previous ankle injuries [3]. On history taking, the main symptoms are persistent posteriorly located ankle pain during plantar flexion movements or while kicking the ball, postexercise ankle swelling and restricted dorsal as well as plantar flexion. With an adapted training programme, most players can continue playing. Sometimes taping the ankle joint can help.

Physical examination reveals recognisable pain on palpation along the posterolateral and/or posteromedial part of the ankle joint. The patient will recognise the pain on palpation or on provocation with plantar flexion provocation test. In plantar flexion the flexor hallucis longus tendon is further entrapped in the retinaculum and the posterior structures are crushed in the narrowed posterior ankle space, inducing pain and possible synovial swelling. The optimal palpation position is at slight ankle plantar flexion.

## 13.5 Diagnostic Imaging

Standard weight-bearing lateral and anteroposterior radiographs can detect a posteriorly located os trigonum but might be false negative. Due to the posterior overlap with the fibula in the standard lateral radiograph, there is a possible over-projection and an os trigonum or an elongated posterior talar process might be missed. The straight lateral ankle (posterior impingement) view is recommended to detect the posterior osseous structures causing impingement. As compared to the standard lateral projection, the beam is tilted into a 90° craniocaudal direction with the leg in neutral position and the foot in neutral flexion (Fig. 13.4). Routine computed tomography (CT) and/or magnetic resonance imaging (MRI) for the posterior impingement syndrome is not advised, although MRI has a high sensitivity to detect the (posteromedial) soft tissue impediments.

Ultrasound and conventional MRI have a debatable role on the footballers' population.

A CT scan is most sensitive in detecting small calcifications in the posterior ligament complex or small loose bodies as well as a small os trigonum or even a Cedell fracture. Ultrasound might be helpful, since hypervascularity of the synovial or meniscoid mass seems to depend on the repetitive injuries and the amount of the fibrosis in it. An additional advantage of sonography over MRI is the possibility to directly infiltrate the inflamed tissue with steroids in a controlled manner, most suitable posterolaterally.

Although some authors have proposed MR arthrography enabling a high sensitivity and specificity as an additional diagnostic tool to diagnose posterior ankle impingement, no radiographic series have been published in literature until now. On an MR arthrography, an irregular or nodular contour of the posterior soft tissues is considered to be pathological and would be highly correlated to ankle scar tissue



**Fig. 13.4** (a) Standard lateral weight-bearing radiograph of the ankle can be negative in relation to a prominent posterior talar process/os trigonum (\*). (b) By tilting the beam into a 90° craniocaudal direction with the leg in neutral position and the foot in neutral flexion, bony anomalies in the posterior ankle can more precisely be exposed (\* os trigonum)

and synovitis at arthroscopy. Our conclusion is that posterior impingement is a clinical diagnosis and imaging assists in understanding the cause of the posterior impingement pain as well as in facilitating the meticulous preoperative planning that is the secret to success.

# 13.6 Classification

Until now there is not a uniform classification system for posterior ankle impingement.

# 13.7 Treatment

## 13.7.1 Conservative Treatment

Conservative treatment, consisting of intra-articular injections and physiotherapy, is recommended in the early stages but has never been systematically studied and might frequently be unsuccessful by itself.



**Fig. 13.5** Arthroscopic image of the posterior ankle compartment in a right ankle indicating the good exposure of the subtalar joint (\*\*). Again proximally to the subtalar joint, the posterior talofibular ligament can be identified (*PTFL*), inserting in the posterolateral talar process (\*\*\*). The flexor hallucis longus (\*) should always be identified prior to treating posterior ankle pathology through posterior ankle arthroscopy

## 13.7.2 Invasive Treatment

Hindfoot endoscopy enables the surgeon to more easily assess the posterior ankle compartment (Fig. 13.5), as compared to open surgery. Also it compares favourably to open surgery with regard to an overall lesser morbidity and quicker recovery [2, 5, 10].

In the initial description of the technique, the main indications to perform a posterior ankle arthroscopy were the treatment of an os trigonum and FHL pathology (Fig. 13.6). Nowadays, however, numerous ankle pathologies in our athletes can be treated through this minimal invasive technique and still indications are added.

The procedure is carried out in an outpatient setting under general or spinal anaesthesia [5, 9]. The patient is positioned in the prone position with a tourniquet above the knee at the affected side. The affected ankle is positioned just over the edge of the operation table and is supported to allow free ankle movement (Fig. 13.7).



**Fig. 13.6** Arthroscopic image in a right ankle treating the os trigonum pathology. (a) The os trigonum is released from the posterior talofibular ligament with the use of a punch. (b) Subsequently the os trigonum is removed with a grasper (\* flexor hallucis longus). (c) Postoperative situation; fraying of the flexor hallucis longus (\*) can be visualised, indicating the prior impingement by the os trigonum

The anatomical landmarks for portal placement are the sole of the foot, the lateral malleolus and the medial and lateral borders of the Achilles tendon. With the ankle in the neutral position (90°), a straight line, parallel to the sole of the foot, is drawn from the tip of the lateral malleolus to the Achilles tendon and is extended over the Achilles tendon to the medial side (Fig. 13.8). The posterolateral portal is located just proximal to – and 5 mm anterior to – the intersection of the straight line with the lateral border of the Achilles tendon (Fig. 13.9).

The posteromedial portal is located at the same level as the posterolateral portal, but on the medial side of the Achilles tendon.



Fig. 13.7 Patient positioning in posterior ankle arthroscopy; the hip at the affected side is supported, a tourniquet is applied and the lower leg is elevated allowing free ankle movement

Fig. 13.8 The lateral malleolus is marked; a hook can be used to identify the level of the posterolateral and medial portal. These are located at the level of the lateral malleolus, lateral and medial to the Achilles tendon



Before addressing any pathology, the FHL tendon should be localised since just medially to it, the posterior neurovascular bundle is located. Therefore, the FHL tendon determines the working area, which is basically only laterally to this tendon. Once this working area is determined, the whole spectrum of posterior pathology can be treated supero-inferiorly from the talocrural over the subtalar joint towards the Achilles tendon insertion and mediolaterally from tarsal tunnel release towards the peroneal tendons (Fig. 13.10).

**Fig. 13.9** Right ankle in which the posterolateral and posteromedial are marked



Now the pathology can be addressed, ranging from debridement of soft tissue to the removal of a hypertrophic posterior talar process (Fig. 13.11), an os trigonum or the release of the FHL tendon from its adjacent structures.

## 13.8 Rehabilitation

Postoperative rehabilitation treatment consists of a compression bandage and partial weight bearing for 3–5 days. The athlete is instructed to actively plantar and dorsi-flex his or her ankle and foot upon awakening and to repeat this exercise a few times



Fig. 13.10 Determining the working area for arthroscopy in posterior ankle pathology. (a) During the insertion, the arthroscope is aiming towards the first webspace, in between the first and second toe. This enables the surgeon to determine the safe working area. (b) The arthroscope is inserted through the posterolateral portal and the working instrument through the posteromedial portal. The shaft of the arthroscope is used to safely introduce the working instrument. Any instrument introduced through the medial portal needs to slide over the shaft anteriorly (*red arrow*) until the posterior ankle compartment is reached to avoid neurovascular injuries

every hour for the first 2–3 days after surgery [7, 8]. The added value of physical therapy has insufficiently been documented. A small retrospective series showed that patients receiving more than 1-month physical therapy scored better on a 7-point ankle scale as compared to the ones who were not treated with physical therapy [1]. In the absence of high-level evidence, in professional football players, physical therapy with a focus on restoration of dorsiflexion, reduction of swelling, functional training and supervised return to sport-specific training after 6–8 weeks is advised.



Fig. 13.11 Arthroscopic image of a left ankle indicating the removal of a hypertrophic posterior talar process. (a) With the use of a chisel, the hypertrophic segment (\*\*) is detached from the talus, (b) detachment of the segment without iatrogenic FHL damage (\*), (c) postoperative situation, the segment is removed without iatrogenic damage to the subtalar joint

## 13.9 Conclusion

Posterior ankle impingement is not always just a bony pathology, frequently a soft tissue impediment with or without a bony component must be considered. Posterior ankle arthroscopy is a challenging, safe, reliable and effective technique in the treatment of posterior ankle impingement. Due to the improved functional outcome after surgery and a quicker rehabilitation time, (football) athletes can hugely benefit from this technique. The initial indications included flexor hallucis longus and os trigonum pathology. Nowadays, however, the technique can be used for an increasing amount of posterior ankle pathologies.

How to Diagnose Posterior Ankle Impingement:

- Ask for sport-specific repetitive ankle movements
- Perform a hyper plantar flexion movement of the ankle
- · Look for palpatory pain along the course of the flexor hallucis longus

How to Treat Posterior Ankle Impingement:

- Perform a diagnostic injection
- Start with the standardised 2-portal hindfoot technique after initial cadaveric training
- Search for the flexor hallucis longus tendon and the posterior talofibular ligament as these are the main anatomical landmarks

## References

- 1. Japour C, Vohra P, Giorgini R et al (1996) Ankle arthroscopy: follow-up study of 33 ankles effect of physical therapy and obesity. J Foot Ankle Surg 35:199–209
- Jerosch J, Fadel M (2006) Endoscopic resection of a symptomatic os trigonum. Knee Surg Sports Traumatol Arthrosc 14:1188–1193
- Junge A, Dvorak J (2013) Injury surveillance in the World Football Tournaments 1998–2012. Br J Sports Med 47:782–788
- Lijoi F, Lughi M, Baccarani G (2003) Posterior arthroscopic approach to the ankle: an anatomic study. Arthroscopy 19:62–67
- Scholten PE, Sierevelt IN, van Dijk CN (2008) Hindfoot endoscopy for posterior ankle impingement. J Bone Joint Surg Am 90:2665–2672
- Sitler DF, Amendola A, Bailey CS et al (2002) Posterior ankle arthroscopy: an anatomic study. J Bone Joint Surg Am 84:763–769
- 7. Tol JL, Verheyen CP, van Dijk CN (2001) Arthroscopic treatment of anterior impingement in the ankle. J Bone Joint Surg Br 83:9–13
- van Dijk CN, Kerkhoffs GM, de Leeuw PA, van Sterkenburg M (2013) Chapter 88. Periarticular endoscopy. In: Johnson DH, Amendola A, Barber AF, Field LD, Richmond JC, Sgaglione NA (eds) Operative Arthroscopy, 4th edn. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins; pp 992–1012
- 9. van Dijk CN, Scholten PE, Krips R (2000) A 2-portal endoscopic approach for diagnosis and treatment of posterior ankle pathology. Arthroscopy 16:871–876
- Willits K, Sonneveld H, Amendola A, Giffin JR, Griffin S, Fowler PJ (2008) Outcome of posterior ankle arthroscopy for hindfoot impingement. Arthroscopy 24:196–202

# Chapter 14 Interview – Ron Spelbos

Pieter P.R.N. d'Hooghe and Gino M.M.J. Kerkhoffs

**Abstract** Spelbos played for several clubs including AZ Alkmaar, Club Brugge, and Ajax Amsterdam. He was a tough defender with a big personality. He won the Dutch Championship twice with AZ and Ajax and was a finalist of the UEFA Cup with AZ and a winner and finalist of the Europa Cup II with Ajax. He was also Dutch international (21 caps, 1 goal) and was part of the team that qualified for the European Championship (an injury prevented his appearance in the championship) in 1988 when the Netherlands won the European Football Championship. As a coach Ron Spelbos worked for FC Twente, NAC, Vitesse, and FC Utrecht. He won the championship of the first league with NAC. Currently he is head scout of the Dutch National Team, in preparation for the World Cup in Brazil. He represents a generation of international top scouts with a successful history as a player in football at the highest level.

P.P.R.N. d'Hooghe, MD (🖂)

G.M.M.J. Kerkhoffs, MD, PhD

Department of Orthopedic Surgery, Academic Medical Centre Amsterdam, University of Amsterdam, Meibergdreef 9, Amsterdam, 1105 AZ, The Netherlands e-mail: g.m.kerkhoffs@amc.uva.nl

Department of Orthopaedic Surgery, Aspetar Hospital, Aspire Zone, Doha, Qatar e-mail: pieter.orthopedie@gmail.com



• Do you notice a difference in attitude of the current young players when compared to your generation?

In our days the young players gathered their experience on the streets in the spontaneous matches between players of different ages. A young player had to adapt to the different circumstances in these matches. Nowadays the young players are with players of their own age the whole year and they are at the club the entire week, so there is no time for difficult street matches against older players. All is taken care of, that makes a big difference. Besides that it is still very difficult to reach the absolute top and it takes a lot of discipline. The altered circumstances for the young players are a fact, but these players still need to invest in themselves and need a lot of intrinsic motivation to be the best.

• What are the criteria to be successful in scouting young players? Obviously there are many aspects to judge in the young players. The player needs to have good technical as well as tactical skills, but there are also the mental and physical aspects that need to be evaluated. A player should have the skills to take the right decisions in ample time and to be able to orientate in small as well as open space. Also it is important that a player is able to restrict himself to certain tasks if this is needed with a certain play that the team chooses to perform; detailed restriction to certain tasks is also important in ball possession, ball loss, and counter play.

- At what age does the scouting start in modern football? In professional football scouting starts at about 6 years. Players are transferred from amateur clubs to professional clubs at a very young age and sometimes even transferred to another country. Also within clubs scouting is started at a young age to provide best education to the young football player.
- What does the organization of the scouting program look like within a professional football club?

Each club has their own system, depending on the club structure and the responsibilities of the scouts. Normally a head scout will do the organization of the scouting program and will give direction to the tasks of the other scouts. The head scout will send his colleagues out with a clear task or without a task, dependent on position and qualities of both scout and club. All scouts will report to the head scout, and he will do the debriefing with the coaches and with medical team.

- What is the difference between your normal work as a scout and your work as a scout for the Dutch national team in preparation for the World Cup? Sorry, I cannot answer this question in all honesty without getting into detail on our opponents, so I will not answer it at this point in time. Ask me again after the World Cup.
- What is the difference between a young talent and a potential top player? A talented young player has shown his qualities to become a good football player. His talents need further development and he has to mature as a player. A potential top player is further in his development and has proven his value on a certain level of play, he has proven to be able to cope with bigger problems on the pitch, and he is ready for the "big matches."
- How important is the physical shape of a player for you as a scout? The physical abilities of a player are of upmost importance, because physical fitness will contribute to the performance on the pitch. A player that is not fit will play less engaged and will most probably fail to perform all tasks that are needed in his position on the field and in the team. This can have a negative influence on the team performance.
- As a scout are you up to date with the medical history/injury stats of a player? To a certain level, yes. I know the injury pattern of a player and why he did or did not play in a certain time period. Also I am used to know all major injuries in a

players' career, but the team doctor is the one to guard all details on the medical history and injury pattern and prognosis; therefore, it is important to have an open communication with the medical staff.

• Who will win the World Cup 2014?

From my position it is obvious to answer the Netherlands, but I believe that there are a number of teams that can go for the title, and there are numerous factors that will make the new champions.

# Chapter 15 Ankle Fractures, Including Avulsion Fractures

Kyriacos I. Eleftheriou, James D.F. Calder, Peter Kloen, and Pieter P.R.N. d'Hooghe

Difficulties break some men but make others.

Robben Island nr. 4664 Nelson Rolihlahla Mandela

**Abstract** The incidence of fractures is small and less than 3 % of all ankle injuries in modern professional football. Optimum management for the higher-level football player has to address the demand for early and safe return to a high level of activity. Sport-specific evidence for best practice is limited. A thorough history, examination and adequate imaging are essential to correctly diagnose injuries and decide on best treatment. Early rehabilitation can allow early return to sport within 2–4 months. Surgical reduction when indicated and provision of stability by fixation can allow this and appear to be advisable to optimise both the outcomes and return to competition in football-related ankle fractures.

Keywords Ankle • Fracture • Football • Avulsion fracture

K.I. Eleftheriou (⊠) Hippocrateon Private Hospital, Nicosia, Cyprus e-mail: akis@dreleftheriou.com

J.D.F. Calder Chelsea and Westminster Hospital, London, UK

Fortius Clinic, London, UK

P. Kloen Academic Medical Centre, Amsterdam, The Netherlands

P.P.R.N. d'Hooghe, MD Department of Orthopaedic Surgery, Aspetar Hospital, Doha, Qatar e-mail: pieter.orthopedie@gmail.com

P.P.R.N. d'Hooghe, G.M.M.J. Kerkhoffs (eds.), *The Ankle in Football*, Sports and Traumatology, DOI 10.1007/978-2-8178-0523-8\_15, © Springer-Verlag France 2014

## 15.1 Introduction

The incidence of ankle fractures in football is fortunately small and less than 3 % of all ankle injuries in the modern professional game (Chap. 2). Their management for the higher-level football player has to address the demand for early and safe return to a high level of activity. This can be challenging but is helped by the generally healthy physiology of the athlete and the availability of a highly supportive team and equipment to enhance rehabilitation.

#### 15.2 General Assessment

A thorough history to ascertain the mechanism of the football ankle injury can help guide the physician to the possibility of a specific injury, as initial presentation is often similar. The use of video technology is now readily available and can help analyse the mechanism of injury (Chap. 2). Most footballers have minimal co-morbidity that needs to be considered, but the pre-injury status of ankle function and symptoms need to be taken into account, providing information with regard to full recovery and the optimal treatment for the athlete, which may involve addressing previous ankle pathology at the same time.

A detailed examination needs to be undertaken, to exclude any other significant injuries and assess limb vascularity, skin viability and injury to nearby structures. Any obvious deformity needs to be reduced and splinted early to minimise further soft tissue compromise and help with pain. In the absence of deformity, clues to the possibility of a fracture include the inability to bear weight both immediately after injury and on later assessment and the presence of bony tenderness around the ankle: other than the malleoli, it is important to palpate the calcaneus and the area of its anterior process, the talus (head, neck and processes) and the base of the fifth metatarsal. The knee joint also needs to be examined for tenderness, especially at the proximal fibula which may be suggestive of a Maisonneuve-type injury. Although not usually possible to assess initially, pain on external rotation is suggestive of an injury to the syndesmosis.

The history and examination will guide further investigations. The Ottawa rules may be applied in deciding when to get plain radiographs, but we feel that especially in the high-level athlete, a lower threshold to image is important in order to avoid any delay in diagnosis and missing any more subtle fractures. We regularly use MRI scans to detect any concomitant injuries (e.g. soft tissue, osteochondral injuries). In the presence of fractures, CT scans can provide a more detailed picture of the configuration and help with preoperative planning.

### **15.3 General Management**

Overall treatment follows the usual principles of fracture management. Further treatment will depend on the actual type of injury sustained, and this is discussed below for both common fractures around the ankle as well as the more subtle injuries that may be missed as a sprain. The evidence on best treatment in ankle injuries in football or sports is discussed when available, but it is obvious that this may be limited in specific injuries, and advice based on general evidence and our own experience is provided. With involvement of even younger people in football, the treatment of paediatric and transitional fractures around the ankle is also discussed. The management of osteochondral fractures (Chap. 4) and stress fractures (Chap. 8) are thoroughly discussed elsewhere.

## 15.4 Malleolar Fractures

The stability of the ankle mortise depends on its bony configuration and surrounding ligaments (inferior tibiofibular complex or syndesmosis, and the lateral and medial ligaments). Nondisplaced, stable malleolar and isolated fractures of the medial malleolus can be treated nonoperatively [28]. Dislocated or unstable malleolar fractures are generally treated with anatomic open reduction and internal fixation (ORIF), as in a weight-bearing joint exposed to 1.25–5.5 times body weight, a malunion will lead to post-traumatic arthritis. Despite anatomic reduction there is still a risk of this [70].

Standard AP, lateral and mortise (20° internal rotation) views are needed for initial diagnosis. The two most common classification systems are the Weber-Danis and the Lauge-Hansen. The former is based on the level of the lateral malleolar fracture: type A (below the tibial plafond), B (oblique or spiral fracture at or near the level of the ankle) and C (fracture higher than the ankle joint). The more proximal the fracture is, the higher the risk of a syndesmotic injury. Although easier to use than the Lauge-Hansen, Weber only described the lateral injury. Lauge-Hansen described four distinct injury patterns based on cadaveric studies and suggested that most ankle fractures are supination-external rotation (SER) injuries and differentiated between SER-II and SER-IV injuries. SER-II can be stable, but in type IV there is medial disruption, either with a fracture of the medial malleolus, or rupture of the deltoid ligament (SER-IV equivalent). Medial tenderness, ecchymosis and swelling may be unreliable clues to diagnose a medial injury, so inversion stress or gravity radiographs can be helpful to determine whether there is medial instability [78].

A systematic review of ankle fractures was recently published [70]. A total of 1,822 fractures were indentified with a mean follow-up of 5.1 years. Outcome was not perfectly related to the Weber classification, with type A fractures having an 82.7 %, type B an 83.8 % and type C a 70.4 % good/excellent outcome. A subclassification of type B injuries showed that SER-II had a 92.2 %, whereas SER-IV an 81.7 % good/excellent outcome. It was suggested that the unexpected finding that Weber A injuries did not do better than type B was due to the former involving a supination-adduction trauma with impaction of the medial compression of the cartilage of talus and tibia. Weber B and C fibula fracture patterns are most often fixed with a lag screw and a neutralisation one-third tubular plate in a lateral or posterior (antiglide) position. In fractures with a medial malleolar involvement, the use of a tension band wire or two bicortical lag screws is a good technique [62]. If the syndesmosis is unstable after fixation of the fibula and medial malleolus, this is fixed.

Direct visualisation or radiographic intraoperative assessment is recommended, as there is high risk of malreduction [47]. There is no consensus on the best technique for syndesmotic stabilisation, with most opting for one bicortical (3.5 mm) screw. The use of TightRope<sup>®</sup> (Arthrex, USA) for syndesmotic fixation has also been introduced, whilst minimally invasive plate osteosynthesis (MIPO) techniques for ankle fracture fixation have also been described. Mostly used for highly comminuted fractures of the fibula using a bridging MIPO, the technique was shown to be safe [29]. The use of small or mini fragment low-profile fixation plates may be a good alternative for medial and lateral malleolus fixation [3, 7].

The treatment of an associated posterior malleolus fracture (part of a trimalleolar ankle fracture) is still a matter of controversy [34]. The posterior malleolus fracture pattern is heterogeneous, and there is no consensus on its optimal treatment. There is poor reliability and accuracy in measuring the size of a posterior fragment on plain films, and CT imaging is much better. Haraguchi et al. described various morphologies of the posterior malleolus fracture [27], but their reproducibility and clinical use remains to be investigated. The posterior malleolus fails in stage III of supinationexternal rotation injuries or in stage III of pronation-abduction injuries. A recent meta-analysis showed no consensus on how to address the posterior malleolus fracture, primarily because there is no standardisation of functional outcomes [78]. Several reports have recommended fixation for involvement of greater than a certain percentage (>25 % or >33 %) of the tibial plafond. If the lateral malleolus is reduced anatomically, the posterior malleolus tends to reduce by means of ligamentotaxis of the posterior inferior tibiofibular ligament (PITFL). Recent studies have suggested that fixation of posterior malleolus fractures might obviate the need for syndesmotic stabilisation altogether [47]. Reduction of a posterior malleolus can be done indirectly (an intact PITFL is necessary) or directly. Screws can be placed percutaneously from anterior to posterior, but lag screws from posterior to anterior are better biomechanically. An open approach using the interval between the flexor hallucis longus (FHL) and the peroneal tendons is common, although others advocate lateral retraction of the FHL or medial retraction of the peroneal tendons. This can be achieved with the patient prone where by external rotation of the leg the medial malleolus fracture can also be fixed [1, 43, 76]. When the medial malleolus is not fractured, the patient can be placed on the lateral position, whilst the use of a medial approach to address both the medial and posterior malleoli was reintroduced by Bois [11].

## 15.5 Distal Metaphyseal Tibial Fractures

A distal metaphyseal tibial fracture is an extra-articular fracture that extends to within 4 cm of the plafond, whilst some also refer to such fractures that have simple extension of a nondisplaced fracture line into the plafond [8]. The distal tibia has three surfaces, the lateral, posterior and medial surface. The medial surface is mainly subcutaneous and forms the medial malleolus distally, whilst posterior and lateral surfaces are for muscle attachments. The cross-section is triangular as

opposed to the circular diaphysis. The blood supply to this area is divided between an outer one-third (periosteal vessels) and inner two-third (nutrient vessels) supply.

The injury mechanism is usually a torsion or compression load. Compression injuries are usually high-energy with substantial soft tissue compromise and comminution, with often an intra-articular extension blurring the distinction between a such a distal metaphyseal tibial fracture and a pilon fracture. Classification of an associated soft tissue injury is important and assessment of the neurovascular status paramount, with a risk for compartment syndrome, but much lower than for diaphyseal fractures. Plain orthogonal films are often sufficient for classification and preoperative planning. To rule out intra-articular fracture extension, CT can be considered. The fracture pattern itself can be classified using the AO or the Robinson classification [65].

There is ongoing controversy regarding the ideal fixation method for such fractures, fuelled by improvements in implant technology and surgical techniques [83]. The extra-articular fracture of the distal tibia can be treated by intramedullary (IM) nailing with very distal locking screws, percutaneous plating/MIPO or external fixation (small wire hybrid or Ilizarov-type fixation). The most recent systematic review of plating vs. IM nailing showed that both techniques can provide adequate treatment, but no studies to date are of sufficient power [33].

Nonoperative treatment with functional cast bracing can be used [15, 66], but outcomes were not impressive. Sarmiento's series had a 13.1 % malunion rate, whilst Bostman's series of 103 patients included 32 cases that required IM nailing for failure to maintain reduction. Malunion is common (26.4 %) with a small group (4.2 %) developing a non-union. Overall, we advise nonoperative treatment only in very few carefully selected cases.

The benefit of IM nailing is preservation of the soft tissue envelope at the fracture site but is more difficult than for midshaft tibial fractures and should be considered with caution. Known difficulties include maintaining alignment distal to the isthmus as the wide metaphysis allows a windshield wiper effect. This can be overcome by the use of strategically placed Poller screws or large K-wires (palisade technique) to narrow the canal. Alternatively, reduction can be maintained during reaming and nailing by use of a femoral distractor, with stability achieved with at least two, or preferably three, very distal interlocking screws. Modern nails have multiple distal locking options to allow better control of the distal fragment in the sagittal and coronal planes, and more recently, locking interlocking screws have been developed that might provide even better holding power. If there is simple extension of the distal fracture into the articular surface, this can be addressed by firstly placing lag screws followed by careful nailing [37, 53]. The use of the largest possible nail after reaming might lower the stresses on the distal interlocking screws with less hardware failure. Adjunctive fixation of the fibula may lower the risk of late malignment [26]. A complication of tibial IM nailing is anterior knee pain (up to 33 %) [65], which is a great concern in an athlete. The recently introduced technique of a suprapatellar entry point might lower this risk, but data is still limited.

Open reduction and internal fixation of distal metaphyseal tibial fractures allows anatomic fixation with stable fixation at the cost of devascularisation and possible complications with skin compromise and infection. The development of MIPO or percutaneous plating has received great interest for the distal tibia, and studies have shown that vascularity is preserved better and infection rate has decreased with MIPO compared to open techniques. The saphenous vein and nerve are at risk, however [48, 55], with resulting burning, numbness or shooting pains, mostly transient and mild, but some patients report a disturbed quality of life. Late infection and impingement have been reported [41], but irritation of the soft tissues overlying the medial malleolus is most common with up to 52 % hardware removal needed [51]. Although most plates currently have locking technology and do not require close apposition, close approximation is preferable to minimise prominence. The best type of screw fixation is unclear, but earlier healing has been reported when combining interfragmentary screws with locking plating [31].

Some authors have advocated definitive fixation of distal metaphyseal tibial fractures with Ilizarov or hybrid frames [6]. In a retrospective review comparing external fixation with IM nailing [63], functional results were similar, but the former seemed to carry a greater risk of secondary intervention. Infections with external fixation are relatively common [35], so in our practice we restrict external fixation to ankle spanning delta frames for temporary stabilisation of high-energy fractures.

In an excellent systematic review of 1,125 distal tibial fractures [83], the overall rate of non-union was 2.4 % and malunion 14.3 %, with a trend towards more malunion in those treated with IM nailing compared to plating. The latest systematic review on nailing vs. plating of distal metaphyseal tibial fracture (2005–2010 literature) showed that both techniques can provide adequate treatment [33], with union rates of 95.5 % vs. 97.7 %, infection rates of 5 % vs. 11.2 % and malunion rates of 25.9 % vs. only 5.3 % after IM nailing and plating, respectively. The MIPO technique has been reviewed by others [51], with open vs. MIS plating showing no differences in a retrospective study [18]. No difference in outcome was shown between low, multidirectional locked nailing and MIPO, although nailing had shorter union times, hospital stay and earlier full weight bearing [42]. In a prospective study comparing open vs. MIPO plating, there were no differences for type A and B fractures, but for type C fractures there was a trend towards shorter healing times using MIPO [84].

In conclusion, most distal tibial fractures need surgery. Ideal treatment depends on multiple factors. The risk of anterior knee pain after nailing is of concern for athletes, whilst perfect anatomic reduction using minimally invasive surgery and fixation with low-profile plates would seem to offer a better chance of early return to sports.

## **15.6 Pilon Fractures**

Destot introduced the term pilon (a pharmacist's pestle) in 1911 for a fracture extending 5 cm off the joint line, involving the dome of the distal articular tibial surface. Pilon fractures are the result of an axial loading force with varying amounts of a torsional component. The fibula may or may not be involved. The limited soft

tissue envelope combined with the often complex (high-energy) fracture pattern requires carefully planned and executed surgery to prevent disastrous complications. Operative treatment of these fractures is widely accepted since the landmark paper by Rüedi and Allgöwer in 1969 [61] and their principles still hold true today: open reduction and fixation of the fibula to restore lateral column length, anatomic restoration of the distal tibia, bone grafting and buttress plating of the varus deformity of the distal tibia. Components of the pilon fracture include the proximal extension, and the fibular and articular surface fragment most often divided into the Tillaux-Chaput (anterolateral), Volkmann (posterolateral) and medial fragments. The first two almost always remain connected to the fibula via the syndesmosis. There is often impaction of the articular surface. The classification by Rüedi and Allgöwer based on articular comminution is commonly used, but the AO/OTA one is an alternative. Topliss studied 126 CTs of pilon fracture and noted six major articular fragments: anterior, posterior, medial, anterolateral (Chaput), posterolateral (Volkmann) and the die-punch [75]. Two patterns were noted, namely, coronal fractures (usually elderly patient with low energy and valgus deformity) and sagittal patterns (usually younger patients with high energy in varus angulation). Others have expanded on this issue focusing on the articular surface, showing that fracture patterns occur in a predictable fashion [19].

The soft tissue should be carefully checked on assessment as open injuries are relatively common (10–30 %), and patient factors affecting soft tissue complications, poor fracture healing and fixation failure should be identified. Plain films (AP, lateral and mortise) will show the severity of the fracture pattern in these types of fractures. Associated fibular fracture, degree of comminution and the direction of talar displacement are important clues to surgical tactics and planning. A relatively simple medial fracture pattern is caused by tension or a valgus injury, whereas a more comminuted medial fracture pattern is caused by a varus injury. Although CT is often performed at the initial workup, more information can be gained if CT imaging is made after primary stabilisation with an ex-fix and/or fibular plating.

Surgical treatment of pilon fractures is difficult and requires careful planning with outcomes difficult to predict. The skill and experience of the surgeon are important, with timing of surgery critical, and a delay of 7–21 days allowing soft tissues to recover is common. The good results shown by Rüedi and Allgöwer could not initially be repeated by others [73], and criticism followed that their series represented low-energy, simple pilon fractures. As a result, many surgeons used external fixators for the more high-energy fractures [4], but this led to more malunions and worse function. A staged approach was the logical result of these changes in surgical techniques.

Similar to distal metaphyseal tibial fractures, treatment of pilon fractures ranges from nonoperative (very rare), hybrid external fixation and open vs. MIS fixation. For the high-energy pilon fractures, the gold standard remains the spanning external fixation (with or without primary fixation of the fibula) followed by definitive internal fixation of the distal tibia once the swelling has resolved. The so-called wrinkle sign is useful in determining that surgery can be done safely. An open technique requires careful soft tissue handling, whilst MIS techniques have recently gained popularity. Visibility of the articular component should not be compromised as the long-term functional outcome is closely related to the ability to restore anatomy at the time of surgery. In an excellent review of the literature, a satisfactory result was seen in 70 % of high-energy and in 80 % of low-energy fractures, with ankle fusions required in 3-27 % of pilon fractures [16].

The surgical approaches for ORIF depend on the fracture pattern and surgeon preference. Most surgeons will use two incisions, one for the fibula (as posterior as possible) and one anteromedial or anterolateral. The classic anteromedial approach used by Rüedi and Allgöwer required a skin bridge of at least 5 cm. Others have modified the anteromedial approach [5] or have used a single anterolateral approach for both the fibula and tibia, with more unusual fracture patterns requiring posterolateral or posteromedial exposures. In general, after good exposure, the reduction is best started posterolaterally, followed by posteromedial, central, anterior and anterolateral reduction. The anterolateral fragment is hinged open laterally to allow access to the posterior and central parts. A laterally based femoral distractor (pins in the talus and in the tibia) will greatly improve visualisation of the tibiotalar joint. Areas of impaction should be carefully disimpacted and bone graft used liberally to fill metaphyseal void. Multiple K-wires can be placed to hold the reduction avoiding obstruction of plate and screw placement. Precontoured locking plates can be used for medial, anterolateral and posterior plating. If the medial fracture pattern is relatively simple, a medial buttress plate can be placed percutaneously. The use of thin flexible plates will minimise medial prominence and still provide adequate buttressing. Minimally invasive plating should be reserved for simple fracture patterns [14]. High complication rates after formal ORIF of pilon fractures led to the introduction of external fixation with or without limited internal fixation of pilon fractures [4], with Ilizarov-type techniques proving powerful in the hands of experts. They are, however, cumbersome for the patient and can have relatively high complication rates.

Complications associated with the treatment of pilon fractures are common and can be the result of both injury and treatment. Infection remains the most common complication, and deep infection rates of 5-55 % have been reported [45]. Numbness on the dorsum of the foot can be prevented by careful protection of the superficial peroneal nerve branches during approach, reduction and fixation and closure. Fortunately, such injuries in football are extremely rare.

### 15.7 Talar Body and Neck Fractures

The major concern in talar body and neck fracture (dislocation) after adequate primary treatment is the early recognition of avascular necrosis (AVN) that usually occurs 6–8 weeks after injury. Incidence is quite similar in fractures of the body and neck, but after body fractures a higher incidence of late subtalar osteoarthrosis is noted. A useful tool in assessment is the 'Hawkins sign' which excludes AVN, represented by an objective thin line of subchondral atrophy on a lateral X-ray along the talar dome indicating the presence of revascularisation. Prevention of AVN is paramount since prolonged non-weight bearing, bone grafting and necrotic tissue excision have not been proven very successful in speeding the revascularisation of the talar body.

#### 15.7.1 Talar Body Fractures

Articular cartilage covers 60 % of the talus providing a very limited surface area for vascular perforation. Since the talar blood vessels enter through the capsule or ligamentous talar attachments, a fracture (dislocation) can easily disrupt vascular access. Inferior to the talar neck, the talar sinus and talar canal vessels form an anastomotic sling and form the primary source of blood supply. With increasing displacement, the rate of AVN also increases. Since talar body fractures usually present in different fracture patterns, no specific classification is commonly used. In general, these injuries present as shearing- or compression-type fractures. Axial compression is the most common mechanism of injury and the location of the primary fracture line is important to evaluate treatment strategy and avoid prolonged disability. Clinical presentation can range from very subtle findings to a markedly swollen and tender ankle. Tenderness and effusion/haematoma are usually noted along the anterior tibiotalar joint line to the talonavicular joint, and associated midfoot injury or peritalar dislocation should be considered. All talar body fractures are considered as intraarticular injuries. Assessing the lateral process is important: fracture lines into or posterior to the lateral process are considered talar body fractures; if the fracture line runs anterior to the lateral process, it becomes a neck fracture.

Talar body fractures are relatively rare and associated with high-energy trauma, so are rarely seen in football, but dislocations can be encountered; these should be recognised and stabilised quickly to avoid the risk of future complications. In the case of severe soft tissue swelling, definitive treatment can be delayed to improve the player's overall condition. In case of a fracture-dislocation, urgent orthopaedic treatment is required and ORIF remains the golden standard. An initial attempt for closed reduction is advocated in order to minimise skin necrosis and potentially improve vascular supply. In case of a surgical intervention, a medial malleolar osteotomy may be necessary to achieve optimal fracture reduction and fixation. Reduction of the fracture can be difficult, and a variety of techniques such as calcaneal traction, distractor and direct manipulation may be used.

## 15.7.2 Talar Neck Fractures

Similarly, talar neck fractures in athletes are renowned for highly unsatisfactory results and later complications such as AVN. Early detection and management are essential in minimising complications (potentially career ending) of such injuries.

After avulsion injuries, fractures of the talar neck are the second most commonly found tarsal injuries [20], usually the result of a hyperdorsiflexion-type mechanism. Described in World War I by Navy surgeon Anderson as the 'aviator's astragalus', it was frequently encountered in pilots during impact. In football, the injury mechanism is more related to axial load than to a specific foot position, although dorsiflexion is usually present. Injuries are usually associated with dislocations, and neurovascular trauma can occur as a result, although most dislocations are posteromedial, 'protecting' the neurovascular bundle. Disruption of vascularity after dislocation frequently leads to AVN, nevertheless, especially in combination with a severe soft tissue injury. Neurovascular assessment is thus the most important initial management.

Other than plain radiographs, subtle talar neck fractures can be visualised by views described by Canale and Kelly, where the plantigrade foot is placed in internal rotation whilst angling the beam at 75 degrees from the ground, a view also frequently used as a fluoroscopic intraoperatively check of adequate reduction. CT is frequently used to assess talar comminution and reduction congruency and is proven superior to plain films in detecting more subtle avulsions in or around the talar neck. MRI is especially helpful in detecting avascular necrosis later on, and with evolving technology the issues with postoperative metallic artefact are now limited. The most common classification for talar neck fractures is by Hawkins: type 1 fractures refer to an undisplaced fracture without associated joint dislocation, type 2 (most common) fractures refer to a combined dislocation at the subtalar joint, whilst type 3 fractures involve a dislocation at the ankle and subtalar joints (and almost always lead to AVN). Type 4 fractures refer to associated dislocation of the talonavicular joint and are rare, especially in football. The grade of comminution is, together with the Hawkins classification, the best predictor for avascular necrosis risk in talar neck fractures. There is no universally used grading system for comminution, but the more severe it is, the worse outcome and rate of complications can be expected.

Treatment of talar neck fractures requires specific attention to meticulous anatomic reduction in order to avoid future AVN. Multiple surgical approaches can be valuable, especially in comminuted cases, or if there is a combination between talar body and talar neck fractures. In case of a combined dislocation, it can be very difficult to reduce the talus, and in these cases a medial malleolar osteotomy is advocated. Infection, malunion or post-traumatic arthritis may be added to career-ending complications for the football player.

## 15.8 Talar and Calcaneal Process Fractures (Often Avulsion Type)

Fractures of both talar and calcaneal processes may be occasionally found and can be mistaken for simple sprains. These are discussed below.

## 15.8.1 Anterior Calcaneal Process Fractures

Although generally thought to be rare, studies have shown such fractures are often missed as ankle sprains constituting 8-13 % of all calcaneal fractures and found in up to 5 % of patients with a history of an ankle sprain and believed to be the result of either an avulsion- or a compression-type injury. Extra-articular avulsion-type fractures are commonly due to inversion plantar flexion and often seen in women which may relate to the plantar-flexed position when wearing high-heeled shoe. The 'nutcracker' compression-type injury is associated with an eversion injury with the foot in dorsiflexion [30]. Associated fractures of the talar neck, body of the calcaneum and the metatarsals have been described, and there is an association with the presence of previous ATFL injury and calcaneonavicular coalition. The latter is thought to predispose to a stress-fracture type, with the relative lack of normal movement resulting in increased stresses on the process, and has been reported in a marathon runner [52] and a professional rugby player [59]. In the athletic population, spontaneous development of foot pain may also be seen in stress-fracture-type injuries [59], and in these patients the presence of pes planus or a stiff hindfoot should raise suspicion. Patients will complain of pain in the dorsolateral aspect of the foot with the possible inability to bear weight and associated swelling. Localisation of pain 1 cm inferior and 3-4 cm anterior to the lateral malleolus is suggestive of such fractures. Applying the 'Gellman grip', with the thumb on the lateral malleolus and the middle finger on the base of the fifth metatarsal, brings the index finger to point out the area of maximum tenderness. A lateral view of the calcaneus would often show such injuries, but specialised views include a 45° inverted oblique view or a 10-15° cephalad and a 10-15° posterior view. In the athlete, we feel CT (and MRI) scans can show the fracture line, as well as associated injuries and the possible presence of coalition (Fig. 15.1). In addition, they exclude the presence of a secondary calcaneus secondarius ossicle, which is found in 2–5 %of the population [64].

Classification is into three types (Table 15.1) [22]. Although good quality evidence for the management of such fractures is limited, delayed diagnosis and treatment has a negative effect on outcomes with regard to symptoms and union rates. Symptoms may be persistent for over a year even when early treatment has been initiated. The presence of non-union (0–40 % of cases) is prognostic of outcome, as well as the presence of a type III injury [22]. Some authors consider ORIF for type III injuries, and we feel that in high-level athletes it would be advantageous for allowing both healing and earlier return to sport. Although in the presence of a stress fracture with associated coalition nonoperative treatment can be successful, we feel that ORIF and excision of the coalition would limit the risk of non-union and reduce the risk of refracture [59]. In the case of a professional rugby player, full match fitness was possible 6 months after surgery [59].

