

Victor Valderrabano  
Mark Easley  
*Editors*

# Foot and Ankle Sports Orthopaedics

---

# Foot and Ankle Sports Orthopaedics

---

Victor Valderrabano • Mark Easley  
Editors

# Foot and Ankle Sports Orthopaedics

 Springer

*Editors*

Victor Valderrabano  
Orthopaedic Department  
SWISS ORTHO CENTER  
Schmerzklinik Basel  
Swiss Medical Network Basel  
Basel Stadt  
Switzerland

Mark Easley  
Department of Orthopaedic Surgery  
Duke University Medical Center  
Durham  
North Carolina  
USA

Lecturing of the book by:

Dr. Ahmed E. Galhoum, M.Sc. MRCS (England), Orthopaedic Surgery Fellow, SWISS ORTHO CENTER, Schmerzklinik Basel, Swiss Medical Network, Basel, Switzerland; Nasser Institute for Research and Treatment, Cairo, Egypt

ISBN 978-3-319-15734-4      ISBN 978-3-319-15735-1 (eBook)  
DOI 10.1007/978-3-319-15735-1

Library of Congress Control Number: 2016963410

© Springer International Publishing Switzerland 2016

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.

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.

The publisher, the authors and the editors are safe to assume that the advice and information in this book are believed to be true and accurate at the date of publication. Neither the publisher nor the authors or the editors give a warranty, express or implied, with respect to the material contained herein or for any errors or omissions that may have been made.

Printed on acid-free paper

This Springer imprint is published by Springer Nature  
The registered company is Springer International Publishing AG  
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

---

# Contents

## Part I General Aspects

<b>1 Clinical Anatomy</b> . . . . .	3
Alexandre Leme Godoy-Santos, Stefan Rammelt, and Alejandro Carri Zoboli	
<b>2 Biomechanical Principles of Foot and Ankle</b> . . . . .	25
Matthias G. Walcher and Ryan du Sart	
<b>3 Overview and Epidemiology of Foot and Ankle Problems in Sports</b> . . . . .	35
John S. Lewis Jr. and Mark E. Easley	
<b>4 Clinical Examination</b> . . . . .	39
Alexander J. Lampley, Christopher E. Gross, Mitchell Klement, and Mark E. Easley	
<b>5 Sports Medicine of the Foot &amp; Ankle, the Role of Imaging</b> . . . . .	49
John Wesley Latting and Charles E. Spritzer	
<b>6 Biomechanical Functional Imaging</b> . . . . .	63
Thomas Jöllenbeck and Juliane Pietschmann	
<b>7 Arthroscopic Supplementation of Imaging Findings: Using Arthroscopy to Detect Abnormalities Missed on Imaging</b> . . . . .	75
Eric Ferkel and Bruce E. Cohen	
<b>8 Management of Injured Athletes at the Field</b> . . . . .	81
Thilo Hotfiel, Hans-Dieter Carl, Casper Grim, and Martin Engelhardt	
<b>9 Sports and Osteoarthritis</b> . . . . .	89
Thomas Schlemmer, Thomas Hügle, Victor Valderrabano, and Jochen Paul	
<b>10 Sports in Children and Young Age</b> . . . . .	97
Bernhard Speth and Carlo Camathias	
<b>11 Sports in Seniors</b> . . . . .	105
Krit Prugsawan and Monika Horisberger	
<b>12 Sports for Disabled and Handicapped People</b> . . . . .	111
Michèle Kläusler and Erich Rutz	
<b>13 Orthobiologics in Foot &amp; Ankle</b> . . . . .	119
Ethan S. Krell, Nicholas J. Montemurro, Kristen P. Pacific, Lionel Emele, and Sheldon S. Lin	
<b>14 Rehabilitation, Back to Sports and Competition</b> . . . . .	127
Abdulhameed Alattar and Shaju Kareem	