Nonoperative treatment does vary greatly [67], but the use of a short-leg cast for 4–6 weeks with an increase in loading as symptoms dictate appears to produce generally satisfactory outcomes. The development of a non-union pseudoarthrosis is
**Fig. 15.1** Axial CT scan slice demonstrating a stress fracture of the anterior process of the calcaneus in a professional rugby player, on a background of a fibrous calcaneonavicular coalition (Image from Pearce et al. [59]. Copyright®2011 by and reprinted with permission of SAGE Publications)



 Table 15.1
 Degan classification of fractures of the anterior process of the calcaneus

Туре	Description
I	Undisplaced fracture of only the tip
Π	Displaced fracture not involving the calcaneocuboid joint
III	Large displaced fracture involving the calcaneocuboid joint

not necessarily painful in most patients. Symptoms can improve for over a year in about a quarter of patients [22], and it is important to wait before considering further surgical management. In the case of a painful pseudoarthrosis, steroid injections do not appear to be of any benefit, but an image-guided injection of local anaesthetic can help decide on excision of the fragment if it proves to relieve symptoms. Although excision was felt to never be indicated and potentially worsen, it was shown to relieve symptoms in five out of seven patients [22], although recovery was prolonged; the two patients that did not improve had a long delay in diagnosis (22–24 months).

Surgical access to the anterior process is through a lateral incision between the tip of the lateral malleolus and the fifth metatarsal base, which can allow reduction and fixation, excision, as well as resection of a coalition [59]. An image intensifier is useful to identify the fracture site and also help with reduction and fixation; for the latter we advise the use of a 3.0-3.2 mm partially threaded cannulated screw with a washer. In high-level athletes we opt for early protected mobilisation. The patient is kept non-weight bearing in a splint for 2 weeks, before a walker boot can be applied and range of motion exercises of the ankle and hindfoot started. In cases where fixation has been performed, the patient is allowed to partially bear weight at this point and fully bear weight at 4 weeks, before free mobilisation is allowed at week 6 based on clinical and radiographic findings. Impact activities are avoided until 12 weeks when a CT scan is performed to confirm union. In our experience, return to full training is possible by 15 weeks and full competition at 24 weeks [59]. When excision has been performed, patients are still protected in a walker boot for 4-6 weeks but allowed to bear weight as comfort allows after the second week once the wound has healed. Patients are warned that resolution of symptoms may be incomplete and prolonged [22].

## 15.8.2 Lateral Talar Process

The lateral process of the talus articulates with both the fibula and the subtalar joint, with its tip forming the origins of the lateral talocalcaneal, cervical and bifurcate, and in particular the anterior talofibular ligament. Although lateral talar process fractures are considered rare, they have received increased attention recently because of their increased frequency in snowboarding with a 2.3 % incidence of all relevant injuries. In the general population, they are often misdiagnosed as lateral ankle sprains, and a retrospective incidence of 0.86 % of ankle and 10.4 % of talar injuries is seen. Although their reported association with football has been scarce, we do occasionally see it in our practice. Stress fractures of the lateral process have also been reported and present with non-resolving lateral ankle pain. Increased supination of the foot is a predisposing factor, with diagnosis made on CT scanning, although bone scans will demonstrate increased activity in the area [9]. Although there appears to be a reduction of symptoms over many months with nonoperative treatment, reported cases did not always return to their previous level of activity even if the fracture had healed. There is little evidence to suggest optimal treatment for such injuries, but we believe delay in identification of such a stress fracture would both delay return to a competitive level of activity and affect the footballer's function in the long term. Early identification using CT scanning is therefore important: an initial trial of nonoperative treatment with non-weight bearing may be attempted with repeat fine-cut CT scans to assess union. For the professional athlete, however, with the need for early return to competition, fixation of the fracture  $(\pm$  bone grafting) should be considered.

Туре	
I	Chip avulsion not extending to the talofibular articulation
II	Single, large fragment extending from the talofibular articular surface to the posterior
	talocalcaneal articular surface of the subtalar joint
III	Comminuted fracture

 Table 15.2
 Classification of lateral talar process fractures and suggested treatment [46]

Such fractures should be considered in any footballer presenting with lateral ankle pain in the acute setting. As a number of these injuries may be initially undetected, it is even more important to exclude them in a footballer that has continuing lateral ankle pain after an injury that has been managed as a lateral ligament complex sprain. A detailed assessment of the mechanism of injury is essential, but there has been controversy on how these injuries are produced. The commonly suggested mechanism is one of forced dorsiflexion of the foot with associated inversion, but a number of other studies are suggestive that dorsiflexion with external rotation or eversion are more likely to cause this, and an inversion component during injury has not been consistently shown. In the acute setting, examination findings are similar to that of acute lateral ankle sprain, with tenderness about 1 cm anterior and inferior to the lateral malleolus and possible inability to bear weight. In the chronic setting there may be stiffness of the subtalar articulation. Although a number of classifications have been put forward, the one put forward by McCrory and Bladin appears to better reflect outcomes and guide treatment [46] (Table 15.2).

Standard AP and lateral ankle radiographs should be obtained; internal rotation  $(10-20^{\circ})$  mortise views may also often visualise fractures better, and it may be optimal to perform these in plantar flexion and inversion [46]. A reverse Broden's view can be helpful to assess articular step-off; asymmetry or crookedness of the normally symmetrical V-shaped contour of the process on the lateral view is consistent with a fracture, and the latter has been designated as a pathognomonic positive V sign. Nevertheless, up to a half of fractures may not be detected, and their extent and displacement may be underestimated. Bone scans may be helpful in chronic symptomatic undetected injuries, but we feel that fine-cut (1 mm) CT scans are essential to detect a fracture and also provide detailed information on its extend, comminution and displacement so as to optimise treatment (Fig. 15.2).

There is a lack of prospective, randomised studies to guide optimal management. Consensus is that delayed diagnosis leads to a poor outcome and can lead to long-term impairment, a high rate of non-union, bony overgrowth and degenerative changes in 15–25 % of patients. Parsons reviewed 40 aggregated cases [57], and a good outcome was seen in 12 of 20 (60 %) of nonoperatively managed patients which included those patients treated conservatively or where the fracture was missed. Conversely, they reported a good outcome in all 21 patients treated with ORIF, closed reduction or initial excision. Non-union was associated with a poor outcome and seen in 60 % of nonoperatively treated patients but only 5 % of aggressively managed fractures. After excluding the missed fractures or early excision cases, they showed that a period of immobilisation was still associated with a higher



**Fig. 15.2** AP and lateral plain films (**a**, **b**) showing a lateral process fracture (*white arrows*), confirmed on coronal and sagittal (**c**, **d**) CT images (Reprinted from Bonvin et al. [13], Copyright (2003) from Elsevier)

non-union rate compared to operatively treated cases. Others similarly showed significantly improved AOFAS hindfoot scores in their 14 patients that were treated operatively, compared to the 6 who did not (97 vs. 85 points) [77].

From these and other reports [79], it is difficult to draw definitive conclusions on the best management for such injuries but is important to consider the following in the management of such injuries in the footballer: firstly, early diagnosis and management appear to optimise outcome and time to return to full activity, with surgical management in displaced and/or comminuted injuries being of benefit. Secondly, associated injuries need to be identified and managed accordingly. Lastly, athletes need to be warned of a potentially long recovery, as well as the possibility that they may be unable to return to their pre-injury level as well as the risk of development of degenerative arthritis. The reported cases of stress fractures of the lateral process provide little information on best management and outcome [9, 49], but early identification may allow either a period of rest to allow fracture healing or surgical intervention to fix the fracture and possibly reduce the time to return to sport.

Operative management involves a curved incision made from the tip of the fibula towards the anterior calcaneal process, and the lateral process is exposed between the anterior talofibular and calcaneofibular ligaments. Assessment of the process and articulation is made, and excision of fixation can be performed. Fixation can be achieved with 2.0 mm lag screws and/or 1.0 mm K-wires; in comminuted fractures a 2.0 mm T-plate has been used successfully as a buttress [77, 79]. The capsule is repaired and any other associated injuries to the surrounding ligaments also addressed. A concern of excising the lateral process is secondary instability of the subtalar joint. We do not feel this is an issue, however: it has not been reported in the literature, cadaveric biomechanical studies did not show this to be an issue [40], and on an anatomic basis the only ligament contributing to subtalar joint stability is the lateral root of the inferior extensor retinaculum that makes little contribution to the latter [57].

# 15.8.3 Posterior Talar Process

The posterior process of the talus articulates with the tibia superiorly and contributes significantly to the subtalar articulation inferiorly at the posterior facet. It consists of a more prominent lateral (Steida's process) and a medial tubercle with the flexor hallucis longus (FHL) tendon running in the sulcus between them. Fractures of either tubercle or the complete posterior process, although rare, have been described and have been associated with sports injuries, including football [50, 58]. It has been common to misdiagnose these as ankle sprains initially, whilst the normal accessory os trigonum just posterior to the lateral tubercle can be erroneously thought to be a fracture. In individuals that have an accessory os trigonum, a fracture of the latter, or more commonly injury to its synchondrosis with the lateral tubercle, can occur after injury that can lead to subsequent 'os trigonum syndrome'.

#### 15.8.3.1 Fractures of the Lateral Tubercle and Os Trigonum Complex

The lateral tubercle serves as an attachment to the talocalcaneal and the posterior talofibular ligament. An avulsion-type (Shepherd's) fracture may be the result of a plantar flexion and inversion force, whilst a compression-type fracture is usually the result of the posterior process being squeezed between the posterior tibia and calcaneum in extreme plantar flexion [58]. The same mechanisms can lead to injuries to the os trigonum in individuals that have it [36]. Athletes will usually present with pain and swelling in the posterolateral ankle. They will often describe the onset of symptoms from kicking the ball and will have tenderness to deep palpation in the area between the tendon of Achilles and the lateral malleolus and exacerbation of

symptoms on plantar flexion of the ankle; compression of the fracture by the FHL by dorsiflexion of the great toe can also be occasionally seen [36, 58]. A plain lateral radiograph provides a good view of the lateral tubercle. Care must be taken when reviewing the radiograph so as not to consider a fracture of the lateral tubercle as a normal os trigonum. The latter when present usually has a smooth cortical rim, compared to an irregular outline in the case of a fracture of the tubercle. Fractures of the os trigonum can also be found, but are rare. A fine-cut CT scan is useful to identify fractures and assess displacement, but an MRI scan is also advisable to identify possible bone bruising or injury to the synchondrosis between the os trigonum and the lateral tubercle.

#### 15.8.3.2 Fractures of the Medial Tubercle

Fractures of the medial tubercle are rare and can occur due to avulsion by the posterior talotibial ligament (posterior aspect of the deltoid ligament) by dorsiflexion and eversion (Cedell fractures), or by direct compression of the process as above or with impingement of the sustentaculum tali in supination. In contrast to lateral tubercle injuries, pain and swelling is usually present between the tendon of Achilles and the medial malleolus, but there may be limited pain on walking or moving the ankle. It is difficult to visualise fractures of the medial tubercle on plain AP and lateral radiographs and has been suggested that the addition of two oblique views at 45° and 70° of external rotation may significantly aid detection prior to resorting to a CT or MR imaging.

#### 15.8.3.3 Fractures of the Entire Posterior Process

Fractures of the entire posterior process of the talus are rare but are significant in that they can affect two articulations, the tibiotalar and the subtalar, with the posterior process contributing a quarter of the articulation with the posterior facet of the calcaneum. Fractures can occur due to direct trauma or compression between the posterior tibia and calcaneum with extreme plantar flexion and tend to be associated with high-energy injuries. An inversion component to the mechanism has also been suggested, as well as injuries occurring with the foot in inversion and plantar flexion. Plain radiographs usually demonstrate these more obvious injuries, but fractures can often be missed, unless there is a high index of suspicion. It is important to assess for associated injuries, such as subtalar dislocation which has been described, and it is felt that CT imaging is essential in order to identify the fracture, assess its extend and displacement, exclude other injuries and identify the presence of small fragments (<1-2 mm) in either articulation that may not be obvious on plain films. Early diagnosis and management is paramount for these injuries as delay in diagnosis appears to be detrimental to the outcome. Avulsion fractures of the tubercles are commonly extra-articular and can be treated nonoperatively if undisplaced or minimally displaced, using a non-weight-bearing below-knee cast in neutral or 15° equinus for 4–6 weeks, followed by mobilisation and weight bearing as tolerated. Nevertheless, in one series, two thirds of patients continued to have pain and less than 10 % responded to further immobilisation and steroid injection. If the fracture site continues to be painful, proprioceptive and muscle-strengthening exercise with custom-made orthotics can help with the resultant posteromedial pain, but late surgical excision (at 4-6 months) can provide good results and return to sport in the majority of patients [17, 38, 68]. Although fractures of the os trigonum are rare, treatment in a below-knee cast for 3 weeks has been shown to be successful with resolution of symptoms at 6 weeks [39]; symptomatic non-unions have been reported which responded well with excision of the os trigonum [2]. From the results above, it is felt that if the fragment is >2 mm and/or interferes with the articulation, operative fixation would be preferable. Significant displacement of the fracture or extension into the talar body would also benefit from this in order to avoid painful non-union, tarsal tunnel syndrome, posterior impingement of the FHL and degenerative changes due to the intra-articular displacement or the presence of small displaced fragment(s) trapped in the joint. Fractures of the entire process, although rare, do not appear to do well with nonoperative treatment with high incidence of non-union and early degenerative changes [54], but in the reported series of four patients, there was significant delay in diagnosis and early unprotected weight bearing was allowed. Early ORIF has, nevertheless, been shown to provide satisfactory outcomes and has been recommended.

Open reduction and internal fixation has been recommended, and fixation with K-wires or interfragmentary compression screws, with cannulated headless compression screws, showed satisfactory results. Both the posterolateral and posteromedial approach may be used. In the former the sural nerve is at risk, whilst others use the posteromedial approach protecting the neurovascular bundle in the tibial tunnel which may provide good access to the fracture as well as the ankle and subtalar joints. The use of a medial malleolar osteotomy has been described, but it is felt that it may cause unnecessary injury to the distal tibia. Late fixation or excision can also be surgically approach in a similar manner, whilst arthroscopic excision is an option in those experienced with posterior ankle arthroscopy. In cases of late fixation, 6 weeks in a non-weight-bearing cast is recommended, whilst in patients who undergo excision, early mobilisation at 7–10 days is recommended once soft tissue healing is achieved to minimise stiffness.

#### **15.9** Paediatric and Adolescent Fractures

The development of the youth game and football academies places significant injury risk to young people involved in the sport, and the potential consequences on the immature growing and the transitional skeleton with regard to development of deformity and premature degenerative changes can be devastating. Particular aspects have to be considered: ligaments and tendons are stronger compared to bone, and injuries result in different injury patterns compared to adults, with often fractures propagating through the relatively weak physeal/apophyseal areas with the relatively stronger ligaments remaining intact. In addition misdiagnosis is not uncommon, as interpretation of plain radiographs can be difficult in the presence of growth plates, with nontraumatic conditions often confusing the clinician Finally, as physeal areas mature and become partially closed, specific patterns of so-called transitional-type fractures are seen. A thorough history and examination are essential but may be difficult, as children may be poor historians and symptoms may not be as localising. The accuracy of Ottawa ankle rules in children over the age of 5 years has been demonstrated, but if interpretation is difficult, comparison views of the contralateral ankle are helpful. Both CT and MRI can be helpful with the latter being advantageous in view of the lower radiation exposure and that its ability to demonstrate concomitant soft tissue, physeal and osteochondral injuries. Fractures around the ankle in children and adolescents can be described by their anatomic location. Dias and Tachdjian [23] have modified the Lauge-Hansen classification for paediatric injuries taking into account the mechanism of injury and the involvement of the physes, whilst the commonly used Salter-Harris classification is helpful in communicating the type of injury and can have prognostic value. Transitional fractures of the distal tibia are classified into either two-plane type with no involvement of the metaphysic (such as Tillaux fractures) or triplanar fractures; the latter are subdivided into 'triplane I' (with a metaphyseal fragment) and 'triplane II' (where both epiphysis and metaphysis are involved). There is limited data on what is the best management for fractures around the ankle in paediatric and adolescent patients with regard to football and sports, but recent reviews from general studies suggest that the following are important aspects in optimising management, and we encourage the reader to consider these evidence and recommendations [10, 82]:

- 1. Decision on management should take into consideration the type of fracture, degree of displacement and restoration of normal alignment.
- 2. Adequate analgesia is important especially in the younger patient.
- 3. Internal fixation is not needed if satisfactory closed reduction is achieved and maintained.
- 4. Displacement and articular gap of greater than 2 mm have been associated with worse outcomes and should be reduced.
- 5. Although there is no significant evidence that multiple closed reduction attempts will increase the risk of physeal injury and growth arrest, it is encouraged to achieve reduction in one attempt under sufficient sedation or anaesthesia. This should be timely done as injuries older than 1 week are at increased risk of physeal injury.
- 6. Inability to reduce the fracture in a closed manner should alert the surgeon to the likelihood of soft tissue interposition; once the latter is removed, the fracture is usually both reducible and stable, with internal fixation often not necessary especially for Salter-Harris type I and II injuries.
- 7. Fractures or severe axial load injuries in the immature ankle should have followup for at least 1 year to detect any growth disturbance.

- 8. If fixation is necessary, K-wires or small fragment screws may be used, but consideration should be taken to minimise further damage to the physis. Removal of metalwork will often be necessary and advisable when transepiphyseal screws are used. Bioabsorbable implants have thus been used by some and been shown to be safe and effective and avoid the need, risk and costs of a second procedure to remove metalwork. The use of screws crossing the physis in transitional injuries has not been shown to have a negative effect on the overall outcome of such injuries.
- 9. Initial protection in a non-weight-bearing cast for 4 weeks followed by the use of a non-weight-bearing removable boot with initiation of range of movement exercise for 2–4 weeks afterwards (depending on the degree of injury) appears to be safe and consistent with good outcomes.

It appears that as long as displacement of >2 mm is addressed in transitional-type fractures, with the majority of these occurring during the last 18 months of growth, these have good to excellent outcomes and the majority of patients were able to return to sport [81]. Follow-up of injuries for at least 1 year is important: growth arrest and physeal bar development can be detected and potentially addressed. Realignment osteotomies to address angular deformity (of which the acceptable limit has not been clearly defined), bar resection when there is <50 % of physeal involvement with >2 years of residual growth and contralateral epiphysiodesis have been shown to be useful procedures to address problems. Such treatments should be decided and provided by surgeons experienced in dealing with such problems. There is limited evidence on the time to return to sport in youth football players; it appears, however, that most can return to sport and in our experience return to training is possible at 2–3 months and return to full contact sport at 4–6 months.

## 15.10 Role of Arthroscopy

Arthroscopic management for chronic pathology after ankle fractures has showed significant benefits, and although evidence on its use in the acute setting is limited, we feel there is an important role for its use as an adjunct to the management of fractures in the injured footballer, in both aiding diagnosis and optimising management [12]. Its proposed advantages in the management of ankle fractures include its minimally invasive nature and its ability to directly visualise the articulation, allowing the diagnosis and treatment of concomitant pathology that may not be obvious on other assessment and which may present with ongoing symptoms and hinder rehabilitation and return to competition.

A substantial proportion of osteochondral injuries after an ankle fracture may not prove symptomatic in the long run. Although one prospective randomised trial showed no difference in outcome between patients that had arthroscopy and management of articular damage at the time of fixation and those that did not [74], others in a larger prospective randomised trial of 72 patients showed a statistically improved AOFAS score (91.0 vs. 87.6) in patients that had arthroscopically assisted fixation [72]. As articular damage following an ankle fracture may be an independent predictor of the development of post-traumatic arthritis, with severity of the defects and their position on the anterior and lateral aspects of the talus and on the medial malleolus having the worst long-term outcomes [69], arthroscopic assessment at the time of fracture would be advantageous in predicting long-term outcome.

Injury to the syndesmosis after an ankle fracture is seen in 47–66 % of patients and can result in ongoing ankle problems. Intraoperative stress views are more reliable compared to plain radiographs at detecting definitive instability, but borderline instability or partial injury to the syndesmotic complex without instability is difficult to detect. MR imaging has been shown to provide accurate information with regard to the presence of a syndesmotic injury but has a significant false-positive rate, whereas arthroscopic assessment has been shown to be more sensitive and specific [71]. In addition, extra-syndesmotic fibres of the ruptured ligaments which may go on to produce chronic pain and impingement can be debrided. Good to excellent results have been reported in a number of studies where arthroscopic assessment (with fixation) and/or debridement was used to manage such injuries, and arthroscopic evaluation can detect sagittal and rotational instability that is not obvious on intraoperative stress radiography [44].

Arthroscopic reduction and internal fixation (ARIF) of various fractures around the ankle has been described in small series or case reports. The obvious benefits would be the reduction in damage to the soft tissue envelope and blood supply to the area, the reduced likelihood of infection, the improved visualisation and the accurate restoration of the articular surface. Concerns certainly exist with regard to the increased surgical time and surgeon-dependent ability and skill to successfully use the technique, as well as the associated soft tissue swelling. Indeed, a leg anterior compartment syndrome following ankle arthroscopy after a Maisonneuve fracture in a football player has been reported [32]. ARIF has been described for fractures of the talar body, talar neck or process, the distal tibia, malleolar and transitional fractures. Hindfoot process fracture excision when symptomatic is also possible.

Fibrous tissue formation related to remnants of torn ligaments and capsular tissue, including those from the syndesmotic, ATFL and medial deltoid ligaments after an ankle fracture can cause impairment in ankle function through impingement. This responds well to arthroscopic resection at a later stage. Arthroscopic evaluation and debridement at the acute stage would likely be beneficial, but evidence to support this at the moment is limited.

## 15.11 Rehabilitation

Rehabilitation is a central aspect of management of ankle fractures in the football athlete. Although some of the evidence for best post-fracture management for specific injuries has been mentioned above and mentioned in detail elsewhere (Chap. 13), we feel that a close relationship with the rehabilitation team is essential to get the athlete back to competitive sport as early and as safely as possible. We encourage strict elevation for the first 2 weeks after injury or an operation as well as early range of motion exercises as soon as safely possible.

# 15.12 General Outcomes and Time to Return to Competition

Outcomes from the general population cannot be directly extrapolated to footballers, who usually receive better and more intense rehabilitation, tend to be a healthier and younger group, and their safe and prompt return to a highly demanding level of activity is paramount. Evidence on outcomes on the rarer fractures around the ankle (i.e. process and talar fractures) in football is scarce and has been discussed earlier. Some evidence on the more common malleolar-type fractures has been documented and allows for some conclusions to be made. It has to be noted that a number of studies reporting time lost from training and competition provide limited information, as they often group ankle injuries depending on severity in general, with that severity of injury often being defined by the time to return to sport (rather than the type of injury, e.g. fracture per se).

Although surgical treatment may allow a more rapid recovery, with earlier weight bearing and functional rehabilitation providing a speedier return to normal daily living and work, a recent systematic review looking at surgical vs. conservative intervention for treating ankle fractures in adults concluded that there is not sufficient evidence to determine whether either type of treatment provides better longterm outcomes [24]. The review, however, only identified four controlled trials (292 adults with displaced ankle fractures) from the general population with significant issues assessing these in view of significant variations and limitations in the types of patients, the surgical and rehabilitation protocols applied, the outcomes reported and the duration of follow-up, whilst the risk of bias was unclear or high in general with studies not being of a sufficient size to detect differences. Colvin et al. looked at the ability of 243 patients who underwent operative fixation of an unstable ankle fracture to return to 'vigorous activity' and sport [21]. Younger, healthier and male patients were more likely to return to sport. At 1 year, although 88 % of recreational athletes were able to return to sport, only 11.6 % of competitive athletes were able to do so, with those with bimalleolar fractures being more likely to do so compared to those with unimalleolar fractures. The study was limited, however, in that it retrospectively looked at self-reported outcomes from a general trauma population and did not control for the type of sport; patients were kept non-weight bearing for 6 weeks and not allowed to return to sport earlier than 3 months [21]. It is suggested, nevertheless, that the surgical management by open reduction and internal fixation of unstable fractures of the ankle in athletes may provide a number of advantages. Firstly, it would avoid the issues with the significant proportion of these that would displace and require surgical treatment anyway, and thus delay recovery. Secondly, it can ensure anatomic fracture reduction and articular surface restoration. Finally, it can allow early range of movement exercises and weight bearing (within 1-2 weeks of fixation) and a more rapid recovery and return to sport [60].

Classification	Ν	Crutches	Boot	Brace	Daily living	Practice	Competition
Lateral malleolus fracture	6	1.3±0.5	3.0±0.9	4.3±3.8	1.2±0.8	5.0±0.9	$6.8 \pm 2.4$
Medial malleolus fracture	2	$2.0 \pm 1.4$	$2.0 \pm 1.4$	7.0±1.4	$2.0 \pm 0.0$	$12.0 \pm 5.7$	$17.0 \pm 9.9$
Bimalleolar fracture and equivalent	10	3.7±1.6	$3.7 \pm 2.0$	4.2.±2.2	$1.0 \pm 0.5$	$10.9 \pm 4.0$	$12.7 \pm 4.0$
Syndesmosis disruption injury	4	$3.3 \pm 1.0$	2.3±1.3	6.8±6.1	$0.8 \pm 0.5$	$13.5 \pm 2.5$	$15.8 \pm 1.7$
Salter-Harris-type fracture	4	$2.0 \pm 0.8$	3.5±1.7	9.0±1.2	$1.0 \pm 0.0$	$6.3 \pm 1.3$	$8.5 \pm 1.0$
Pilon fracture	1	4.0	2.0	2.0	1.0	8.0	16.0
Energy Destant of all [(0]	0	1.4000	00 1		1 1 1		TT D. 11'

**Table 15.3** Time (in weeks) athletes required the use of rehabilitative devices and time when athletes were able to resume activities in 27 athletes with ankle fractures that underwent ORIF

From Porter et al. [60]. Copyright<sup>©</sup>2008 by and reproduced with permission of SAGE Publications

Studies specifically looking at ankle fractures in athletes are limited [25, 56, 60, 80] but appear to demonstrate that a successful return to high-level competition should be expected. One study of three professional American football players showed that all returned to their pre-injury level [25], and this was the case in another reporting the results of surgical treatment of ankle fractures in three American football players and one football player [80]. In another study looking at the time lost from play in foot and ankle injuries of Turkish professional football players, the three patients with surgically treated ankle fractures were all able to return to sport [56]. Time lost was 150 days for two footballers (one with a Maisonneuve fracture and one with a lateral malleolar fracture with deltoid rupture), whilst the patient that was treated for a lateral malleolus pseudoarthrosis took 200 days.

Porter et al. detailed their management, rehabilitation and outcomes in 27 athletes with ankle fractures that underwent ORIF (including repair of any injured ligaments) either for displacement of  $\geq 3$  mm or if the athlete was 'especially enthusiastic' for an early return to sports [60]. The most common sport injuries were in American football (10 athletes) and baseball (3 athletes), but included two athletes involved in football. At an average follow-up of 2.4 years (12 months to 3.7 years), athletes reported an average 96.4 % functional rating compared to their pre-injury level, with 12 athletes rating their ankle as 100 %. Early rehabilitation and ambulation was encouraged, which included the use of an ankle Cryo/Cuff<sup>TM</sup> and walking boot, with athletes encouraged to bear weight within a week but protected in the latter. The ability of athletes to be weaned of their rehabilitative devices and the time required to reach activity goals are shown in Table 15.3. Those athletes with isolated Weber A and B isolated lateral malleolar fractures were able to return to sport the quickest, with return to full activity as early as 4 weeks; only two of the six athletes did not rate their ankle 100 % in view of either flexibility or decreased stability issues. There were only two athletes with isolated medial malleolar fractures, who both had their deltoid ligament repaired at the same time; return to competition for them took longer on average, with one patient taking 24 weeks to return to motocross racing. Athletes with bimalleolar fractures required  $12.7 \pm 4.0$  weeks to return to competition, with syndesmotic and pilon fractures taking slightly longer. The authors did not document the recovery of patients with stable and undisplaced fractures that underwent nonoperative treatment, and although there is a lack of guidance evidence with regard to outcomes and return to competition in athletes with such injuries, we feel that as early rehabilitation and ambulation would be possible in such cases, similar return to sport should be expected.

# 15.13 Conclusion

The incidence of fractures is small and less than 3 % of all ankle injuries in the modern professional game. Optimum management for the higher-level football player has to address the demand for early and safe return to a high level of activity, but as discussed above, the evidence for best practice in for the sport is limited. A thorough history, examination and adequate imaging are essential to correctly diagnose injuries and decide on best treatment. Early rehabilitation can allow early return to sport within 2–4 months. Surgical reduction when indicated and provision of stability by fixation can allow this and appear to be advisable to optimise both the outcomes and return to completion in football-related ankle fractures.

## References

- Abdelgawad AA et al (2011) Posterolateral approach for treatment of posterior malleolus fracture of the ankle. J Foot Ankle Surg 50(5):607–611. doi:10.1053/j.jfas.2011.04.022
- Abramowitz Y et al (2003) Outcome of resection of a symptomatic os trigonum. J Bone Joint Surg Am 85-A(6):1051–1057
- Amanatullah DF et al (2012) Effect of mini-fragment fixation on the stabilization of medial malleolus fractures. J Trauma Acute Care Surg 72(4):948–953. doi:10.1097/ TA.0b013e318249697d
- Anglen JO (1999) Early outcome of hybrid external fixation for fracture of the distal tibia. J Orthop Trauma 13(2):92–97
- 5. Assal M et al (2007) The extensile approach for the operative treatment of high-energy pilon fractures: surgical technique and soft-tissue healing. J Orthop Trauma 21(3):198–206. doi:10.1097/BOT.0b013e3180316780
- 6. Babis GC et al (2010) Distal tibial fractures treated with hybrid external fixation. Injury 41(3):253–258. doi:10.1016/j.injury.2009.09.014
- Bariteau JT et al (2013) Biomechanical evaluation of mini-fragment hardware for supination external rotation fractures of the distal fibula. Foot Ankle Spec 6(2):88–93. doi:10.1177/1938640013477130
- Bedi A et al (2006) Surgical treatment of nonarticular distal tibia fractures. J Am Acad Orthop Surg 14(7):406–416
- 9. Black KP, Ehlert KJ (1994) A stress fracture of the lateral process of the talus in a runner. A case report. J Bone Joint Surg Am 76(3):441–443
- 10. Blackburn EW et al (2012) Ankle fractures in children. J Bone Joint Surg Am 94(13):1234–1244. doi:10.2106/JBJS.K.00682

- Bois AJ, Dust W (2008) Posterior fracture dislocation of the ankle: technique and clinical experience using a posteromedial surgical approach. J Orthop Trauma 22(9):629–636. doi:10.1097/BOT.0b013e318184ba4e
- Bonasia DE et al (2011) The role of arthroscopy in the management of fractures about the ankle. J Am Acad Orthop Surg 19(4):226–235
- Bonvin F et al (2003) Imaging of fractures of the lateral process of the talus, a frequently missed diagnosis. Eur J Radiol 47(1):64–70
- Borens O et al (2009) Minimally invasive treatment of pilon fractures with a low profile plate: preliminary results in 17 cases. Arch Orthop Trauma Surg 129(5):649–659. doi:10.1007/ s00402-006-0219-1
- 15. Bostman O et al (1984) Infra-isthmal longitudinal fractures of the tibial diaphysis: results of treatment using closed intramedullary compression nailing. J Trauma 24(11):964–969
- 16. Calori GM et al (2010) Tibial pilon fractures: which method of treatment? Injury 41(11): 1183–1190. doi:10.1016/j.injury.2010.08.041
- Cedell CA (1974) Rupture of the posterior talotibial ligament with the avulsion of a bone fragment from the talus. Acta Orthop Scand 45(3):454–461
- Cheng W et al (2011) Comparison study of two surgical options for distal tibia fractureminimally invasive plate osteosynthesis vs. open reduction and internal fixation. Int Orthop 35(5):737–742. doi:10.1007/s00264-010-1052-2
- 19. Cole PA et al (2013) The pilon map: fracture lines and comminution zones in OTA/AO type 43C3 pilon fractures. J Orthop Trauma 27(7):e152–e156. doi:10.1097/BOT.0b013e318288a7e9
- 20. Coltart WD (1952) Aviator's astragalus. J Bone Joint Surg Br 34-B(4):545-566
- Colvin AC et al (2009) Return to sports following operatively treated ankle fractures. Foot Ankle Int 30(4):292–296. doi:10.3113/FAI.2009.0292
- Degan TJ et al (1982) Surgical excision for anterior-process fractures of the calcaneus. J Bone Joint Surg Am 64(4):519–524
- Dias LS, Tachdjian MO (1978) Physeal injuries of the ankle in children: classification. Clin Orthop Relat Res 136:230–233
- 24. Donken CC, et al (2012) Surgical versus conservative interventions for treating ankle fractures in adults. Cochrane Database Syst Rev 8:CD008470. doi:10.1002/14651858. CD008470.pub2
- 25. Donley BG et al (2005) Pronation-external rotation ankle fractures in 3 professional football players. Am J Orthop (Belle Mead NJ) 34(11):547–550
- 26. Egol KA et al (2006) Does fibular plating improve alignment after intramedullary nailing of distal metaphyseal tibia fractures? J Orthop Trauma 20(2):94–103. doi:10.1097/01. bot.0000199118.61229.70
- Haraguchi N et al (2006) Pathoanatomy of posterior malleolar fractures of the ankle. J Bone Joint Surg Am 88(5):1085–1092. doi:10.2106/JBJS.E.00856
- Herscovici D Jr et al (2007) Conservative treatment of isolated fractures of the medial malleolus. J Bone Joint Surg Br 89(1):89–93. doi:10.1302/0301-620X.89B1.18349
- Hess F, Sommer C (2011) Minimally invasive plate osteosynthesis of the distal fibula with the locking compression plate: first experience of 20 cases. J Orthop Trauma 25(2):110–115. doi:10.1097/BOT.0b013e3181d9e875
- Hodge JC (1999) Anterior process fracture or calcaneus secundarius: a case report. J Emerg Med 17(2):305–309
- Horn C et al (2011) Combination of interfragmentary screws and locking plates in distal metadiaphyseal fractures of the tibia: a retrospective, single-centre pilot study. Injury 42(10):1031– 1037. doi:10.1016/j.injury.2011.05.010
- 32. Imade S et al (2009) Leg anterior compartment syndrome following ankle arthroscopy after Maisonneuve fracture. Arthroscopy 25(2):215–218. doi:10.1016/j.arthro.2007.08.027
- 33. Iqbal HJ, Pidikiti P (2013) Treatment of distal tibia metaphyseal fractures; plating versus intramedullary nailing: a systematic review of recent evidence. Foot Ankle Surg 19(3):143–147. doi:10.1016/j.fas.2013.04.007

- 34. Irwin TA et al (2013) Posterior malleolus fracture. J Am Acad Orthop Surg 21(1):32–40. doi:10.5435/JAAOS-21-01-32
- 35. Joveniaux P et al (2010) Distal tibia fractures: management and complications of 101 cases. Int Orthop 34(4):583–588. doi:10.1007/s00264-009-0832-z
- Judd DB, Kim DH (2002) Foot fractures frequently misdiagnosed as ankle sprains. Am Fam Physician 66(5):785–794
- 37. Katsenis D et al (2013) The results of closed intramedullary nailing for intra-articular distal tibial fractures. J Orthop Trauma. doi:10.1097/BOT.0b013e31829e7358
- 38. Kim DH et al (2003) Avulsion fractures of the medial tubercle of the posterior process of the talus. Foot Ankle Int 24(2):172–175
- 39. Kose O et al (2006) Fracture of the os trigonum: a case report. J Orthop Surg (Hong Kong) 14(3):354–356
- Langer P et al (2009) Effect of simulated lateral process talus "fracture excision" on its ligamentous attachments. Am J Orthop (Belle Mead NJ) 38(5):222–226
- 41. Lau TW et al (2008) Wound complication of minimally invasive plate osteosynthesis in distal tibia fractures. Int Orthop 32(5):697–703. doi:10.1007/s00264-007-0384-z
- 42. Li Y et al (2012) Comparison of low, multidirectional locked nailing and plating in the treatment of distal tibial metadiaphyseal fractures. Int Orthop 36(7):1457–1462. doi:10.1007/ s00264-012-1494-9
- 43. Little MT et al (2013) Complications following treatment of supination external rotation ankle fractures through the posterolateral approach. Foot Ankle Int 34(4):523–529. doi:10.1177/1071100713477626
- 44. Lui TH et al (2005) Comparison of radiologic and arthroscopic diagnoses of distal tibiofibular syndesmosis disruption in acute ankle fracture. Arthroscopy 21(11):1370. doi:10.1016/j. arthro.2005.08.016
- 45. McCann PA et al (2011) Complications of definitive open reduction and internal fixation of pilon fractures of the distal tibia. Int Orthop 35(3):413–418. doi:10.1007/s00264-010-1005-9
- 46. McCrory P, Bladin C (1996) Fractures of the lateral process of the talus: a clinical review. "Snowboarder's ankle". Clin J Sport Med 6(2):124–128
- 47. Miller AN et al (2010) Posterior malleolar stabilization of syndesmotic injuries is equivalent to screw fixation. Clin Orthop Relat Res 468(4):1129–1135. doi:10.1007/s11999-009-1111-4
- 48. Mirza A et al (2010) Percutaneous plating of the distal tibia and fibula: risk of injury to the saphenous and superficial peroneal nerves. J Orthop Trauma 24(8):495–498. doi:10.1097/BOT.0b013e3181cb584f
- Motto SG (1993) Stress fracture of the lateral process of the talus–a case report. Br J Sports Med 27(4):275–276
- 50. Nakai T et al (2005) Painful nonunion of fracture of the entire posterior process of the talus: a case report. Arch Orthop Trauma Surg 125(10):721–724. doi:10.1007/s00402-005-0055-8
- 51. Newman SD et al (2011) Distal metadiaphyseal tibial fractures. Injury 42(10):975–984
- Nilsson LJ, Coetzee JC (2006) Stress fracture in the presence of a calcaneonavicular coalition: a case report. Foot Ankle Int 27(5):373–374
- 53. Nork SE et al (2005) Intramedullary nailing of distal metaphyseal tibial fractures. J Bone Joint Surg Am 87(6):1213–1221. doi:10.2106/JBJS.C.01135
- 54. Nyska M et al (1998) Fracture of the posterior body of the talus-the hidden fracture. Arch Orthop Trauma Surg 117(1-2):114-117
- 55. Ozsoy MH et al (2009) Minimally invasive plating of the distal tibia: do we really sacrifice saphenous vein and nerve? A cadaver study. J Orthop Trauma 23(2):132–138. doi:10.1097/ BOT.0b013e3181969993
- 56. Oztekin HH et al (2009) Foot and ankle injuries and time lost from play in professional soccer players. Foot (Edinb) 19(1):22–28. doi:10.1016/j.foot.2008.07.003
- 57. Parsons SJ (2003) Relation between the occurrence of bony union and outcome for fractures of the lateral process of the talus: a case report and analysis of published reports. Br J Sports Med 37(3):274–276

- Paulos LE et al (1983) Posterior compartment fractures of the ankle. A commonly missed athletic injury. Am J Sports Med 11(6):439–443
- 59. Pearce CJ et al (2011) Stress fracture of the anterior process of the calcaneus associated with a calcaneonavicular coalition: a case report. Foot Ankle Int 32(1):85–88. doi:10.3113/ FAI.2011.0085
- 60. Porter DA et al (2008) Functional outcome after operative treatment for ankle fractures in young athletes: a retrospective case series. Foot Ankle Int 29(9):887–894. doi:10.3113/ FAI.2008.0887
- 61. Rüedi T, Allgöwer M (1969) Fractures of the lower end of the tibia into the ankle-joint. Injury 1(2):92–99
- 62. Ricci WM et al (2012) Lag screw fixation of medial malleolar fractures: a biomechanical, radiographic, and clinical comparison of unicortical partially threaded lag screws and bicortical fully threaded lag screws. J Orthop Trauma 26(10):602–606. doi:10.1097/BOT.0b013e3182404512
- Ristiniemi J et al (2011) Surgical treatment of extra-articular or simple intra-articular distal tibial fractures: external fixation versus intramedullary nailing. J Orthop Trauma 25(2):101– 105. doi:10.1097/BOT.0b013e3181da4682
- 64. Robbins MI et al (1999) MR imaging of anterosuperior calcaneal process fractures. AJR Am J Roentgenol 172(2):475–479
- 65. Robinson CM et al (1995) Distal metaphyseal fractures of the tibia with minimal involvement of the ankle. Classification and treatment by locked intramedullary nailing. J Bone Joint Surg Br 77(5):781–787
- 66. Sarmiento A, Latta LL (2004) 450 closed fractures of the distal third of the tibia treated with a functional brace. Clin Orthop Relat Res 428:261–271
- 67. Schepers T et al (2008) Demographics of extra-articular calcaneal fractures: including a review of the literature on treatment and outcome. Arch Orthop Trauma Surg 128(10):1099–1106. doi:10.1007/s00402-007-0517-2
- 68. Stefko RM et al (1994) Tarsal tunnel syndrome caused by an unrecognized fracture of the posterior process of the talus (Cedell fracture). A case report. J Bone Joint Surg Am 76(1): 116–118
- Stufkens SA et al (2010) Cartilage lesions and the development of osteoarthritis after internal fixation of ankle fractures: a prospective study. J Bone Joint Surg Am 92(2):279–286. doi:10.2106/JBJS.H.01635
- 70. Stufkens SA et al (2011) Long-term outcome after 1822 operatively treated ankle fractures: a systematic review of the literature. Injury 42(2):119–127. doi:10.1016/j.injury.2010.04.006
- 71. Takao M et al (2001) Arthroscopic diagnosis of tibiofibular syndesmosis disruption. Arthroscopy 17(8):836–843
- Takao M et al (2004) Diagnosis and treatment of combined intra-articular disorders in acute distal fibular fractures. J Trauma 57(6):1303–1307
- Teeny SM, Wiss DA (1993) Open reduction and internal fixation of tibial plafond fractures. Variables contributing to poor results and complications. Clin Orthop Relat Res 292:108–117
- 74. Thordarson DB et al (2001) The role of ankle arthroscopy on the surgical management of ankle fractures. Foot Ankle Int 22(2):123–125
- 75. Topliss CJ et al (2005) Anatomy of pilon fractures of the distal tibia. J Bone Joint Surg Br 87(5):692–697. doi:10.1302/0301-620X.87B5.15982
- 76. Tornetta P 3rd et al (2011) The posterolateral approach to the tibia for displaced posterior malleolar injuries. J Orthop Trauma 25(2):123–126. doi:10.1097/BOT.0b013e3181e47d29
- Valderrabano V et al (2005) Snowboarder's talus fracture: treatment outcome of 20 cases after 3.5 years. Am J Sports Med 33(6):871–880. doi:10.1177/0363546504271001
- van den Bekerom MP et al (2009) Biomechanical and clinical evaluation of posterior malleolar fractures. A systematic review of the literature. J Trauma 66(1):279–284. doi:10.1097/ TA.0b013e318187eb16
- 79. von Knoch F et al (2007) Fracture of the lateral process of the talus in snowboarders. J Bone Joint Surg Br 89(6):772–777. doi:10.1302/0301-620X.89B6.18813

- Walsh WM, Hughston JC (1976) Unstable ankle fractures in athletes. Am J Sports Med 4(4):173–183
- Weinberg AM et al (2005) Transitional fractures of the distal tibia. Injury 36(11):1371–1378. doi:10.1016/j.injury.2005.04.004
- Wuerz TH, Gurd DP (2013) Pediatric physeal ankle fracture. J Am Acad Orthop Surg 21(4):234–244. doi:10.5435/JAAOS-21-04-234
- 83. Zelle BA et al (2006) Treatment of distal tibia fractures without articular involvement: a systematic review of 1125 fractures. J Orthop Trauma 20(1):76–79
- 84. Zou J et al (2013) Comparison of minimally invasive percutaneous plate osteosynthesis with open reduction and internal fixation for treatment of extra-articular distal tibia fractures. Injury 44(8):1102–1106. doi:10.1016/j.injury.2013.02.006

# Chapter 16 Interview – Leonne Stentler

Pieter P.R.N. d'Hooghe and Gino M.M.J. Kerkhoffs

**Abstract** Leonne Stentler is a Dutch female football player that played for SV Bolnes and with RVVH before she changed to ADO Den Haag in the season 2007/2008 to play in the Dutch professional league. In the season 2011/2012, she won the "double," the championship, and the national cup with ADO Den Haag. She changed to play with Ajax in the season 2012/2013. Leonne made her debut in the national team in 2009.

Next to her football career, she is a successful master in communication science at the University of Amsterdam and writes a thesis on successful communication measures in prevention of injuries in female football.

P.P.R.N.d'Hooghe, MD (🖂)

G.M.M.J. Kerkhoffs, MD, PhD

Department of Orthopaedic Surgery, Aspetar Hospital, Aspire Zone, Doha, Qatar e-mail: pieter.orthopedie@gmail.com

Department of Orthopedic Surgery, Academic Medical Centre Amsterdam, University of Amsterdam, Meibergdreef 9, Amsterdam, AZ 1105, The Netherlands e-mail: g.m.kerkhoffs@amc.uva.nl



• What is the most important for an ankle-injured player to experience from his staff to be able to cope with his rehabilitation? What advice can you give to ankle-injured players?

The most important to experience from the staff is the feeling that the injury is taken very seriously. They should give you the idea that they would do anything possible to help you recover; this leads to the fact that you as an injured player will feel confident that you will fully recover. Whether it is a relatively small or big injury does not really matter in this case. As an injured player, you have to rehabilitate by yourself, but the physiotherapist and doctor can really add a lot of positivity by taking your injury seriously. Some players are more touchy than others, but overall the main thing that a physiotherapist should do is give him or her the idea that they're not alone. Beside this, I always like to have clarity about the injury and the time it will take to recover. Physiotherapists and doctors can improve the mental state of a player in a later stadium by creating a personal recovery plan based on the growth and goals. My advice to injured players is to always communicate about the aches and feelings that you have. That way the doctor and physiotherapist can help you recover most efficiently. Also when you're having a day in which you feel mentally lost, the physiotherapist can always try to help with different exercises or some extra rest whenever you inform him or her. Clarity is important so when you have the feeling that you're not fully informed, just make sure you'll ask the doctor or physiotherapist.

Being injured for a longer period can really be a mental battle. Some periods will be easier than others. Physiotherapists and doctors can, besides providing medical care, really help by supporting players in general during these times.

• What is a good team doctor/medical staff for you, and what do you expect from them?

A good medical staff makes sure that they will always be approachable, helpful, and forward thinking. Whenever they're not sure about specific complaints, they should be open to ask help from specialists. Clarity toward injured players is also highly important, to make sure the players feel that they are being taken seriously.

• How do you experience your current ankle rehab, and how did you manage to stay positive?

I have had some minor setbacks during my rehab so far. Of course I had some bad days finding it difficult to accept the next periods in which I would not be able to practice my sport. I managed to stay positive by not thinking in longer periods anymore. I have really learned to live day by day and hold on to really small improvements. Thereby I always had the feeling that the medical team around me did everything they could to help me recover. Everyone involved with my treatment supports me in the best possible way. As an injured player, that is really what you wish for, being taken seriously by the doctor and physiotherapist.

• Did the medical setup in female football change the last 10 years, and what do you think about the evolution seen? Do you experience differences in the medical setup if you compare this to the male football?

The medical setup in female football changed because the whole competition setup changed. Instead of amateur clubs, the top league consists of professional clubs. Therefore, financially there is more possible, but this still depends on which club you represent. My former club did not have the same medical facilities as Ajax has. The medical staff that I work with now is similar to the medical staff of a national women squad, a doctor and two physiotherapists. A few club teams have the same facilities as Ajax. But in general the medical staff in female football is very much less professional comparing to male football. This has everything to do with the financial state. For example, some clubs in the top league have difficulties finding a decent physiotherapist because they cannot provide a normal salary.

• Do you think you are performing enough ankle injury prevention strategies for your ankle in your daily workout?

I think I can perform more injury prevention strategies for my ankle in my daily workout. But I also think that the main injury prevention strategies that I do

perform are based on the different physical characteristics comparing to the male body. The attention in prevention strategies is now particularly based on knee injuries, and we are performing a lot of core stability.

• What's your philosophy on ankle injury prevention?

My philosophy has definitely changed in the last few years. In female soccer, injury prevention strategies are still in a state of development. That is mainly due to the soccer environment in the Netherlands that is getting more and more professional. I play with Ajax now, where we have a really professional medical staff. Performing your injury prevention programs is now normal, especially comparing to a few years ago, when I did not perform any of these strategies. I now believe that I can prevent a lot of injuries by doing these programs, especially when these are developed based on personal characteristics and goals. At Ajax, we have this possibility, and I hope that this will be a normal environment in all the top league clubs where female soccer is established.

• You're a student on the University of Amsterdam, and in your master in communication science, you're also involved in communication on injury prevention; could you update us on your project and give your ideas on this important topic?

So far, my research will be based on neuromuscular exercises. I will examine whether there is a difference in effectiveness between messages that instruct these exercises. The differences of the messages will be based on the use of a soccer celebrity versus noncelebrity x gain or loss frame. The dependent variables will inter alia contain neuromuscular behavioral attitude and intention. I will definitely share my conclusions when I have finished this research.

• Are the new generations of artificial grass, newer football shoe designs, and new training strategies beneficial to avoid ankle injuries in your opinion? When I have to choose between artificial grass and natural grass, I will always choose natural grass. I believe that despite the latest improvements, artificial grass is not beneficial to avoid injuries. My body has a lot more difficulties to recover after playing a match on artificial grass. Artificial grass sure has a lack of natural cushioning that your body needs during a soccer game or practice.

I believe the newer football shoe designs can contribute. Earlier I would play on shoes that didn't completely fit my feet. Nowadays the designers have really improved the fit of soccer shoes. Some designs do not provide the support you sometimes need, but every player has got his or her own wishes. The increased number of choice makes it possible for everyone to get a shoe that really meets all your needs. Some brands even provide specific female soccer shoes, way to go!

In my opinion the latest developments in research and associated training strategies in female soccer are really beneficial. There are some specific female soccer injuries which can be more avoided now. The more physical characteristics are known, the more specific training strategies can be developed. Through this, specific injuries can be avoided even more in the near future. • Do you believe that your sport will cause early joint problems (in your ankle) after your career?

Yes I do believe that my sport will cause early joint problems; in my opinion you can't completely avoid this. Sometimes you take risks by, e.g., continuing to play while you are suffering from injuries. Football simply is a sport with increased risk, and I keep in mind that I possibly continue to suffer from old injuries. On the other hand, in my case, the facilities that I can use help me to be as fit as possible. When I would not play at the highest level and did not have these facilities, the probability that my sport would cause early problems would certainly increase. Therefore, the differences in facilities are way too large within female soccer.

- Who will win WC 2014 in Brazil? *The Netherlands*
- Who will win WC 2015 in Canada? United States

# Chapter 17 Fifth Metatarsal Stress Fractures in Football

Hanneke Weel, Simon Goedegebuure, Pieter P.R.N. d'Hooghe, and Gino M.M.J. Kerkhoffs

**Abstract** Fifth metatarsal stress fractures are potentially career-ending injuries for professional football players and are therefore considered a high-risk stress fracture. Reported incidence among football players is relatively high. This metatarsal stress fracture usually appears in the proximal part of the bone. The lack of vascularization in the proximal part of the bone could be a reason for the healing problems. Surgical treatment seems preferable with a median time to return to sports after 14 weeks.

Keywords MT5 fracture • Football

# 17.1 Introduction Including Epidemiology

Stress fractures of the proximal fifth metatarsal (MT-5) are reported in football [1, 2] accounting up to 78 % of all stress fractures in football players [3]. A survey among club doctors of premier and first league professional football clubs in the

H. Weel • G.M.M.J. Kerkhoffs, MD, PhD

Department of Orthopedic Surgery, Academic Medical Centre Amsterdam, University of Amsterdam, Meibergdreef 9, Amsterdam 1105 AZ, The Netherlands e-mail: g.m.kerkhoffs@amc.uva.nl

S. Goedegebuure

De Sportartsen Groep, Department of Sportsmedicine, St. Lucas Andreas Hospital, Academic Medical Center, 22700, Amsterdam 1100 DE, The Netherlands

P.P.R.N. d'Hooghe, MD (⊠) Department of Orthopaedic Surgery, Aspetar Hospital, Aspire Zone, Doha, Qatar e-mail: pieter.orthopedie@gmail.com Netherlands showed that every professional football club can expect a fifth metatarsal stress fracture each 6 years. This is comparable to the number of fifth metatarsal stress fractures found in the UEFA football players database where the expectation is one-fifth metatarsal stress fracture among the players of a professional football club each 5 years [2]. Mostly young adolescents are affected, possibly due to a rapid rise of exercise intensity. The impact of this fracture can be severe in professional athletes, and it is potentially career ending. Union problems and refractures are frequent. From a treatment point of view, the most important is, especially in professional football players, a quick return to sports at former level. Knowing this, a stress fracture of the fifth metatarsal needs to be well understood by medical and paramedical individuals involved in football.

## 17.2 Functional Anatomy

The fifth metatarsal is a rather non-curved toe, marking the lateral part of the foot [4]. The tuberosity is very well recognizable on the outside of the foot. In this proximal area of the bone, a lot of adjacent structures are present. Some are firmly connected to the proximal fifth metatarsal. The most described structures of interest in this area are the ligaments to the surrounding bony structures, e.g., with the cuboid bone and the fourth metatarsal. Also attachments of the peroneal tendons are described (e.g., peroneus brevis, tertius, and sometimes fibers of the longus) and so are the insertions of the m. abductor digiti minimi and m. flexor digiti minimi [5]. Sometimes fibers of one or more of these structures are woven with parts of the lateral plantar fascia, which is also connected to the proximal fifth metatarsal [5, 6]. In the distal part of the fifth metatarsal, only weak muscles insertions and transverse ligaments are presented [7].

The vascularization of the bone has been identified in a cadaver study of 56 ft [8]. This study verified the results of previous studies by describing a so-called watershed area [9]. This watershed area is a zone just proximal of the tuberosity, in the metaphysic, where the blood supply is not optimal. The proximal part is provided of vascularization by +/-3 different arteries. One single nutrient vessel enters the diaphysis on an average of 27 mm from the medial aspect of the base of the fifth metatarsal and provides the intraosseous blood supply distally from there. Retrograde branches are responsible to provide the proximal metaphysis of blood. In this study, 100 % of the nutrient arteries arose from the fourth plantar metatarsal artery.

## 17.3 Etiology and Injury Mechanism

Stress fractures are overuse injuries and also called fatigue fractures. The etiology differs from a "normal" or traumatic fracture. A stress fracture develops over time and is therefore more like an overload injury. The damage caused by repeated forces

Fig. 17.1 Typical fifth metatarsal stress fracture



on the bone outruns the remodeling capacity, resulting in microfractures, a weak spot, and eventually a fracture [10-12]. Compared to normal fracture, stress fractures do not seem to heal via callus formation, but more by primary bone healing with remodeling across the fracture line [10, 13, 14] which is a slower process, sometimes even progressing to complete fractures and refractory healing as similar to nonunion [10, 14].

Several theories about anatomical risk factors like cavovarus foot [15, 16] and curved type MT5 bone in the medial oblique radiograph [16] have been reported. Although, firm evidence for either one of these anatomical variations is still lacking.

Stress fractures usually appear in the proximal metaphysic of the bone (Fig. 17.1). This is the location where the watershed area is present. Distinction from other

fractures of the proximal part of the bone is important, because of the discussed differences in genesis of traumatic fractures and stress fractures. This implicates a different approach, treatment, and time to return to sport [17].

## **17.4** Clinical Examination

Patients suffering a fifth metatarsal stress fracture often do have a history with prodromal symptoms. These symptoms include a dull pain in the lateral part of the foot around the tuberosity which is usually aggravated during walking or sportive actions. The prodromal symptoms eventually worsen after an acute moment of severe pain; in other cases they have a more chronic character and are worsening gradually. The athletes who recently increased their training intensity and are of adolescent age are at risk to develop a stress fracture.

When performing physical examination, notice that the tuberosity of the fifth metatarsal is a marker of the lateral foot. Patients could have a swollen area, sometimes colored red. They could suffer from pain when performing axial forces in the metatarsal bone and local pressure on the tuberosity of the fifth metatarsal.

## 17.5 Diagnostic Imaging

A stress reaction and sometimes a stress fracture are not always seen on standard AP-3/4 footprint views. In the early stage of the injury, the fracture line could be too vague to distinguish. An MRI or CT scan can be of help at this stage. When prodromal symptoms exist for a longer period of time, the fracture can have progressed into a stress fracture well recognizable on an X-ray by a radiolucent fracture line and evidence of a sclerotic intramedullary cavity, with or without a thickening of the affected cortex. The fifth metatarsal stress fracture is usually located just distal from its tuberosity, in the proximal metaphysic of the bone (Fig. 17.1).

# 17.6 Classification

The distinction and interpretation of stress fractures and other fractures to the proximal part of the fifth metatarsal can be difficult. Besides stress fractures, avulsion and Jones' fractures can occur in the proximal part, both with an acute origin. Though some claim that differentiation between these fractures is not necessary [18], a recent review showed a difference in outcomes between these fracture types [17].

Туре	Radiographic characteristics
T1, Torg 1	No intramedullary sclerosis, a fracture line with sharp margins, no widening or radiolucency, minimum cortical hypertrophy, or evidence of periosteal reaction – acute fracture
T2, Torg 2	Fracture line involving both cortices, associated periosteal new bone, a widened fracture line with adjacent radiolucency and evidence of intramedullary sclerosis – delayed union
T3, Torg 3	Wide fracture line with periosteal new bone and radiolucency, complete obliteration of the medullary canal at the fracture site by sclerotic bone – nonunion

Table 17.1 Torg classification for proximal fifth metatarsal fractures (Torg 84)

Existing classifications of fractures of the proximal fifth metatarsal are the Torg classification [19], zones according to Lawrence and Botte [20], and the plantar gap [21]. These classifications might help to predict outcome and guide treatment. For example, a plantar gap of more than 1 mm or Torg 2 or 3 fractures are likely to have a prolonged healing time [21]. It seems that the Torg et al. classification [19] is the most easy to use in clinical practice (Table 17.1); the Torg classification makes a clear distinction between acute fractures (T1), a delayed union or stress fracture (T2), and a nonunion (T3). Another radiological classification for stress fractures has recently been introduced [22] and has yet to prove its value. This system is not location specific, but useful to understand the fracture, although a particular stage is not associated with treatment.

#### 17.7 Treatment

#### 17.7.1 Conservative

In the developing stage, rest from activities can provide the bone some time to remodel and heal the microfractures before a definite stress fracture exists. After all the origin of a stress fracture is overloading; training intensity should be deliberated in order to prevent this type of fracture. Unfortunately, this preventive approach is not always possible, because of match-play pressure or because the stress reaction has already turned into a stress fracture. Conservative treatment is possible if the football player can manage the pain and can still play despite it. According to the biology of a stress fracture, a possible theory would be that when one keeps on playing, a total fracture could occur. This means a fracture more like a traumatic fracture and thus with more intention to heal. Keep in mind that the proximal fifth metatarsal is a high-risk stress fracture due to the localization in a watershed area. When not treated, healing problems could occur due to the lack of vascularization at the spot. Alongside continuation of playing football, conservative treatment varies from limitations of activities to non-weight-bearing cast immobilization. Average time to return to sports is reported to be 24 weeks [17].

## 17.7.2 Operative

In a recent review [17] about weighted averages of time to return to football and combined with the opinion of an expert panel, surgical treatment is suggested as first-choice treatment for professional football players. In this review an average time to return to sports of 12 weeks was found after surgery. A specific prospective study among professional football players [2] supported these results. When surgically treated, a faster healing, less refractures, and nonunions were found. Advice is to revitalize the fracture surfaces prior to fixation. Performed surgical techniques varied but are mostly based on providing compression by means of an intramedulary screw fixation, tension band wiring, or plating. None of these techniques has proven superiority in research.

## 17.8 Rehabilitation

Not much evidence is available about rehabilitation of fifth metatarsal stress fractures in football players after surgery. Theoretically the fracture is stabilized and thus ready for function after care. When fixated and stabilized, the fracture can be loaded even when complete union is not fully achieved. This advantage of surgical treatment shortens the time to return to sports and is of great importance for the demanding professional football player.

Because the hypo-vascularization zone in which the stress fracture of the fifth metatarsal is located, keep in mind that possible healing problems can occur. Due to this, a stress fracture of the fifth metatarsal still has career-ending potentials. This information should be explained to all patients in order to avoid disappointments.

#### References

- 1. DeLee JC, Evans JP, Julian J (1983) Stress fracture of the fifth metatarsal. Am J Sports Med 11(5):349–353
- Ekstrand J, van Dijk CN (2013) Fifth metatarsal fractures among male professional footballers: a potential career-ending disease. Br J Sports Med 47(12):754–758
- Ekstrand J, Torstveit MK (2012) Stress fractures in elite male football players. Scand J Med Sci Sports 22:341–346
- Ebraheim NA, Haman SP, Lu J, Padanilam TG, Yeasting RA (2000) Anatomical and radiological considerations of the fifth metatarsal bone. Foot Ankle Int 21(3):212–215
- Theodorou DJ, Theodorou SJ, Kakitsubata Y, Botte MJ, Resnick D (2003) Fractures of proximal portion of fifth metatarsal bone: anatomic and imaging evidence of a pathogenesis of avulsion of the plantar aponeurosis and the short peroneal muscle tendon. Radiology 226(3):857–865
- Moraes do Carmo CC, de Fonseca de Almeida Melão LI, Valle de Lemos Weber MF, Trudell D, Resnick D (2008) Anatomical features of plantar aponeurosis: cadaveric study using ultrasonography and magnetic resonance imaging. Skeletal Radiol 37(10):929–935

- 7. Putz R, Pabst R (2000) Sobotta, Atlas van de menselijke anatomie. Bohn Stafleu van Loghum, Houten/Diegem
- McKeon KE, Johnson JE, McCormick JJ, Klein SE (2013) The intraosseous and extraosseous vascular supply of the fifth metatarsal: implications for fifth metatarsal osteotomy. Foot Ankle Int 34(1):117–123
- Smith JW, Arnoczky SP, Hersh A (1992) The intraosseous blood supply of the fifth metatarsal: implications for proximal fracture healing. Foot Ankle 13(3):143–152
- 10. Fazzalari NL (2011) Bone fracture and bone fracture repair. Osteoporos Int 22(6):2003-2006
- Kaeding CC, Yu JR, Wright R, Amendola A, Spindler KP (2005) Management and return to play of stress fractures. Clin J Sport Med 15(6):442–447
- 12. Warden SJ, Burr DB, Brukner PD (2006) Stress fractures: pathophysiology, epidemiology, and risk factors. Curr Osteoporos Rep 4(3):103–109
- Dimitriou R, Tsiridis E, Giannoudis PV (2005) Current concepts of molecular aspects of bone healing. Injury 36(12):1392–1404
- 14. Kidd LJ, Stephens AS, Kuliwaba JS, Fazzalari NL, Wu AC, Forwood MR (2010) Temporal pattern of gene expression and histology of stress fracture healing. Bone 46(2):369–378
- Hens J, Martens M (1990) Surgical treatment of Jones fractures. Arch Orthop Trauma Surg 109(5):277–279
- Lee KT, Kim KC, Park YU, Kim TW, Lee YK (2011) Radiographic evaluation of foot structure following fifth metatarsal stress fracture. Foot Ankle Int 32(8):796–801
- Kerkhoffs GM, Versteegh VE, Sierevelt IN, Kloen P, van Dijk CN (2012) Treatment of proximal metatarsal V fractures in athletes and non-athletes. Br J Sports Med 46(9):644–648
- Chuckpaiwong B, Queen RM, Easley ME, Nunley JA (2008) Distinguishing Jones and proximal diaphyseal fractures of the fifth metatarsal. Clin Orthop Relat Res 466(8):1966–1970
- Torg JS, Balduini FC, Zelko RR, Pavlov H, Peff T, Das M (1984) Fractures of the base of the fifth metatarsal distal to the tuberosity. Classification and guidelines for non-surgical and surgical management. J Bone Joint Surg Am 66(2):209–214
- Lawrence SJ, Botte MJ (1993) Jones' fractures and related fractures of the proximal fifth metatarsal. Foot Ankle 14(6):358–365
- Lee KT, Park YU, Young KW, Kim JS, Kim JB (2011) The plantar gap: another prognostic factor for fifth metatarsal stress fracture. Am J Sports Med 39(10):2206–2211
- Kaeding CC, Miller T (2013) The comprehensive description of stress fractures: a new classification system. J Bone Joint Surg Am 95(13):1214–1220

# Chapter 18 Acute Achilles Tendon Rupture

Jon Karlsson, Nicklas Olsson, and Katarina Nilsson Helander

**Abstract** Total Achilles tendon ruptures are most common in racket sports, and the incidence in football is low. Most footballers who sustain a total Achilles tendon rupture are recreational players around 40 years of age. This injury also occurs in top-level players, although not frequent. The diagnosis is made by clinical evaluation, and only in few cases MRI and/or ultrasonography are needed to establish the diagnosis. Treatment can be either nonsurgical or surgical, and in active football players surgical treatment is recommended. The main reason for surgical treatment is restoration of tendon length and improved muscle strength. The recommended surgical treatment is mini-invasive approach, with end-to-end suture using stable sutures and local adaptation of the tendon gap. Early range of motion (in some cases, even without plaster or brace) is recommended. A recent study revealed a rerupture rate of 0 % using this technique. During the last decade, major improvements in terms of rehabilitation have shown promising results.

# 18.1 Background

Achilles is the heroic Greek warrior of Homer's Iliad, son of Peleus and the nymph Thetis. In the classical version his mother Thetis made Achilles immortal by immersing him in the river Styx. Since she was holding him by the heel, this part remained vulnerable. Another less famous story tells that Thetis anointed him in ambrosia and put him into the fire to burn all the mortal parts of his body. Peleus interrupted Thetis before she had completed the mission of burning all mortal parts and leaving the heel vulnerable. In the Trojan War, Achilles killed Hector, the prince

J. Karlsson, MD, PhD (⊠) • N. Olsson, MD, PhD • K.N. Helander, MD, PhD Institute of Clinical Sciences at Sahlgrenska Academy, Sahlgrenska University Hospital, University of Gothenburg, Gothenburg, Sweden e-mail: jon.karlsson@telia.com

Fig. 18.1 Acute Achilles tendon rupture



of Troy. Hector's brother Paris kills Achilles, with assistance from Apollo, in revenge by shooting a poisoned arrow into Achilles unprotected heel (Fig. 18.1).

The incidence of Achilles tendon rupture appears to be rising, and approximately 75 % of all ruptures occur during sport activities, especially related to racket sports. Total rupture is, however, not frequent among active football players. Today, an Achilles tendon rupture is treated surgically, using either standard open technique or mini-invasive (percutaneous) technique or nonsurgically with different mobilization alternatives [35]. Surgical treatment is recommended in young and highly active sports persons, e.g., football players. One main reason is the shorter layoff and earlier and safer return to sports. There is a wide variation of immobilization methods after both surgical and nonsurgical treatment including cast and functional brace with or without weight-bearing and range-of-motion training. The main focus in the literature has been to compare the outcome of different treatment options in terms of rerupture and surgical complications.

#### 18.2 Anatomy

The superficial group of muscles in the posterior crural compartment consists of the gastrocnemius, soleus, and plantaris muscles. The most superficial muscle, the gastrocnemius, has two heads of origin. The medial head that arises from the medial condyle of the femur is slightly larger and extends a little more distally than the lateral head. The lateral head arises from the lateral surface of the lateral condyle of the femur. The origin of the soleus muscle is entirely below the knee at the posterior aspect of the head and superior fourth of the fibula, the soleal line, and middle third of medial border of the tibia. The gastrocnemius and soleus muscles are sometimes together called the triceps surae muscle. The plantaris muscle is very small and absent in approximately 10 % in the population, and its tendon lies in close proximity to the Achilles tendon [21]. The plantaris tendon can be used as a graft for reinforcement during Achilles tendon surgery but has no function when it comes to calf muscle strength.

The Achilles tendon is formed by three flat and broad aponeuroses from each muscle in the triceps surae. The Achilles tendon becomes progressively rounder in shape until it reaches 4 cm from the insertion site at the superior calcaneal tuberosity where it becomes flatter again [21]. Kager's fat pad is located in the Kager's triangle between the anterior aspect of the Achilles tendon, the posterior aspect of the tibia, and the superior aspect of the calcaneus. It has been hypothesized that this fat pad lubricates the anterior part of the Achilles tendon and also reduces pressure of the tendon. There is a retrocalcaneal bursa that is located between the tendon and the calcaneus. Between the skin and the tendon, there is a subcutaneous bursa, which reduces the friction between the tendon and the surrounding tissues.

The fibers of the Achilles tendon rotate approximately  $90^{\circ}$  during its descent, such that the fibers that lie medially in the proximal portion becomes more posterior further distally. This spiraling of the tendon contributes to the elongation and elastic recoiling within the tendon. The gastrocnemius muscle acts on both the knee and ankle joint by flexion of the knee and plantar flexion of the ankle but also supination of the foot. The soleus muscle acts only over the ankle joint and therefore produces a plantar flexion and to the same extent a supination of the foot. The gastrocnemius muscle contains a greater number of white, type II fibers and produces fast action that is important during activities like running. The soleus muscle contains more of the slow, red type I fibers and is important to maintain posture. The triceps surae muscles are innervated by the tibial nerve.

#### 18.3 Tendon Structure

The Achilles tendon is in similarity to all tendons dominated by type I collagen, which explains its high strength. The collagen accounts for 65–80 % of the dry weight and elastin approximately of 1–2 %. The collagen is embedded in a proteoglycan-water matrix. Collagen is produced by fibroblasts and fibrocytes that lie between the collagen fibers in a complex structure [11]. Tendon stem cells have recently been found in human tendons. The synthesis of collagen fibrils follows first as an intracellular step assembling and secretion of procollagen. The extracellular step converts the procollagen into tropocollagen. Five tropocollagen molecules (or microfibril) are cross-linked and aggregated into collagen fibrils. The stability and quality of the collagen is largely based on the cross-links [15]. Multiple collagen fibrils are embedded in the extracellular matrix and forming collagen fibers. This is the basic unit of a tendon and the smallest visible (light microscopy) tendon unit (Fig. 18.2).

The structure of the tendon forming *the primary, secondary, and tertiary* bundles, even though the nomenclature may vary in the literature [15]. The collagen fibers vary but could be as long as the tendon. Tendons in the hand and foot are covered with a synovial sheet. The Achilles tendon does not have a true synovial sheet, but instead a peritendinous sheet often called paratenon. The paratenon functions as a sleeve that allows free movement of the tendon. A fine connective tissue sheath called epitenon surrounds the tendon, and the outer surface is contiguous



Fig. 18.2 Stress-strain curve showing elongation, minor rupture, and finally total rupture of the Achilles tendon

with the paratenon. Inside the Achilles tendon, the endotenon surrounds the different bundles of the tendon. Blood vessels and nerves run inside the endotenon.

## 18.4 Epidemiology

Jozsa and co-workers [9] reported that, of the tendons requiring surgery, the Achilles tendon is the most frequently ruptured. The incidence of Achilles tendon ruptures in the population is increasing. Leppilahti and co-workers [17] showed an annual incidence rising from 2/100,000 between the years 1979 and 1986 to 12/100,000 between the years 1979 and 1986 to 12/100,000 between the years 1987 and 1994, and Houshian and co-workers reported in a more recent study an annual incidence rising from 18/100 000 in the year 1984 to 37/100,000 in the year 1996. The incidence was highest in the age group 30–39 years. Houshian and co-workers showed that 73 % of the injuries were sport related and the peak of sport-related injuries occurred in the 30–49 age group. This age group is somewhat older than most active football players. This means that acute Achilles tendon rupture is relatively non-frequent in the elite football player, between 20 and 30 years of age, but much more frequent in recreational football players, 30 years and older.

There is a second non-sport-related peak in incidence occurring at a mean age of 53 years [17]. The risk of a contralateral tendon rupture is significantly increased in patients who have previously suffered an Achilles tendon rupture. Most Achilles

tendon ruptures occur in men, and the ratio between men and women is between 3:1 and 18:1 (in meta-analyses 9–10:1)

#### 18.5 Mechanism of Rupture

The most common injury mechanisms for Achilles tendon ruptures have been classified into three main categories all with a very distinct patient history [1]. In the first mechanism, the patient is pushing off with the weight-bearing forefoot while the knee is extended. This mechanism is described by the majority of patients and is seen in sprint starts, jumping, and racket sports. The second mechanism is a sudden, unexpected dorsiflexion of the ankle, which occurs when the patient slips into a hole or falls downstairs. The third mechanism is a violent dorsiflexion of a plantar-flexed foot, such as after a fall from a height.

#### 18.6 Presentation and Diagnosis

Patients who sustain an Achilles tendon rupture have a characteristic history of a sudden pain in the Achilles tendon without any previous symptoms. It is often reported from patients that it felt like they had been struck by something/someone from behind and often accompanied by an audible snap. In the typical case the diagnosis is clear. The diagnosis is clinical, and there is a palpable gap at the site of the rupture during the first week. The ability to plantar flex the ankle is absent or very weak. In the literature there are numerous different clinical diagnostic tests described [20]. Sensitivity and specificity have been evaluated for these various clinical tests. The calf-squeeze test and Matle's test had the highest sensitivity and specificity, and these tests are also noninvasive, simple, and inexpensive [20].

The calf-squeeze test is also known as Simmonds' or Thompson's. The patient lies in prone position, and the examiner squeezes the affected calf muscle, and if the tendon is intact, the foot will plantar flex, but if the tendon is ruptured, there will be no or minimal reaction on the foot and the test is said to be positive. In the Matle's test, the patient actively flexes both knees, and the change in position of the ankles is observed. The test is positive if the foot on the injured side moves into neutral or dorsiflexion. Imaging examinations by either ultrasonography (US) or magnetic resonance imaging (MRI) are not recommended to be used routinely to establish the diagnosis in acute ruptures.

#### **18.7** Surgical or Nonsurgical Treatment

There is an ongoing debate of optimal treatment after an acute Achilles tendon rupture, especially when dealing with sport-related injuries [3, 5, 8, 13, 14, 16, 18, 23, 24, 25, 28, 30, 33, 34, 36]. Several randomized controlled studies and meta-analysis have been made in the literature; however, they are not related to football players in the first place. The general discussion has been between surgical and nonsurgical treatment. Each type of treatment is having its own drawbacks. One can conclude from the literature that open surgical repair has two to four times lesser risk of rerupture compared to nonsurgical treatment, but on the other hand having the risk of surgery-related complications [3, 14, 23, 25, 27, 28, 33, 36]. These complications are superficial or deep infection, n. suralis injury, and scar problems. From a functional outcome perspective, no treatment has been shown to be superior to the other. The knowledge in the literature has shown that patients are not fully recovered, concerning symptoms and function, and these deficits seem to be permanent [29].

There are nonsignificant differences between surgical and nonsurgical treatment. An exception is the heel-rise work test in the nonsurgical group having significantly lower LSI values after both the first and second year [32]. The nonsurgical group did, however, improve significantly in this test between the 1- and 2-year follow-up [25, 29]. This supports the interpretation that it appears to be a slower recovery after nonsurgical treatment, and this may be of major importance, for instance, for active football players, in which timely return to sports may be important for the future carrier.

Due to the deficits in function seen in previous studies and the indication that tendon elongation might be a major issue [10], a randomized controlled study showed that a very stable surgical technique that could tolerate an accelerated rehabilitation protocol showed excellent results, with 0 % rerupture rate [30]. The tendon was directly postoperatively exposed to mechanical load, which has been shown to be beneficial for tendon healing in animal studies.

This type of stable surgical technique and accelerated rehabilitation, including immediate weight-bearing, range-of-motion, and strength training, is a safe, well-tolerated method. There is some evidence that this treatment was superior to nonsurgical treatment with immediate weight bearing in a brace, when comparing functional results [30]. In this randomized controlled study with stable repair and accelerated rehabilitation, the LSI variables in the surgically treated group were numerically higher when assessed by the functional evaluation tests including jump, power, and endurance at the 12-month follow-up compared with the nonsurgical group (Figs. 18.3 and 18.4). Favorable functional results after surgical treatment at 12 months have also been reported in the study by Nilsson-Helander and co-workers [25]. In that study, the same functional evaluation was used, and the absolute values in the different tests were 4–11 % higher in favor of surgical treatment compared with nonsurgical treatment (both groups had identical rehabilitation). There are approximately the same results in the two studies, indicating a tendency of superior results for surgical treatment.

This surgical treatment had no reruptures (0 %), which is in strong contrast to other previous randomized studies comparing open surgical technique to nonsurgical technique. Therefore, it is concluded that this method of treatment is safe (Figs. 18.3 and 18.4) [30].

Animal studies show the importance of mechanical load in tendon healing [2, 4, 7, 31]. In the literature there are no convincing results from randomized controlled studies that show that mechanical load is superior, even though from a historical perspective, the outcome seems to improve over time, and we consider this as a





result of more functional bracing rehabilitation. The AAOS guidelines recommend early protected postoperative weight bearing. Presently, a treatment protocol that promoted immediate weight bearing and also including controlled range-of-motion and strength training early in the surgical group is recommended in young active athletes, e.g., football players [22]. This indicates and supports a shift toward early loading of the tendon. Percutaneous suture has also been shown to be a valuable method (Fig. 18.5) [19].

There are general advantages of surgical repair that includes the opportunity of putting the tendon ends in the optimal length, even though the optimal length is not known. The surgeon can nevertheless put the tendon in tension, and the correct length of the tendon is probably easier to achieve by surgical treatment compared with nonsurgical, and this could explain the difference in function in favor of surgical treatment.


In the treatment of Achilles tendon ruptures, there is a difficult balance between loading the tendon for optimal stimulation and the risk of complications such as rerupture [20, 25, 30]. There is another advantage of stable surgical repair that it can allow the tendon to be early mechanically loaded in an accelerated rehab protocol that is not acceptable for nonsurgical treatment. In this randomized controlled study, one should notice that the accelerated rehabilitation did not only mobilize the patient, but the early range-of-motion and strength training gave the tendon a direct mechanical load [30]. There are difficulties in quantifying the actual tendon load, and there is probably a wide variation between patients in mechanical load to the tendon. Weight bearing is possible to measure in pressure-sensitive soles and can be used in future studies, but there are still difficulties in measuring the actual tendon load.

Fig. 18.4 Enforcement sutures



# 18.8 Physical Activity and Return to Football

Since patients with an Achilles tendon rupture have been found to have long-term functional deficits, it could be that this decrease in activity level is an adaptation and adjustment to fit their current functional capacity. The adjustments to a lower activity level may occur not only in sport and recreational activities but also in activities of daily life. The long-term effects of these limitations of physical activity level are uncertain, but in this middle-aged population and not high-demanding athletes, the interruption of daily training and the deficits in function may lead to persistently decreased physical activity in the future. Consequently this could lead to increased risk of general health-related disorders, and, therefore, an acute Achilles tendon rupture could be detrimental to the general health.

There is a variation of definition of return to sport and previous physical activity level among studies, but nevertheless in all studies, approximately half or two-thirds of the patients return to previous activity level 1 and 2 years after an acute Achilles tendon rupture [6, 12, 23, 28, 30]. These studies include a span of 30 years, and the results remain nevertheless fairly stable over time despite different treatment protocols.

# 18.9 Conclusion

It is concluded that stable surgical repair with early accelerated tendon loading is a safe method with a low risk of rerupture. However, this treatment might not be significantly superior to nonsurgical treatment in terms of functional results, similar to what have been previously reported in the literature. Nevertheless this kind of treatment shows an important tendency of superior functional results, less degree of symptoms, and no reruptures in patients treated with this type of stable surgical repair and accelerated rehabilitation. This kind of treatment might also lead to less risk of tendon lengthening and reduced strength during the first 6 months after the injury. This kind of treatment will also lead to shorter rehabilitation period and safer return to football play.