---

**Part II Clinics, Lesions, and Diseases**

<b>15 Acute Fractures (Lower Leg, Ankle, Hindfoot, Midfoot, Forefoot)</b> . . . . .	147
Milena M. Ploeger, Christof Burger, and Matthias D. Wimmer	
<b>16 Acute Ankle Osteochondral and Chondral Lesions</b> . . . . .	157
Martin Wiewiorski, Alexej Barg, Markus Wurm, and Victor Valderrabano	
<b>17 Stress Fractures of the Foot and Ankle</b> . . . . .	161
William Melton and J. Benjamin Jackson III	
<b>18 Compartment Syndromes (Acute &amp; Chronic)</b> . . . . .	171
Jonathan A. Godin, Travis J. Dekker, and C.T. Moorman III	
<b>19 Shin Splints</b> . . . . .	181
Sampat S. Dumbre Patil	
<b>20 Achilles Tendon</b> . . . . .	187
Matthijs Jacxsens, Lukas Weisskopf, Victor Valderrabano, and Claudio Rosso	
<b>21 Plantar Fasciitis in Sport</b> . . . . .	201
Yousef Alrashidi, Alexej Barg, Manuel Kampmann, and Victor Valderrabano	
<b>22 Exostosis (Osteochondrosis, Apophysites, and Haglund's Deformity)</b> . . . . .	209
Abdulaziz Almaawi, Andrzej Marcin Boszczyk, and Timothy R. Daniels	
<b>23 Posterior Tibial Tendon Lesions and Insufficiency</b> . . . . .	219
Yousef Alrashidi, Hasan N. Alsayed, Hamza M. Alrabai, and Victor Valderrabano	
<b>24 Peroneal Tendons</b> . . . . .	231
Mario Herrera-Perez and Anna Oller-Boix	
<b>25 Anterior Tibial Tendon Lesion</b> . . . . .	239
Ahmed Nabil Abdulazim, Victor Valderrabano, and Jochen Paul	
<b>26 Flexor Hallucis Longus Tendon</b> . . . . .	243
May Fong Mak and Mathieu Assal	
<b>27 Acute Ankle Instability/Ankle Sprains in Athletes</b> . . . . .	249
Travis J. Dekker, Alexander J. Lampley, Jonathan A. Godin, and Mark E. Easley	
<b>28 Chronic Ankle Instability</b> . . . . .	257
Jochen Paul, Christian Stelzenbach, and Victor Valderrabano	
<b>29 Acute Syndesmotic Injuries</b> . . . . .	265
Craig R. Lareau, Andrew R. Hsu, and Bruce E. Cohen	
<b>30 Acute and Chronic Subtalar, Chopart- and Lisfranc Instability</b> . . . . .	279
Sergio Tejero	
<b>31 Chronic Ankle Osteochondral and Chondral Lesions</b> . . . . .	291
Martin Wiewiorski, Alexej Barg, Beat Hintermann, and Victor Valderrabano	
<b>32 Anterior and Posterior Ankle Impingement</b> . . . . .	299
Norman Espinosa, Ana Fajardo-Ruiz, and Anita Hasler	
<b>33 Ankle Osteoarthritis</b> . . . . .	307
Alexej Barg, Christian J. Gaffney, and Victor Valderrabano	
<b>34 Subtalar, Chopart, and Lisfranc Joint Degeneration</b> . . . . .	329
Jeannie Huh, Christopher E. Gross, and James K. DeOrio	