# References

- 1. Arner O, Lindholm A (1959) Subcutaneous rupture of the Achilles tendon; a study of 92 cases. Acta Chir Scand Suppl 116(Supp 239):1–51
- 2. Aspenberg P (2007) Stimulation of tendon repair: mechanical loading, GDFs and platelets. A mini-review. Int Orthop 31(6):783–789
- 3. Bhandari M, Guyatt GH, Siddiqui F et al (2002) Treatment of acute Achilles tendon ruptures: a systematic overview and metaanalysis. Clin Orthop Relat Res 400:190–200
- Bring DK, Kreicbergs A, Renstrom PA, Ackermann PW (2007) Physical activity modulates nerve plasticity and stimulates repair after Achilles tendon rupture. J Orthop Res 25(2):164–172
- Cetti R, Christensen SE, Ejsted R, Jensen NM, Jorgensen U (1993) Operative versus nonoperative treatment of Achilles tendon rupture. A prospective randomized study and review of the literature. Am J Sports Med 21(6):791–799
- Cetti R, Henriksen LO, Jacobsen KS (1994) A new treatment of ruptured Achilles tendons. A prospective randomized study. Clin Orthop Relat Res 308:155–165
- Eliasson P, Andersson T, Aspenberg P (2012) Achilles tendon healing in rats is improved by intermittent mechanical loading during the inflammatory phase. J Orthop Res 30(2):274–279
- Ingvar J, Tagil M, Eneroth M (2005) Nonoperative treatment of Achilles tendon rupture: 196 consecutive patients with a 7% re-rupture rate. Acta Orthop 76(4):597–601
- Jozsa L, Kannus P (1997) Histopathological findings in spontaneous tendon ruptures. Scand J Med Sci Sports 7(2):113–118
- Kangas J, Pajala A, Ohtonen P, Leppilahti J (2007) Achilles tendon elongation after rupture repair: a randomized comparison of 2 postoperative regimens. Am J Sports Med 35(1):59–64
- 11. Kannus P (2000) Structure of the tendon connective tissue. Scand J Med Sci Sports 10(6):312–320
- 12. Kearney RS, Achten J, Lamb SE, Parsons N, Costa ML (2012) The Achilles tendon total rupture score: a study of responsiveness, internal consistency and convergent validity on patients with acute Achilles tendon ruptures. Health Qual Life Outcomes 10:24
- Kerkhoffs GM, Struijs PA, Raaymakers EL, Marti RK (2002) Functional treatment after surgical repair of acute Achilles tendon rupture: wrap vs walking cast. Arch Orthop Trauma Surg 122(2):102–105
- Khan RJ, Fick D, Keogh A, Crawford J, Brammar T, Parker M (2005) Treatment of acute achilles tendon ruptures. A meta-analysis of randomized, controlled trials. J Bone Joint Surg Am 87(10):2202–2210

- 18 Acute Achilles Tendon Rupture
- Kjaer M (2004) Role of extracellular matrix in adaptation of tendon and skeletal muscle to mechanical loading. Physiol Rev 84(2):649–698
- Kocher MS, Bishop J, Marshall R, Briggs KK, Hawkins RJ (2002) Operative versus nonoperative management of acute Achilles tendon rupture: expected-value decision analysis. Am J Sports Med 30(6):783–790
- 17. Leppilahti J, Forsman K, Puranen J, Orava S (1998) Outcome and prognostic factors of Achilles rupture repair using a new scoring method. Clin Orthop Relat Res 346:152–161
- Lo IK, Kirkley A, Nonweiler B, Kumbhare DA (1997) Operative versus nonoperative treatment of acute Achilles tendon ruptures: a quantitative review. Clin J Sport Med 7(3):207–211
- Ma GW, Griffith TG (1977) percutaneous repair of acute closed ruptured achilles tendon: a new technique. Clin Orthop Relat Res 128:247–255
- Maffulli N (1998) The clinical diagnosis of subcutaneous tear of the Achilles tendon. A prospective study in 174 patients. Am J Sports Med 26(2):266–270
- 21. Maffulli N (1999) Rupture of the Achilles tendon. J Bone Joint Surg Am 81(7):1019-1036
- 22. Maffulli N, Tallon C, Wong J, Lim KP, Bleakney R (2003) Early weightbearing and ankle mobilization after open repair of acute midsubstance tears of the achilles tendon. Am J Sports Med 31(5):692–700
- Moller M, Movin T, Granhed H, Lind K, Faxen E, Karlsson J (2001) Acute rupture of tendon Achilles. A prospective randomised study of comparison between surgical and non-surgical treatment. J Bone Joint Surg Br 83(6):843–848
- 24. Nilsson-Helander KN-K (2008) A new surgical method to treat chronic ruptures and reruptures of the Achilles tendon. Knee Surg Sports Traumatol Arthrosc 16(6):614
- 25. Nilsson-Helander K, Silbernagel KG, Thomee R et al (2010) Acute achilles tendon rupture: a randomized, controlled study comparing surgical and nonsurgical treatments using validated outcome measures. Am J Sports Med 38(11):2186–2193
- 26. Nilsson-Helander K, Thomee R, Gravare-Silbernagel K et al (2007) The Achilles tendon Total Rupture Score (ATRS): development and validation. Am J Sports Med 35(3):421–426
- Nilsson-Helander K, Thurin A, Karlsson J, Eriksson BI (2009) High incidence of deep venous thrombosis after Achilles tendon rupture: a prospective study. Knee Surg Sports Traumatol Arthrosc 17(10):1234–1238
- Nistor L (1981) Surgical and non-surgical treatment of Achilles Tendon rupture. A prospective randomized study. J Bone Joint Surg Am 63(3):394–399
- Olsson N, Nilsson-Helander K, Karlsson J et al (2011) Major functional deficits persist 2 years after acute Achilles tendon rupture. Knee Surg Sports Traumatol Arthrosc 19(8):1385–1393
- Olsson N, Silbernagel KG, Eriksson BI, et al. (2013) Stable surgical repair with accelerated rehabilitation versus nonsurgical treatment for acute achilles tendon ruptures: a randomized controlled study. Am J Sports Med. 41(12):2867–2876
- 31. Schepull T, Kvist J, Andersson C, Aspenberg P (2007) Mechanical properties during healing of Achilles tendon ruptures to predict final outcome: a pilot Roentgen stereophotogrammetric analysis in 10 patients. BMC Musculoskelet Disord 8:116
- 32. Silbernagel KG, Nilsson-Helander K, Thomee R, Eriksson BI, Karlsson J (2010) A new measurement of heel-rise endurance with the ability to detect functional deficits in patients with Achilles tendon rupture. Knee Surg Sports Traumatol Arthrosc 18(2):258–264
- Soroceanu A, Sidhwa F, Aarabi S, Kaufman A, Glazebrook M (2012) Surgical versus nonsurgical treatment of acute achilles tendon rupture: a meta-analysis of randomized trials. J Bone Joint Surg Am 94(23):2136–2143
- 34. Thermann H, Zwipp H, Tscherne H (1995) Functional treatment concept of acute rupture of the Achilles tendon. 2 years results of a prospective randomized study. Unfallchirurg 98(1):21–32
- van Dijk CN, van Sterkenburg MN, Wiegerinck JI, Karlsson J, Maffulli N (2011) Terminology for Achilles tendon related disorders. Knee Surg Sports Traumatol Arthrosc 19(5):835–841
- 36. Willits K, Amendola A, Bryant D et al (2010) Operative versus nonoperative treatment of acute Achilles tendon ruptures: a multicenter randomized trial using accelerated functional rehabilitation. J Bone Joint Surg Am 92(17):2767–2775

# Chapter 19 Achilles Tendinopathy

Robert Jan de Vos, Pieter P.R.N. d'Hooghe, Peter de Leeuw, and Gino M.M.J. Kerkhoffs

I always think about what I missed, and I think that was my driving force - never be satisfied with what I've done.

Thierry Henry

Abstract Overuse Achilles tendon disorders are frequently seen in middleaged, active people. Achilles tendon injuries are also common in elite-level football and result in the longest absences from playing. These tendon injuries are currently referred to as 'tendinopathy', a clinical diagnosis that can be made in the presence of pain, swelling of the Achilles tendon and an impaired load-bearing capacity. Histopathological studies have shown that chronic tendinopathy is frequently characterised by degeneration of the tendon tissue and not inflammation. When evaluating tendinopathies, it is important to differentiate between midportion and insertional disorders and to separate acute from chronic injuries. These injury characteristics will mainly guide treatment options and affect prognosis. For both insertional and midportion acute tendinopathies, the first treatment of choice is a symptom-based reduction in load and NSAIDs. For chronic midportion Achilles tendinopathy, the cornerstone of treatment is currently

R.J. de Vos, MD, PhD (⊠) Department of Sports Medicine, Aspetar Hospital, SportsCity Street – Aspire Zone, Doha, Qatar

The Hague Medical Centre, Burgemeester Banninglaan 1, Leidschendam AK 2260, The Netherlands e-mail: r.devos@eramsumc.nl

P.P.R.N. d'Hooghe, MD Department of Orthopaedic Surgery, Aspetar Hospital, Aspire Zone, Doha, Qatar e-mail: pieter.orthopedie@gmail.com

P. de Leeuw • G.M.M.J. Kerkhoffs, MD, PhD

Department of Orthopedic Surgery, Academic Medical Centre Amsterdam, University of Amsterdam, Meibergdreef 9, Amsterdam 1105 AZ, The Netherlands e-mail: p.a.deleeuw@amc.uva.nl; g.m.kerkhoffs@amc.uva.nl

eccentric exercise therapy. Efficacy of other treatment options needs to be proven. Chronic insertional tendinopathy can be treated with anti-inflammatory treatment if a bursitis is present. In cases of intratendinous pathology, eccentric exercise therapy from a flat ground is proposed and extracorporeal shock wave therapy might be another promising option. Surgery might be considered for both insertional and midportion tendinopathies if conservative treatment fails. The prognosis for chronic tendinopathies is currently disappointing and difficult to predict, regardless of treatment.

**Keywords** Tendon overuse injuries • Tendinosis • Tendinitis • Histopathology • Imaging • Treatment • Surgery

### **19.1 Introduction**

Overuse Achilles tendon disorders are frequently seen in middle-aged, active people. Approximately 2-3/1,000 patients consult a general practitioner because of pain in the Achilles region [1]. This probably only represents the tip of the iceberg though, as it is known that in elite running athletes, there is a lifetime risk of sustaining an Achilles tendon injury of 52 % [2]. In elite-level football, Achilles tendon disorders are also common as this injury represents 2.4 % of all injuries [3]. Moreover, when evaluating the most frequent injuries in these football players, Achilles tendon disorders were found to result in the longest absences from playing. These overuse tendon injuries are therefore important to consider in football.

In recent decades, the popularity of football has increased in the general population, and there are increased physical demands placed on high-level competitive players. This might result in an increased risk of overuse injuries. The aetiology of these overuse injuries is largely unknown. It is likely that a combination of ageing and increased activity levels leads to an increased risk of overuse injuries.

The phrase 'Too much, too soon, too often' is frequently heard in the patient's history.

The terminology for describing tendon disorders has changed over the past decades [4]. Previously, this condition was referred to as 'tendinitis', implying an inflammation of the tendon tissue. However, as there are no signs of inflammation in chronic painful tendons, this term has been abandoned [4, 5]. The term 'tendinopathy' as a descriptor of the clinical picture is now used to redress this confusing terminology. Tendinopathy is a clinical diagnosis that can be established in the presence of pain, swelling of the Achilles tendon and an impaired load-bearing capacity [4, 6]. Histopathological studies have shown that chronic tendinopathy is frequently characterised by degeneration of the tendon tissue, also denoted as 'tendinosis' [7]. However, the term tendinosis is based on histopathological characteristics and should only be used after

histopathological confirmation [6]. The treatment of tendinopathy has always been a challenge in sports medicine and orthopaedics, and therefore it is becoming a major problem in this field.

# 19.2 Anatomy

The Achilles tendon connects the gastrocnemius muscles and soleus muscle with the calcaneus and is the strongest and largest tendon in the human body. The plantaris tendon was previously thought to be absent in many cases [8]; however, a recent study showed that the plantaris tendon contributed to the Achilles tendon in all cases [9]. The musculotendinous junction of the Achilles tendon originates in the middle of the lower leg, and the tendon is approximately 15 cm in length.

The tendon inserts on the posterior surface of the calcaneus and contains fibrocartilage tissue in this insertional site. Two bursae are present adjacent to the insertion. The innate retrocalcaneal bursa is located proximal and ventral to the Achilles tendon and on the posterior surface of the calcaneus [8]. The superficial Achilles bursa is an acquired bursa that may be present between the distal Achilles tendon insertion and the subcutaneous tissues [10]. The area between the proximal part of the calcaneus, the posterior margin of the tibia and the ventral side of the Achilles tendon is known as Kager's triangle and contains a fat pad. This structure is thought to play a role in the blood supply of the tendon through small vincula, although it is well known that vascularisation in this area of the Achilles tendon is physiologically the lowest [11]. This midportion area of the Achilles tendon, located 2-7 cm proximal to the insertion, shows a spiral rotation around its axis that works as an elastic coil, functioning as an energy-storing tendon to improve the efficiency of locomotion [12]. In vivo measurements revealed that Achilles tendons of athletes are sometimes subjected to tensile loads up to 12.5 times their body weight during running [13]. These forces are transmitted through the tendon collagen bundles. Microscopically, the tendon collagen tissue is hierarchical in structure and well ordered, predominantly in a longitudinal direction. The collagen is densely packed, and macroscopically it provides a typical white, glistening appearance. Tendon collagen type I is the main extracellular matrix (ECM) constituent, which is produced by tenocytes [7]. The major non-collagenous ECM constituents are water-attracting proteoglycans. The ECM does not expand due to this water attraction in healthy tendons which may provide increased internal strength due to a greater resistance to tensile and compressive forces [14]. The collagen fibres in the ECM of healthy tendons are in a state of dynamic equilibrium between synthesis and degradation, regulated by matrix metalloproteinases and their inhibitors [7]. The tendon is enveloped by the peritendineum, a thin membrane of loose areolar tissue. This tissue comprises more blood vessels and nerves, and its main function is to allow tendon gliding without restrictions against the surrounding tissues [8].



**Fig. 19.1** Histology and histopathology of tendon tissue. On the *left image*, tissue of a healthy tendon is presented. The collagen fibres are *purple* coloured. This collagen is produced by sparsely distributed tenocytes and oriented in the direction of the tensile forces. On the *right image*, a tissue sample of a patient with chronic Achilles tendinopathy is displayed. There is a clear loss of a well-organised tendon tissue structure and the tenocyte nuclei may become more rounded, and there are areas of hypercellularity and areas of hypocellularity. These characteristics might be an expression of cellular dysfunction

# 19.3 Histopathology

Achilles tendon disorders can be divided into abnormalities of the intratendinous tissues, peritendineum and bursae [15]. Tendinosis is a general descriptor of intratendinous disorders, which encompasses a wide range of histopathological entities (Fig. 19.1). The main abnormalities observed are the loss of a well-organised tendon tissue structure, changes in morphology and distribution of tenocytes and increased interfibrillar proteoglycans [6, 16]. Macroscopically, degenerative tendon has a soft appearance with a greyish, white colour (Fig. 19.2). Another feature of tendinosis is an increased number of blood vessels which are thought to be newly formed. This 'neovascularisation' is characterised by a tortuous phenotype and a small lumen, but its functional relevance is still unknown [17].

Another feature that can be observed macroscopically is thickening of the peritendineum [6, 18]. Adhesions between the tendon tissue and peritendineum may be present, which may result in contractions around the tendon due to proliferation of the connective tissue. Increased friction between the tendon and the surrounding structures can be the consequence [15]. This condition is frequently referred to as paratendinopathy if there is clinical or radiological suspicion. Recently, adhesions with the plantaris tendon have also been postulated as a source of pain [9].

The above-mentioned abnormalities are well defined; however, there remain some gaps in scientific knowledge regarding pain generation in tendinopathies. Histological abnormalities are present in more than 50 % of previously asymptomatic Achilles tendons of older individuals [19]. This indicates that a certain degree of degenerative tendon changes may be more frequent with increasing age. A clear association between tendon structure organisation and reported patient symptoms



**Fig. 19.2** Macroscopic view on an incised degenerative Achilles tendon during surgery. The skin and subcutaneous tissues are incised, and the peritendineum has been removed. An incision is made in the Achilles tendon to identify degenerative tissue, which is macroscopically characterised by a *greyish colour* and loss of *white* glistening appearance

has not been found using current imaging modalities [20, 21]. One might therefore conclude that there is a missing link between the pain experienced and the presence of tendon structure disorganisation. Several biochemical agents and changes in peritendinous structures have been advocated as pain generators in chronic tendinopathies, but to date we can only speculate as to whether they are causative or a result of tendon degeneration, since their association with the severity of symptoms is largely unknown. There is also scarce scientific knowledge on the progression of tendon tissue disorders in time. Recently, a continuum of tendon pathology model has been proposed. Cook and Purdam [22] suggested that tendon overuse initially results in a reactive phase, characterised by cell proliferation and increase in proteoglycans with intact tendon collagen fibres. If overuse continues or recurs, tendon dysrepair might be the next phase. This phase is characterised by tenocyte proliferation, disorganised tendon tissue structure and neovascularisation. These first two phases are thought to be more or less reversible. Degenerative tendinopathy is regarded as the end stage of overuse tendon disorders, and this stage is obviously the area in which most research is performed. The histopathological changes described in the reactive and dysrepair phases are based on findings in animal studies and imaging studies in humans. This is currently the best evidence available. However, the stages preceding this degenerative state might be very important in the understanding, prevention and treatment of tendinopathies. Furthermore, the absence of inflammatory cells in chronic painful tendons does not exclude the possibility of inflammatory mediators involved in the development of tendinopathies. Several inflammatory markers have been proposed to influence tendon cell activity. This could theoretically result in a change to growth factor homeostasis and disturbance in tendon matrix turnover as a result of the disturbance in proteolytic matrix metalloproteinase activity [23]. Animal studies have previously shown that repetitive exposure of prostaglandins to tendons eventually

results in the degeneration of the collagen tissue [24]. These inflammatory markers could not be the cause of pain in chronic tendinopathy because they have been shown to be absent in this phase. Knowledge about pain physiology has advanced significantly in musculoskeletal medicine, and new research is expected in the field of central sensitisation mechanisms [25].

The histopathology of insertional disorders is not as well researched as midportion disorders. It is therefore important to address the relevant structures that might be involved at the insertional site. Three main anatomical structures can be involved, and abnormalities of these structures can coexist: the calcaneus, the Achilles tendon insertion (which also includes the first 2 cm of the tendon proximal to the insertion) and the two bursae [26]. It is thought that these structures are more susceptible to compressive, rather than tensile forces. This may be caused by external compression or due to impingement from repetitive dorsiflexion of the ankle. The superolateral portion of the posterior part of the calcaneus can be enlarged, which may predispose to higher compressive forces. This anatomical variant is designated as Haglund deformity. However, this deformity is also frequently present with an absence of symptoms [27]. Degeneration and calcifications can be present within the Achilles tendon insertional site. However, these findings have also been reported in asymptomatic individuals, so the clinical relevance of these findings still needs to be addressed. The retrocalcaneal bursa can show histopathological signs of bursal fluid accumulation, degeneration of the fibro-cartilaginous bursal walls and hypertrophy of the synovial folds [28]. These signs on histopathology examination have also been found in the superficial Achilles bursa where fluid accumulation and synovial hypertrophy have been reported [27].

### **19.4** Clinical Features

As stated previously, tendinopathy is a clinical diagnosis characterised by pain, swelling and impaired load-bearing capacity of the tendon. This diagnosis is straightforward, but a thorough history taking and physical examination are essential to the establishment of a correct diagnosis and subsequent management strategy.

There are several important injury characteristics to evaluate initially. This is not only because these characteristics influence the terminology used, but they also affect management. The location of the symptoms is one important feature. True tendon pain is almost always confined to the tendon itself, although other pathologies in the ankle and Achilles region should always be considered. The tendon pain can be either located in the Achilles tendon insertion or in the Achilles tendon midportion, located 2–7 cm proximal to the calcaneus [26]. These conditions should be distinguished clinically, because of the differing pathology, clinical decision-making and prognosis. Musculotendinous disorders are located more proximally. These injuries are less frequent and not within the focus of this chapter. Other important features are the onset and duration of symptoms. An acute onset is suggestive of tendon rupture or partial rupture, and this is discussed elsewhere (Chap. 18). The same accounts for neglected ruptures, which may be characterised by chronic pain, but following thorough examination of the patient, an acute history and considerably enlarged tendon can be found. A gradual onset is more suggestive of tendon overuse injury, and this can be classified as either acute or chronic. There are no specific time criteria for this classification; some advocate a cut-off for chronic injuries as symptoms lasting greater than 6 weeks, but others define chronic injuries as the presence of symptoms for more than 12 weeks [15]. However, these distinctions are arbitrary and not based on histopathological features.

Characteristics that are more reminiscent of tendinopathy are the presence of morning stiffness and the progress of activity-related symptoms. Initially pain is often only present during the warming-up period or after activity. In this phase, rest may have a positive influence, but symptoms will frequently recur with an increase in activity. Finally, the tendon may even become painful during rest and activities of daily living. Characteristics that may help to identify contributing factors to the development and persistence of tendinopathy are change in training intensity, technique, equipment or training surfaces [26].

It is important to address the possibility of inflammatory arthropathy involvement (e.g. rheumatoid arthritis, M. Bechterew), presence of familial hypercholesterolaemia, diabetes mellitus and preceding use of fluoroquinolone antibiotics, as these are associated with tendinopathy [18, 29].

Physical examination is needed to confirm the clinical diagnosis, but it is also helpful to address potential contributing factors. Many modifiable factors that have been proposed in the literature are obesity, an excessive foot pronation, decreased ankle dorsiflexion, decreased subtalar joint motion and decreased flexibility and strength of the calf muscles [18, 26, 29]. The clinician can decide to take these factors into account when treating patients; however, scientific evidence for the contribution of these factors is relatively scarce.

The Thompson calf squeeze test is used to evaluate the possibility of tendon rupture. Neuromeningeal provocation tests can be used if there is suspicion of referred pain from the spine. Classically, a nodular swelling can be palpated in the Achilles tendon midportion in the presence of midportion tendinopathy. In cases of acute paratendinopathy, crepitations may be felt during passive plantar flexion and dorsiflexion [30]. Insertional tendinopathy may be characterised by swelling at the central posterior aspect of the calcaneus in case of intratendinous involvement. In cases of retrocalcaneal bursa enlargement, a painful soft tissue swelling may be palpable lateral and medial to the Achilles tendon insertional site [31].

# 19.5 Imaging

There are several modalities available for imaging of the Achilles tendon. Radiographic imaging is not the prime imaging method to evaluate acute or chronic tendinopathy in the midportion of the Achilles. Ultrasonography (US) and magnetic resonance imaging (MRI) are the additional diagnostics that can be used because of



**Fig. 19.3** Ultrasonography in patients with chronic midportion Achilles tendinopathy. On the *left image* (**a**), a moderate thickening in the Achilles tendon midportion is observed. It shows a lower degree of neovascularisation, observed with power Doppler ultrasonography. On the *right image* (**b**), a high grade of neovascularisation within a thickened Achilles tendon can be observed

their excellent visualisation of soft tissue. The question is whether these imaging modalities will add beneficial information to the clinical diagnosis, resulting in better recovery prediction or guidance of treatment.

US is readily accessible in many clinical centres, and it is quick and patient friendly. The addition of power or colour Doppler can be helpful in examining the blood flow within and around the tendon (Fig. 19.3). The major ultrasonographic findings in chronic midportion Achilles tendinopathy are tendon thickening, presence of hypo-echogenic areas representing areas of disorganised tendon tissue structure and increased Doppler flow [10].

**Fig. 19.4** MRI image of a 'normal' Achilles tendon presentation in an elite football player. Note the dark signal in the flat Achilles tendon, which is a normal representation



The presence of blood flow in Achilles tendinopathy is frequently referred to as 'neovascularisation' because it is thought to represent a formation of new blood vessels which can also observed on histopathology. This neovascularisation is frequently located at the ventral side of the tendon, and a diffuse distribution within the tendon can also be observed. However, to date it is unknown whether this phenomenon is due to the underlying pathology of the tendon tissue or a physiological increased tendon Doppler flow. Tendon Doppler flow is significantly more frequent in patients with Achilles tendinopathy compared to healthy controls [32]. However, there is no association between the amount of reported symptoms and the severity of neovascularisation [33], and it is not possible to predict recovery with evaluation of tendon Doppler flow [34].

Hypoechoic areas and disorganised tendon structure are frequently observed in patients with chronic Achilles tendinopathy. The structural arrangement of the collagenous matrix determines the echogenicity of tendons. Disorganisation of tendon structure leads to a decreased echogenicity. As a consequence, structural abnormalities and hypoechoic lesions can be visualised. However, even with standardised settings, no association could be found between the amount of symptoms and the quantity of tendon structure disorganisation [21, 35]. Likewise, severity of tendon structure abnormalities is not predictive for recovery [20, 21]. Therefore, US is less valuable in daily clinical practice if the physician is confident with the clinical diagnosis.

The advantages of MRI are the three-dimensional view offered and the excellent soft tissue contrast imaging. A normal, healthy tendon is displayed as a dark signal on MRI as a result of the parallel arrangement of the densely packed collagen and the low intrinsic water content of the tendon. T1 sequences deliver a good delineation of anatomic elements, and T2-weighted sequences are sensitive to an increase in water signal (Fig. 19.4). In cases of chronic midportion Achilles tendinopathy, tendon thickening and an intratendinous increased water signal can be observed as a result of

e

Fig. 19.5 MRI image of a symptomatic midportion Achilles tendinopathy in an elite football player. Note the increased thickness of the Achilles tendon midportion with slightly increased signal intensity in the peritendineum

the increased interfibrillar water-attracting proteoglycans (Fig. 19.5). This increased signal intensity on MRI is, in contrast to US, associated with the amount of pain [36]. In addition, a lower amount of signal abnormalities was shown to be associated with better clinical outcomes and thus indicative of a more favourable prognosis [37]. For chronic midportion tendinopathies, the MRI result does not guide treatment, but for the elite football player, it might be valuable as an indicator of injury recovery time.

Attempts to assess the reliability of peritendineum abnormalities using US and MRI have previously been conducted. In this study, the demarcation of the peritendineum was subjectively classified as either normal or abnormal. With histological findings as the reference, the assessment of the peritendineum seemed to be unreliable with both methods [38]. Another study suggested that paratendinopathy might also manifest as linear areas of increased signal on the ventral side of the Achilles tendon and in Kager's triangle on T2-weighted images [10]. These findings are, however, not related to histology.

Conventional radiography can be performed in cases of insertional tendinopathy. The information provided to assess the soft tissue structures is limited, but X-ray can show abnormalities that are frequently found with insertional tendinopathy. A prominent superior portion of the posterior part of the calcaneus and the presence of calcifications within the tendon can be visualised [10]. A loss of radiolucency in the bursal recess located at the supero-posterior part of the calcaneus may be present in cases of retrocalcaneal bursitis. All these findings might predispose an athlete to compressive forces, but they do not necessarily cause symptoms. Furthermore, the association between the presence of intratendinous calcifications and treatment success needs to be established in future studies [31].

With US, insertional tendon abnormalities can be visualised as described for midportion tendinopathies. However, its relation to symptoms and prognosis is Fig. 19.6 MRI image of a symptomatic retrocalcaneal bursitis in an elite football player. Note an increased signal intensity in the retrocalcaneal bursa in the upper image. The lower image is of another elite football player who sustained insertional symptoms. Note an increased intratendinous signal intensity in the Achilles tendon insertion



unknown. The retrocalcaneal bursa is found frequently in normal subjects, and there is a substantial variation in ultrasonographic appearance [10]. The superficial Achilles bursa is absent in healthy individuals, and its presence is thought to be a sign of either post-traumatic or frictional forces.

MRI can also depict an enlarged retrocalcaneal or superficial Achilles bursa. Especially on the T2-weighted sequences, the retrocalcaneal bursa normally contains detectable high-signal-intensity fluid. As with US, the presence of a retrocalcaneal bursa is not per se related to pathology, and the size of the bursa is variable. It generally does not extend to 1 mm anteroposteriorly, 11 mm transversely or 7 mm craniocaudally [39]. Presence of retrocalcaneal bursitis (Fig. 19.6) or superficial Achilles bursitis might influence treatment strategies, and therefore US and MRI can be of value in patients with insertional Achilles tendinopathy.



**Fig. 19.7** X-ray of an asymptomatic elite football player. Note the calcifications located at the Achilles tendon insertion, which are sometimes found in asymptomatic athletes

As stated above, there are some limitations to imaging of tendons. Correlation with the clinical picture is essential, because imaging abnormalities are also sometimes observed in asymptomatic athletes (Fig. 19.7). Furthermore, the imaging findings of athletes with subacute pain are less researched. Ultrasonographic studies on asymptomatic elite football players showed that Achilles tendon thickening may be present without the presence of symptoms [40]. Almost half of the soccer players with ultrasonographic abnormalities developed symptoms within 1 year, while the other half did not. This finding emphasises that imaging results are hard to interpret if not correlated with the clinical picture.

# **19.6** Treatment Options

# **19.6.1** Conservative Treatment

Treatment of tendinopathies in football remains challenging for the involved care providers. It has changed considerably in the past decade due to changing knowledge of the underlying pathology. Nonetheless, the treatment of choice can vary from country to country, from clinic to clinic and from clinician to clinician.

The management of acute tendinopathies in football is rarely described in the scientific literature, whether the condition is acute insertional or midportion

tendinopathy. The reactive and dysrepair phases in the continuum model are probably representative for acute tendinopathy. The optimal intervention for each phase of pathology is unknown, and therefore a more basic approach with differentiation between acute and chronic tendinopathies is displayed in this chapter. It is generally accepted that load reduction is the first method to apply in athletes with acute Achilles tendinopathy. Cycling and strength training that does not affect the tendon energy storage and release are encouraged within this period [22]. This will result in a decrease in pain and will allow the tendon tissue to adapt. Usually, a few days of adjusted training programme will result in reduced symptoms. However, longer recovery times may be warranted if symptoms persist. There is limited evidence that nonsteroidal anti-inflammatory drugs (NSAIDs) can provide a reduction in symptoms in patients with acute Achilles tendinopathy [41]. The NSAIDs have been shown to reduce tendon repair, and in acute tendinopathy this may be preferable because upregulation of tenocytes and increase in proteoglycans are present in this phase [22]. If NSAIDs are considered in acute tendinopathy, ibuprofen and related agents may be favoured. The final tendon repair seemed to be unaffected by these pharmacologicals.

Treatment of acute paratendinopathy does not differ from acute tendinopathy, although it is recognised as a separate entity [30]. Symptom-based load reduction and NSAIDs are also the first treatment of choice in this condition. Injection with corticosteroids in the peritendinous space performed by an experienced clinician might be an option if symptoms persist. However, even with this indication, scientific evidence for this treatment is absent.

Treatment options for chronic Achilles tendinopathies are increasingly described in scientific literature; especially the midportion location has frequently been a subject for research purposes. Patients with chronic Achilles tendinopathy initially need a comprehensive education about the injury, which may help then to adapt better coping mechanisms. Rest and NSAIDs are less favourable in cases of chronic tendinopathy. Rest can have an initial positive effect on symptoms, but it has also been shown to induce a reduction in the amount of collagen. NSAIDs have fallen out of favour because of the absence of inflammation in this chronic phase and the possible reduction in tendon repair [22, 42].

The same accounts for the effects of corticosteroids, which do not have a rationale because of the absence of inflammatory cells. The efficacy of corticosteroids have only been described in anecdotal reports or based on expert opinion, but no large studies with long-term follow-up have been performed. A recent systematic review showed that the effects of corticosteroids on patient symptoms are detrimental on the longer term and, moreover, the effects on tendon tissue in the football player are detrimental as well [43]. Therefore, intratendinous injections are discouraged for the treatment of chronic Achilles tendinopathy.

There are many conservative treatment options for chronic midportion tendinopathy. After decades of increasing research in this field, exercise therapy remains the cornerstone of treatment [42]. Exercise therapy may result in cellular responses with subsequent change in extracellular matrix turnover, a process called



**Fig. 19.8** Eccentric exercise therapy for patients with chronic midportion Achilles tendinopathy. The exercises can be performed on a step or staircase to enable full dorsiflexion of the ankle. Patients are instructed to start with a bilateral toe raise (*left image*), and after that the eccentric drop can be performed on the injured leg with a straight knee (*middle image*). Patients are also instructed to exercise with flexed knee in order to train the soleus muscle (*right image*). If the exercises can be performed pain-free, the patients are instructed to increase the load (e.g. by putting weights in a backpack)

mechanotransduction [26]. In the previous decade, eccentric exercise therapy gained attention as a treatment for tendon injuries (Fig. 19.8). Several systematic reviews have shown beneficial effects of eccentric exercises on perceived pain [44, 45]. However, it does not seem to matter which dosage of exercises are prescribed, and other exercise therapies might be as beneficial as eccentric exercise [46, 47]. Nonetheless, eccentric exercises are currently the mainstay of treatment for chronic midportion tendinopathy as it induces clinically meaningful treatment effects. It may take up to 12 weeks or longer to notice substantial improvement in symptoms, and it is currently difficult to predict which category of patients will respond well to this treatment.

Less commonly used treatment options are orthotics, extracorporeal shock wave therapy (ESWT), glyceryl trinitrate (GTN) patches and various injection therapies.

Orthotics may be helpful if a malalignment is observed. One small randomised controlled trial (RCT) showed a positive effect of orthotics after 4 weeks when compared to a 'wait and see policy'. Referral to a qualified podiatrist could be considered in cases of excessive foot pronation, although there is little evidence to suggest that orthotics will be effective as the sole treatment for this condition [48]. ESWT delivers energy flux to the tendon tissue which can be achieved with several machines. ESWT is thought to initiate biological responses and tissue regeneration. In a recent systematic review, it was concluded that this treatment can result in an improvement in symptoms over time [49]. However, it remains inconclusive if it is superior to other treatments. GTN patches are applied locally to the Achilles tendon and are originally thought to improve vascularisation and enhance collagen production through nitric oxide delivery. Initial studies showed promising effects, but more recent studies could not reproduce these effects [42]. A variety of injection therapies have been proposed. Injection agents that have been used

include corticosteroids, polidocanol (sclerosing therapy), dextrose (prolotherapy), aprotinin, high volume injections, autologous blood and platelet-rich plasma (PRP). The injection therapies that have been well explored are sclerosing injections and PRP. Sclerosing injections with polidocanol were introduced based on new findings that the nerves were located close to the area of neovascularisation ventral to the Achilles tendon. Polidocanol has the ability to destroy this neovascularisation and accompanying nerves. The first RCTs with this treatment showed promising results with high patient satisfaction after multiple injections by a Swedish research group. However, these results could not be reproduced by other research groups [50].

The method is technically demanding and therefore it is less suitable if there is no experience with this procedure. Other research groups have been focussing on injecting autologous platelets which may secrete growth factors with regenerative effects on the tendon collagen tissue. The use of this PRP treatment has been evaluated in one RCT, and it did not show a beneficial effect compared to placebo [51]. Therefore, PRP injections are currently discouraged. For the other injectable agents mentioned, there is also a lack of good evidence for their efficacy [50]. Treatments with autologous fibroblasts or tenocytes are currently developing, but these are not routinely used in daily clinical practice and need further investigation.

Conservative treatment of chronic insertional disorders is even more challenging as it is not as common or as well researched as midportion tendinopathy. As stated previously, it is important to differentiate between insertional Achilles tendinopathy with and without associated retrocalcaneal or superficial bursitis because this can change treatment options. In cases of bursitis, a generally accepted approach is to start with NSAIDs. If this medication does not reduce symptoms, a corticosteroid injection into the bursa could be an option. However, intrabursal corticosteroid injections have been shown to decrease tendon tissue properties in one animal study [52]. Furthermore, there are no trials providing evidence for its effect. These disadvantages should be considered when opting for an intrabursal corticosteroid injection. If there is an absence of bursitis, the insertional tendinopathy should be managed as compressive tendinopathy. Previously, eccentric exercises with full dorsiflexion were studied in this patient group with disappointing results. More recently, it was advocated to perform the eccentric exercises from a flat ground, thereby decreasing the impingement of the Achilles tendon insertion with the retrocalcaneal bursa and calcaneus [53] (Fig. 19.9). The results of this loading programme are promising and are currently the first treatment of choice. Another treatment option is ESWT, which was demonstrated to be efficacious in two studies evaluating patients with insertional Achilles tendinopathy [31]. More research is needed to draw definite conclusions, but based on the current knowledge, ESWT might be a reasonable option for this indication.

If conservative treatment fails, surgery can be a final option for patients with long-standing insertional or midportion tendinopathy. There are no specific timebased criteria to progress to surgery, but it is generally accepted that the completion of 6 months of conservative treatment is recommended prior to deciding on surgery. However, surgery might be an earlier option for elite-level football athletes.



**Fig. 19.9** Eccentric exercise therapy for patients with chronic insertional Achilles tendinopathy. The exercises are performed from a flat ground to limit dorsiflexion of the ankle and thereby preventing impingement between the calcaneus, retrocalcaneal bursa and Achilles tendon. Patients are instructed to start with a bilateral toe raise (*left image*), and after that the eccentric drop can be performed on the injured leg with a straight knee until the heel touches the ground (*right image*). If the exercises can be performed pain-free, the patients are instructed to increase the load (e.g. by putting weights in a backpack)

# 19.6.2 Surgical Treatment

In the surgical treatment of a midportion Achilles tendinopathy, an attempt must be made to distinguish an isolated paratendinopathy from a combined tendinopathy and paratendinopathy. Open surgery can be performed; however, this strategy remains controversial in the professional football athlete.

Preferably, they are being treated minimally invasive [54, 55]. Patient positioning in the minimally invasive treatment of isolated or combined paratendinopathy is identical: in the prone position with the ankle elevated and at the end of the operation table to allow full range of motion. Routinely a 2.7 mm arthroscope is used. The location of the portals for both entities is different.

In isolated paratendinopathy, the distal portal is made first, located about 3 cm distally from the clinically palpable nodule, at the lateral aspect of the Achilles tendon. The peritendineum is released first with the use of a blunt trocar, subsequently followed by the introduction of the arthroscope. Under direct view the second proximal portal is made, about 3 cm proximal to the nodule but then on the medial aspect of the Achilles tendon. Typically the peritendineum, the Achilles

tendon and the plantaris tendon are melted together and should be released and partly debrided accordingly [55].

In combined tendinopathy and paratendinopathy, both portals are located at the medial aspect of the Achilles tendon, one about 10 cm proximal to the calcaneal tuberosity, used mainly for the arthroscope, and the second portal just superior to the tuberosity for the introduction of surgical instruments. The peritendineum should be released from the Achilles tendon, mainly at the ventral side, since here the former described neovascularisation and ingrowth of sensory nerves can be present. The intratendinous disorders can be treated with the use of a retrograde knife blade [54]. In the first described technique, the patient is allowed full weight bearing directly postoperative, whether for the second technique this is allowed after removal of the stitches 2 weeks postoperatively.

Chronic and/or a retrocalcaneal bursitis not responding to the conservative treatment options can be treated with invasive surgery like a dorsal calcaneal wedge osteotomy, although in the professional football athlete a minimally invasive endoscopic treatment is preferred [56]. The patient is positioned in the prone position, a tourniquet applied on the upper leg and the affected ankle is supported, allowing free movement throughout the procedure. The lateral portal is located at the posterosuperior aspect of calcaneus, just lateral to the Achilles tendon. After introducing the 4.0 mm/30°-angled arthroscope into the retrocalcaneal space, the location of the medial portal is determined under direct view. The supero-posterior calcaneal process can be reduced with the use of a bonecutter shaver or a burr; it is important to change portals frequently to assure sufficient bone removal. In doubt fluoroscopy can be used. Postoperatively patients are allowed full weight bearing with a compression bandage for up to 3 days; regular shoe wear can be used when tolerated.

A recent systematic review showed a high patient satisfaction in all surgical studies regarding the treatment of insertional Achilles tendinopathy, despite differences in outcome and complication ratios [31]. Furthermore, there are no randomised studies comparing surgery to other treatment options. In general, an open surgical debridement is indicated if conservative treatment options fail. Many surgical approaches have been described, of which the central longitudinal splitting tendon approach seems preferable for not disturbing too much of the Achilles tendon blood supply with optimal functional outcomes [57]. The goal of the invasive treatment is to expose the pathological tendon, the calcifications, the retrocalcaneal bursa and the postero-superior calcaneal process. Subsequently these need to be resected adequately, if present. Up to 50 % of the Achilles tendon can safely be elevated or excised without disturbing early full weight bearing [58]. If up to 75 % of the tendon is elevated, it should be reattached by transosseous sutures or anchors at the appropriate length [59]. If over 75 % of the tendon is released, an augmentation is advised, most frequently with the use of the flexor hallucis longus tendon [60]. After treatment will depend on the extent of the surgery, in general a cast immobilisation is necessary in case the Achilles tendon is reinserted, and in all other cases early full weight bearing is allowed.

# 19.7 Prognosis

Although tendinopathy may seem like a relatively innocent injury, recovery times are difficult to predict, and in many cases it follows a prolonged time course. There are no prognostic data of acute tendinopathies because many remain under the radar for the treating physician in a hospital.

Chronic midportion tendinopathies were thought to have a reasonable prognosis. An 8-year observational study showed that 94 % of patients were almost asymptomatic and could run normally [61]. However, a recent study showed less reassuring results. Approximately 60 % of a mainly conservatively treated patient group had symptoms remaining at a 5-year follow-up [62]. This fact can be an element to share with the patient before starting treatment. Management of the reasonable expectations from treatment can be very helpful for both the football player and the doctor.