<b>35 Coalition and Sports</b> . . . . .	341
Tetsuro Yasui	
<b>36 Hallux Valgus and Hallux Rigidus in Sports</b> . . . . .	347
Arno Frigg	
<b>37 Sesamoid and Capsule Lesions of the First Metatarsophalangeal Joint</b> . . . . .	355
Andrew R. Hsu and Robert B. Anderson	
<b>38 Metatarsophalangeal Problems II-V</b> . . . . .	365
Erik C. Nilssen and William K. Whiteside	
<b>39 Forefoot Sports Injuries</b> . . . . .	371
Johnny Lau and David Santone	
<b>40 Tarsal Tunnel Syndrome in Sport</b> . . . . .	377
Tim Schneider and Elango Selvarajah	
<b>41 Nerve Entrapments</b> . . . . .	383
Christopher E. Gross and James A. Nunley II	
<b>42 Morton's Neuroma in Sports</b> . . . . .	391
Hamza M. Alrabai, Yousef Alrashidi, Victor Valderrabano, and Marino Delmi	
<b>43 Soft Tissue Management in the Foot and Ankle</b> . . . . .	397
Rik Osinga, Andreas Gohritz, Martin D. Haug, and Dirk J. Schaefer	
 <b>Part III Sports Specific Injuries</b>	
<b>44 Aerobic and Fitness Sports</b> . . . . .	407
Christian Plaass, Christoph Becher, and Hauke Horstmann	
<b>45 American Football</b> . . . . .	413
Mark E. Magill and Robert B. Anderson	
<b>46 Athletic Injuries</b> . . . . .	421
Cristian Ortiz, Emilio Wagner, and Gonzalo Fernandez	
<b>47 Basketball</b> . . . . .	427
Christopher E. Gross, Jeannie Huh, and James A. Nunley II	
<b>48 Rock Climbing</b> . . . . .	437
Volker Schöffl	
<b>49 Cycling</b> . . . . .	441
Andreas Gösele-Koppenburg	
<b>50 Dance Injuries/Ballet</b> . . . . .	447
Martin Wiewiorski and Christie-Joy Cunningham	
<b>51 Equestrian Sports</b> . . . . .	453
Monika Horisberger, Martin Wiewiorski, and Alexej Barg	
<b>52 Football/Soccer</b> . . . . .	459
Martin Wiewiorski, Markus Wurm, Alexej Barg, Markus Weber, and Victor Valderrabano	
<b>53 Floorball</b> . . . . .	465
Markus Wurm and T. Schlemmer	
<b>54 Golf</b> . . . . .	469
Erik C. Nilssen and William K. Whiteside	

<b>55</b>	<b>Gymnastics</b> . . . . .	473
	Larry Nassar	
<b>56</b>	<b>Handball</b> . . . . .	479
	Christian Stelzenbach and Jochen Paul	
<b>57</b>	<b>Hiking, Mountaineering, Canyoning</b> . . . . .	485
	Thomas Schlemmer, Andreas von Roll, Markus Wurm, and Victor Valderrabano	
<b>58</b>	<b>Ice Hockey</b> . . . . .	491
	Lukas Weisskopf, Julian Röhm, and Thomas Hesse	
<b>59</b>	<b>In-Line and Roller Skating</b> . . . . .	497
	Christopher E. Gross and James K. DeOrio	
<b>60</b>	<b>Martial Arts</b> . . . . .	501
	Sebastian Mueller, Matthijs Jacxsens, and Claudio Rosso	
<b>61</b>	<b>Motorsports</b> . . . . .	505
	Christian Stelzenbach and Victor Valderrabano	
<b>62</b>	<b>Orienteering</b> . . . . .	509
	Daniel Bianco	
<b>63</b>	<b>Rugby</b> . . . . .	513
	Elango Selvarajah and Timothy Schneider	
<b>64</b>	<b>Running</b> . . . . .	521
	Dave Santone and Timothy R. Daniels	
<b>65</b>	<b>Alpine Sports</b> . . . . .	527
	Jannis Sailer, Martin Majewski, Matthias Gilgien, and Victor Valderrabano	
<b>66</b>	<b>Surfing: Board, Wind, Kite</b> . . . . .	535
	Karl-Heinz Kristen	
<b>67</b>	<b>Swimming/Aquatics</b> . . . . .	539
	Matthias D. Wimmer and Milena M. Ploeger	
<b>68</b>	<b>Snowboarding</b> . . . . .	541
	Klaus Dann	
<b>69</b>	<b>Tennis and Racquet Sports</b> . . . . .	549
	Christian Egloff and Victor Valderrabano	
<b>70</b>	<b>Volleyball</b> . . . . .	553
	Oliver Miltner and Markus Wurm	
<b>71</b>	<b>Water Sports</b> . . . . .	557
	Mario Herrera-Perez and Anna Oller-Boix	
	<b>Index</b> . . . . .	561



---

## Contributors

**Ahmed Nabil Abdulazim, MD** Orthopaedic Department, University Hospital, University of Basel, Basel, Switzerland

**Daniel Bianco Adames, MD** Orthopaedic Department, University Hospital Basel, Basel, Switzerland

**Abdulhameed Alattar, BA, MBBCh, MRCP, ABFM, MSc SEM** Department of Physical Medicine & Rehabilitation, Rashid Hospital, Dubai, United Arab Emirates