# References

- 1. De Jonge S, van den Berg C, de Vos RJ, van der Heide HJL, Weir A, Verhaar JAN et al (2011) Incidence of midportion Achilles tendinopathy in the general population. Br J Sports Med 45(13):1026–1028
- Kujala UM, Sarna S, Kaprio J (2005) Cumulative incidence of Achilles tendon rupture and tendinopathy in male former elite athletes. Clin J Sport Med 15(3):133–135
- Ekstrand J, Hägglund M, Kristenson K, Magnusson H, Waldén M (2013) Fewer ligament injuries but no preventive effect on muscle injuries and severe injuries: an 11-year follow-up of the UEFA Champions League injury study. Br J Sports Med 47(12):732–737
- Maffulli N, Khan KM, Puddu G (1998) Overuse tendon conditions: time to change a confusing terminology. Arthroscopy 14(8):840–843
- Khan KM, Cook JL, Kannus P, Maffulli N, Bonar SF (2002) Time to abandon the "tendinitis" myth. BMJ 324(7338):626–627
- 6. Alfredson H (2003) Chronic midportion Achilles tendinopathy: an update on research and treatment. Clin Sports Med 22(4):727–741
- 7. Khan KM, Cook JL, Bonar F, Harcourt P, Astrom M (1999) Histopathology of common tendinopathies. Update and implications for clinical management. Sports Med 27(6):393–408
- 8. O'Brien M (2005) The anatomy of the Achilles tendon. Foot Ankle Clin 10(2):225-238
- Van Sterkenburg MN, Kerkhoffs GMMJ, Kleipool RP, Niek van Dijk C (2011) The plantaris tendon and a potential role in mid-portion Achilles tendinopathy: an observational anatomical study. J Anat 218(3):336–341
- 10. Bleakney RR, White LM (2005) Imaging of the Achilles tendon. Foot Ankle Clin 10(2):239-254
- Leadbetter WB, Mooar PA, Lane GJ, Lee SJ (1992) The surgical treatment of tendinitis. Clinical rationale and biologic basis. Clin Sports Med 11(4):679–712
- Lichtwark GA, Wilson AM (2005) In vivo mechanical properties of the human Achilles tendon during one-legged hopping. J Exp Biol 208(Pt 24):4715–4725
- Komi PV (1990) Relevance of in vivo force measurements to human biomechanics. J Biomech 23(Suppl 1):23–34
- Parkinson J, Samiric T, Ilic MZ, Cook J, Handley CJ (2011) Involvement of proteoglycans in tendinopathy. J Musculoskelet Neuronal Interact 11(2):86–93

#### 19 Achilles Tendinopathy

- Paavola M, Kannus P, Järvinen TAH, Khan K, Józsa L, Järvinen M (2002) Achilles tendinopathy. J Bone Joint Surg Am 84-A(11):2062–2076
- 16. Leadbetter WB (1992) Cell-matrix response in tendon injury. Clin Sports Med 11(3):533-578
- 17. Cook JL, Khan KM, Purdam C (2002) Achilles tendinopathy. Man Ther 7(3):121-130
- Maffulli N, Wong J, Almekinders LC (2003) Types and epidemiology of tendinopathy. Clin Sports Med 22(4):675–692
- Kannus P, Józsa L (1991) Histopathological changes preceding spontaneous rupture of a tendon. A controlled study of 891 patients. J Bone Joint Surg Am 73(10):1507–1525
- 20. De Vos RJ, Weir A, Tol JL, Verhaar JAN, Weinans H, van Schie HTM (2011) No effects of PRP on ultrasonographic tendon structure and neovascularisation in chronic midportion Achilles tendinopathy. Br J Sports Med 45(5):387–392
- De Vos RJ, Heijboer MP, Weinans H, Verhaar JAN, van Schie JTM (2012) Tendon structure's lack of relation to clinical outcome after eccentric exercises in chronic midportion Achilles tendinopathy. J Sport Rehabil 21(1):34–43
- 22. Cook JL, Purdam CR (2009) Is tendon pathology a continuum? A pathology model to explain the clinical presentation of load-induced tendinopathy. Br J Sports Med 43(6):409–416
- 23. Kaux J-F, Forthomme B, Goff CL, Crielaard J-M, Croisier J-L (2011) Current opinions on tendinopathy. J Sports Sci Med 10(2):238–253
- 24. Khan MH, Li Z, Wang JH-C (2005) Repeated exposure of tendon to prostaglandin-E2 leads to localized tendon degeneration. Clin J Sport Med 15(1):27–33
- 25. Rio E, Moseley L, Purdam C, Samiric T, Kidgell D, Pearce AJ et al (2013) The pain of tendinopathy: physiological or pathophysiological? Sports Med 44(1):9–23
- 26. Brukner P, Khan K (2012) Clinical sports medicine, 4th edn. McGraw-Hill, Sydney/New York
- Van Dijk CN, van Sterkenburg MN, Wiegerinck JI, Karlsson J, Maffulli N (2011) Terminology for Achilles tendon related disorders. Knee Surg Sports Traumatol Arthrosc 19(5):835–841
- 28. Stephens MM (1994) Haglund's deformity and retrocal caneal bursitis. Orthop Clin North Am  $25(1){:}41{-}46$
- 29. Kvist M (1994) Achilles tendon injuries in athletes. Sports Med 18(3):173-201
- 30. Paavola M, Järvinen TAH (2005) Paratendinopathy. Foot Ankle Clin 10(2):279-292
- Wiegerinck JI, Kerkhoffs GM, van Sterkenburg MN, Sierevelt IN, van Dijk CN (2013) Treatment for insertional Achilles tendinopathy: a systematic review. Knee Surg Sports Traumatol Arthrosc 21(6):1345–1355
- Sengkerij PM, de Vos R-J, Weir A, van Weelde BJG, Tol JL (2009) Interobserver reliability of neovascularization score using power Doppler ultrasonography in midportion achilles tendinopathy. Am J Sports Med 37(8):1627–1631
- 33. De Jonge S, Warnaars JLF, De Vos RJ, Weir A, van Schie HTM, Bierma-Zeinstra SMA, et al (2013) Relationship between neovascularization and clinical severity in Achilles tendinopathy in 556 paired measurements. Scand J Med Sci Sports
- 34. De Vos R-J, Weir A, Cobben LPJ, Tol JL (2007) The value of power Doppler ultrasonography in Achilles tendinopathy: a prospective study. Am J Sports Med 35(10): 1696–1701
- 35. Van Schie HTM, de Vos RJ, de Jonge S, Bakker EM, Heijboer MP, Verhaar JAN et al (2010) Ultrasonographic tissue characterisation of human Achilles tendons: quantification of tendon structure through a novel non-invasive approach. Br J Sports Med 44(16): 1153–1159
- 36. Gärdin A, Bruno J, Movin T, Kristoffersen-Wiberg M, Shalabi A (2006) Magnetic resonance signal, rather than tendon volume, correlates to pain and functional impairment in chronic Achilles tendinopathy. Acta Radiol 47(7):718–724
- 37. Khan KM, Forster BB, Robinson J, Cheong Y, Louis L, Maclean L et al (2003) Are ultrasound and magnetic resonance imaging of value in assessment of Achilles tendon disorders? A two year prospective study. Br J Sports Med 37(2):149–153
- 38. Aström M, Gentz CF, Nilsson P, Rausing A, Sjöberg S, Westlin N (1996) Imaging in chronic achilles tendinopathy: a comparison of ultrasonography, magnetic resonance imaging and surgical findings in 27 histologically verified cases. Skeletal Radiol 25(7):615–620

- Bottger BA, Schweitzer ME, El-Noueam KI, Desai M (1998) MR imaging of the normal and abnormal retrocalcaneal bursae. AJR Am J Roentgenol 170(5):1239–1241
- Fredberg U, Bolvig L (2002) Significance of ultrasonographically detected asymptomatic tendinosis in the patellar and achilles tendons of elite soccer players: a longitudinal study. Am J Sports Med 30(4):488–491
- McLauchlan GJ, Handoll HH (2001) Interventions for treating acute and chronic Achilles tendinitis. Cochrane Database Syst Rev (2):CD000232
- Scott A, Huisman E, Khan K (2011) Conservative treatment of chronic Achilles tendinopathy. Can Med Assoc J 183(10):1159–1165
- 43. Coombes BK, Bisset L, Vicenzino B (2010) Efficacy and safety of corticosteroid injections and other injections for management of tendinopathy: a systematic review of randomised controlled trials. Lancet 376(9754):1751–1767
- Woodley BL, Newsham-West RJ, Baxter GD (2007) Chronic tendinopathy: effectiveness of eccentric exercise. Br J Sports Med 41(4):188–198; discussion 199
- 45. Wasielewski NJ, Kotsko KM (2007) Does eccentric exercise reduce pain and improve strength in physically active adults with symptomatic lower extremity tendinosis? A systematic review. J Athl Train 42(3):409–421
- 46. Malliaras P, Barton CJ, Reeves ND, Langberg H (2013) Achilles and patellar tendinopathy loading programmes: a systematic review comparing clinical outcomes and identifying potential mechanisms for effectiveness. Sports Med 43(4):267–286
- 47. Stevens M, Tan C-W (2014) Effectiveness of the Alfredson's protocol compared with a lower repetition volume protocol for mid-portion achilles tendinopathy: a randomized controlled trial. J Orthop Sports Phys Ther 44(2):59–67
- Mayer F, Hirschmüller A, Müller S, Schuberth M, Baur H (2007) Effects of short-term treatment strategies over 4 weeks in Achilles tendinopathy. Br J Sports Med 41(7):e6
- 49. Al-Abbad H, Simon JV (2013) The effectiveness of extracorporeal shock wave therapy on chronic achilles tendinopathy: a systematic review. Foot Ankle Int 34(1):33–41
- Gross CE, Hsu AR, Chahal J, Holmes GB Jr (2013) Injectable treatments for noninsertional achilles tendinosis: a systematic review. Foot Ankle Int 34(5):619–628
- 51. De Vos RJ, Weir A, van Schie HTM, Bierma-Zeinstra SMA, Verhaar JAN, Weinans H et al (2010) Platelet-rich plasma injection for chronic Achilles tendinopathy: a randomized controlled trial. JAMA 303(2):144–149
- 52. Hugate R, Pennypacker J, Saunders M, Juliano P (2004) The effects of intratendinous and retrocalcaneal intrabursal injections of corticosteroid on the biomechanical properties of rabbit Achilles tendons. J Bone Joint Surg Am 86-A(4):794–801
- 53. Jonsson P, Alfredson H, Sunding K, Fahlström M, Cook J (2008) New regimen for eccentric calf-muscle training in patients with chronic insertional Achilles tendinopathy: results of a pilot study. Br J Sports Med 42(9):746–749
- 54. Thermann H, Benetos IS, Panelli C et al (2009) Endoscopic treatment of chronic mid-portion Achilles tendinopathy : novel technique with short term results. Knee Surg Sports Traumatol Arthrosc 17:264–269
- 55. Steenstra F, van Dijk CN (2006) Achilles tendoscopy. Foot Ankle Clin 11:429-438
- 56. Scholten PE, van Dijk CN (2006) Endoscopic calcaneoplasty. Foot Ankle Clin 11:439-446
- DeOrio MJ, Easley MW (2008) Surgical strategies: insertional achilles tendinopathy. Foot Ankle Int 29:542–550
- Kolodziej P, Glisson RR, Nunley JA (1999) Risk of avulsion of Achilles tendon after partial excision for treatment of insertional tendonitis and Haglund's deformity: a biomechanical study. Foot Ankle Int 20:433–437
- 59. Maffuli N, Testa V, Capasso G, Sullo A (2004) Calcific insertional Achilles tendinopathy: reattachment with bone anchors. Am J Sports Med 32:174–182
- Den Hartog BD (2003) Flexor hallucis longus transfer for chronic Achilles tendinosis. Foot Ankle Int 24:233–237

- Paavola M, Kannus P, Paakkala T, Pasanen M, Järvinen M (2000) Long-term prognosis of patients with achilles tendinopathy. An observational 8-year follow-up study. Am J Sports Med 28(5):634–642
- 62. Van der Plas A, de Jonge S, de Vos RJ, van der Heide HJL, Verhaar JAN, Weir A et al (2012) A 5-year follow-up study of Alfredson's heel-drop exercise programme in chronic midportion Achilles tendinopathy. Br J Sports Med 46(3):214–218

# Chapter 20 Peroneal and Posterior Tibial Tendon Pathology

## Hélder Pereira, Pedro Luís Ripoll, Joaquim M. Oliveira, Rui L. Reis, João Espregueira-Mendes, and C. Niek van Dijk

Success is no accident. It is hard work, perseverance, learning, studying, sacrifice and most of all, love of what you are doing or learning to do.

Pele

ICVS/3B's – PT Government Associated Laboratory, Guimarães, Portugal e-mail: heldermdpereira@gmail.com; jem@espregueira.com

#### P.L. Ripoll

Ripoll y De Prado Sports Clinic - FIFA Medical Centre of Excellence, Murcia-Madrid, Spain e-mail: pedrolripoll@gmail.com

J.M. Oliveira • R.L. Reis • J. Espregueira-Mendes Clínica Espregueira-Mendes F.C. Porto Stadium – FIFA Medical Centre of Excellence, Porto, Portugal

3B's Research Group – Biomaterials, Biodegradables and Biomimetics, Headquarters of the European Institute of Excellence on Tissue Engineering and Regenerative Medicine, University of Minho, Ave Park, S. Cláudio de Barco, 4806-909 Taipas, Guimarães, Portugal

ICVS/3B's – PT Government Associated Laboratory, Guimarães, Portugal e-mail: miguel.oliveira@dep.uminho.pt; rgreis@dep.uminho.pt

C.N. van Dijk Department of Orthopaedic Surgery, Academic Medical Center, Amsterdam, The Netherlands e-mail: c.n.vandijk@amc.uva.nl

P.P.R.N. d'Hooghe, G.M.M.J. Kerkhoffs (eds.), *The Ankle in Football*, Sports and Traumatology, DOI 10.1007/978-2-8178-0523-8\_20, © Springer-Verlag France 2014

H. Pereira (🖂)

<sup>3</sup>B's Research Group – Biomaterials, Biodegradables and Biomimetics, Headquarters of the European Institute of Excellence on Tissue Engineering and Regenerative Medicine, University of Minho, Ave Park, S. Cláudio de Barco, 4806-909 Taipas, Guimarães, Portugal

**Abstract** Football is a demanding sport requiring specific repetitive technical gestures involving the foot and ankle. This must be taken into account in the understanding of complaints, the search for diagnostics, and the selection of the appropriate action. Patient's expectations usually include full recovery with fast return to previous activity level. Overuse and post-trauma events (isolated or in association) are the most frequent etiologic factors of tendon-related problems. The goal of this chapter is to present an overview of peroneal tendons (evertors) and posterior tibial tendon (invertor) pathologies in football. The therapeutic options herein discuss the aim to reflect the requirements of football players in their active period. Complementary data including a broad analysis of ankle anatomy, biomechanics, physiopathology, and treatment including a complete surgical manual can be found on http://www.ankleplatform.com.

**Keywords** Groove deepening • Peroneal tendons • Posterior tibial tendon • Tendinopathy • Tendon rupture • Tendoscopy • Tenosynovitis

# 20.1 Introduction

Ball control during practice of football is far from being a simple achievement. Enhanced proprioception and fine-tuned neuromuscular control during all foot and ankle positions are required and demand training strategies [3].

The balance between pronation and supination is mandatory for normal gait but even more solicited during football (e.g., ball reception or dribbling). It mostly depends on adjustments of the subtalar joint, but the ankle joint and foot participate at different levels. Foot pronation is a complex movement which combines abduction of the forefoot, eversion of the hindfoot, and dorsiflexion [8]. Supination also occurs in three planes while combining internal ankle rotation, hindfoot adduction, forefoot inversion, and medial arch elevation. Pronation provides some degree of flexibility opposing to supination which is associated to increased foot stability [3]. The invertors of the foot comprise posterior and anterior tibial muscles [24]. Conversely, peroneal muscles (PMs), including peroneus longus, brevis, and tertius, are the active evertors of the foot [35]. These muscles play a key role in controlling pronation-supination.

Understanding the biomechanics of the foot and ankle is mandatory in understanding physiopathology. The navicular bone is the "key bone" providing distal support to the talus. In standing position, it represents the higher structure of the longitudinal medial arch. The harmony of these structures in static position relies on the surrounding bones and the spring ligament (calcaneonavicular). In motion, the action of the posterior tibial muscle (PTM) maintains the superior position of the navicular supporting the medial arch. Thus, a weak PTM or diseased posterior tibial tendon (PTT) is unable to keep the navicular in place and a fall of the medial arch might occur (acquired flat foot condition) [17]. Conversely, hindfoot varus or ankle instability creates increased persistent/repeated strain on the peroneal tendons (PTs) which will predispose to pathology [35]. These basic examples are representative of the paramount relevance of biomechanical phenomena in this field.

In the light of the previously exposed, some therapeutic options implicate a surgical modification of this complex biomechanics (e.g., tendon transfers, osteotomies, or fusion of selected joints). Such procedures have been proposed through time with strict selection criteria and usually addressing the general population and not high-level athletes [16, 30, 41]. They are considered out of the scope of this text and are properly described elsewhere (http://www.ankleplatform.com). Dealing with patient's expectations is mandatory. Implications of surgical options on function, including sports-specific skills, must be considered on an individual basis.

History taking and physical examination including gait analysis are the first and most important step in the diagnostic process. In some cases, evaluation on the pitch during football practice can be useful. History of medication consumption must be addressed once some (e.g., fluoroquinolones – FQs) have been recognized as risk factors for tendinopathies. FQs should indeed be used cautiously in athletes [14].

Radiological study must always include standing foot and ankle x-rays. Ultrasound and MRI might be useful (Fig. 20.1); however, limitations exist and must be acknowledged. Ultrasound is known to be operator dependent while providing dynamic evaluation [20]. MRI is considered diagnostically specific but not sensitive [26]. It has presented fair sensitivity for diagnosing chronic peroneus



Fig. 20.1 MRI showing peroneus brevis tendinopathy (*yellow arrow*). Notice flattening of the ruptured tendon surrounding peroneus longus and increased fluid within the synovial sheath surrounding both tendons

longus pathology, but not in the peroneus brevis [26, 34]. Similar limitations have been described for PTT [48]. Local anesthetic injection can be considered to confirm the origin of pain [2]. CT scans are particularly useful in the study of bony structures which might be suspected (e.g., prominent peroneal tubercle, navicular deformities, bone ossicles/spurs). Endoscopic/tendoscopic evaluation provides direct inspection and is gaining popularity in either diagnostic or therapeutic approaches [26, 48].

Morphological constitutional conditions can sometimes be favorably overcome with adapted shoes, insoles, or orthoses when it brings athlete's comfort without negative implication in skills ([15], see also book Chap. 21).

One needs to listen and identify properly the nature of the complaints. The goals are as follows: identify its etiology, understand how it affects the patient (in his/her specific needs), and try to find a therapeutic option which might provide relief while being compatible to football participation at the same level. These are the "golden rules." Sometimes, the surgeon might consider an option which suits demands on general population might even provide more "anatomic" outcome but can endanger the short career of a professional football player. All this information must be shared with patients. In selected cases, patients might choose partial or temporary relief of complaints with delay of more "aggressive" approaches.

Herein will be discussed the fundamentals of the most common pathological conditions of PTs and PTT while considering the usual author's approach when dealing with these conditions within this specific group of patients.

# 20.2 Peroneal Tendons Pathology

As previously stated, the three muscles that evert the ankle are the peroneus longus, brevis, and tertius. The peroneus longus (PL) arises from the proximal fibula, while the peroneus brevis (PB) arises from the distal fibula. The deep peroneal nerve passes under the upper end of the PL.

At the level of the ankle, the peroneus longus tendon (PLT) and peroneus brevis tendon (PBT) pass behind the lateral malleolus and beneath the peroneal retinaculum. PLT runs behind and PBT in front of it. PBT inserts on the base of the fifth metatarsal. PLT runs around the cuboid bone, and along a deeply placed fibrous tunnel, to insert on the base of the first metatarsal.

The PTs receive their nutritive irrigation from vincula supplied by posterior peroneal and medial tarsal arteries. However, three relatively hypovascular zones have been described and considered when dealing with degenerative changes: one in PBT as it surrounds the malleolus and two in PLT (as it curves the malleolus also and when it surrounds the cuboid) [27].

In front of PBT and PLT, there is the peroneus tertius. The peroneus tertius also arises from the fibula. The tendon of peroneus tertius passes under the extensor retinaculum and in front of the lateral malleolus to insert on the base of the fifth meta-tarsal, next to PBT. It is absent in nearly 10 % of subjects but has been implicated in

cases of anterolateral pain and/or "snapping ankle" [13]. Peroneus quartus is a rare anatomic variant. The consequences of a peroneus quartus tendon are unknown. In case of pain or snapping at this level without further pathology, resection can be considered. A prominent peroneal tubercle is another anatomic variant which has been implicated in persistent lateral pain.

The main action of all three of the peroneal muscles is to evert the foot. The PL locks the transversal arch and plantarflexes the first ray [35].

Os peroneum can be found within the length of PLT at the level of calcaneocuboid joint. It has been described in 5–26 % of people and sometimes associated with lateral pain [37]. When observed, a proximal migration of this bony or cartilaginous structure suggests PLT rupture.

All peroneal muscles assist in dorsiflexion of the ankle. We reinforce the relevance of all muscles participating in inversion-eversion once they enable us to stay balanced and upright on an uneven or unstable surface.

### 20.2.1 Peroneal Tendinopathy

The term "tendinopathy" generally describes a disease of a tendon with complaint of pain and swelling. More specifically, it can refer to terms that were used in the past but are now obsolete [46].

- Tendinitis: tendon injuries which involve predominantly acute injuries accompanied by inflammation.
- Tendinosis: chronic tendon injury with damage to a tendon at a cellular level.
- When it involves a synovial sheath covering, a tendon is then called tenosynovitis.

When there is no synovial envelope but instead a paratenon-covered tendon (e.g., Achilles tendon), then pathological changes are referred as "paratendinopathy" (acute or chronic) [46].

In football athletes, most tendon-related problems are associated to overuse [34]. Traumatic events (contusions, sprains, fractures) are the second leading cause, but morphological foot/ankle issues and football-specific gestures must also be taken into account. It is always mandatory – in the presence of lateral foot and ankle pain on a football player – to rule out concomitant ankle instability.

Clinical examination will show tenderness on palpation, local edema, and intensification of symptoms on active eversion against resistance.

The first option in treatment is conservative treatment: rest, ice, massage therapy, eccentric exercise, NSAIDs, ultrasound therapy, LIPUS, electrotherapy, taping, glyceryl trinitrate patches, last-generation extracorporeal shockwave therapy (ESWT), insoles, and shoe changes.

If conservative treatment fails, one can consider peroneal tendoscopy [43]. This approach permits confirmation of uncertain findings after imaging study including dynamic evaluation, treatment of hypertrophic tendinopathy, minimally invasive

Fig. 20.2 Two needles point proximal (2–2.5 cm) and (1.5–2.0 cm) distal portals using lateral malleolus as superficial landmarks (a). Notice that the little finger of the surgeon's left hand is slightly supported in the patient facilitating small motion control (b). Postoperative look after closure of the wounds (c)



cleaning of synovitis, removal of debris and inflammatory fluid, or regularization of prominent peroneal tubercle [33]. In properly selected cases, particularly in elite football players, it "breaks" the vicious circle associated to repetition of complaints after football gameplay (Figs. 20.2 and 20.3).



**Fig. 20.3** Peroneal tendoscopy showing both tendons and vincula (*red arrow* – **a**); rupture of peroneus brevis noticed (**b**); further inspection revealed a length rupture (*blue arrow* – **c**); notice the flattened aspect of the tendon on the right surrounding the tendon on the left bottom of the image (**d**) similarly to what was suggested in MRI of Fig. 20.1

Compared to open surgery, tendoscopy presents several advantages: lower aggression (minimizing scar tissue), lower morbidity, reduction in postoperative pain, functional aftertreatment, and outpatient surgery.

### **20.2.1.1 Peroneal Tendoscopy Technique** (Figs. 20.2 and 20.3)

The patient lies supine with a beanbag under the buttock to help endorotate the foot or in lateral decubitus. A bloodless field is used. Two main portals are located directly over the peroneal tendons. One portal is created 1.5-2 cm distal and

2-2.5 cm proximal to the posterior edge of the lateral malleolus. The distal portal is made first. A skin-only incision is made and then the tendon sheath is opened using a blunt trocar within the scope's sleeve.

The  $30^{\circ}$  2.7-mm arthroscope (or sometimes 4.5 mm) can be used according to patient profile, availability, and personal experience. It is possible to use 1.8-mm scope; however, it must be considered as a general rule that the smaller the tools, the more "aggressive"/sharp it tends to be comparing to relative bluntness of larger devices. Furthermore, it provides more limited visualization capacity and fluid supply. We recommend keeping the fifth finger supported while controlling the arthroscope with the remaining four for fine control.

Postoperative treatment consists of dressings with partial weight-bearing for 2-3 days. Active motion is encouraged from the first day.

### 20.2.2 Peroneal Tendon Ruptures

Peroneal tendons have been misdiagnosed during several years [40]. Peroneal tears might result from acute trauma, associated to ankle instability and retinaculum insufficiency with or without subluxation. A cavovarus foot produces increased strain on lateral structures. A plantarflexed first ray must be ruled out opposing to calcaneus varus by means of clinical examination including the Coleman block test [9].

Peroneal ruptures have been described as "Zone I" (proximal injuries usually involving PBT) or "Zone II" (distal injuries involving PLT pathology sometimes associated to *os peroneum*) [38].

Patients complain of lateral pain and swelling, many times following an inversion injury. Sometimes the feeling of a "pop" and subsequent ankle weakness is described.

Radiographies should search for small ossicles, avulsions, or *os peroneum* changes. US and MRI (Fig. 20.1) might help. As previously stated, local anesthetic injections can confirm the origin of complaints.

Conservative treatment includes medication, physiotherapy, bracing, orthoses, or temporary immobilization. However, particularly in high-level footballers, this might fail on the long term.

The author's preferred surgical approach is the tendoscopy one. Besides confirming the diagnosis with dynamic and precise knowledge of the characteristics of the injury, it might permit definitive treatment of some partial tears. Furthermore, it helps on determining the precise location of injuries, thus helping to minimize the surgical aggression if open surgery is required. Basic principles of the technique were formerly described.

### 20.2.2.1 Peroneal Tendons Open-Surgery Technique

The position of the patient can usually be the same following tendoscopy. A limited lateral incision is performed according to the previously determined (tendoscopy)



Fig. 20.4 Peroneus brevis length rupture exposed (a), tubularization suture of PBT (b), concomitant peroneus longus length rupture (c), repair of PLT using the same method (d), dynamic inspection of both tendons in place (e), closure of peroneal retinaculum (f)

injury position or need to approach the peroneal retinaculum. The common peroneal sheath is opened and both tendons inspected as well as concomitant bony structures (Fig. 20.4).

If a degenerated portion of the tendon is detected, it should be excised. A lowriding muscle belly can be excised. In case of a clear length rupture, direct repair is possible. Tubularization repair is performed in case of flattening of the tendon using absorbable sutures (Fig. 20.4). Closure is performed by planes with re-tensioning of the retinaculum when appropriate.

Postoperative care includes Walker boot immobilization with protected partial weight-bearing for 4–6 weeks. Full weight-bearing follows. Closed-chain exercises

with heel support are permitted if pain and swelling disappear at the same period. Protected ankle range of motion exercises are permitted 3–4 weeks.

Several authors recommend tenodesis when more than 50 % of tendon thickness or more than 2-cm length is affected; however, this represents a higher aggression to consider carefully on an active soccer player [9]. If tendons are found to be irreparable, tendon grafts (e.g., plantaris tendon) or transfers (e.g., flexor hallucis longus tendon) might also constitute valuable options. If the *os peroneum* needs to be removed, a tenodesis of PLT to PBT is required [9]. Selected cases might benefit from bony procedures (e.g., osteotomies); however, this subject is considered out of the scope of this chapter.

# 20.2.3 Peroneal Tendons Dislocation

Peroneal tendon instability was first described in the early nineteenth century in a ballet dancer [19]. A peroneal tendon dislocation is a rare event which often occurs after a single traumatic event which creates a sudden resisted contraction of the peroneal muscles [32]. Most frequently the foot is dorsiflexed, abducted, and everted but has also been described during forced dorsiflexion while the foot is everted [22]. Usually, this condition happens during sports participations (football, ski, American football, running, gymnastics, tennis, ballet, basketball, or ice skating) [32]. The superior peroneal retinaculum (SPR) is the primary restraint to PTs dislocation; however, recurrent dislocation correlates with the groove depth [23]. Eckert and Davis described that the superior retinaculum and the periosteum insertion can be detached from the fibula [4]. They described a classification in which grade I represents detachment of SPR from the collagenous lip; in grade II, both structures are separated from the fibula; and in grade III, a small bony avulsion accompanies the detachment of both previous structures. A grade IV was added later, describing a retinaculum rupture from its posterior attachment [23]. In general, grade I comprises more than 50 % of all cases.

When a dislocation occurs, this predisposes to recurrence. Furthermore, it has been shown that lateral ankle ligament laxity increases the strain transmitted to the retinaculum which explains the association between both conditions [7]. Differential diagnosis with "snapping ankle" caused for peroneus tertius is required. Several anatomic variants (tendinous or bony), concomitant ankle instability, or varus malalignment predisposes to this condition.

Conservative treatment (physiotherapy, bracing, taping, orthoses, immobilization) has been associated to a high recurrence rate [22] opposing to excellent results reported with surgical treatment [32]. Several procedures have been described either directed to repair or reinforcement of the SPR or bony procedures aiming groove deepening (including bone-block procedures) [28].

There is a lack for large controlled series comparing results of several techniques. It is therefore not possible to claim superiority of any technique over another concerning re-dislocation rate.



**Fig. 20.5** A probe is used to remove the peroneals from their native grove (**a**). A needle assists in the correct placement of accessory portal (**b**). A 5-mm shaver is used to deepen the groove (*red rectangle*) (**c**). Final result with peroneals in place (**d**)

Considering a football population, the author's preferred technique (Fig. 20.5) comprises endoscopic groove deepening [32, 50]. Despite reported excellent results, it constitutes outpatient surgery and enables functional treatment from day 1 enabling a faster rehabilitation. Open surgery comprises a higher risk of complications (sural nerve injury, secondary scar, and infection) [31]. Moreover, a plaster cast is indicated after an open procedure which delays the rehabilitation process.

#### 20.2.3.1 Technique for Endoscopic Peroneal Tendon Groove Deepening

The patient is placed prone as described for posterior ankle arthroscopy [45]. The tendons are inspected after opening the peroneal tendon sheath (Fig. 20.5). A probe is used to dislocate both tendons from the groove. An accessory portal is created to introduce a 5-mm bone-cutter blade to deepen the groove. Tendons are then relocated within the groove by removing the probe. The patient is discharged in the same day. Immediate weight-bearing is permitted as tolerated as well as activities of daily living. An ankle brace is applied during 6 weeks.
#### 20.3 Posterior Tibial Tendon Pathology

The tibialis posterior muscle arises from the back of the tibia, the back of the fibula, and the interosseous membrane. PTT passes immediately behind the medial malleolus, through a fibrous tunnel which is covered by the flexor retinaculum. Beyond the malleolus, the tendon begins to fan out. It has a wide insertion including the navicular and first cuneiform bones and the bases of the second, third, and fourth metatarsals. The PTT does not have a mesotenon.

Immediately distal to the medial malleolus, it has an area with poorer vascularity which can be implicated in degenerative changes of the tendon [6]. The PTT is an important dynamic stabilizer of the medial arch and the most powerful invertor of the foot [8].

A patient suffering from PTT tendinopathy might refer posteromedial ankle pain alone. However, on clinical examination, one can find local tenderness, a positive PPT provocation test, and inability to walk on tiptoes. Radiographies assess global foot and ankle morphology. MRI and US – despite their known limitations – are useful to identify pathologies, such as tenosynovitis, (longitudinal) ruptures, degenerative changes, or adherences [2, 17].

Johnson and Strom proposed a classification system correlating the severity of PTT dysfunction and subsequent adaptations of the foot (to the collapse of the medial longitudinal arch) along with combined treatment recommendations [12]. Considering the football player, only grade I dysfunction will be herein considered. This includes PTT tendinopathies without major deformity.

Conservative treatment comprises physiotherapy, medication, insoles, and corrective shoes (medial heel and sole wedge). However, when it fails overtime, with limited function, particularly on an athletic population, the author's option is once more tendoscopy.

Indications for posterior tibial tendon tendoscopy [44]:

- Tenosynovectomy
- Tendon sheath release
- · Tendon debridement and cleaning of partial rupture
- Resection of pathological vincula
- · Removal of exostosis/irregularity of posterior tibial sliding channel
- · Endoscopic removal of implants (screws/anchors) from medial malleolus
- · Adhesiolysis
- Diagnostic procedure

Posterior tibial tendoscopy enables diagnostic confirmation for patients with a suspected or radiologically diagnosed partial tendon tear (once either false positives and false negatives have been described) [48]. Partial tendon tears can be diagnosed and treated while avoiding a large incision, increased postoperative pain, and prolonged rehabilitation. When a tendon tear suitable for reconstruction is identified, the tendoscopic procedure can be converted to a mini-open approach, which is still less invasive than the standard open procedure.



**Fig. 20.6** Creation of the distal portal 2.5 cm distal to the posterior edge of medial malleolus (**a**), use of 2.7-mm shaver from proximal portal (**b**), inspection of all the tendon sheath surrounding the tendon with the arthroscope (**c**), cleaning of tenosynovitis using the shaver blade (**d**)

## 20.3.1 Technique for Posterior Tibial Tendoscopy

The patient is positioned supine and a bloodless field used. For superficial landmarks, the patient is asked before anesthesia to actively invert the foot, to facilitate palpating the PTT to mark the portals. The level of maximum pain should also be marked. The distal portal (Fig. 20.6) is performed first, 2.5 cm distal to the posterior edge of the medial malleolus. A 30° 2.7-mm arthroscope is introduced, and the complete tendon sheath should be inspected. The proximal portal is made under direct vision using a needle, after which an incision is created in the tendon sheath and a 2.7-mm shaver can be used. At the end of the procedure, the portals are sutured, and a bandage is applied. Active range of motion exercises are performed from day 1. Partial weight-bearing is advised for 2–3 days and gradually resumption of daily activities as supported. Sutures are removed from the 10th to 14th days.

### 20.4 Anterior Tibial Tendon

The tibialis anterior acts as an ankle dorsiflexor. Tibialis anterior tendinitis is rare. However, a direct trauma in a footballer might produce local tenderness and swelling.

Insertional tendinopathy has been described, more often in ballet dancers or jumpers. Patients describe pain on the medial cuneiform particularly when loading the foot immediately after heel strike or during swing phase of gait [29]. Spontaneous rupture is also rare and usually occurs in elderly people causing a foot drop and subsequent gait abnormality.

## 20.5 Injection Therapy

Percutaneous treatment of pain conditions has been gaining increased popularity, particularly among football players.

Injection treatments have been including several agents such as corticosteroids [11], polidocanol, platelet-rich plasma (PRP), high-volume injections, hyperosmolar dextrose, brisement, aprotinin, and low-dose heparin [49].

Corticosteroids have a powerful anti-inflammatory effect. Its use in tendon pathology is currently discouraged given the risk of subsequent tendon degeneration and/or rupture [39].

Polidocanol has been proposed as a method to abolish neovascularity within and around inflamed tendons [21]. However, the role of this neovascularization phenomenon is not fully understood which is in line with inconsistent results obtained from the method [47].

One must understand that each agent and technique presents its specific implications, and until now there is no such thing as a securely effective and harmless percutaneous "panacea" capable to cure all sources of pain.

There is controversial data in literature involving percutaneous treatment/prolotherapy, and sometimes the rationale supporting its application, particularly in tendons, is not completely understood.

These are promising and increasingly developing techniques and, while considering them as valuable options in specific conditions, one should keep some caution and critical spirit.

Currently, the most popular is probably PRP in one of its different preparations (with inherent different effects). It has been promoted for injection therapy as minimally invasive, nonoperative approach for several conditions [25]. It proposes a wide range of favorable defects in several tissues including tendon pathologies, muscle injuries, or even cartilage and joint arthritis. However, there has been some controversy on its clinical use [42]. Results have been obtained from pathologies, considering different tissues and different preparations of PRP (e.g., with/without leukocytes, platelet concentration, etc.) which limits the possibility for further conclusions or guidelines for its application [18]. It should be acknowledged that this is

a promising technique. Growth factors provide consistent laboratorial results; however, extrapolation to clinical setting is demanding [1]. Improved methodology in related research design and more clinical trials assessing outcome of PRP technology are still required [5].

It is necessary to develop appropriate guidelines and increase evidence level prior to its widespread application as a treatment option for joint diseases [36].

Many limitations persist concerning clinical and basic science aspects of tendon healing. The critical goal is tendon repair that leads to faster rehabilitation with regeneration of tissue with similar or better characteristics than those of the normal tendon. Tissue engineering and regenerative medicine research envisions new answers for the future [10].

#### 20.6 Conclusions

Tendinopathies around the foot and ankle in footballers are frequently associated to overuse and/or traumatic events. Fine control of pronation-supination has been implicated in technical skills required for ball control. Dealing with patient's expectations is particularly demanding in elite football players. Tendoscopy of posterior tibial tendon and peroneal tendons has been significantly developed in recent years. It has shown to provide definitive diagnosis in previously dubious situations, enables definitive therapeutic approach in an important percentage of cases, and assists in diminishing surgical aggression when open surgery is required. Despite being a demanding approach with challenging learning curve, it provides shorter recovery with faster return to football participation in selected cases. Future developments from biology and tissue engineering might further improve our course of action. Peroneal tendons and posterior tibial tendon pathologies must be understood combining a comprehensive approach of foot and ankle anatomy and biomechanics.

### References

- Boswell SG, Schnabel LV, Mohammed HO, Sundman EA, Minas T, Fortier LA (2014) Increasing platelet concentrations in leukocyte-reduced platelet-rich plasma decrease collagen gene synthesis in tendons. Am J Sports Med 42(1):42–49
- Cooper AJ, Mizel MS, Patel PD, Steinmetz ND, Clifford PD (2007) Comparison of MRI and local anesthetic tendon sheath injection in the diagnosis of posterior tibial tendon tenosynovitis. Foot Ankle Int 28:1124–1127
- Cote KP, Brunet ME, Gansneder BM, Shultz SJ (2005) Effects of pronated and supinated foot postures on static and dynamic postural stability. J Athl Train 40:41–46
- Eckert WR, Davis EA Jr (1976) Acute rupture of the peroneal retinaculum. J Bone Joint Surg Am 58:670–672
- Engebretsen L, Schamasch S (2012) The use of platelet-rich plasma in sports medicine—the International Olympic Committee Opinion. Oper Tech Orthop 22:43–48
- Frey C, Shereff M, Greenidge N (1990) Vascularity of the posterior tibial tendon. J Bone Joint Surg Am 72:884–888

- Geppert MJ, Sobel M, Bohne WH (1993) Lateral ankle instability as a cause of superior peroneal retinacular laxity: an anatomic and biomechanical study of cadaveric feet. Foot Ankle 14:330–334
- Gluck GS, Heckman DS, Parekh SG (2010) Tendon disorders of the foot and ankle, part 3: the posterior tibial tendon. Am J Sports Med 38:2133–2144
- Heckman DS, Reddy S, Pedowitz D, Wapner KL, Parekh SG (2008) Operative treatment for peroneal tendon disorders. J Bone Joint Surg Am 90:404–418
- Hogan MV, Bagayoko N, James R, Starnes T, Katz A, Chhabra AB (2011) Tissue engineering solutions for tendon repair. J Am Acad Orthop Surg 19:134–142
- Johnson JE, Klein SE, Putnam RM (2011) Corticosteroid injections in the treatment of foot & ankle disorders: an AOFAS survey. Foot Ankle Int 32:394–399
- Johnson KA, Strom DE (1989) Tibialis posterior tendon dysfunction. Clin Orthop Relat Res (239):196–206
- 13. Joshi SD, Joshi SS, Athavale SA (2006) Morphology of peroneus tertius muscle. Clin Anat 19:611–614
- 14. Kim GK (2010) The risk of fluoroquinolone-induced tendinopathy and tendon rupture: what does the clinician need to know? J Clin Aesthet Dermatol 3:49–54
- 15. Kulig K, Reischl SF, Pomrantz AB, Burnfield JM, Mais-Requejo S, Thordarson DB et al (2009) Nonsurgical management of posterior tibial tendon dysfunction with orthoses and resistive exercise: a randomized controlled trial. Phys Ther 89:26–37
- Lee MS (2005) Posterior calcaneal displacement osteotomy for the adult acquired flatfoot. Clin Podiatr Med Surg 22:277–289, vii
- 17. Lhoste-Trouilloud A (2012) The tibialis posterior tendon. J Ultrasound 15:2-6
- Martinez-Zapata MJ, Marti-Carvajal AJ, Sola I, Exposito JA, Bolibar I, Rodriguez L et al (2012) Autologous platelet-rich plasma for treating chronic wounds. Cochrane Database Syst Rev (10):CD006899
- 19. Monteggia GS (1803) Parte secondu. Instituzini Chirurgiche. In: vol. Milan, pp 336-341
- Nallamshetty L, Nazarian LN, Schweitzer ME, Morrison WB, Parellada JA, Articolo GA et al (2005) Evaluation of posterior tibial pathology: comparison of sonography and MR imaging. Skeletal Radiol 34:375–380
- Ohberg L, Alfredson H (2002) Ultrasound guided sclerosis of neovessels in painful chronic Achilles tendinosis: pilot study of a new treatment. Br J Sports Med 36:173–175; discussion 6–7
- 22. Oliva F, Del Frate D, Ferran NA, Maffulli N (2009) Peroneal tendons subluxation. Sports Med Arthrosc 17:105–111
- 23. Orthner E, Polcik J, Schabus R (1989) Dislocation of peroneal tendons. Unfallchirurg 92:589–594
- 24. Otis JC, Gage T (2001) Function of the posterior tibial tendon muscle. Foot Ankle Clin 6:1–14, v
- 25. Papalia R, Vasta S, Zampogna B, Tecam A, Maffulli N (2012) Platelet-rich plasma injections and surgery: short-term outcomes and long-term prognosis. Oper Tech Orthop 22:71–77
- Park HJ, Lee SY, Park NH, Rho MH, Chung EC, Kwag HJ (2012) Accuracy of MR findings in characterizing peroneal tendons disorders in comparison with surgery. Acta Radiol 53:795–801
- Petersen W, Bobka T, Stein V, Tillmann B (2000) Blood supply of the peroneal tendons: injection and immunohistochemical studies of cadaver tendons. Acta Orthop Scand 71:168–174
- Porter D, McCarroll J, Knapp E, Torma J (2005) Peroneal tendon subluxation in athletes: fibular groove deepening and retinacular reconstruction. Foot Ankle Int 26:436–441
- 29. Ritter S, Moore M (2008) The relationship between lateral ankle sprain and ankle tendinitis in ballet dancers. J Dance Med Sci 12:23–31
- 30. Roukis TS (2004) Corrective ankle osteotomies. Clin Podiatr Med Surg 21:353-370, vi
- Saxena A, Ewen B (2010) Peroneal subluxation: surgical results in 31 athletic patients. J Foot Ankle Surg 49:238–241

- Scholten PE, Breugem SJ, van Dijk CN (2013) Tendoscopic treatment of recurrent peroneal tendon dislocation. Knee Surg Sports Traumatol Arthrosc 21:1304–1306
- 33. Scholten PE, van Dijk CN (2006) Tendoscopy of the peroneal tendons. Foot Ankle Clin 11:415–420, vii
- Schubert R (2013) MRI of peroneal tendinopathies resulting from trauma or overuse. Br J Radiol 86:20110750
- Selmani E, Gjata V, Gjika E (2006) Current concepts review: peroneal tendon disorders. Foot Ankle Int 27:221–228
- 36. Sheth U, Simunovic N, Klein G, Fu F, Einhorn TA, Schemitsch E et al (2012) Efficacy of autologous platelet-rich plasma use for orthopaedic indications: a meta-analysis. J Bone Joint Surg Am 94:298–307
- Smith JT, Johnson AH, Heckman JD (2011) Nonoperative treatment of an os peroneum fracture in a high-level athlete: a case report. Clin Orthop Relat Res 469:1498–1501
- Sobel M, Mitzel M (1993) Peroneal tendon injury in current practice in foot and ankle surgery, vol 1. Mc-Graw Hill, Inc., New York, pp 30–56
- Speed CA (2001) Fortnightly review: corticosteroid injections in tendon lesions. BMJ 323:382–386
- Squires N, Myerson MS, Gamba C (2007) Surgical treatment of peroneal tendon tears. Foot Ankle Clin 12:675–695, vii
- Stapleton JJ, DiDomenico LA, Zgonis T (2008) Corrective midfoot osteotomies. Clin Podiatr Med Surg 25:681–690, ix
- Tinsley BA, Ferreira JV, Dukas AG, Mazzocca AD (2012) Platelet-rich plasma nonoperative injection therapy—a review of indications and evidence. Oper Tech Sports Med 20:192–200
- 43. van Dijk CN, Kort N (1998) Tendoscopy of the peroneal tendons. Arthroscopy 14:471-478
- 44. van Dijk CN, Kort N, Scholten PE (1997) Tendoscopy of the posterior tibial tendon. Arthroscopy 13:692–698
- 45. van Dijk CN, Scholten PE, Krips R (2000) A 2-portal endoscopic approach for diagnosis and treatment of posterior ankle pathology. Arthroscopy 16:871–876
- 46. van Dijk CN, van Sterkenburg MN, Wiegerinck JI, Karlsson J, Maffulli N (2011) Terminology for Achilles tendon related disorders. Knee Surg Sports Traumatol Arthrosc 19:835–841
- 47. van Sterkenburg MN, de Jonge MC, Sierevelt IN, van Dijk CN (2010) Less promising results with sclerosing ethoxysclerol injections for midportion Achilles tendinopathy: a retrospective study. Am J Sports Med 38:2226–2232
- van Sterkenburg MN, Haverkamp D, van Dijk CN, Kerkhoffs GM (2010) A posterior tibial tendon skipping rope. Knee Surg Sports Traumatol Arthrosc 18:1664–1666
- 49. van Sterkenburg MN, van Dijk CN (2011) Injection treatment for chronic midportion Achilles tendinopathy: do we need that many alternatives? Knee Surg Sports Traumatol Arthrosc 19:513–515
- Vega J, Golano P, Dalmau A, Viladot R (2011) Tendoscopic treatment of intrasheath subluxation of the peroneal tendons. Foot Ankle Int 32:1147–1151

# Chapter 21 The Footballer's Inlay Sole: An Individualised Approach

D.P. Francisco Escobar Ruiz and Pieter P.R.N. d'Hooghe

One of the problems in Football is that the only people who really know how to play the game... are sitting in the pressroom.