**Abdulaziz Almaawi, MD, FRCSC** Department of Surgery, University of Toronto, St. Michael's Hospital, Toronto, ON, Canada

**Hamza M. Alrabai, MD** Department of Orthopaedics, King Saud University Hospital, King Saud University, College of Medicine, Riyadh, Saudi Arabia

**Yousef Alrashidi, MBBS, SB-Orth** Orthopedic Surgery Department, Taibah University, College of Medicine, Almadinah Almunawwrah, Saudi Arabia

Orthopaedic Department, SWISS ORTHO CENTER, Schmerzlinik Basel, Swiss Medical Network, Basel, Switzerland

**Hasan N. Alsayed, Saudi Board in Orthopedics** Department of Orthopedic Surgery, King Fahd Hospital of the University – University of Dammam, Dammam, Eastern Province, Saudi Arabia

**Robert B. Anderson, MD** Department of Orthopaedic Surgery, OrthoCarolina Foot & Ankle Institute, Charlotte, NC, USA

**Mathieu Assal, MD** Clinique La Colline, Center for Surgery of the Foot and Ankle, Geneva, Switzerland

**Alexej Barg, MD** Department of Orthopaedics, University of Utah, Salt Lake City, UT, USA

**Christoph Becher, MD** Department for Orthopaedic Surgery, Hannover Medical School, Hannover, Germany

**Andrzej Marcin Boszczyk, MD, PhD** Department of Traumatology and Orthopaedics, Adam Gruca Autonomous Public Clinical Hospital, Otwock, Poland

**Christof Burger, MD** Department of Orthopedics and Trauma Surgery, University Clinic of Bonn, Bonn, Germany

**Carlo Camathias, MD** Department of Pediatric Orthopedic Surgery, University Children's Hospital Basel (UKBB), Basel, Switzerland

**Hans-Dieter Carl** Division of Orthopedic Rheumatology, University of Erlangen-Nuremberg, Erlangen, Germany

**Alejandro Carri Zoboli, MD** Department of Orthopaedic Surgery, Orthopaedics and Traumatology Institute, São Paulo, São Paulo, SP, Brazil

**Bruce E. Cohen, MD** OrthoCarolina Foot and Ankle Institute, Charlotte, NC, USA

**Christie-Joy Cunningham, BA** Universitätsspital Basel, Osteoarthritis Research Center Basel, Basel, Switzerland

**Timothy R. Daniels, MD, FRCSC** Professor, Department of Orthopaedic Surgery, St. Michael's Hospital, University of Toronto, Toronto, ON, Canada

**Klaus Dann, MD** Department of Trauma Surgery, Arthroscopy, Sports Medicine, Sports Trauma, Rudolfiner Hospital, TOP-MED, Zentrum f. Traumatologie-Orthopädie-Plastische Chirurgie, Vienna, Austria

**Travis J. Dekker, MD** Department of Orthopaedics, Duke University Hospital, Durham, NC, USA

**Marino Delmi, MD** Clinique des Grangettes, Foot & Ankle Center, Chêne-Bougeries, Geneva, Switzerland

**James K. DeOrto, MD** Department of Orthopaedic Surgery, Duke University Medical Center, Durham, NC, USA

**Ryan du Sart, MBChB** Department of Orthopaedic Surgery, Sir Charles Gardner Hospital, Nedlands, Perth, WA, Australia

**Mark E. Easley, MD** Department of Orthopaedic Surgery, Duke University Medical Center, Durham, NC, USA

**Christian Egloff, MD** Department of Orthopaedic Surgery, University Hospital Basel, Basel, Switzerland

**Lionel Emele, BA** Department of Orthopaedics, New Jersey Medical School, Newark, NJ, USA

**Martin Engelhardt** Orthopädie, Klinikum Osnabrück, Osnabrück, Germany

**Norman Espinosa, MD** Institute for Foot and Ankle Reconstruction Zurich, Zurich, Switzerland

**Eric Ferkel, MD** OrthoCarolina Foot and Ankle Institute, Charlotte, NC, USA

**Gonzalo Fernandez, MD** Staff, Foot and Ankle Unit, Department of Orthopedics and Traumatology, Clinica Alemana, Santiago, Vitacura, Chile

**Arno Frigg, MD** Foot and Ankle Surgery Zürich, Zürich, Switzerland

**Christian J. Gaffney, MD MSc** Department of Orthopaedics, University of Utah, Salt Lake City, UT, USA

**Matthias Gilgien, PhD** Center for Alpine Sports Biomechanics, Samedan, Switzerland  
Department for Physical Performance, Norwegian School of Sport Sciences, Oslo, Norway

**Jonathan A. Godin, MD, MBA** Department of Orthopedic Surgery, Duke University Hospital, Durham, NC, USA

**Alexandre Leme Godoy-Santos, MD, PhD** Department of Orthopaedic Surgery, University of São Paulo, Orthopaedics and Traumatology Institute, São Paulo, SP, Brazil

**Andreas Gohritz, MD** Department of Plastic, Reconstructive, Aesthetic and Hand Surgery, University Hospital of Basel, Basel, BS, Switzerland

- Andreas Gösele-Koppenburg, MD** Crossklinik, Swiss Olympic Medical Center, Basel, BS, Switzerland
- Casper Grim** Klinikum Osnabrück, Orthopädie, Unfall- und Handchirurgie, Osnabrück, Germany
- Christopher E. Gross, MD** Department of Orthopaedic Surgery, Duke University Medical Center, Durham, NC, USA
- Anita Hasler** Department of Orthopaedics, Universitätsklinik Balgrist, Zürich, Switzerland
- Martin D. Haug, MD** Department of Plastic, Reconstructive, Aesthetic and Hand Surgery, University Hospital of Basel, Basel, BS, Switzerland
- Mario Herrera-Perez, MD, PhD** Department of Orthopaedics, University Hospital of Canary Islands, Carretera De La Cuesta S/N, La Laguna, Tenerife, Spain
- Thomas Hesse, MD** ALTIUS Swiss Sportmed Center AG, Rheinfelden, Aargau, Switzerland
- Beat Hintermann, MD** Kantonsspital Baselland, Orthopaedic Clinic, Liestal, BL, Switzerland
- Monika Horisberger, MD** Orthopaedic Department, University Hospital Basel, Basel, Switzerland
- Hauke Horstmann** Hannover Medical School, Institut of Sports Medicine, Hannover, Niedersachsen, Germany
- Thilo Hotfiel, MD** Division of Orthopaedic Rheumatology, University of Erlangen-Nuremberg, Erlangen, Germany
- Andrew R. Hsu, MD** Department of Orthopaedic Surgery, OrthoCarolina Foot & Ankle Institute, Charlotte, NC, USA
- Thomas Hügle, MD, PhD** Department of Orthopaedic Surgery/Rheumatology, University Hospital Basel, Basel, Switzerland
- Jeannie Huh, MD** Department of Orthopaedic Surgery, Duke University Medical Center, Durham, NC, USA
- J. Benjamin Jackson III, MD** Department of Orthopaedics, University of South Carolina, Columbia, SC, USA
- Matthijs Jacxsens, MD** Department of Orthopaedic Surgery, University Hospital Basel, Basel, Switzerland
- Thomas Jöllenbeck** Klinik Lindenplatz, Institut für Biomechanik, Bad Sassendorf, Germany
- Manuel Kampmann** Department of Radiology, University of Basel, Basel, BS, Switzerland
- Shaju Kareem, BSc Physiotherapy** NAS Sports Medicine & Rehabilitation Centre, Nad Al Sheba, Dubai, United Arab Emirates
- Karl-Heinz Kristen, MD** Department of Orthopaedic Sports Medicine, Sportklinik, Wien, Austria
- Michèle Kläusler, MD** Pediatric Orthopaedic Department, University Children's Hospital, Basel, Switzerland
- Mitchell R. Klement, MD** Department of Orthopaedics, Duke University Hospital, Durham, NC, USA
- Ethan S. Krell, BS** Department of Orthopaedics, New Jersey Medical School, Newark, NJ, USA