Robert Lembke - German journalist (1919)

**Abstract** Football is one of the most popular sports and one in which the lower extremities are most exposed to injury. Statistics show that between 2 and 9.4 players per 1,000 h of exposure suffer injury to their lower limbs. Of these injuries, we most commonly find sprains, fractures, tear ligaments as well as various blows and bruises. The type of foot is considered to be an intrinsic factor when taking injury into consideration while at the same time the football boot is considered an extrinsic factor.

The inlay sole or insole (foot orthotic, FO) has been used for many years as a tool for health professionals in the treatment and prevention of injury to the feet and lower limbs among football players. The principal objectives are improvement in sports performance, optimisation of biomechanics and the reduction of pain.

The materials used in FO – and at the same time the making of the moulds and the techniques employed in their manufacturing – have evolved at a rapid pace over the last few years. It's not so long ago that metal inlays were beaten into shape with a hammer. Compare that to the carbon fibre ones (or the composites formed under high temperatures) we have nowadays and you know what an evolution football inlay soles have gone through over the last decennia.

D.P. Francisco Escobar Ruiz (🖂) Sport Podiatry Center PODOAXIS, Madrid, Spain

F.C Getafe, Spain

Spartak of Moscow, Moscow, Russia http://www.podoaxis.com

P.P.R.N. d'Hooghe, MD Department of Orthopaedic Surgery, Aspetar Hospital, Aspire Zone, Doha, Qatar e-mail: pieter.orthopedie@gmail.com

P.P.R.N. d'Hooghe, G.M.M.J. Kerkhoffs (eds.), *The Ankle in Football*, Sports and Traumatology, DOI 10.1007/978-2-8178-0523-8\_21, © Springer-Verlag France 2014

Acknowledgment: D.P Manuel Romero Soto

The advances in technology and the materials used in the fabrication have enabled more and better results thanks to the wide range of options now available. Furthermore, weight and size are now no longer a problem for the adaptation of the football boot.

The importance of the foot in the mechanics of the lower extremities is an undeniable reality, and custom-made foot orthoses (FOs) are possibly the best option for the treatment and prevention of injuries, through the optimisation of the biomechanics of the football player.

Keywords Football • Inlay sole • Orthosis

#### 21.1 Introduction

No clear definition of foot orthosis or inlay sole exists. The definition taken from *Dorland's Medical Dictionary* 'an orthopaedic appliance or apparatus used to support, align, prevent or correct deformities or to improve the function of movable parts of the body' is the most widely used, but it is not altogether complete.

*Ray Anthony* describes the inlay soles this way: 'the functional orthosis is an orthopaedic device which is designed to promote structural integrity of the joints of the foot and lower limb, by resisting ground reaction forces that cause abnormal skeletal motion to occur during the stance phase of gait'. This definition introduces new concepts mentioning functional orthosis, reaction forces and the dynamic phase.

The description given by *Kirby* could possibly be the most complete and currently accepted: 'An in-shoe medical device which is designed to alter the magnitudes and temporal patterns of the reaction forces acting on the plantar aspect of the foot in order to allow more normal foot and lower extremity function and to decrease pathologic loading forces on the structural components of the foot and lower extremity during weightbearing activities'.

Since its beginnings in 1845 boot makers and all through the twentieth century, different podiatrists and medical practitioners tried to develop an orthopaedic treatment for painful pathologies and deformations of the feet.

Midway through the twentieth century, *Merton Root* and collaborators produced one of the first valid theories for a new lower extremity biomechanical classification system based on STL neutral position and eight biophysical criteria of the foot and lower extremity with the idea of finding the ideal or normal foot. Working further on this theory, they began to work up thermoplastic sole inlays adapted from a plaster mould in the attempt to find the neutrality of the foot. At this moment, a new era in the development of FOs started really off.

It was later when newer investigations began to appear, investigations which tried to refine and potentiate the therapeutic effectiveness and prescribe treatments for new pathologies. Campbell, Blake and Kirby among others were the maximum exponents of these new theories and orthopaedic treatments.

#### **21.2** Efficacy of Custom-Made Foot Orthoses (Inlay Soles)

In the world of sports and above all in football, a wide-ranging effectiveness is demanded, meaning that we must provide greater scientific evidence that justifies the most effective treatment for our football player. Because of this and thanks to the technological advances in the last few decades – accompanied by greater clinical experience – we now know more about the provision of scientific validation for each and every treatment/prevention of injury through FO in football.

No criteria in the evaluation of the effectiveness of the FO exist through consultation of scientific literature. We can find multitudinous articles which try to demonstrate the effectiveness based on the reduction of pain, mechanical change, reduction of injury treatment times, and patient satisfaction. The importance however of comparing the effectiveness of custom-made FOs versus those of a standard fabrication (as the results can be quite different) is related to whether the FOs are based on a prior study of the patient or not.

Often, it helps in football to show the results of studies performed on different populations, due to the lack of further sufficient evidence. As sprinting and running are integral parts of football, we quite often find ourselves obliged to base our treatments upon studies that involve professional runners despite knowing that the movements are not exactly the same. The study of the mechanical changes in footballers can be quite difficult to perform due to the complexity of movements and the multiple external factors involved in these movements.

The comparison between the effectiveness of different FOs is often attempted to be demonstrated through the control of certain movements. In 2000, *Mc Poil and Cornwall* compared the effectiveness of FOs in relation to the control of tibial rotation. This study concluded that the use of soft, accommodative, pre-moulded FOs – in addition to shoes – can reduce the magnitude and acceleration of transverse internal tibial rotation. They also showed that the use of rigid materials in the construction of FOs (and the use of posting or wedging) is not more effective than the aforementioned.

In 2006, *MacLean* et al. demonstrated that custom-made FOs reduce the maximum angle of rearfoot eversion and speed together with the moment of internal inversion of the ankle in the short-term intervention in runners. This finding is usually seen in the initial midstance during the running cycle.

In 2007, *Stacoff and Col* demonstrated that a combined moulding and posting orthosis in the treatment of the valgus foot reduces eversion best. They also showed that individual variations may be due to subject-dependent proprioception, internal foot mechanics and/or a combination of both.

Anderson and Stanek in 2013 revised the efficiency of FOs in the treatment of plantar fasciitis – a common injury among football players – in which they proved the effectiveness of different types of FOs for this condition. However, it was also concluded that the reduction of symptoms was not complete in the majority of cases.

The clinical experience gained and the improved results obtained have led us to base our treatments on personalised FOs together with a previously performed clinical assessment of the patient. There are many intrinsic and extrinsic factors that we should consider in a football player's FO. The study and analysis of these factors combined with experience in football lead us to a therapeutic successful outcome.

## 21.3 Theories

Different biomechanical theories search for the possible causative mechanisms that produce injury. At the same time, these theories can be used in the study on the effect of FOs related to the football player. Thorough background in these theories, understanding the new technologies of Foot Function and dynamic ankle/foot behaviour allow us to explain the biomechanical causes of the pathology/injury to the ankle. This furthermore enables us then to successfully create a mechanical design and use it in the personalisation of the FO for our football players.

These theoretical insights have always served as the basis to choose the most appropriate FO for each player, tailored individually. This approach serves as the basis for our daily clinical assessment, diagnostics and treatment strategy in football.

The use of force plates, pressure platforms, in-shoe plantar pressure analysis and computerised three-dimensional motion analysis allows a more in-depth study of the biomechanics involved. Newer computer modelling techniques enable us to create a personalised design, tailored to the biomechanical needs of the football player. In this way, we try to alter the extrinsic pathological effects on the ankle in football.

One of the first theories was established by *Morton* in 1935. He concluded that the 'hypermobile first metatarsal segment' would produce pain in the foot and he proposed the fabrication of a compensating insole with a plantar extension of the first metatarsal. This is known today as the 'Morton's extension'.

In the 1960s, *Root and collaborators* developed a classification for the ideal ankle based on the neutrality of the subtalar joint with reference to the foot. In their belief, FOs were designed to neutralise malalignment to the subtalar joint and thus avoid further pathological compensation mechanisms towards the knee and foot.

In 1987, *Kirby* concluded that abnormal subtalar joint (STJ) rotational forces (moments) were responsible for many mechanically based pathologies in the foot and lower extremities. They also stated that the abnormal STJ axis spatial location was the primary cause of pathological STJ moments. As an example, an ankle with a medially deviated STJ axis was more likely to suffer from pronation-related symptoms, since the ground reaction force (GRF) would cause increased magnitudes of external STJ pronation moments. An ankle with a laterally deviated STJ axis will in the same way show supination-related syndromes since the GRF would cause increased magnitudes of external STJ supination moments. Kirby concluded that medial and lateral deviations of the STJ axis in the ankle will produce a change in magnitudes and directions of the moments of the STJ. They showed that this finding

consequently leads to a higher contractile activity in the musculature around the ankle and foot. Kirby and Green hypothesised that FOs could control the pronation of the STJ during weightlifting activities using a deep inverted heel cup known as the Blake inverted orthosis. This orthosis has been shown to be particularly interesting in the treatment of paediatric flatfoot deformity, posterior tibialis dysfunction, sinus tarsi syndrome and the magnitude control of external STJ supination moments.

A new alternative of great importance at a biomechanical level – and one which questions the validity of some of the current theories or standards (paradigms) in relation to the treatment of foot and ankle disorders – is the 'tissue stress model' as described by *McPoil and Hunt* in 1995. In their work, they presented assessment and treatment strategies for tissues that are damaged through excessive mechanical tension. Their model is based upon the 'load/deformation curve': as increasing load is placed on a body tissue, deformation or change in the dimensions of that structure gradually occurs. This 'stress tension model' generates a curve of deformation through tension in two regions, an elastic and a plastic one. The area between the two regions is the elastic limit and the final area is the rupture point. The normal or abnormal foot does not exist in this concept. What does exist is that each individual tissue has its own level of tolerance to the amount of tension it can withstand over time, and the author's belief is that the FO will aim to reduce this tension.

The *Fuller* model described in 1996 describes how to locate the pressure centres of the sole of the foot and how to spatially orientate the STJ in order to assist in the prevention and therapy of ankle injuries through the use of FO.

One of the latest theories of foot orthosis function, 'The preferred Movement Pathway Model', is recently being proposed by *Nigg*. The authors state that if the preferred ankle movement pathway is counteracted – e.g. with a combination of insoles and shoes – an increase in muscular activity will be noted, whereas if the movement is permitted, a reduction of muscular activity will be seen. Therefore, they conclude that the function of the FO should not be to realign the skeleton but to conduct the 'muscular tuning' during the sports.

These newer insights in functional ankle/foot models are receiving considerable recognition within the biomechanics international community. Nevertheless, more research is necessary to understand the efficacy of FOs in football particularly.

## 21.4 Biomechanical Effects of the FOs in Football

We often tend to believe that football is a sport where the player is in constant contact with the ball, but the reality is quite different. If we measure the intensity of exertion – represented by the total distance run and divided into separate actions – a defender will run between 8 and 12 km per match divided into 1,000 separate actions and with a pause every 6 s.

In total, the distance run is performed walking (25 %), at a slow jog (37 %), at cruising speed (submaximum) (20 %), performing tackles (11 %), backwards (7 %) and backwards and to the sides (16 %). The distance run with the ball is around 2 %

of the total for which reason we must not consider this the predominant action of the player. Also to the action of running with the ball, we must add jumps (1 every 5-6 min) and the hitting or kicking of the ball.

With regard to the lower extremities, the hitting or kicking of the ball is important in as much for its frequency as for its aggression. The kicking or striking of the ball has been studied on many occasions. However, the many different situations and individual peculiarities make the establishment of a standard mechanical pattern identical to all football players difficult.

In 2010, *Juarez* studied the starting speed of the ball when kicked with the instep. The author observed that the speed of the foot at the moment of striking the ball was of  $24.59 \pm 1.33$  m/s similar facts to the  $20.4 \pm 1.3$  m/s observed by Barfield and collaborators in 2002.

The diversity of the movements and gestures in football make it a singular sport. In the first place, the player's profile should be known: how is the player like, and what are the player's needs and requirements? It can also be very helpful to understand their performance personality, the position and behaviour in which they play on the pitch, quirks, footwear habits, etc.

Added to the importance of understanding the biomechanics of the player is understanding the effects of FO on the ankle/foot and the lower extremity. This will enable us to produce an integral personalised support that offers the desired outcome to the player.

The first investigations into the effects of FOs on athletes showed little or no changes in the gait kinematics and questioned whether FOs could have any demonstrable effect on the ankle/foot and lower extremities. However, technological advances and the ability to carry out research have shown how FOs can alter the mechanical function of the ankle/foot and how they assist in the prevention of lower limb football injuries.

It has been shown that FO intervenes in the *kinetic control of the foot and lower extremities*. This control is produced through changes such as the reduction in the angle of eversion and the speed of the hindfoot, angle of dorsiflexion of the ankle, moment of maximum inversion of the ankle, changes in knee external rotation moments and changes in knee abduction moment.

If we take, for example, an anterior tibialis tendinopathy in a football player, a custom-made FO can shift the most medial forces of reaction in the heel and longitudinal arch resulting in an increase in external STJ supination moment. This will lower the contractile forces of the muscle and in turn the tensile forces in the tendon. This example shows how an FO can improve the symptoms of an ankle/foot tendinopathy and alleviate the player in the frontal tibia. It should be noted that every FO has an immediate impact on the kinematics of the athletic football movement and therefore should be used with caution.

Research has shown that EMG alters the activity of the muscles in the lower extremities with athletic activity. Since FOs alter the *contractile activity of lower extremity muscles*, they can also alter EMG activity and this should be taken into account in further gait EMG research.

Evidence of improvement in postural stability through the use of FOs exists. With the help of 3D platforms, ankle inversion-eversion movements in subjects with ankle instability were measured. The use of FOs showed to improve the mobility of the foot and ankle. With unipodal standing, a decrease of medial control in the hind-foot (pronated feet) can be seen. The importance of stability in unipodal standing in the football player is important due to the necessary action of striking the ball. Having good stability increases the postural control abilities and there is a reduction in medial-lateral sway during bipodal standing.

The forces and pressures on the sole of the foot can also be modified by another therapeutic mechanism for injured and painful areas. In players suffering from metatarsalgia, the pressure peaks at the metatarsal heads and loading forces can be diminished by FOs. With football players, we must also add the intrinsic pressure on the metatarsal heads by the studs from the football shoes. At the moment of ankle propulsion – owing to the poor distribution of the studs – an increase in the pressure on the metatarsal heads may be produced, causing possible injury to the bone and soft tissues in the joint. A double action on the redistribution of the loads on adjacent metatarsals – with reduced loads combined with the correct distribution of the studs – will diminish the overload. This redistribution of FOs in football.

## 21.5 Custom-Moulded Foot Orthoses (Custom-Fitted)

In recent years, the greater efficacy of custom-fitted FOs has been advocated compared to standard ones. Considering the advances in gait kinematics, FO customisation and technological knowledge over the past few years, this should be considered further. Some of the following studies demonstrate the effectiveness of customfitted FOs for other ankle/foot treatments.

The personalisation of insoles improves in the following:

- · Assessing ankle/foot pain reduction or functional limitation of deformity.
- Metatarsal padding was shown to relieve sesamoid overload in 80 % of patients (*Axe and Ray* 1988).
- Impact reduction in plantar fasciitis or calcaneal spurs, compared to other treatments such as anti-inflammatory treatment (corticosteroid injection plus NSAIDs) or silicon heel cups (*Lynch* 1998).
- *Saggini* studied in 1996 the effectiveness of the custom dynamic FO versus heel lift on myofascial trigger points in the peroneus longus muscle, in patients with an anatomical leg-length discrepancy. The patients who wore custom-fitted insoles experienced a significant reduction in pain. The author stated that controlling the biomechanics with a custom-fitted FO was more important than using a heel lift.
- They improve and reduce the plantar pressure in the forefoot by up to 1/3 in relation to sham insoles.

- Greater control over skin lesions, callus, etc. where an increase of pressure produced these lesions.
- The efficacy of FOs on stress fractures in the military was proven already in 1989 by *Simkin*. The effect was demonstrated through the FO reduction of lesions in low arched feet and the reduced stress in high arched feet.

Clinical experience has brought us to the conclusion that the use of custom-fitted FOs is much more effective compared to the prefabricated ones. Prefabricated insoles (less expensive devices) have solved above all mild injuries but have not been as effective in the prevention of elite football player injuries.

Still, there is only limited data demonstrating the efficacy of custom-fitted FOs being more effective than the prefabricated variety. For example, *Sasaki and Yasuda* proved in 1987 the effectiveness of a prefabricated wedge (lateral heel/valgus wedge) in order to reduce the symptoms of a mild medial osteoarthritis in the knee. This shows that less expensive methods can help to reduce symptoms with a slight wedge or prefabricated device in daily life activities. A study by *Mc Court* in 1990 demonstrated no difference in the reduction of symptoms between prefabricated FOs and the custom-fitted types. There was no significant difference between the controlled groups, costs and relief timing, associated with custom FOs compared to the non-custom types. Further credible research is necessary to delineate specifically the differences between them, especially in football.

## 21.6 Capture Mould (CAD-CAM)

The search for a reliable technique to obtain a correct mould of the foot – to enable the most exact and trustworthy reproduction as possible – is one of the challenges faced by modern orthopaedic technicians. First, a fitted cast allows us to create the necessary corrections and enables us to compensate for the necessary mechanical alterations. Then – with utmost precision – one can start the FO personalisation process. Further, this personalisation phase is of high importance because the therapeutic success of the FO depends upon the method chosen.

One of the most commonly used techniques for the capture of the foot is the pedigraphy method, where plaster casting or foam boxes are used with good objective and subjective outcome. In Europe, the method of direct adaptation was also commonly used. Nowadays, advances in the latest 3D-scan technologies make it possible to capture the foot in the most precise and anatomical way.

The essence for all used methods is to find a reliable neutral mechanical model that reproduces the ankle/foot parameters as closely as possible matching with the clinical measurements. The most commonly used method to create a correct and neutral mould is the forefoot aligned with the hindfoot, the foot contour and plantar surface in the correct angle along with the height and length of the internal longitudinal arch.

The choice of the 3D scanner system as a protocol method for capturing the foot has been shown to be valid, reliable and a good tool in our clinical daily life activities. This 3D system is mainly used by commercial brands for the customisation of shoes for football players.

In the last decennium, clinical results have been shown the best using 3D scanning when comparing all the different methods and techniques of mould capture in football.

The advantage of 3D surface scanning in foot capture is that it offers reliable information and reduces the negative variables in the ankle/foot measurement. This technique is independent of clinical experience in relation to other techniques such as plaster casting or foam box moulding. In casting or foam box techniques, clinical experience is essential, but a lack of it can evidently affect the reliability of the mould.

In plaster moulding, clinical measurements of the ankle/foot can be affected, for example, by the height of the arch or by the alignment of the forefoot to hindfoot. Other affecting variables in the handling of the plaster mould are the amount of water used (setting time), the ambient temperature and quantity of plaster. All of these variables can influence the difficulty of casting with a prolonged immobilisation in a neutral position, a good grip of the ankle/foot or even the tiredness in the arms of the castmaster. The result of a good casting mould is determined by the correct handling and control over these above-mentioned variables.

With foam box moulding, there are errors in the pressure of the foot over the foam that can increase the measurements due to the expansion of soft tissue areas and of the lateral-to-medial ankle/foot movements. The width of the forefoot, for example, is 1.5 cm greater with this technique than in comparison with other techniques.

A good mould that is casted in the most optimal conditions remains the golden standard in worldwide practice. With the use of the latest generation 3D scanners, however, more reliable outcome is seen in sports such as in elite football.

To reduce possible errors and because of the flexibility of the foot, the correct position of the foot on the scan is with the knees and ankles resting on a controlled load support on the scanner mirror. Any increase in the load on the forefoot should be avoided, and the aim is to position the foot muscularly relaxed and easy to manipulate. This allows us to find the point of neutrality in the mould and adjust to the clinical measurements obtained. Through this method, we can repeat the scan as many times as necessary until we can document the correct scanned image (Fig. 21.1).

To minimise errors in the scan, we must place the foot on the glass in order to gain precision (to thousandths of a millimetre) compared to a foot position at a higher distance from the scanner. A correct calibration of the scanner and a good resolution are extremely important to generate a virtual mould with the highest accuracy possible so that the correct foot's length, width and height can be measured. Evidently, the clinician needs to use the correct technique in order to minimise errors also and this is where experience is key.



Fig. 21.1 3D partial weightbearing digital scanner

One of the major disadvantages in the use of these above-mentioned techniques is the difference in costs between them. The use of plaster casting or a foam box is much lower than that of digitalisation. However, casts and moulds have a higher cost in storage and materials compared to the digital materials that are cheaper in a daily use setting.

# 21.7 The Design of FOs (Computer-Aided Design, CAD, and Computer-Aided Manufacturing, CAM)

The precision of the digital image captured by the 3D scanner is an important tool for the digital software to personalise the FOs. It also allows us to make the necessary corrections and to treat the existing mechanical alterations with great accuracy. Other methods (e.g. the manual ones) do not permit this speed of design in their elaboration.

Once the digital mould is captured, the necessary points of reference for the design are taken. The points of reference taken are those provided by the reliability of this method of measurement of the foot, and in this way, a neutral mould is obtained. The points of reference taken are the centre of the heel, the 1st and 5th metatarsal heads and the medial tubercle point of the navicular bone at the longitudinal internal arch. These are the points that determine the contour and measurements of the 3D-based custom-fitted FO.

The advantage of this method is that we can modify the captured digital mould and every individually generated image of the FO. It is possible to work with a multitude of parameters such as changing the inclinations of the mould, the measurements of the width of forefoot or hindfoot, invert or evert the mould to the exact form, arch fill, forefoot tapers, flanges, raise or lower the first or fifth metatarsal, heel cups, advance or postpone with relation to the FO, etc.



Fig. 21.2 Software to design and type of corrective or accommodating custom orthotic insoles

It is also possible to make modifications on the FO directly. It is simple to add intrinsic and extrinsic forefoot or hindfoot posts in order to adjust foot alignment, posts like cutouts, kinetic wedge extensions, heel raise or lifts (that can be applied in the event of leg-length discrepancies) and a wide range of mechanical corrections with a high degree of accuracy. Even every effective personal design can be saved with a high degree of control and adaptability. These options can be used for corrective, as well as accommodative, applications (Fig. 21.2).

The true difficulty with FOs for football players is the space between the insole and the shoe. In football, 3D image capturing is the preferred FO tool for perfect fit in the shoe since it can create an exact design to the form of the shoe and also change the width of the heel, midfoot and forefoot without the loss of any correction or fitting.

A football player expects to have a light and comfortable FO. To create the correct width, shape and thickness in the FO production process is indeed the biggest challenge today. FOs can be elaborated in a single layer of just 1.5 mm including the necessary mechanical corrections, without the need to add any additional materials that might affect the FO's thickness and lightness. In this way, FOs can be made lightweight and flexible without losing the necessary stability to maintain a correct alignment.

Furthermore, the moulds and designs can be saved easily on computer and used in a multifunctional way.

The advantages of these systems – aside from their ease of use and reliability – are that the common problems of storing moulds and materials are avoided. Today, it also still requires a technical craftsmanship – regardless of the digitalisation era – as the outcome of the design is based upon a skilled experience that remains completely personalised.



Fig. 21.3 Manufacturing four pairs of foot orthoses (homopolymer polypropylene for CAD-CAM)

#### 21.8 Machining Custom Orthotic Insoles

The next step in the process after the scanning and design phase is the machining of the different materials. In this process, the use of a digital laboratory and a system capable of reading the designs is necessary. By doing so, the design is matched to the selected materials through the use of a cutting machine of numerical control (CNC) (Fig. 21.3).

The milling machine uses a sharp rotary blade, and through subtractive manufacturing, the different materials are formed into an FO. These materials can be EVAS or plastic. Depending on the power and/or speed of the milling machine, we can elaborate an FO in 15 min (if it is machined on just one side and in 25 min if it is machined on both sides). The milling machine has an error margin of just 0.05 mm.

The performance time of a plaster of foam box mould is quite different since it relies on thermo-adaptive materials that require time to cool down. Hence, because we are working with different layers of materials, this process can take up to over 1 h.

# 21.9 Materials to Customise the FOs

Correct material selection and correct design are the most important modalities to create a football inlay sole that matches the high demand needs of the player.

At present, we are living in an advanced technological era that provides us with the opportunity to access the best next-generation materials and even enables us to use high-profile robotic systems. For these mechanised systems, blocks of raw material are necessary. For the elaboration of FOs in football, we look for a series



Fig. 21.4 CAD-CAM blocks. Different densities and colours to suit your requirements

of very specific materials that provides the necessary requirements. Mostly used materials nowadays in elite football are for double- or triple-density *EVA CAD-CAM (ethylene vinyl acetate)*. These materials offer the orthopaedic technician a rapid and efficient mechanisation process that also creates a personalised and multifunctional FO. The higher densities are used in the areas where we want most control whereas the lower densities provide comfort in the other zones of the foot. These FOs are lightweight, shock absorbing and very hard wearing and also retain their shape. They also reflect body heat with thicknesses that we can adapt to, taking into account the reduced space of a football shoe. We can personalise FOs with singular characteristics and easy adaptability. Here, we can see one of the major differences between the digital and the manual techniques. If a similar effect would be expected by the manual moulding techniques, we would need more different layers of material that would require a thicker and heavier FO, thus, consequently leading to more discomfort and less fitting for the player.

Another new material related to the advances in design software (which allows us to machine on both sides) is polypropylene (homopolymer polypropylene). This is a thermoplastic polymer that can be found mixed with amalgamations of fibre glass or carbon. By doing so, it offers the FO its specific properties for its personalization and adaptation process. The capacity of this material to yield pressure and torque – yet consistently return its form without fatiguing or eliminating distortion – we are provided with FOs of an excellent and solid production that remain rigid even in thin applications. Thanks to the FO's stability, good mechanical control, slim design, lightweight status, comfortable feeling in gameplay and durability, it is widely used in elite football (Fig. 21.4).

There is a great variety of available materials on the market to make a comfortable FO, but the choice of use remains very sport-specific, especially in football (Fig. 21.5).

**Fig. 21.5** Inlay sole fit inside a football boot (multidensity)



# 21.10 Conclusion

The manufacturing of foot orthoses (FOs) has been susceptive to major changes in the last decennium with the emergence of 3D scanners and software designs. They offer a variety of options to improve the design of corrective or accommodative orthotics insoles. Particularly, the latest generation of customised FOs is widely recognised and is used in elite football. Digitalisation facilitates further the FO design and manufacturing processes. It also offers a reliable, precise and comfortable FO to the player, compared to the thermo-formed moulded techniques. We use nowadays FO materials with special mechanical properties that offer the player flexible, lightweight and comfortable FOs. The challenge remains to reduce the FO thickness even more in the future, occupying minimal space in the football shoe (enabling the player to maintain a natural feeling) without compromising the FO's therapeutic goals.

# References

- Burns J, Landorf KB, Ryan MM, Crosbie J, Ouvrier RA (2008) Intervenciones para la prevención y el tratamiento del pie cavo (Revisión Cochrane traducida). En: La Biblioteca Cochrane Plus, Número 4. Update Software Ltd, Oxford. Disponible en: http://www.update-software. com. (Traducida de The Cochrane Library, 2008 Issue 3. Wiley, Chichester)
- 2. Carroll M, Annabell ME, Rome K (2011) Reliability of capturing foot parameters using digital scanning and the neutral suspension casting technique. J Foot Ankle Res 4(1):9
- 3. Hawke F, Burns J, Radford JA, du Toit V (2008) Ortesis de pie hecha a medida para el tratamiento del dolor de pie (Revisión Cochrane traducida). En: La Biblioteca Cochrane Plus, Número 4. Update Software Ltd, Oxford. Disponible en: http://www.update-software.com. (Traducida de The Cochrane Library, 2008 Issue 3. Wiley, Chichester)
- 4. Hume P, Hopkins W, Keith R, Maulder P, Coyle G, Nigg B (2008) Effectiveness of foot orthoses for treatment and prevention of lower limb injuries: a review. Sport Med 38(9):759–779
- Kirby KA (2010) Chapter 2: Evolution of foot orthoses in sports. In: Werd MB, Knight EL (eds) Athletic footwear and orthoses in sport medicine. Springer, New York, pp 19–32

- 6. Kirby KA, Spooner SK, Sherer PR, Schuberth JM (2012) Foot and ankle specialist. Foot Ankle Spec 5:334; originally published online 10 September 2012
- 7. Koller A, Rosenbloom KB, Schweinberger MH, Smith S (2009) Orthotics and shoes. Foot Ankle Spec 2:294–8
- Landorf KB, Keenan AM (2000) Efficacy of foot orthoses. What does the literature tell us? J Am Podiatr Med Assoc 90(3):149–158 (Aust J Pediatr Med 32(3):105–113)
- 9. Laugthon C, McClay Davis I, Williams DS (2002) A comparison of four methods of obtaining a negative impression of the foot. J Am Podiatr Med Assoc 92(5):261–8
- Llana S, Pérez P, Lledó E (2010) La epidemiología del fútbol: una revisión sistemática. Revista Internacional de Medicina y Ciencias de la Actividad Física y el Deporte 10(37):22–40
- Percy ML, Menz HB (2001) Effects of prefabricated foot orthoses and soft insoles on postural stability in professional soccer players. J Am Podiatr Med Assoc 91(4):194–202
- Reilly T (1996) Aspectos Fisiológicos del Fútbol. Actualización en Ciencias del Deporte. 4(13)
- Štajer T, Burger H, Vidmar G (2011) Influence of casting method on effectiveness of foot orthoses using plantar pressure distribution: a preliminary study. Prosthet Orthot Int 35(4):411–7
- Telfer S, Woodburn J (2010) The use of 3D surface scanning for the measurement and assessment of the human foot. J Foot Ankle Res 3:19

# Chapter 22 Rehabilitation After Ankle Football Injuries

**Mark Feger and Jay Hertel** 

Football: a feast!

Dr. Michel d'Hooghe – a great (grand)father and SportsMedicine mentor

**Abstract** Rehabilitation for foot and ankle injuries in the football athlete can provide unique challenges for a sports medicine clinician. The majority of the book focuses on specific foot and ankle injuries and highlights important rehabilitation considerations for each of those injuries. This chapter aims to highlight important rehabilitation of all foot and ankle injuries. Due to the unique challenges associated with foot and ankle injuries in the football athlete, a comprehensive and multidisciplinary approach to rehabilitation is required. This chapter outlines the key members of the sports medicine team as well as how to structure a rehabilitation program around important short- and long-term goals that will need to be achieved on the road to recovery. We also highlight the importance of a return to participation progression and the benefit of having a consensus among the sports medicine team about the appropriateness of returning an injured athlete back to participation.

Keywords Rehabilitation • Functional exercise • Neuromuscular training

M. Feger, MEd, ATC (🖂) • J. Hertel, PhD, ATC

Department of Kinesiology, University of Virginia,

<sup>210</sup> Emmet St. South, Charlottesville, VA 22901, UK e-mail: mf3de@virginia.edu

## 22.1 The Sports Medicine Team

The rehabilitation process for all but the most severe ankle injuries begins immediately after the injury has been sustained. However, the rehabilitation process should not be considered complete once a player returns to the pitch. The physical demands competitive football places on the foot and ankle during competition necessitates that the rehabilitation process be multidisciplinary and comprehensive in nature. The multidisciplinary sports medicine team at a minimum includes a physician, an athletic trainer or sports physiotherapist, coaches, and the injured player. Regardless of the specific personnel, the sports medicine team should collectively have an intimate knowledge of the following:

- · Pathomechanics of the injury
- · Healing process of the injured tissue
- Techniques to assess tissue integrity
- · Techniques and tools to aid in rehabilitation
- Physical and psychological demands of the injury, rehabilitation process, and the progression of returning to sport

Having the collective knowledge required to facilitate a safe return to sport is only beneficial when collaboration and effective communication is observed between the members of the sports medicine team. Coaches and players often do not understand the pathophysiology of the injury or healing process and thus must be informed of clinical signs and symptoms utilized by the clinicians when making decisions on treatment or functional progressions. Conversely, many clinicians may not understand the physical demands placed on the foot and ankle such as when striking a ball from 18 m out with a defender slide-tackling from the rear. Therefore, the final return to play decision requires a consensus from the sports medicine team to ensure the injured tissue can withstand the loads placed on it during sport and that the physical and mental state of the athlete leaves no apprehension about reinjury during participation.

### 22.2 Setting Goals

Setting goals is an integral part in the rehabilitation process and often facilitates communication between the members of the sports medicine team when trying to establish safe and realistic time frames. Goals should include objective measurements of clinical progress as well as subjective assessments associated with the athlete's perception of physical and psychological concerns.

### 22.2.1 Short-Term Goals

Short-term goals during the rehabilitation process are often designed to address clinical signs or symptoms and restore physical limitations such as range of motion, neuromuscular control, muscle function, and balance. The immediate short-term



Fig. 22.1 Flow chart of goals of general rehabilitation process and common techniques to achieve individual short- and long-term goals

goal for any injury should always be accurate identification, protection, and management as to not cause further injury or exacerbate the inflammatory response. Subsequent short-term goals should be documented with objective measurements and realistic time frames of which the goal is to be achieved. The majority of this chapter focuses on rehabilitation considerations for many of the physical limitations of which short-term goals will likely be based upon (Fig. 22.1).

## 22.2.2 Long-Term Goals

Long-term goals are almost always associated with "milestones" within the rehabilitation process and will be different for each injury. A long-term goal following a Grade I ankle sprain may be to return to full competition within 2 weeks without a subsequent sprain. However, long-term goals following an Achilles' tendon rupture may include multiple "milestones" such as walking without support, jogging with no pain or tightness, and eventually returning to full competition with no subsequent complications. Depending on the level of competition and the point in an athlete's career, the final long-term goal may or may not be to return to the same level of competition as soon as safely possible, but that will likely be the long-term goal of emphasis.

# 22.3 Early Phases of Rehabilitation and Treatment

Early phases of rehabilitation are inevitably always focused around aiding the body in its natural healing process. Injuries can be broken into two broad categories, including macro-trauma and microtrauma. Macro-trauma is often associated with a specific event, of which can be identified as the mechanism of injury and this is considered an acute injury. Chronic injury is a cumulative process that results from multiple microtraumas over time, and the mechanism of injury cannot be attributed to a specific event.

Regardless of the type of trauma, the body will undergo a cascade of events at the cellular level, of which the primary purpose of this process is the restoration of normal tissue function. This healing process is chemically mediated and an influx of specialized cells to the site of injury will occur to aid in the removal of damaged tissue and to repair or regenerate new tissue. This process is known as the inflammatory response phase and is characterized by the clinical signs and symptoms of redness, pain, swelling, increased temperature, and decreased function [1].

The signs and symptoms of the inflammatory response are associated with an alteration in the local rate of metabolism in response to injury. Rehabilitation during this period should focus on protecting the injured area as to do no further harm, minimize the extent of inflammation, and decrease the level of pain the athlete is experiencing. The duration and extent of the inflammatory response will be specific to the type of tissue injured, severity of the injury, and many other intrinsic factors that the clinician will be unable to control for. Therefore, it is important to focus on the modifiable aspects such as protecting the site of injury, minimizing inflammation, and reducing pain. Successful management during the inflammatory response phase is crucial to the overall success of the rehabilitation process.

#### 22.3.1 Protection

Protection of the injury is vital as to prevent further damage to the injured tissue itself or to surrounding tissues in the presence of a comprised joint. Casting, walking boots, or braces are often used to immobilize, restrict range of motion, or provide mechanical support. External support is often required in conjunction with activity modification or rest. A key component of protecting the injured tissue is restricting the level of activity to only those activities that are safe for the stage of healing that the damaged tissue is presently in.

The most common method of activity modification for protection of the foot and ankle when weight-bearing ambulation is contraindicated is crutch walking. Weight bearing should only be performed when pain, range of motion, flexibility, and muscle function are adequate to allow for a normal gait pattern. Antalgic gait patterns often lead to compensatory strategies that may have long-term consequences and predispose athletes to further injury.

#### 22.3.1.1 Non-weight Bearing

Non-weight bearing is indicated when it is unsafe to place any load on the injured foot and ankle. During this stage, two crutches should be used to aid in ambulation. When it becomes safe to apply minimal loading to the injured limb but residual swelling or pain limits a full range of motion during gait, non-weight bearing is often progressed to toe-touch weight bearing.

#### 22.3.1.2 Toe-touch Weight Bearing

Toe-touch weight-bearing stage of ambulation is also performed with two crutches, and this stage permits the athlete to touch the toes of their injured limb on the ground while supporting the majority of their weight on the crutches. When residual swelling and/or pain no longer restrict range of motion during gait, a progression from toe-touch weight bearing to partial weight bearing can be initiated.

#### 22.3.1.3 Partial Weight Bearing

During partial weight bearing, the injured athlete will initially use two crutches to support a portion of their body weight while allowing the injured limb to support the remaining body weight. During this stage, the athlete should be maintaining a normal gait pattern with each step, and this should dictate how much support is required to be placed on the crutches and subsequently how much weight the injured limb can support. When the athlete is able to support the majority of their body weight while maintaining a normal gait pattern, but the athlete still requires minimal support or stability, a progression from two crutches to a single crutch or cane can occur. During this transition, the cane or single crutch will be placed on the contralateral side of the injury and will move concurrently with the injured limb. Finally, a progression to full weight bearing is indicated when the athlete is able to demonstrate a normal gait pattern without external support.

#### 22.3.1.4 Walking Boot

Utilization of a walking boot is often supplemented for crutch use with injuries that would require prolonged or extensive crutch use. Allowing the athlete to ambulate in the walking boot provides adequate protection of foot and ankle injuries while still allowing for the physiologic benefits of bearing weight and joint loading.

#### 22.3.1.5 Benefits of Bearing Weight

Bones, articular surfaces, ligaments, muscles, and tendons may all undergo deleterious effects during prolonged periods of immobilization or non-weight bearing. Partial weight bearing with crutches or walking boots can retard muscle atrophy and soft-tissue contractures, maintain bone mineral density, and facilitate movement of joint synovial fluid. Additionally, systematically loading tissue during the phases of healing will allow the tissue to respond linearly with the loads placed on it [1].

# 22.3.2 Minimize Inflammation

The acronym RICE is often used in conjunction with protection to remind athletes and clinicians of effective methods at minimizing inflammation in the acute phases of rehabilitation. RICE represents the concepts rest, ice, compression, and elevation.

Rest is a method of protection, as limiting activity will prevent further injury. However, rest also plays an important role in minimizing the inflammatory response, as inappropriately stressing the damaged tissue can exacerbate the inflammatory response, increase degradative activity, and reduce cell synthesis [1].

Lowering tissue temperature for therapeutic effects is often referred to as cryotherapy. Ice is a physical modality that is used to slow the rate of cellular metabolism at the site of injury. The reduction in the metabolic process is believed to reduce the demand for oxygen in tissues that are in a hypoxic state due to increased hydrostatic pressure as a result of inflammation [2]. Reducing the amount of secondary cell death via hypoxia will decrease the overall extent of the injury and allow for a quicker progression to active phases of rehabilitation [2]. While the current evidence on treatment recommendations for cryotherapy is not conclusive, repeated application (three times) of ice for 10 min per application with 10 min rest will allow for the benefits of ice without causing damage to superficial tissues due to excessive cooling of skin and superficial nerves [3].

Compression is the application of an external force to the area where swelling is likely to or has already accumulated. The mechanical pressure is believed to decrease the available space for swelling to accumulate in the tissues adjacent to the site of injury. The most common form of compression is an elastic bandage that can be used for slight external support as well as continuous compression throughout the day when the athlete is in a gravity-dependent position. Intermittent compression with inflatable cuffs may also be used in an elevated position to systematically increase and decrease pressure as to allow the circulatory system during periods of low pressure to remove swelling as the pressure increases.

Elevation is important during periods of rest as the foot and ankle are in the most gravity-dependent position in the body when sitting or during ambulation. Allowing gravity to work in conjunction with compression may be beneficial at reducing swelling, but benefits may be negated once gravity-dependent positions are resumed [4].

## 22.3.3 Pain Reduction

Pain should be considered as a natural form of protection to inhibit unnecessary or unsafe tissue loading during the rehabilitation process. Clinicians should be cautious when prescribing rehabilitative exercises when pain has been artificially reduced by analgesic medications or following periods of cryotherapy application. With that in mind, minimizing and controlling pain are almost always required at some point through the various phases of healing, and many methods of pain reduction are available for clinicians. Pain will often dictate progress during rehabilitation, and modulating pain safely and effectively will allow efficient achievement of short-term goals.

Cryotherapy, thermotherapy, electrical modalities, and manual therapy techniques are common methods used to modulate pain during rehabilitation. During acute phases when the inflammatory response is active, cryotherapy serves a dual purpose in slowing cell metabolism and modulating pain by slowing nerve conduction velocity and providing a counterirritant to the noxious stimulus. Electrical currents such as TENS may also be used throughout the rehabilitation phase to modulate pain via gate control and noxious pain theories. Thermotherapy is often used at later stages of rehabilitation to address chronic pain or discomfort when dual benefits of cryotherapy are not desired. Grade I and II joint mobilizations are often used as analgesic treatments for pain associated with movement.

Addressing pain with pain relievers, NSAIDs, or other pharmacological agents should be prescribed on an individual basis. However, communication between the team physician, athlete, and athletic trainer is imperative to ensure the progression of rehabilitation is in accordance with tissue integrity and not the pharmacological reduction or masking of pain.

### 22.4 Basic Neuromuscular Control

Effective neuromuscular control requires the integration of sensory information into motor output. Vestibular, visual, and somatosensory information are all required for coordinated movement during football. However, following an ankle injury the somatosensory system is the only sensory system that will likely be compromised. Somatosensory information can come from cutaneous receptors in the skin and mechanoreceptors in joint capsules, ligaments, muscles, and tendons. The somatosensory information can result in reflexive activation of muscle or provide proprioceptive information that can aid the body in coordinated movements.

Following an injury, immobilization, pain, and inflammation all contribute to altered neuromuscular control. Initial restoration of basic neuromuscular function may require conscious activation of muscle during electrically induced contractions with currents such as neuromuscular (NMES) or Russian electrical stimulation. Joint repositioning and proprioceptive neuromuscular facilitation (PNF) exercises are also effective methods of restoring basic neuromuscular control. More functional neuromuscular control exercises, as described in Sect. 22.7, should only be performed once range of motion and muscle function is adequate to perform the exercises safely.

#### 22.5 Range of Motion and Flexibility

Restoration of normal range of motion and flexibility are important and measurable short-term goals that will be required for normal ambulation and the progression back to the pitch. A decrease in range of motion to some degree will accompany almost any injury that elicited the symptoms of pain and swelling. Many other factors can contribute to a loss of motion including muscle tightness, arthrokinematic impairments, and adverse neural tension.

Arthrokinematic motions are small amplitude motions that occur at the joint interface between adjacent bones. Arthrokinematic motions help facilitate the gross movements known as osteokinematic motions. Basic osteokinematic motions of the ankle include plantar flexion, dorsiflexion, inversion, and eversion. Pronation and supination are more complex movements of the foot and ankle that aid in shock absorption and propulsion during gait, respectively.

To restore normal ranges of motion, it is important to identify the restriction responsible for the functional limitation. If the limitation is a symptom of the inflammatory response phase such as pain or swelling, addressing the pain or swelling with ice and/or compression and elevation should be the first step in restoring normal joint motion. Joint mobilizations, stretching, and other manual therapy techniques may be beneficial in addressing mechanical sources of range of motion deficits. A thorough bilateral assessment of arthrokinematic and osteokinematic motion, as well as muscular flexibility and neuro-mobility, will help the clinician differentiate between specific mechanical restrictions in range of motion. Grade III-V joint mobilizations may facilitate increases in range of motion associated with arthrokinematic impairments. Posterior talar glides are effective for dorsiflexion deficits associated with arthrokinematic impairments of the talocrural joint (Fig. 22.2). Following joint mobilizations, therapeutic exercises such as muscle stretching and strengthening should be performed to maintain functional improvements of the manual therapy techniques. Isolated soleus and gastrocnemius stretching are common stretching techniques used to restore dorsiflexion deficits due to calf muscle tightness (Fig. 22.3). Neuromobilizations for the sciatic nerve can address adverse neurodynamics of the tibial and deep and superficial peroneal nerves. Towel crunches, mini squats, and stationary bicycling are all beneficial at increasing range of motion deficits associated with residual swelling.

#### 22.6 Muscular Strength, Power, and Endurance

Restoring muscle function of the entire lower extremity is a key component in a rehabilitation program of foot and ankle injuries. Following immobilization or periods of decreased activity, muscle atrophy can occur and must be restored before



Fig. 22.2 Posterior talar glides with movement (*top images*) and posterior talar glides (*bottom images*)



Fig. 22.3 Gastrocnemius (*left images* – knee extended) and soleus (*right images* – knee flexed) stretching

more functional rehabilitation can be performed. Muscle strength, power, and endurance are all important characteristics that need to be addressed in the rehabilitation of football athletes.

During periods of immobilization or when the joint is unable to generate force over the entire ROM, isometric exercises are performed to decrease the extent of atrophy that may occur. Isometric exercises, however, are only able to facilitate strength gains at a small range of joint angles around the angle of which the



Fig. 22.4 Manual (*top images*) and thera-band (*bottom images*) resistance exercises for inversion, eversion, plantar flexion, and dorsiflexion (*left to right*)



**Fig. 22.5** Intrinsic foot muscle rehabilitation: marble pickups (*top images*), towel crunches and towel slides (*middle images, left to right*), short foot exercises (*bottom images*)

isometric exercise was performed. Progressive resistive exercise is the most common method utilized in rehabilitation programs to restore strength through the full ROM. Concentric and eccentric exercises can be performed and the intensity progressively increases linearly with strength gains. Thera-bands and tubing, weights, and manual resistance are all effective methods of applying resistance during progressive resistance exercises for the foot and ankle (Fig. 22.4). Towel crunches, towel slides, and short foot exercises should also be performed to strengthen the intrinsic foot musculature (Fig. 22.5). Heel and toe walks are great weight-bearing exercises for restoring strength and neuromuscular control (Fig. 22.6).

Strength and endurance gains will initially improve concurrently as increases in neuromuscular control are observed [5]. However, the intensity and volume of the progressive resistive exercises can be modified to address endurance or strength in isolation as



Fig. 22.6 Toe (top images) and heel (bottom images) walks

well. When strength and endurance have been restored in all planes of motion, plyometric exercises are useful in the rehabilitation setting to aid in restoring muscular power. For all exercises, the clinician should monitor the quality of movement, and proper technique should never be sacrificed to increase the volume performed.

Muscle tissue is comprised of fast-twitch and slow-twitch muscle fibers. Fasttwitch muscle fibers are used to generate large amounts of force for short periods of time, and slow-twitch fibers are used to generate less force for greater periods of time. These two muscle fiber types collectively allow muscles to have the properties of strength and endurance. Muscle fibers will respond and adapt only when the individual muscle fibers have been systematically overloaded. The overload principle states that for an adaptation to occur, a load greater than normal loading must be placed on a tissue during training or rehabilitation. Therefore, if the desire is to overload fast-twitch muscle fibers for strength gains, 8-10 repetitions for 3 sets at 80 % of an athlete's 1 rep max will accomplish this goal. Additionally, if the desire is to overload slow-twitch muscle fibers for muscular endurance gains, 15-20 repetitions for 3 sets at 40-60 % of an athlete's 1 rep max may be required. It is important to note that the specific intensity and number of repetitions and sets should be tailored to the specific goal of the exercise and the requirement of the athlete's position in football. Generally, an emphasis on lower weight and higher repetitions will facilitate gains in muscular endurance whereas lower repetitions with heavier weight will facilitate strength gains [5].

# 22.7 Functional Neuromuscular Control: Postural Stability and Balance

Static and dynamic balance requires the integration of sensory information about postural sway into reflexive and corrective muscle recruitment. Therefore, basic neuromuscular control must be adequate, and muscle strength, power, and



Fig. 22.7 Dynamic balance exercises. (*Top images*) Y-balance. (*Bottom images*) star excursion balance test/exercise

endurance must be great enough as to not result in injury during postural stability rehabilitation. However, it is not necessary to have fully restored muscle function to pre-injury status before beginning controlled static balance exercises.

Static balance tasks are those in which individuals are attempting to maintain balance on a single limb or both limbs without any functional adjuncts. Progression to dynamic balance is acceptable when the athlete has mastered all static balance conditions with various constraints such as eyes closed or foam pad conditions. Dynamic balance often incorporates a similar stance to static balance but functional components are added. Single leg balance with a maximal reach in various directions with the contralateral limb is a common dynamic balance task (Fig. 22.7). For the football athlete, a sport-specific dynamic balance task may be a single limb balance trial while volleying a ball back to a partner with the contralateral limb or heading the ball back while in the same single limb stance balance position (Fig. 22.8). As the athlete progresses, a common dynamic balance task that is also a useful functional rehabilitation exercise is a hop to stabilization (Fig. 22.9). This task requires the athlete to hop in various directions as far as possible and land on a single limb, while attempting to regain postural control as quickly as possible after landing. In this task, the hopping distance will be progressively increased when the athlete is able to successfully regain balance at shorter hopping distances. Hopping



Fig. 22.8 Balance progression: firm  $\rightarrow$  foam  $\rightarrow$  foam volley  $\rightarrow$  foam header

can be performed in anterior/posterior, medial/lateral, anteromedial/posterolateral, and posteromedial/anterolateral directions. The athlete will jump from the start position to the target position, regain balance as quickly as possible, and then complete the repetition by jumping back to the start position and subsequently regaining their balance in the start position for repetition number 2. Each series of directions should be successfully completed ten consecutive repetitions prior to increasing the distance or adding an unstable surface to land on [6].

## 22.8 Functional Rehabilitation

Functional rehabilitation is a gradual process that allows the athlete to master isolated functional tasks and slowly progress into sport-specific exercises prior to being evaluated for their ability to return to sport. A functional progression will almost always include basic exercises such as lunges, hopping, running, cutting, and other universal functional exercises. However, specific to the football athlete, the functional progression must also include sport-specific aspects of football that will be encountered when returning to the pitch. Shooting, passing, juggling, tackling, heading, and other sport-specific activities will have to be mastered in controlled situations to ensure the athlete is fully prepared to complete these tasks in a



Fig. 22.9 Hop to stabilization task: anterior/posterior hop direction, medial/lateral hop direction, anteromedial/posterolateral hop direction, and respective directions for foam pad placement (descending order of images)

competitive environment. It is beneficial during this stage for the clinician to communicate with the coaches and athlete about the demands of football specific to the athlete's position.

# 22.9 Maintaining Cardiovascular Fitness

Depending on the position of the injured player, football players can run greater than 7 miles per match [7]. However, the game of football requires periods of maximal sprinting, slower recovery runs, and short periods of rest. Therefore, the athlete must maintain cardiovascular fitness specific to the demands of football while recovering from an injury or the return to play process will be much longer.

When dealing with a foot and ankle injury, cardiovascular fitness becomes more challenging, especially when weight-bearing tasks are not appropriate during certain phases of tissue healing. Upper body ergometers, swimming, rowing, or other upper body intensive exercises can be good ways to maintain cardiovascular fitness, but ultimately, the goal should be to include as much lower body cardiovascular conditioning as possible. Once weight-bearing tasks are appropriate, cycling, elliptical training, and eventually running will provide the most sport-specific retraining for the football athlete. When restoring cardiovascular fitness for football players, one should consider the position of the athlete and the type of fitness required to compete. Completing interval training with periods of high-intensity sprinting and shorter periods of rest can be performed on nearly any cardiovascular exercise machine and will likely be the most sport-specific cardiovascular training for these athletes [7]. An example of an effective sprint interval training program for football athletes would include four sets of 4 min of sprinting (90-95 % of maximal heart rate) followed by 3 min of jogging (50–60 % of heart rate max) 2–3 times per week for 8 weeks [8].

### 22.10 Functional Testing and Return to Play Considerations

The rehabilitation process is broken up by the short-term goals that aim to restore ROM and flexibility, neuromuscular control, muscle function, and postural stability while the injured tissue is healing. During the healing process, our intention is to apply specific loads to the tissue during each phase of healing that will stress the tissue enough for positive adaptations to occur but ensure loads are not too great as to increase the extent of tissue damage. This philosophy follows the SAID principle, which represents the idea of specific adaptations to imposed demands. Throughout the entire rehabilitation process, we place various demands on the bones, ligaments, muscles, tendons, and nervous system to ensure the injured player adapts in a way that prepares them to return to the pitch.

When assessing an athlete's ability to return, all functional limitations as a result of the injury must be restored, cardiovascular fitness should be equal to or greater than pre-injury status, and there should be no apprehension from the player of other members of the sports medicine team about the athlete's safety. The final assessment should include all areas of previous functional limitations as described above.

- Is range of motion and flexibility normal when compared bilaterally?
- Is neuromuscular control efficient and effective as to prevent injury and perform at a competitive level?
- Has muscle strength, power, and endurance been restored to pre-injury status?
- Has the athlete successfully completed all static and dynamic balance trials as well as a complete a rigorous functional progression?
- Does any member of the sports medicine team or the athlete have any apprehension about returning the athlete to competition?
The return to play process itself will often be progressive as well. For the football athlete, early stages may include completing drills with the team that do not include contact, full intensity sprinting, or shooting with the injured limb. Progressing into short-sided low-intensity matches with the team is a great way to build confidence for more competitive situations. Finally, returning to the pitch in a competitive environment will achieve the long-term goal of emphasis that is more often than not to return to play at or above pre-injury status. However, as stated previously, the rehabilitation process should never abruptly stop once the athlete returns to the pitch. Occasionally, deficits will be erroneously overlooked during the return to play evaluation, as some deficits may only be present after the athlete has been thoroughly fatigued. Continuing functional and sport-specific rehabilitation will help to minimize the risk of the clinically undetectable residual limitations the athlete may still have.

# 22.11 Prophylactic Support

During the early phases of return to play, the use of external support may help facilitate a smooth transition and decrease the risk of reinjury. Ankle bracing and taping are the most commonly used prophylactic supports used following ankle injuries. Both have been shown to be effective at reducing the risk of injury [9–12]. However, comfort and functionality are key concerns when employing prophylactic support for football players, as the intervention will only be effective if adherence is adequate. Many football players are unwilling to wear anything more than what is required during participation, in hopes to maximize their "touch" on the ball. Poor adherence to prophylactic mechanical support in the football athlete makes a comprehensive rehabilitation process and a rigorous return to play evaluation that much more important.

### 22.12 Chapter Summary

The rehabilitation process for foot and ankle injuries requires a multidisciplinary and comprehensive approach by a knowledgeable sports medicine team. Setting short-term goals will provide a checklist to follow that will ensure all functional limitations are addressed systematically during the rehabilitation process and longterm goals will keep the sports medicine team focused on the best possible outcome for the injured athlete. After functional limitations are addressed, a comprehensive and sport-specific return to play progression should be performed. Following a rigorous functional progression, the final return to play decision should be made only when all members of the sports medicine team conclusively determine participation is safe and reinjury is no longer a concern.

# References

- Lee C (2007) Injury, inflammation, and repair: tissue mechanics, the healing process, and their impact on the musculoskeletal system. In: Magee D, Zachazewski J, Quillen W (eds) Scientific foundations and principles of practice in musculoskeletal rehabilitation. Saunders Elsevier, St. Louis, pp 1–22
- Swenson C, Swärd L, Karlsson J (1996) Cryotherapy in sports medicine. Scand J Med Sci Sports 6(4):193–200
- 3. Mac Auley DC (2001) Ice therapy: how good is the evidence? Int J Sports Med 22(05):379-384
- Tsang KK, Hertel J, Denegar CR (2003) Volume decreases after elevation and intermittent compression of postacute ankle sprains are negated by gravity-dependent positioning. J Athl Train 38(4):320
- Anderson T, Kearney JT (1982) Effects of three resistance training programs on muscular strength and absolute and relative endurance. Res Q Exerc Sport 53(1):1–7
- Mckeon P, Ingersoll C, Kerrigan DC, Saliba E, Bennett B, Hertel J (2008) Balance training improves function and postural control in those with chronic ankle instability. Med Sci Sports Exerc 40(10):1810
- Rampinini E, Bishop D, Marcora S, Ferrari Bravo D, Sassi R, Impellizzeri F (2007) Validity of simple field tests as indicators of match-related physical performance in top-level professional soccer players. Int J Sports Med 28(3):228
- Helgerud J, Engen LC, Wisloff U, Hoff J (2001) Aerobic endurance training improves soccer performance. Med Sci Sports Exerc 33(11):1925–1931
- 9. Verhagen E, Bay K (2010) Optimising ankle sprain prevention: a critical review and practical appraisal of the literature. Br J Sports Med 44(15):1082–1088
- 10. Olmsted LC, Vela LI, Denegar CR, Hertel J (2004) Prophylactic ankle taping and bracing: a numbers-needed-to-treat and cost-benefit analysis. J Athl Train 39(1):95
- 11. McGuine TA, Hetzel S, Wilson J, Brooks A (2012) The effect of lace-up ankle braces on injury rates in high school football players. Am J Sports Med 40(1):49–57
- 12. McGuine TA, Brooks A, Hetzel S (2011) The effect of lace-up ankle braces on injury rates in high school basketball players. Am J Sports Med 39(9):1840–1848

# **Chapter 23 Taping Techniques and Braces in Football**

Ruben Zwiers, Leendert Blankevoort, Chris W.A. Swier, A. Claire M. Verheul, and Gino M.M.J. Kerkhoffs

If you do not believe you can do it then you have no chance at all.

Arsene Wenger

**Abstract** The aim of the chapter is to give an overview of the different taping techniques and types of braces used in football, conjunct with the evidence in literature of their use as treatment and prevention of ankle ligament injuries. The prevention and treatment of ankle ligament lesions are extensively discussed in the Chapters "Prevention of ankle ligament lesions" and "Treatment of acute ankle ligament lesions".

Keywords Ankle • Taping • Bracing • Prevention • Treatment • Sprain

# 23.1 Role of Taping and Braces in Football

Lateral ligament injury of the ankle is the most common injury in football. Taping is frequently used in football players during the recovery from lateral ligament injuries of the ankle or preventing first-time or recurrent sprains. Often the choice of taping technique depends on the personal preference of the athlete. Ankle braces have been developed as an alternative to ankle taping. The numerous different ankle braces that have been designed to prevent ankle sprains can be divided in three groups: soft braces, semi-rigid and rigid braces.

University of Amsterdam, 22700, Amsterdam 1100 DE, The Netherlands e-mail: r.zwiers@amc.uva.nl; g.m.kerkhoffs@amc.uva.nl

R. Zwiers (🖂) • L. Blankevoort • G.M.M.J. Kerkhoffs, MD, PhD

Department of Orthopaedic Surgery, Academic Medical Center,

C.W.A. Swier • A.C.M. Verheul Manual Fysion, Burgerweeshuispad 54, Amsterdam 1076 EP, The Netherlands

Since in football the foot and ankle are directly involved in shooting and passing, it is more likely that a tape or a brace used as preventive measure could influence performance than in other sports. The question is whether the benefits of tape or brace outweigh the possible impairments in performance.

### 23.2 Taping

Numerous taping techniques were described over time. The choice is often governed by personal preference and the experience of the person applying the tape. In 1885 Virgil Gibney described his basket weave technique using strips of adhesive plaster as a treatment for ankle sprains [1]. Later Galland described a technique based on the technique by Gibney, augmented by additional oblique strips of tape with the aim of limiting lateral movements of the calcaneus into either supination or pronation [2]. His argument limiting lateral calcaneal motion would reduce the strain on the injured calcaneofibular or calcaneotibial ligaments.

Ever since, numerous taping techniques are used in literature. However, almost every contemporary ankle-taping procedure incorporates the basic components of the procedure described by Gibney. Due to the lack of comparative studies between different taping techniques, no technique has been proven to be superior to one other. The choice of tape is often governed by personal preference and the experience of the person applying the tape.

### 23.2.1 Taping Methods

An overview is provided of the most commonly used taping techniques used in football players. All techniques are composed of variations of several basic taping techniques.

#### 23.2.1.1 Basic Tape Elements

A taping technique consists of a number of taping elements, i.e. anchor, stirrup, spur, figure of 6, full heel lock, lock off, heel sling, figure of 8 and subtalar sling.

• Anchor

An anchor is used as fixation point for the other tapes. In the basic ankle-taping technique, the proximal anchor is placed around the lower leg, below the bellies of the calf muscles. To prevent circulatory problems the tape has to be applied gently. The distal anchor is placed around the midfoot. Caution is required regarding the head of the fifth metatarsal bone. To prevent irritation the tape has to be applied proximal of the metatarsal head or by starting at the plantar side of the foot (Fig. 23.1).



Fig. 23.1 Anchors. (a) The medial view of the proximal and distal anchors. (b) Anterior view of the proximal anchor



Fig. 23.2 Stirrup and spur. (a) Medial view of the first stirrup. (b) Medial view of the combination of the first stirrup and spur

• Stirrup

A stirrup is a vertical U-shaped strip supporting both sides of the ankle. With the foot in neutral position, the tape is started at the medial side of the ankle at the level of the proximal anchor. The tape is directed behind the medial malleolus, under the foot behind the lateral malleolus concluding at the posterolateral aspect of the anchor. Rationale for starting medially is the preference to tape the ankle in slight eversion rather than in inversion (Fig. 23.2a).

• Spur

A spur is a horizontal U-shaped strip, starting laterally along the fifth metatarsal bone, directing posterior behind the calcaneus, anterior to the first metatarsal bone (Fig. 23.2b).



**Fig. 23.3** Figure of 6. (a) The first step of a figure of 6, at the medial side of the proximal anchor. (b) The second step, a cross over the foot sole in lateral direction. (c) The third step, the return to the medial malleolus. (d) Medial view of a figure of 6. (e) Lateral view of a figure of 6

• Figure of 6

The figure-of-6 tape starts at the medial side of the ankle at the level of the anchor the tape and follows the course of the stirrups. When reaching the lateral side of the ankle, the tape is directed over the dorsum of the foot concluding just above the medial malleolus (Fig. 23.3). *Reverse Figure of 6* 

The reverse figure-of-6 tape is opposite to the figure of 6, start and finish are lateral.



Fig. 23.4 Heel sling. (a) The start of the heel sling anterolateral. (b) The final step. (c) Medial view of the heel sling. (d) Posterior view of the heel sling

• *Heel Sling (Lateral)* 

The heel sling starts anterolateral at the level of the proximal anchor. It directs posteriorly over the Achilles tendon, obliquely crossing the lateral side of the calcaneus, under the plantar surface of the foot towards the starting point at the medial side (Fig. 23.4).

• Full Heel Lock

The full heel lock is a combination of a medial and lateral heel sling. It starts anterolateral at the level of the proximal anchor with the lateral heel sling. The lateral heel sling ends at the dorsal site of the foot. The tape then directs medially under the plantar surface of the foot, obliquely crossing the calcaneus at the medial side and continues posteriorly around the calcaneus to end at the anterior joint line (Fig. 23.5).

• Lock Off

A lock off is a tape strip superimposing the strip of a stirrup, heel spur and other functional strips.



**Fig. 23.5** Heel lock. (a) The first step of the heel lock. (b) The second step, the return to the starting point. (c) The final step, locking of the medial side of the heel. (d) Medial view of the heel lock

• Figure of 8

The figure-of-8 tape starts at the dorsal side of the foot and runs medially around the sole of the foot back to the dorsum. It proceeds around the posterior side of the leg back to the starting point (Fig. 23.6).

• Subtalar Sling

The subtalar sling starts at the plantar surface of the forefoot; runs over the lateral border of the foot, over the lateral malleolus; and is wrapped around the leg above the malleoli (Fig. 23.7).

# 23.2.1.2 Commonly Used Techniques

```
• Closed Basket Weave Technique
The original basket weave taping technique consists of anchors, stirrups, spurs, (reverse) figure of 6s, heel lock or figure of 8s and lock offs (Fig. 23.8).
```

```
• Open Basket Weave Technique
In acute ankle sprains where severe oedema is present, an open basket weave
technique is advised. It provides support and compression on the lateral and
medial side whilst allowing room for expanding oedema. This tape configuration
may be useful in the first 48 h. There is no evidence to substantiate this claim.
```



**Fig. 23.6** Figure of 8. (a) The start of the figure of 8 at the dorsum of the foot. (b) The second step, directing the tape posteriorly. (c) The third step, the return to the dorsal side of the foot. (d) Medial view of the figure of 8. (e) Dorsal view of the figure of 8

• Hinton-Boswell

This taping method is an adjustment on the basket weave technique. The anchors and stirrups are applied in dorsiflexion. Then with the ankle in a relaxed plantar flexion, the remaining techniques, four overlapping figure of 6, are applied. The tape is finished with circular strips.

• Anti-inversion Taping (Many Variations) A large number of taping methods for the ankle has been described. The methods aiming to prevent inversion trauma are often adaptations of the basket weave technique by adding or omitting one or more of the basic tape elements. The



Fig. 23.7 Subtalar sling. (a) The first step of the subtalar sling, obliquely crossing the lateral side of the ankle. (b) The lateral view of the subtalar sling



Fig. 23.8 Basket weave technique. (a) The double anchors and the first stirrup. (b) The combination of the first stirrup and spur. (c) The situation after the alternated application of the stirrups and spurs (d) Medial view of the basket weave technique

combination of techniques that is used often depends on the preferences of the player or experiences of the therapist.

The lack of comparative studies between the different taping techniques makes that the choice of technique mainly is based on experience and preference.

Rarick et al. compared basket weave technique with and without heel lock and concluded favoured a basket weave with stirrup and heel lock to be more supportive [3]. Frankeny et al. performed a biomechanical analysis of taping methods and concluded that the Hinton-Boswell method provided greatest resistance to inversion [4].

Metcalfe et al. compared closed basket weave with heel locks and figure of 8, reinforced with moleskin tape to a Swede-O universal brace and found no difference between the three methods in terms of talocrural and subtalar range of motion [5]. Vaes et al. performed a radiological study to evaluate the effectiveness of the closed basket weave taping technique with no heel lock. They found that the basket weave was superior to elastic adhesive bandage in reducing talar tilt when an inversion load was applied to the foot [6].

#### 23.2.2 Type of Tape

#### 23.2.2.1 Elastic Adhesive Tape

Elastic adhesive tape follows easily the contours of the ankle and allows soft tissue to expand. Hence, this type of tape is useful for compression and support of the soft tissue in the acute phase. It will not give mechanical support to ligaments but can be an addition to non-elastic tape, for example, to apply the proximal anchor, allowing the calf muscles to expand.

#### 23.2.2.2 Non-elastic Adhesive Tape

Non-elastic adhesive tape is used to support structures like ligaments or joint capsule. It is generally thought to limit joint movement and enhance propriocepsis more than elastic tape. The most common type of tape used is the non-elastic zincoxide tape.

Care must be taken when non-elastic tape is used, especially when the technique involves applying the tape circumferentially around a limb. If the tape is applied too tightly, the risk of impeding blood flow is increased. This could lead to tissue damage and even tissue necrosis. At best, comfort and performance could be detrimentally affected by taping that is too restrictive.

To avoid these potential problems, an alternative taping technique using elastic adhesive bandaging has been developed. The theory is that the elastic tape not only provides adequate support but also allows functional movement without restricting blood flow to and from the tissues distal to the taping, but at the cost of mechanical effectiveness. Tregouet et al. showed non-elastic tape to reduce the inversion velocity more than elastic tape [7]. However, Abian-Vicen et al. recommend the use of elastic tape as the first choice because it is perceived as more comfortable and less restrictive by the users, whilst it produces the same restriction in the ROM as non-elastic tape [8].

Since there are several types of tape with varying degree of elasticity and no type of tape is proven to be superior to one other, the choice of tape should be made based on preference of the player.

### 23.2.2.3 Prewrap

Prewrap is a thin foam-like material. In players which use tape frequently, often a layer of prewrap is applied prior to application of the tape. Others may prefer to shave the skin around the ankle so the tape is applied directly on the skin. The use of prewrap under the tape seems to be as effective as applying the tape directly to the skin.

### 23.2.2.4 Hypoallergenic Tape

In case of an allergy for zinc oxide tape, a hypoallergenic underlay tape can be used under the conventional adhesive tape. This tape is generally manufactured from a stretchable non-woven tape with a breathable porous adhesive material.

#### 23.2.2.5 Cohesive Bandages

Cohesive bandages may be useful instead of elastic tape. The product sticks to itself and not to the skin, is waterproof and is reusable. However, no literature about its use is yet available.

# 23.3 Bracing

Multiple types of braces are developed over the years. These braces are manufactured of various materials and therefore providing varying amounts of support and stability. Broadly these braces are divided in three types: soft braces, semi-rigid and rigid braces. This classification is not strict nor generally accepted and may vary between studies, making it hard to compare the types of braces. Generally, the soft and semi-rigid braces are suitable for use during sports. A rigid brace is appropriate for use in the treatment of an acute lateral ankle ligament lesion or a stable ankle fracture.

# 23.3.1 Type of Braces

#### 23.3.1.1 Soft Braces

Soft braces are characterised by the absence of rigid or stiffening elements. They are fully composed of textile material.

#### **Compression Stocking**

A compression stocking is generally used in aiding the treatment of mild ankle sprains and tendinitis. It does not provide any mechanical support to the ankle. It consists of a light weight stretchable material and provides the ankle joint only with compression. This type of brace does not limit ankle range of motion nor does it constrain inversion or eversion (Fig. 23.9a).



Fig. 23.9 Soft Braces. (a) Compression stocking. (b) Soft brace with elastic strap



Fig. 23.10 Semi-rigid braces. (a) Lace-up brace. (b) Semi-rigid brace with elastic straps

#### Soft Brace with Elastic or Non-elastic Velcro Straps

This brace is characterised by straps that are applied such as to simulate tape bandage. The straps are usually crossing anterior of the ankle in the shape of a half or full figure-of-8 configuration. There is moderate support, characterised by some constraint to inversion, sometimes combined with eversion constraint, and some constraint to plantar flexion (Fig. 23.9b).

#### 23.3.1.2 Semi-rigid

Semi-rigid braces are characterised by one or more semi-rigid shells or other similarly stiff elements, lateral or lateral and medial of the ankle and connected by a flexible stirrup, either under the heel and/or under the foot. These braces are used for mild to moderate ankle sprains and as a measure to prevent recurrent ankle sprains.

#### Lace-Up Brace

Historically, the lace-up brace is a common type of semi-rigid ankle brace. It is designed to limit the ankle from inversion-eversion as well as providing some constraint to plantar flexion and dorsiflexion (Fig. 23.10a).

#### Brace with Elastic or Non-elastic Velcro Straps

Contrary to the lace-up brace, this type of brace is fixated by using elastic or nonelastic straps. Straps are used to fix the brace to the shank. Crossed straps, or figure-of-6 or figure-of-8 straps anterior of the ankle are applied to provide for inversion and/or eversion stability, thereby also constraining to some extent plantar flexion (Fig. 23.10b).

#### 23.3.1.3 Rigid Braces

Rigid braces are characterised by rigid shells enveloping the shank and hindfoot. The main use is in the early acute phase of an ankle injury and not meant to be worn during sports activities. The rigid brace provides more side-to-side support than a semi-rigid brace. The full rigid brace also constrains foot flexion. It may be difficult to fit in some shoes. After the acute phase, a more functional lace-up brace or semi-rigid brace is recommended.

Rigid Stirrup Ankle Brace

The rigid stirrup ankle brace is perhaps the most widely used brace in sports medicine because of the effectiveness and versatility in treating ankle sprains. They are made of two plastic shells that cover both sides of the ankle, connected by a stirrup under the heel and are strapped into place with easily adjustable Velcro straps (Fig. 23.11a).

Rigid Brace with Hinge Connection Between Shank and Hindfoot

Consisting of rigid shells for the shank and hindfoot that are connected by a hinge, this brace prevents inversion-eversion, whilst it does not affect the plantar-dorsiflexion range of motion (Fig. 23.11b).

*Rigid Brace Consisting of a Unilateral Shell Covering the Shank and Hindfoot* The rigid brace covering the shank and hindfoot on one side is fully constraining the motions of the hindfoot. It can be considered as an alternative to a splint, primarily used in cases of acute ligament injuries or of stable ankle fractures (Fig. 23.11c).

Rigid Brace in One Piece Enveloping the Shank, Ankle and Foot

If supplied with a foot sole to walk on, the rigid brace that is fully enveloping the shank, ankle and foot is also called an ankle walker. It is the alternative to a full circumferential cast, mainly used for treating ankle fractures (Fig. 23.11d).

Many comparative studies have evaluated the efficacy of the different types of braces on range of motion, functional performance and neuromuscular effects. Most studies compare one or two ankle supports. Since a wide variety of braces is developed, it is hard to draw conclusions on which brace is superior to one other. Only a few studies evaluated the mechanical characteristics of multiple braces.



Fig. 23.11 Rigid braces. (a). Rigid stirrup brace. (b) Rigid brace with hinge connection. (c) Rigid brace consisting of unilateral shell. (d) Rigid brace with bilateral shell

#### **Correct Application of an Ankle Brace: No Function Without Force**

Whilst tape is usually applied by an experienced professional or instructions to the athlete are likewise adequate, an ankle brace that is supplied to the athlete usually comes with some basic instructions. Braces are supplied in various sizes to fit the ankle properly. Despite the correct size and instructions, two mistakes are usually made whilst applying a brace. The first is that not enough force is applied whilst tightening the laces or straps. Obviously, there is no function without force. Also the laces of the shoe should be tightened, as the shoe supplements the function of the ankle brace. The second mistake that is made is the incorrect positioning of the foot whilst tightening the laces or straps. One can even find this incorrect positioning of the foot is usually on the ground a little forward. The foot is in plantar flexion whilst tightening the brace (Fig. 23.12a). Once the foot is in neutral flexion, the straps loosen



Fig. 23.12 Application of ankle brace in plantar and dorsiflexion

thereby diminishing their function (Fig. 23.12b). An ankle inversion injury normally occurs with the foot in plantar flexion, where loose straps cannot control the motion. The proper way to tighten the laces or straps is with the foot in some dorsal flexion (Fig. 23.12c). After tightening the brace, bringing the foot in neutral flexion will tighten the straps more (Fig. 23.12d), thereby increasing the controlling function of the straps. Like with the tape, because of stress relaxation, the laces or straps of a brace will loosen over time. Therefore, it is important to retighten the brace regularly during sports activity.

## 23.4 Evidence on Treatment of Lateral Ligament Injuries

### 23.4.1 Acute Phase

The PRICE treatment protocol is commonly used for acute ankle sprain. PRICE stands for protection, rest, ice, compression and elevation [9]. During the first week, the Dutch College of general practitioners guideline for the treatment of ankle injuries recommends treatment consisting of ICE, followed by ankle taping or bracing

for 6 weeks. Thereafter, sports participants are advised to use an ankle brace whilst engaging in sports to prevent recurrence.

In the acute and sub-acute phases of injury, compression of the injured ankle is used to control swelling. Compression can be applied in a variety of modes, including elastic wraps, adhesive tape, braces or cold compression units. In addition, clinicians apply adhesive tape using the open basket weave, leaving the dorsal aspect open to allow additional swelling. However, evidence to support or refute this technique is non-existent. On the other hand, no reports of detrimental effects related to application of compression after acute ankle sprain is reported.

Although compression is used in nearly all clinical practices, limited research and conflicting results make it impossible to provide specific treatment recommendations [10].

## 23.4.2 Rehabilitation

Functional treatment appears to be a superior treatment strategy compared with long-term immobilisation. The effectiveness of different functional treatment modalities for acute ankle ligament injuries was evaluated in a systematic review. It was found that a lace-up brace or a semi-rigid brace gave better results in terms of reduction of swelling and speed of recovery than bandage [11]. These findings were supported by more recent studies [12]. Lardenoye compared a semi-rigid brace with tape. Although functional outcome and pain were similar in the two groups, some skin complications were found in the tape group [13]. Beynnon et al. reported subjects with grade I or II returned to normal walking and stair climbing in half the time when treated with a combination of semi-rigid brace and elastic wrap compared with a semi-rigid brace alone [14]. A recent systematic review concluded that during the proliferation phase the ankle is most effectively protected against inversion by a semi-rigid ankle brace [12].

A review by Kemler et al. found good evidence for a better functional outcome when using an ankle brace. In addition, treatment with ankle braces did not show any unfavourable effects [15]. Since it is also more cost effective, the use of an ankle brace for treatment of acute ankle sprains can be considered, rather than tape.

#### 23.5 Evidence on Prevention of Sprains

Several studies on the effect of prophylactic taping or bracing in preventing ankle sprains in athletes have been conducted. Generally, there is consensus that external ankle support is useful in the prevention of sprains. Inconclusive evidence is reported on which type of external support is more effective, brace or tape. In a systematic review of the literature on prevention strategies for ankle sprains, it was reported that there is clear evidence to support the use of external support such as semi-rigid or lace-up braces and taping for the prevention of ankle sprains. Taping and bracing have been shown to decrease the risk of ankle sprain by 50–70 % in those who have a history of ankle sprain. A study by Mickel et al. showed taping and bracing to be equally effective in preventing sprains [16]. Olmsted et al. used the number needed to treat (NNT) to evaluate the effectiveness. They reported a significantly lower NNT for bracing as compared to taping (39–57 versus 143). Since only three studies were eligible for the pooling of data, no distinction was made for type of brace or method of taping [17].

Taping and bracing appear to be most effective in those who have sustained a previous ankle sprain. In those who have not sustained a previous ankle sprain, the prophylactic effects are not as conclusive [12, 17, 18]. Therefore, providing individuals with a history of ankle sprain who participate in activities associated with a high risk of ankle sprain such as basketball, volleyball, football and football appears to be a valid prophylactic measure for the prevention of recurrent sprains.

Petersen et al. concluded that there is good evidence from high-level randomised trials that the use of a brace is effective for the prevention of ankle sprains. Three of the four identified studies showed that the use of lace-up braces reduced the incidence, but not the severity of acute ankle injuries in football and basketball players [12]. A reduction of ankle sprains by 69 % with the use of ankle brace and reduction of ankle sprain by 71 % with the use of ankle tape among previously injured athletes was found in a recent systematic review. No type of ankle support was found to be superior [19].

### 23.5.1 Studies on Football Players

Four studies on ankle sprain prevention specifically in football players have been conducted. Tropp et al. studied the effect of an ankle orthosis on ankle injury incidence in a 6-month prospective randomised study of male football players. Of the subjects in the group using the ankle orthosis, 3 % sustained an ankle sprain, whereas in the control group, this percentage was much higher (17 %). In the study by Surve et al., a semi-rigid orthosis significantly reduced the incidence of recurrent ankle sprains in football players with previous history of ankle sprains [20]. In addition, Sharpe et al. concluded that ankle bracing is effective in reducing ankle sprain recurrences during in female football players during a season [21]. On the contrary, the results reported in a study by Mohammadi showed no significant difference in ankle sprain reinjury frequency between the orthosis group and the control group [22].

# 23.6 Mechanisms of Beneficial Effects

The mechanisms underlying the preventive effect of external ankle support is not fully understood. Several mechanisms have been studied, of which the most commonly proposed theory is that mechanical support is provided by tape or brace. This support is achieved by preventing extreme range of motions or abnormal movement. Other theories on the effects of tape or braces focus on neuromuscular mechanisms. It is hypothesised that an increase in the proprioceptive input by activating the cutaneous mechanoreceptors around the ankle joint might influence the peroneal muscle activity and postural stability [19, 23]. Additionally, there might be a psychological effect of using tape or brace.

## 23.6.1 Mechanical

One of the most frequently mentioned mechanisms of external ankle support is the reduction of the range of motion, particularly in the frontal plane. The effectiveness of external support in limiting the range of motion is extensively investigated. However, most studies were performed in a static situation.

In a meta-analysis tape, lace-up braces and semi-rigid braces were compared both before and after exercise. The semi-rigid braces restricted the inversion more than tape and lace-up braces both before and after exercise. The semi-rigid braces maintained their restrictive support, whereas lace-up and tape lose their effect. No difference was found in the reduction of the inversion range of motion between the tape and lace-up brace conditions, before or after exercise. Regarding eversion, semi-rigid braces provided greater restriction compared with the tape and lace-up brace. Range of motion of dorsiflexion was restricted more with tape compared with lace-up braces, whereas in terms of plantar flexion, no difference was found [24].

Like this meta-analysis, several other studies have shown tape to lose their restrictive properties during exercise. One study described a 40 % effect loss after 10 min of vigorous exercise, and another study reported up to 50 % loosening of restriction after 15 min of standard vigorous exercise [3, 4]. However, both tape and braces provide more restriction in comparison with no ankle support. It is questionable whether this remaining restrictive effect could account for the preventive effect or that other factors are of more importance.

Apparently less evidence exists on how tape and braces may act to control joint motion and attenuate joint forces during dynamic activities. Therefore, the role of ankle support in reducing forces and loads placed on the ankle remains unclear [24]. Studies have shown brace and tape to reduce the inversion velocity, maximum inversion and time to maximum inversion. It is thought that the delay provided by the ankle support may allow the peroneal muscle additional time to respond. However, real-time inversion injury occurs with a much higher velocity than used in these study settings [7].

Eils et al. compared ten different ankle braces of the three categories. They aimed to evaluate the effects of a brace on the ankle subjected to passive and rapidly induced loading conditions in a population suffering from chronic ankle instability. Rapidly induced stability refers to a situation where subjects are subjected to a fast inversion event on a tilting platform simulating an ankle sprain. This method reflects a more realistic condition because the foot is loaded with body weight and the inversion instant is unknown to the subjects. All braces restricted the range of motion significantly in all directions compared to the no-brace condition. There were differences between the semi-rigid and soft braces. For eversion and plantar flexion the rigid and semi-rigid braces showed differences in total excursion. The authors recommend semi-rigid braces with stirrup design if restriction of inversion under passive and rapidly induced conditions is the primary goal. A variation in restriction of plantar flexion was found between different braces. For prophylactic use in sports, the brace should ensure necessary stability without limiting performance. Semirigid models with a stirrup design can then be recommended. However, it has to be considered that the construct of the stirrup braces may wear out the shoes and is often not compatible to other equipment like shin guards [25].