**Alexander J. Lampley, MD** Department of Orthopaedic Surgery, Duke University Medical Center, Durham, NC, USA

**Craig R. Lareau, MD** Department of Orthopaedic Surgery, OrthoCarolina Foot & Ankle Institute, Charlotte, NC, USA

**John Wesley Latting, MD** Department of Radiology, Duke University Hospital, Durham, NC, USA

**Johnny Lau, MD, MSc, FRCSC** Department of Orthopedic Surgery, University Health Network – Toronto Western Division, Toronto, ON, Canada

**John S. Lewis, MD** Department of Orthopaedic Surgery, Duke University, Duke University Medical Center, Durham, NC, USA

**Sheldon S. Lin, MD** Department of Orthopaedics, New Jersey Medical School, Newark, NJ, USA

**Mark E. Magill, MD** Department of Orthopedic Surgery, Charlotte Medical Center, Charlotte, NC, USA

**Martin Majewski, MD, MBA** Department of Orthopedic Surgery, University Hospital Basel, Basel, Switzerland

**May Fong Mak, FRCSEd (Ortho)** Khoo Teck Puat Hospital, Singapore, Singapore

**William Melton, MD** Department of Orthopaedics, Palmetto Health Richland, Columbia, SC, USA

**Oliver Miltner** DocOrtho – Die Bewegungsprofis, Berlin, Germany

**Nicholas J. Montemurro, BS** Department of Orthopaedics, New Jersey Medical School, Newark, NJ, USA

**C.T. Moorman III, MD** Department of Orthopaedics, Duke University Medical Center, Durham, NC, USA

**Sebastian Müller, MD, Dr. med.** Department of Orthopaedics and Traumatology, University Hospital Basel, Basel, Switzerland

**Larry Nassar, DO, FAOASM** Department of Radiology, Division of Sports Medicine, Michigan State University, East Lansing, MI, USA

**Erik C. Nilssen, MD** Orthopaedic Foot and Ankle Department, Gulf Breeze Hospital of Baptist Healthcare, Andrews Orthopaedic & Sports Medicine Center, Gulf Breeze, FL, USA

**James A. Nunley II, MD** Department of Orthopaedic Surgery, Duke University Medical Center, Durham, NC, USA

**Anna Oller-Boix, MD** Department of Orthopaedics, University Hospital of Canary Islands, La Laguna, Tenerife, Spain

**Cristian Ortiz, MD** Foot and Ankle Unit, Department of Orthopedics and Traumatology, Clinica Alemana, Vitacura, Santiago, Chile

**Rik Osinga, MD** Department of Plastic, Reconstructive, Aesthetic and Hand Surgery, University Hospital of Basel, Basel, BS, Switzerland

**Kristen P. Pacific, BA** Department of Orthopaedics, New Jersey Medical School, Newark, NJ, USA

**Sampat S. Dumbre Patil, MBBS, D, DNB, MNAMS** Consultant Orthopedic Surgeon, Noble Hospital, Magarpatta City Road, Pune, Maharashtra, India