A recent study evaluated the effects of tape and brace before, during and after a football-match simulation protocol. The conclusion was that tape lost its restrictive benefits after 15 min of football-specific exercise, whilst the brace maintained some effect until the 45th minute [26].

### 23.6.2 Neuromuscular Effects

Besides the mechanical mechanism, another theory on the effect of ankle support was evaluated, although to a lesser degree. Since it remains uncertain that ankle sprains are fully prevented by mechanical effects of tape or brace, it is suggested that increased proprioception and awareness of joint position play a role.

Studies were focused on joint position and movement sense or reproduction, peroneal muscle activity, reflex, reaction time and postural stability.

#### 23.6.2.1 Joint Position and Movement Sense (Propriocepsis)

It is hypothesised that due to damage to mechanoreceptors in and around the ankle joint as a result of sprain, the propriocepsis is impaired. After a first sprain the disruption of afferent information affects the ability to detect sense of movement and joint position. Application of tape or use of a brace would improve the propriocepsis by enhancing proprioceptive acuity as a result of increased stimulation of the cutaneous mechanoreceptors.

This theory explains the high recurrence rate of sprains and the fact that the preventive effect of tape and brace seems to be higher after first-time ligament injury.

Although, several studies were published on the effect of ankle support on joint position and movement sense, the contribution of enhancement of proprioception to the preventive effect remains unclear. In a systematic review Raymond et al. concluded that using an ankle brace or ankle tape has no effect on proprioceptive acuity in participants with recurrent ankle sprain or who have functional ankle instability. This finding was consistent when the two aspects of proprioception (sense of movement or joint position) were considered separately. Although the majority of the included studies reported a positive effect, the authors failed to find a statistically significant difference after pooling of data [27].

#### 23.6.2.2 Peroneal Muscle Activity

As the most important everter of the ankle, the role of peroneal longus muscle in preventing ankle sprains was studied extensively. Both muscle function and reflex latency were subjects of several studies. The theory that ankle support would shorten the reaction time was not clarified. Studies report conflicting results regarding the peroneal latency, some reporting on decreased latency [28, 29], where others did find no difference [30, 31]. One study even showed an increase in personal latency [32]. However, these conflicting findings may be the result of methodological differences, since these studies used a setting simulating a real inversion injury. Furthermore, the velocity and amplitude of the inversion used during these simulations may not reflect the situation of a true inversion trauma.

#### 23.6.2.3 Postural Control or Stability

It is thought that players with a decreased postural control more are prone to an ankle sprain injury. Multiple studies have investigated possible influence of tape and brace on postural control. These studies most often focused on centre-of-pressure or centre-of-gravity displacement.

Two studies evaluated the effect of tape on postural control in football players. Tropp et al. found that taping did not influence stabilometric values [33].

Lohkamp et al. reported that any potential benefit of tape was negated after 15 min of football-specific activity. Hence, they concluded that taping to prevent lateral ligament injury in healthy football players may only be effective in during the first 15 min [34].

### 23.6.3 Psychological

A more alternative and less extensively described theory regarding beneficial effects of ankle support is the psychological factor. Subjects reported feeling more stable, confident and reassured with the application of the standard taping technique (stirrups, figure of 8, heel lock) or a single strip of tape applied to the calf compared with the untaped condition whilst performing functional tasks such as hopping and balancing [17].

Manfroy et al. stated that athletes who believe that a certain type of ankle support will protect them from injury will participate in sport with greater confidence [35].

Sawkins et al. demonstrated a placebo effect of tape. Although they failed to find a difference in functional performance tests between real tape and placebo tape, both tapes did affect the participants' perceptions of stability, confidence and reassurance when performing functional tests [36].

# 23.7 Detrimental Effects

### 23.7.1 Effects on Performance

An important factor of the use of external ankle supports in sports is the impact of these devices on the functional performance of the athletes. Although tape and brace seem to be useful in preventing (recurrent) ankle sprains, players are not likely to use them if they perceive that their performance will be hampered.

One of the main purposes of brace and tape is limiting the range of motion; however, a restriction of motion in both the frontal and sagittal plane could interfere with the execution of functional tasks. The three skills that were widely investigated are running speed, agility and vertical jump height. Of these, running speed and agility are important in football players.

#### 23.7.1.1 Sprinting

Since running speed is important for football players, an ankle support that impedes a football player in running will not become popular.

In running, players must be able to quickly dorsiflex the ankle before the stance phase, before they forcefully plantarflex the ankle during push off. For speed generation this combination of movements has to occur within thousands of seconds. It seems clear that when tape or brace restrict the dorsiflexion and/or plantar flexion, this will affect the generation of speed.

#### 23.7.1.2 Agility

Ability to quickly change direction at top speed is an important factor in football.

For this sideward cutting movement, the ankle is placed under an inversion stress to be able to push off and return to top speed. Hence, for optimal agility skills the maximum amount of inversion as well as plantar and dorsiflexion is necessary. Especially, reaching the maximum amount of dorsiflexion is required to provide explosive propulsion for push off for a change in direction. It seems obvious that a brace or tape that restricts the amount of inversion will affect the agility of a player.

#### 23.7.1.3 Vertical Jump

Vertical jump height can certainly become impaired if the external ankle support decreases the functional ROM. Like with running, talocrural motion and torque production in the sagittal plane is a critical component of any jumping task, as it assists in propulsion from the ground.

Several studies have evaluated the effects of ankle support on functional performance.

There is a lack of consistent effects among these studies. In a review by Bot et al., it was concluded that results of the studies are contradictory. However, they found that the majority of the studies indicate that ankle bracing or taping has no or only a minor effect on vertical jump height, agility, running speed and broad jump distance and may even improve agility in subjects with functional ankle instability [37]. Cordova et al. conducted a meta-analysis to evaluate the effect of external ankle support on the functional performance. They concluded that the average effects of external ankle support on sprint, agility and vertical jump performance ranged from trivial to small in subjects who are not elite athletes, i.e. a 1 % performance change for each of the tests evaluated. Moreover, it is reasonable to surmise that the effects observed in this study may have greater performance implications in the elite athlete.

Overall, based on the results of this meta-analysis, it could be concluded that external ankle support has a small effect on agility performance, but not in all settings.

In their opinion the benefit in preventing injury outweighs the possibility of substantial but small impairment of performance when athletes use external ankle support [38].

#### 23.7.1.4 Football-Specific Aspects

Almost all aforementioned studies evaluated performance in sports where the upper extremity is used to handle the ball. In football the ankle and foot are directly involved in handling the ball. The use of external support may influence the performance more in comparison with sports like basketball and volleyball. Since there is contact between the brace or tape and the ball, it is more likely to affect the performance of shooting and passing. Only a few studies were performed on footballspecific skills.

In the study by Paris, the basket weave taping technique and three types of laceup braces were compared. There were no significant differences in speed, balance and agility between the braced, taped or unprotected football players. Only the vertical jump performance was significantly reduced in players using one of the lace-up braces [39].

Putnam et al. evaluated in a recent study the kicking accuracy in addition to running speeds and agility between players wearing a brace with elastic wraps and players without a brace. This study of healthy recreational football players suggests that an ankle brace has no significant effect on performance in kicking accuracy, speed and agility [40].

## 23.8 Discussion

It can be concluded that there is evidence to support the use of brace and tape in both rehabilitation of acute lateral ligament injury and prevention. The mechanisms underlying this beneficial effect are, however, not fully understood. It seems evident that tape and brace limit the range of motion of the ankle joint in the sagittal and frontal plane. The degree of limitation and direction of limitation depends highly on type of brace or taping technique. Whether this limitation of range of motion and thereby prevention of extreme movements is the main mechanism is questionable since it is proved that both tape and brace lose their restrictive effect during exercise. In addition, there is also no conclusive evidence for the alternative mechanisms like improved propriocepsis.

In addition to the preventive effect, there are disadvantages of wearing a brace or tape during football. It is frequently stated that ankle support would impede the functional performance of the player. Although the literature is not consistent, it seems that restriction of range of motion at least has some effect on running speed and agility. Only one single study on football-specific performance is conducted, which concluded that tape or brace will not affect the shooting or passing accuracy [40]. In particular for prophylactic use of ankle support, the question is whether the benefits outweigh the possible impairments in performance. Based on the current evidence, this question should be answered positively.

It is difficult to compare braces with tape, since there is a wide range of both types of brace and taping techniques. However, the literature shows neither brace nor tape to be obviously superior to the other. The use of tape seems to be more popular among athletes. Both have their advantages and disadvantages. Tape is often less bulky and therefore possibly more comfortable, which could be important for football players. In addition, tape could be applied according to the preferences of the player. Braces have the advantages of being reusable, readjustable and non-allergic. Also important is that minimal expertise is required for correct application. Since its known that tape and brace lose their restrictive effect during exercise, the possibility of readjusting could be important for maintaining the function. An important advantage of bracing is the lower cost. Olmsted et al. performed costbenefit analyses and found that taping was more than three times as expensive as bracing over the course of a competitive season [17].

Apparently, a brace not worn or incorrectly applied will not reduce the chance of recurrent inversion injury. The compliance of using an ankle brace by athletes depends on the looks of the brace and to minor extent comfort and costs. Awareness of the odds of incurring an inversion injury and stimulation by a team coach positively affects the compliance [41].

#### References

- Gibney VP (1895) Sprained ankle: a treatment that involves no loss of time, requires no crutches, and is not attended with an ultimate impairment of function. NY Med J 61:193–197
- 2. Walter GI (1940) An adhesive strapping for the ankle sprain. J Bone Joint Surg 22(1):211–215
- Rarick GL, Bigley G, Karst R, Malina RM (1962) The measurable support of the ankle joint by conventional methods of taping. J Bone Joint Surg 44(6):1183–1190
- Frankeny JR, Jewett DL, Hanks GA, Sebastianelli WJ (1993) A comparison of ankle-taping methods. Clin J Sport Med 3(1):20–25

- Metcalfe RC, Schlabach GA, Looney MA, Renehan EJ (1997) A comparison of moleskin tape, linen tape, and lace-up brace on joint restriction and movement performance. J Athl Train 32(2):136
- Vaes P, De Boeck H, Handelberg F, Opdecam P (1985) Comparative radiologic study of the influence of ankle joint bandages on ankle stability. Am J Sports Med 13(1):46–50
- 7. Tregouet P, Merland F, Horodyski MB (2013) A comparison of the effects of ankle taping styles on biomechanics during ankle inversion. Ann Phys Rehabil Med 56(2):113–122
- Abian-Vic'n J, Alegre LM, Ferníndez-Rodriguez JM, Aguado X (2009) Prophylactic ankle taping: elastic versus inelastic taping. Foot Ankle Int 30(3):218–225
- 9. Ivins D (2006) Acute ankle sprain: an update. Am Fam Physician 74(10):1714
- Kaminski TW, Hertel J, Amendola N, Docherty CL, Dolan MG, Hopkins JT et al (2013) National Athletic Trainers' Association position statement: conservative management and prevention of ankle sprains in athletes. J Athl Train 48(4):528–545
- 11. Kerkhoffs GM, Rowe BH, Assendelft WJ, Kelly K, Struijs PA, Van Dijk CN (2002) Immobilisation and functional treatment for acute lateral ankle ligament injuries in adults. Cochrane Database Syst Rev 3(3):CD003762
- Petersen W, Rembitzki IV, Koppenburg AG, Ellermann A, Liebau C, Brüggemann GP et al (2013) Treatment of acute ankle ligament injuries: a systematic review. Arch OrthopTrauma Surg 133(8):1129–41
- Lardenoye S, Cleffken B, Brink PR, de Bie RA, Poeze M (2012) The effect of taping versus semi-rigid bracing on patient outcome and satisfaction in ankle sprains: a prospective, randomized controlled trial. BMC Musculoskelet Disord 13(1):81
- Beynnon BD, Renström PA, Haugh L, Uh BS, Barker H (2006) A prospective, randomized clinical investigation of the treatment of first-time ankle sprains. Am J Sports Med 34(9):1401–1412
- Kemler E, van de Port I, Backx F, van Dijk CN (2011) A systematic review on the treatment of acute ankle sprain: brace versus other functional treatment types. Sports Med 41(3):185–197
- Mickel TJ, Bottoni CR, Tsuji G, Chang K, Baum L, Tokushige KA (2006) Prophylactic bracing versus taping for the prevention of ankle sprains in high school athletes: a prospective, randomized trial. J Foot Ankle Surg 45(6):360–365
- Olmsted LC, Vela LI, Denegar CR, Hertel J (2004) Prophylactic ankle taping and bracing: a numbers-needed-to-treat and cost-benefit analysis. J Athl Train 39(1):95
- Handoll HH, Rowe BH, Quinn KM, De Bie R (2001) Interventions for preventing ankle ligament injuries. Cochrane Database Syst Rev 3(3):CD000018
- Dizon JM, Reyes JJ (2010) A systematic review on the effectiveness of external ankle supports in the prevention of inversion ankle sprains among elite and recreational players. J Sci Med Sport 13(3):309–317
- Surve I, Schwellnus MP, Noakes T, Lombard C (1994) A fivefold reduction in the incidence of recurrent ankle sprains in soccer players using the sport-stirrup orthosis. Am J Sports Med 22(5):601–606
- 21. Sharpe SR, Knapik J, Jones B (1997) Ankle braces effectively reduce recurrence of ankle sprains in female soccer players. J Athl Train 32(1):21
- 22. Mohammadi F (2007) Comparison of 3 preventive methods to reduce the recurrence of ankle inversion sprains in male soccer players. Am J Sports Med 35(6):922–926
- 23. Callaghan MJ (1997) Role of ankle taping and bracing in the athlete. Br J Sports Med 31(2):102-108
- Cordova ML, Ingersoll CD, Palmieri RM (2002) Efficacy of prophylactic ankle support: an experimental perspective. J Athl Train 37(4):446
- 25. Eils E, Demming C, Kollmeier G, Thorwesten L, Völker K, Rosenbaum D (2002) Comprehensive testing of 10 different ankle braces: evaluation of passive and rapidly induced stability in subjects with chronic ankle instability. Clin Biomech (Bristol, Avon) 17(7):526–535
- Forbes H, Thrussell S, Haycock N, Lohkamp M, White M (2013) The effect of prophylactic ankle support during simulated soccer activity. J Sport Rehabil 22:170–176

- Raymond J, Nicholson LL, Hiller CE, Refshauge KM (2012) The effect of ankle taping or bracing on proprioception in functional ankle instability: a systematic review and metaanalysis. J Sci Med Sport 15(5):386–392
- Karlsson J, Andreasson GO (1992) The effect of external ankle support in chronic lateral ankle joint instability an electromyographic study. Am J Sports Med 20(3):257–261
- 29. Knight AC, Weimar WH (2011) Difference in response latency of the peroneus longus between the dominant and nondominant legs. J Sport Rehabil 20(3):321
- 30. Cordova ML, Takahashi Y, Kress GM, Brucker JB, Finch AE (2010) Influence of external ankle support on lower extremity joint mechanics during drop landings. J Sport Rehabil 19(2):136
- Jerosch J, Hoffstetter I, Bork H, Bischof M (1995) The influence of orthoses on the proprioception of the ankle joint. Knee Surg Sports Traumatol Arthrosc 3(1):39–46
- 32. Shima N, Maeda A, Hirohashi K (2005) Delayed latency of peroneal reflex to sudden inversion with ankle taping or bracing. Int J Sports Med 26(06):476–480
- 33. Tropp H, Askling C, Gillquist J (1985) Prevention of ankle sprains. Am J Sports Med 13(4):259-262
- 34. Lohkamp M, Craven S, Walker-Johnson C, Greig M (2009) The influence of ankle taping on changes in postural stability during soccer-specific activity. J Sport Rehabil 18(4):482
- 35. Manfroy PP, Ashton-Miller JA, Wojtys EM (1997) The effect of exercise, prewrap, and athletic tape on the maximal active and passive ankle resistance to ankle inversion. Am J Sports Med 25(2):156–163
- 36. Sawkins K (2007) The placebo effect of ankle taping on ankle instability. Med Sci Sports Exerc 39:781–7
- Bot MSD, van Mechelen W (2003) The effect of ankle bracing and taping on functional performance: a review of the literature. Int Sports Med J 4(5):1–14
- Cordova ML, Scott BD, Ingersoll CD, LeBlanc MJ (2005) Effects of ankle support on lowerextremity functional performance: a meta-analysis. Med Sci Sports Exerc 37(4):635–641
- 39. Paris DL (1992) The effects of the Swede-O, New Cross, and McDavid ankle braces and adhesive ankle taping on speed, balance, agility, and vertical jump. J Athl Train 27(3):253
- Putnam AR, Bandolin SN, Krabak BJ (2012) Impact of ankle bracing on skill performance in recreational soccer players. PM R 4(8):574–579
- 41. Cusimano MD, Faress A, Luong WP, Amin K, Eid J, Abdelshaheed T et al (2013) Factors affecting ankle support device usage in young basketball players. J Clin Med 2(2):22–31

# **Chapter 24 Ankle Osteoarthritis in Former Elite Football Players: What Do We Know?**

Vincent Gouttebarge and Monique H.W. Frings-Dresen

You don't stop playing Football because you're getting old. You get old because you stop playing Football.

Sir Stanley Matthews

Abstract Due to cumulative exposure to heavy physical demands and to the occurrence of traumatic ankle injuries during their career, former professional football players are likely to suffer in their post-sport life from ankle osteoarthritis. Ankle osteoarthritis involves a progressive degeneration of articular cartilage in the ankle joint that might lead to joint pain, reduced function, instability, deformity and swelling. The recent scientific literature has showed that the prevalence of ankle osteoarthritis in former professional football players was high (9-17 %) compared to former athletes from other sport disciplines and to the general population. Most of the former professional football players suffering from ankle osteoarthritis reported to experience moderate to severe problems related to joint pain/discomfort, mobility and performing usual activities (work, study, house, etc.). In addition, 37 % of them reported moderate or severe problems with anxiety/depression because of the pain/ discomfort and impairments caused by ankle osteoarthritis. Future directions related to the medical care and support of former professional football players facing l ankle osteoarthritis might involve self-awareness (information provision) system, self-management programme and/or end-career socio-medical consultation.

M.H.W. Frings-Dresen Coronel Institute of Occupational Health, Academic Medical Center, Amsterdam, The Netherlands

V. Gouttebarge (⊠)

Academic Medical Center, Amsterdam, The Netherlands

e-mail: vgouttebarge@gmail.com

# 24.1 Introduction

During both training and competition, professional football players are highly exposed to intense and prolonged physical demands such as running, sprinting, jumping and landing, dribbling and passing and sliding or other duel forms with opponents. In addition, players are also exposed to high energetic demands (aerobic and anaerobic). Exceeding regularly normal health capacities, these physical and energetic demands, in combination with insufficient time to recover, lead regularly to the occurrence of injuries of the musculoskeletal system, especially in the ankle joint.

As elite athletes from other sport disciplines, former professional football players are keen to have on the long term a lower risk of chronic diseases (heart disease, diabetes, asthma and bronchitis) compared to healthy individuals of similar age. On the other hand, former professional football players are also keen to suffer after their career from long-term health problems. Especially, due to the cumulative physical exposure during training and competition, and to the high risk for severe, i.e. recurrent ankle injuries and related surgery, former professional football players are likely to suffer from ankle osteoarthritis in their post-career life.

# 24.2 Ankle Osteoarthritis

Osteoarthritis (OA) is a 'degenerative joint disease' resulting from the interaction of several factors such as joint integrity, genetics, local inflammation, mechanical forces and cellular and biochemical processes [7, 10, 17]. Being a normal consequence of ageing, OA is caused by overuse of the joint's cartilage and results in irreversible pathologic changes in the affected joints [7, 17].

Ankle OA involves a progressive degeneration of articular cartilage characterized by the formation of impinging bone spurs, loose bodies and joint space narrowing. The progression of ankle OA might lead to the decrease of articular cartilage thickness and to the enhancement of cartilage deterioration. As the ankle joint is formed by three bones (tibia, fibula and talus), OA might occur in three sets of articular surfaces: the medial, lateral and central articular surfaces.

Distinction is made between primary and secondary ankle OA. Primary ankle OA is seen as an idiopathic phenomenon related to the ageing process including previously healthy joints and having no apparent cause or initiating factor. Primary ankle OA occurs typically in older individuals. Secondary ankle OA is easier to understand and refers to a joint disease resulting from clear predisposing and initiating factors such as obesity, prior traumatic event that causes cartilage damage or excessive repetitive injury (especially in athletes). In contrast to primary OA, secondary ankle OA can occur in relatively young individuals.

Among reduced function, instability and deformity, the principal symptom associated with ankle OA is joint pain. Being exacerbated by activity and relieved by rest in an early phase, joint pain occurs both at rest and at night in a more advanced disease stage. A common complaint among patients with ankle OA is also stiffness: after a patient awakens, morning stiffness typically resolves within 30 min but may recur following periods of inactivity. A distinction between noninflammatory and inflammatory OA is made. Aside from pain and disability-related complaints that may be seen in both types, patients with inflammatory OA might have joint swelling, morning stiffness lasting for more than 30 min, night pain and signs of inflammation. Most of these symptoms and signs can be detected by physical examination.

# 24.3 Prevalence of Ankle Osteoarthritis in Former Professional Football Players

As degenerative changes in the joint is not consistently associated with clinical OA, determining the prevalence of ankle OA remains difficult. Nearly 20 years ago, 6 % of former professional football players were found to have been admitted to the hospital for OA of the weight-bearing joints of the lower limbs among which hips, knees and ankles [12]. While epidemiological researches related to hip and knee OA have been largely performed among various study populations, studies related to ankle OA are scarce, especially among former professional football players as acknowledged in a systematic literature review.

Only two empirical studies were retrieved from the scientific literature in which the prevalence of ankle OA was presented [11]. Among 185 retired English professional football players who had played professional football on average for nearly 14 years, the prevalence of ankle OA (diagnosed by clinician) was found to be 5.7 % in the right ankle and 6 % in the left ankle. In this study, the mean age at diagnosis was 30 years for the right ankle and 32 years for the left ankle. A second study explored 284 former professional football players from the United Kingdom who had played professional football on average for nearly 14 years. Forty-nine percent of these former professional football players indicated that they had been diagnosed (by clinician) at an average age of 40 years with OA on at least one anatomical site, 29 % in two or more joints and 15 % in three or more joints. Especially, the ankle joints accounted for 17 % of all 314 OA diagnoses among these 284 former professional football players, whereof 11 % in the right ankle and 6 % in the left ankle [11]. A latest published study by Armenis et al. [2] explored the prevalence of ankle/foot OA in a group of 105 former Greek professional football players (older than 40 years) who had played professional football for 8-10 years. Clinical signs of OA were found in 4 % of these former professional football players, while radiographic OA was found in nearly 9 %.

Whether the occurrence of ankle OA in former professional football players is alarming can be put into perspective when compared to the general population or to athletes from other sport disciplines. An older study from Kujala et al. [12] acknowledged that the prevalence of ankle OA ranged from 0 to 2 % in former elite athletes (44 years old or older) that were involved during their career in different sport

<b>Table 24.1</b> Prevalence (%) of ankle osteoarthritis: overview among former professional football players ( $\geq$ 30 years of age), former elite athletes from various sport disciplines ( $\geq$ 44 years of age) and the general population ( $\geq$ 30 years of age)	Football		9–17		
	Athletics <sup>a</sup>		0.2		
	Basketball		0		
	Boxing		0.4		
	Cross-country skiing		0		
	Ice hockey		1.8		
	Long-distance running		0		
	Weight lifting		0.9		
	General population		1–4		
	<sup>a</sup> Jumping, sprinting, running	hurdling,	decathlon,	middle	distance

disciplines (long-distance running, cross-country skiing, ice hockey, boxing or weight lifting; see Table 24.1). Worldwide, approximately 1-4 % of the adult general population has OA of the ankle. Then, despite the limited information available from the scientific literature, it is clear that the prevalence of ankle OA in former professional football players is higher than in the general population or former athletes from other sport disciplines.

# 24.4 Consequences of Ankle Osteoarthritis in Former Professional Football Players

Being primarily associated with previous traumatic injuries, ankle OA is a frequent health concern among former professional football players, a condition that might even appear in the early years after the end of a football career. The adverse impacts of ankle OA on the quality of life and functioning (work and daily living) of former professional football players cannot be neglected, even if empirical evidence about the long-term consequences of this health condition is limited.

In a recent systematic review [5], only two original studies exploring the consequences of ankle OA in former professional football players were identified. A cross-sectional survey was conducted in the United Kingdom (UK) among 284 former professional football players who had played professional football on average for nearly 14 years. One hundred and thirty-eight of these former professional football players suffered from OA in a lower limb joint (hip, knee, ankle and/or foot), from which 33 from OA in the right ankle and 20 from OA in the left ankle. From the former professional football players suffering from OA (not solely of the ankle), nearly 90 % reported to have moderate or severe joint pain and discomfort, while around 65 % indicated to experience moderate or severe problems with mobility and performing usual activities (work, study, house). In addition, 37 % of them reported moderate or severe problems with anxiety/depression because of the pain/ discomfort and impairments caused by ankle osteoarthritis. Based on this study, the authors conducted 2 years later a qualitative study by interviewing 12 former professional football players who were suffering from hip, knee and/or ankle OA. With regard to pain, some former professional football players reported that their conditions were chronically very painful, and that the pain was significantly affecting their lives. With regard to restricted mobility and movement, some former professional football players reported that the lack of mobility was a major issue in their lives, moving being hardly possible, especially bending, kneeling and long standing. With regard to employment, some former professional football players reported that no employer wanted to employ them with their conditions and that they abandoned their jobs for this reason.

# 24.5 Origin of Ankle Osteoarthritis in Former Professional Football Players

The origin of ankle OA on the long term among former professional football players is expected to be related to physical exposure and occurrence of ankle injury during a career. Within professional football, it remains unknown whether the cumulative exposure to intense and prolonged physical demands involving the ankle joint (running, sprinting, jumping and landing, dribbling and passing, duel forms with opponents) during both training and competition contributes solely to the high prevalence of ankle OA among former players. Despite the lack of empirical studies involving large sample sizes and suitable controls matched for football exposure, ankle OA in former professional football players seems principally attributed to the occurrence of ankle injury during a football career [7, 10, 17].

### 24.5.1 Picture Sliding in Football if Available?

In (professional) football, ankle ligament (medial and lateral bands) and cartilage injuries are common during training and competition, accounting approximately for 20–30 % of all injuries. Especially, having more drastic consequences during a football career in terms of related surgery, rehabilitation and long-term disability, recurrent or severe ankle injuries, in combination with their surgical treatment, are seen as determinants for ankle OA in former professional football players. Recent empirical studies in which causality between previous injury and ankle OA was investigated among former professional football players (both in study and control groups) are lacking. However, older studies or studies performed among athletes from other sports disciplines indicate that the occurrence of ankle injuries is a relevant determinant for ankle OA.

Larsen et al. [13] examined the incidence ankle OA in injured and uninjured elite football players, with a mean time from injury of 25 years. OA was present in 33 % of the injured ankles, whereas the incidence of OA in uninjured players was 18 % in

the ankle. On a series of more than 300 ankle fractures treated with open reduction and internal fixation, Lindsjö [14] found that the prevalence of posttraumatic OA was 14 %, which was directly correlated with the fracture pattern. Reviewing retrospectively data from 30 patients (mean age: 59 years, 33 ankles) with ankle OA, Valderrabano et al. [27] found that 55 % had a history of sports injuries (33 % from soccer) and 85 % had a lateral ankle ligament injury. Even more, the same author [28] found in a study of 406 ankles with end-stage OA that the underlying aetiology in this group was posttraumatic ankle OA in 78 % of cases. Within these posttraumatic OA cases, 62 % were attributable to fracture events (malleolar fractures and tibial plafond fractures) and 16 % to ligamentous injuries. Some studies indicate that severity of the initial injury and the initial cartilage damage may play a role in the development of ankle OA.

# 24.6 Diagnosis

In order to diagnose ankle OA and assess thoroughly the extent of the disease, anamnesis, physical examination and radiographic evaluation should be combined [7, 10, 17]. While the presence of OA might be suggested by anamnesis and physical examination, ankle OA diagnosis is usually confirmed by routine radiographic evaluation. In addition, radiological diagnostic assesses OA severity and serves as an initial evaluation to monitor the worsening of the disease.

### 24.6.1 Anamnesis and Physical Examination

During the anamnesis, the physician strives to retrieve important information related to ankle symptoms, exploring several aspects such as joint pain, joint stiffness in the morning, duration of the symptoms, past (recurrent) trauma, sprain and related surgery, family history and any general symptoms (fatigue, weight loss, fever, etc.) affecting the whole body. Also, the contribution of sport activities, occupational activities and daily living activities to the symptoms is explored. In order to assess the overall functional level, the American Orthopaedic Foot and Ankle Society (AOFAS) hindfoot score can be used. This valid questionnaire (available in different languages) consists of 42 items (5-point Likert scale) divided into subscales assessing symptoms, stiffness, pain, quality of life and function, resulting in a score from 0 to 100 with higher scores indicating fewer problems.

During the physical examination, the physician strives to identify important signs related to ankle OA such as:

- · Presence of abnormal skin and soft tissues
- · Presence of tender areas, synovitis, effusion, bony knobs and loose bodies
- Ankles pattern (if only one is affected)

- Ankle instability (anterior draw and talar tilt test)
- Ankle impingement (Molloy impingement test)
- · Remaining movement in the subtalar and midtarsal joints
- Range of motion
- Muscle weakness or atrophy
- Unequal leg lengths
- Alignment of the tibia to the hindfoot, the midfoot and the forefoot
- Other joints and limb alignment
- Gait

### 24.6.2 Radiography

A routine radiographic evaluation of the ankle consisting of a weight-bearing AP view, a mortise view and a lateral view (Fig. 24.1) is made in order to identify radiological signs of OA, including asymmetrical narrowing of the joint space (indicating loss of cartilage), development of osteophytes and subchondral sclerosis [10, 17, 25]. On a true AP view, the talus overlaps a portion of the lateral malleolus, obscuring the lateral aspect of the ankle joint. The mortise view is obtained with the foot in 15–20° endorotation, making visualization of both the lateral and medial joint spaces possible. An additional hindfoot alignment radiograph can be considered in situations where the ankle has coronal plane tilting and the heel is in varus or valgus position.

The Kellgren and Lawrence (K&L) criteria have been widely used to grade OA and were chosen as reference by the World Health Organization to characterize OA in the hip and knee joints [8]. By now, the K&L criteria have been also validated for the ankle joints [6], consisting in the assessment of three radiological ankle features (osteophyte formation, joint space narrowing and bone end sclerosis). According to the K&L criteria, ankle OA can be classified as follows:

- Grade 0: normal joint
- · Grade 1: unlikely or doubtful narrowing of joint space and possible osteophytes
- Grade 2: definite osteophytes and possible narrowing of joint space
- Grade 3: multiple moderately sized osteophytes, definite narrowing of joint space, some sclerotic areas and possible deformation of bone contour
- Grade 4: large osteophytes, marked narrowing of joint space, severe sclerosis and define deformation of bone contour

### 24.7 Treatment

While the treatment of hip and knee OA has been sufficiently based on scientific knowledge, evidence to support the effective treatment of ankle OA is lacking. Nevertheless, analogously to OA in other joints, conservative and surgical treatment



Mortise view OA

Mortise view healthy

 $\label{eq:Fig. 24.1} \begin{array}{l} \mbox{Radiographs of a healthy ankle joint and an ankle joint affected by osteoarthritis (a)} \\ \mbox{Lateral view OA (b) Lateral view healthy (c) Mortise view OA (d) Mortise view healthy} \end{array}$ 

have been suggested to treat ankle OA in order to prevent and slow down OA worsening, relieve symptoms and improve joint function [9, 10, 17, 21, 22, 24]. With regard to safety, invasiveness and costs, conservative treatment might be preferred to surgical treatment. Surgical treatment should be reserved to the patients who do not improve with conservative treatment and who have seriously affected quality of life.

# 24.7.1 Conservative Treatment

Aiming to relieve and control the pain associated with ankle OA and improve the function of the joint, conservative treatment relies on various pharmacological or non-pharmacological options involving [9, 15, 16, 18, 21, 25]:

- Short-term use (because of side effects) of nonsteroidal anti-inflammatory drugs (NSAIDs) to relieve and control ankle pain
- Glucosamine and chondroitin supplement as a safe and potential effective option for the relief of ankle OA symptoms
- Injection of viscosupplementation into the ankle joint to alleviate joint symptoms
- Judiciously timed injection of corticosteroids into the ankle joint to decrease pain for the enjoyment of a particularly important life event
- Modified footwear (rocker-bottom sole, solid ankle cushion heel, polypropylene ankle/foot orthosis, lace-up ankle support, ankle brace)
- Physical therapy to preserve joint mobility and range of motion
- Specific exercises to increase muscle and neuromuscular, i.e. proprioceptive functions in order to enhance ankle functions and stability
- Healthy lifestyle, especially related to weight control through general physical, i.e. fitness programmes

# 24.7.2 Surgical Treatment

The decision for surgical treatment of ankle OA requires a grounded evaluation of the patient's functional needs and problems. As surgical techniques continuously change and evidence for effectiveness accumulates, the indications for surgery treatment of ankle OA have been evolving in time. For ankle OA, surgery is seen as a treatment option when conservative treatment has failed to control the patient's symptoms in such a way that the patient's quality of life and daily living or work activities are seriously affected. Surgical options for ankle OA include joint-preserving surgery, arthrodesis and (total) ankle replacement [10, 17, 20, 23, 25, 26].

Joint preserving surgery, including arthroscopic debridement and articular distraction, aims to delay more invasive and extensive surgery. Being commonly achieved through arthroscopy, ankle debridement is performed in case of impinging osteophytes, loose bodies and chondral defects. For severe end stage of OA, articular distraction based on an external articulated fixation frame and a distraction force applied across the ankle has been recently advocated for patients being candidate for arthrodesis in order to decrease joint pain and improve movements.

Ankle arthrodesis has been seen for several decades as the gold standard treatment of end-stage ankle OA. Ankle arthrodesis can be done with numerous techniques and approaches. Several methods of stabilization can be used such as external fixation; internal fixation with screws, plates and on-lay or dowel bone grafts; and cast fixation alone. Despite several limitations such as a disturbed gait pattern and reduced functionality after ankle fusion, most patients are satisfied with ankle arthrodesis. However, it remains unclear to which extent ankle arthrodesis contributes to progressive degeneration of adjacent joints.

# 24.8 Future Directions: Medical Care and Support of Former Professional Football Players

Professional football players should be seen as any other employees from any occupational sectors. Consequently, as stated by the World Health Organization (WHO) and the International Labour Organization (ILO), professional football clubs and responsible (inter)national bodies are responsible for 'the protection, promotion, surveillance and maintenance of the highest degree of physical, mental and social well-being of players during their career but also long after their retirement years'. Consequently, the expectation is that the medical care and support offered to professional football players would be related to long-term health problems such as ankle OA because it may impair their sustainable health and functioning in their postsport life.

Nevertheless, recent findings suggested that the current medical care and support in professional football is exclusively directed towards the short-term health issues during a career, namely, the injuries of the musculoskeletal system [1]. Despite the occurrence after retirement of long-term health problems such as ankle OA, any form of socio-medical counselling aiming to empower sustainable health and functioning of players in their post-sport life has been up till now neglected [1, 4]. Both current and former professional football players, as well as physicians working in professional clubs, have recently acknowledged that information provision about long-term adverse effects was lacking, advocating the development and implementation of a proper socio-medical counselling for these long-term adverse effects [1]. These needs are consistent with past findings in which the necessity to develop a long-term strategy for (forced) retired football players was underlined [4].

Consequently, several future directions related to the medical care and support of former professional football players facing long-term adverse effects such as ankle OA might be proposed. A first step could be to raise the self-awareness (information provision) of professional football players at the time around their retirement with regard to ankle OA. Especially, relevant information could be disseminated about potential risk determinants, disease process, pain mechanisms, treatment options and strategies, consequences for well-being, sport and work functioning.

For chronic health conditions such as rheumatic diseases (including OA), selfmanagement programmes have been identified as effective in order to engage and promote a healthful and active behaviour of patients in managing their disease [19]. Consequently, the development and implementation of a self-management programme related to the particular characteristics and specific needs of former
professional football players facing ankle OA at the time around their retirement might be helpful as cognitive and behavioural therapy in order to manage ankle OA, prevent its worsening and empower sustainable health and functioning. Also, an end-career socio-medical consultation could be an optimal innovation in order to empower former players and give them advice about active lifestyles and relevant physical activities that might be performed (non-weight-bearing rather than weightbearing activities) to prevent an increase in body mass and a decrease in muscular and neuromuscular function and to increase functional abilities in (working) life [3].

## References

- 1. Akturk A, Inklaar H, Gouttebarge V et al Medical examination in Dutch professional football (soccer): a qualitative study (in press)
- 2. Armenis E, Pefanis N, Tsiganos G et al (2011) Osteoarthritis of the ankle and foot complex in former Greek soccer players. Foot Ankle Spec 4:338–343
- Bennell K, Hunter DJ, Vicenzo B (2012) Long-term effects of sport: preventing and managing OA in the athlete. Nat Rev Rheumatol 8:747–752
- Drawer S, Fuller CW (2002) Perceptions of retired professional soccer players about the provision of support services before and after retirement. Br J Sports Med 36:33–38
- Gouttebarge V, Inklaar H, Frings-Dresen MHW (2013) Risk and consequences of osteoarthritis after a professional football career: a systematic review of the recent literature. J Sports Med Phys Fitness (in press)
- Holzer N, Salvo D, Marijnissen AK et al (2012) How to assess ankle osteoarthritis: comparison of the Kellgren and Lawrence scale with functional outcome and digital image analysis. J Bone Surg Br 94-B(Supp XXXVII):64
- Hunter DJ, McDougall JJ, Keefe FJ (2008) The symptoms of OA and the genesis of pain. Rheum Dis Clin North Am 34:623–643
- Kellgren JH, Lawrence JS (1957) Radiological assessment of osteo-arthosis. Ann Rheum Dis 16:494–502
- Koh J, Dietz J (2005) Osteoarthritis in other joints (hip, elbow, foot, ankle, toes, wrist) after sports injuries. Clin Sports Med 24:57–70
- Koopman WJ, Moreland LW (eds) (2004) Arthritis and allied conditions: a textbook of rheumatology. Lippincott Williams & Wilkins, Philadelphia
- 11. Kuijt M-TK, Inklaar H, Gouttebarge V et al (2012) Knee and ankle osteoarthritis in former elite soccer players: a systematic review. J Sci Med Sport 15:480–487
- 12. Kujala UM, Kaprio J, Sarna S (1994) Osteoarthritis of weight bearing joints of lower limbs in former elite male athletes. BMJ 308:231–234
- Larsen E, Jensen PK, Jensen PR (1999) Long-term outcome of knee and ankle injuries in elite football. Scand J Med Sci Sports 9:285–289
- Lindsjö U (1985) Operative treatment of ankle fracture-dislocations. A follow-up study of 306/321 consecutive cases. Clin Orthop Relat Res 199:28–38
- McAlindon TE, LaValley MP, Gulin JP et al (2000) Glucosamine and chondroitin for treatment of osteoarthritis: a systematic quality assessment and meta-analysis. JAMA 283:1469–1475
- Migliore A, Giovannangeli F, Bizzi E et al (2011) Viscosupplementation in the management of ankle osteoarthritis: a review. Arch Orthop Trauma Surg 131:139–147
- Moskowitz RW, Altman RD, Hochberg MC et al (eds) (2007) Osteoarthritis. Wolters Kluwer/ Lippincott Williams & Wilkins, Philadelphia
- 18. Nakasone Y, Watabe K, Watanabe K et al (2011) Effect of glucosamine-based combination supplement containing chondroitin sulphate and antioxidant micronutrients in subjects with symptomatic knee osteoarthritis: a pilot study. Exp Ther Med 2:893–899

- Nolte S, Osborne RH (2012) A systematic review of outcomes of chronic disease selfmanagement interventions. Qual Life Res 22:1805–1816
- 20. Pagenstert G, Leumann A, Hintermann B et al (2008) Sports and recreation activity of varus and valgus ankle osteoarthritis before and after realignment surgery. Foot Ankle Int 29:985–993
- 21. Rao S, Ellis SJ, Deland JT et al (2010) Nonmedical therapy in the management of ankle osteoarthritis. Cur Opin Rheumatol 22:223–228
- 22. Roos EM, Juhl CB (2012) Osteoarthritis 2012 year in review: rehabilitation and outcomes. Osteoarthritis Cartilage 20:1477–1483
- 23. Saltzman CL, Hillis SL, Stolley MP et al (2012) Motion versus fixed distraction of the joint in the treatment of ankle osteoarthritis. J Bone Joint Surg Am 94:961–970
- 24. Semanik PA, Chang RW, Dunlop DD (2012) Aerobic activity in prevention and symptom control of osteoarthritis. PM R 4:S37–S44
- 25. Sinusas K (2012) Osteoarthritis: diagnosis and treatment. Am Fam Physician 85:49-56
- 26. Thomas RH, Daniels TR (2003) Ankle arthritis. J Bone Joint Surg 85:923-936
- Valderrabano V, Hintermann B, Horisberger M et al (2006) Ligamentous posttraumatic ankle osteoarthritis. Am J Sports Med 34:612–620
- Valderrabano V, Horisberger M, Russel I et al (2009) Etiology of ankle osteoarthritis. Clin Orthop Relat Res 467:1800–1806