- Jochen Paul, MD** Rennbahnklinik, MuttENZ, Basel, Switzerland
- Juliane Pietschmann, MA** Klinik Lindenplatz, Institut für Biomechanik, Bad Sassendorf, Germany
- Christian Plaass, MD** Department for Foot and Ankle Surgery, Orthopedic Clinic, Hannover Medical School, Hannover, Germany
- Milena M. Ploeger, MD** Department of Orthopedics and Trauma Surgery, University Clinic of Bonn, Bonn, Germany
- Krit Prugsawan, MD** Department of Orthopaedics, Vejthani Hospital, Bangkok, Thailand
- Stefan Rammelt, MD, PhD** University Hospital Carl Gustav Carus, Dresden, University Center of Orthopaedics and Traumatology, Dresden, Germany
- Julian Röhm, MD** Department of Orthopaedic Surgery and Traumatology, Kantonsspital, Liestal, Baselland, Switzerland
- Claudio Rosso, MD, MSc, PD Dr. med.** Shoulder and Elbow Center, Arthro Medics, Basel, Switzerland
- Ana Fajardo-Ruiz, MD** Department of Traumatology and Orthopaedic Surgery, University Hospital of Torrejon, Torrejon de Ardoz, Madrid, Spain
- Erich Rutz, MD** Pediatric Orthopaedic Department, University Children's Hospital, Basel, Basel, Switzerland
- Jannis Sailer, MD** Department of Traumatology and Orthopedic Surgery, University Hospital Basel, Basel, BS, Switzerland
- David Santone, MSc, MD, FRCSC** Department of Orthopedic Surgery, Markham Stouffville Hospital, Markham, ON, Canada
- Dirk J. Schaefer, MD** Department of Plastic, Reconstructive, Aesthetic and Hand Surgery, University Hospital of Basel, Basel, BS, Switzerland
- Thomas Schlemmer, MD** Orthopaedic Department, University Hospital Basel, Basel, Switzerland
- Timothy Schneider, MBBS, FRACS** Melbourne Orthopaedic Group, Windsor, VIC, Australia
- Volker Schöffl, MD, PhD, MHBA** Klinikum Bamberg, Sportorthopedics, Sportsmedicine, Shoulder and Ellbow Surgery, Bamberg, Bavaria, Germany
- Elango Selvarajah, FRACS MB ChB** Foot and Ankle Department, Melbourne Orthopaedic Group, Melbourne, VIC, Australia
- Bernhard Speth, MD** Department of Pediatric Orthopedic Surgery, University Children's Hospital Basel (UKBB), Basel, Switzerland
- Charles E. Spritzer, MD** Department of Radiology, Chief MSK Division, Duke University School of Medicine, Duke University Medical Center, Durham, NC, USA
- Christian Stelzenbach, MD** Orthopaedic Department, University Hospital, University Basel, Basel, Switzerland
- Sergio Tejero, PhD, MD** Foot and Ankle Unit, Virgen del Rocío, Bormujos, Sevilla, Spain
- Victor Valderrabano, MD, PhD** Orthopaedic Department, SWISS ORTHO CENTER, Schmerzlinik Basel, Swiss Medical Network, Basel, Switzerland

**Andreas von Roll, MD** Department of Orthopedic Surgery and Sports Medicine,  
Privatklinik Obach, Solothurn, Switzerland

**Emilio Wagner, MD** Foot and Ankle Unit, Department of Orthopedics and Traumatology,  
Clinica Alemana, Vitacura, Santiago, Chile

**Matthias G. Walcher, MD** Head of Foot and Ankle Unit, Department of Orthopaedic  
Surgery, University of Würzburg, König-Ludwig-Haus, Würzburg, Germany

**Markus Weber,** Club Physician FC Basel 1893, Praxis Neumatt, Aesch, Switzerland

**Lukas Weisskopf, MD** Altius Swiss Sportmed Center, Rheinfelden, AG, Switzerland

**William K. Whiteside, MD** Orthopaedic Foot and Ankle Department,  
Gulf Breeze Hospital of Baptist Healthcare, Andrews Orthopaedic & Sports Medicine Center,  
Gulf Breeze, FL, USA

**Martin Wiewiorski, MD** Department of Orthopedics and Traumatology,  
Kantonsspital Winterthur, Winterthur, Switzerland

**Matthias D. Wimmer, MD** Department of Orthopedics and Trauma Surgery,  
University Clinic of Bonn, Bonn, Germany

**Markus Wurm, MD** Department of Orthopaedics and Traumatology,  
University Hospital Basel, Basel, Switzerland

**Tetsuro Yasui, MD, PhD** Department of Orthopaedic Surgery,  
University of Tokyo Hospital, Tokyo, Japan

---

**Part I**

**General Aspects**

Alexandre Leme Godoy-Santos, Stefan Rammelt,  
and Alejandro Carri Zoboli

## Abstract

This chapter presents the most frequent approaches in foot and ankle surgery based on a summarized session of surface anatomy with landmarks: anteroposterior view, posteroanterior view, lateral view, medial view; it is followed by an illustrated step-by-step description of ten extended incisions: anterolateral suprafibular approach, medial ankle approach, anterior ankle approach, posterolateral ankle approach, posteromedial ankle approach, midfoot approaches – dorsomedial approach, dorsal intermediate approach, dorsolateral approach, first metatarsophalangeal joint approach, dorsal intermetatarsal space approach, which include 15 different approaches.

## Keywords

Anatomy • Dissection • Surgery • Incision • Approach • Foot • Ankle

The surgical safety depends on a thorough knowledge of the anatomy and surgical skills, both go together. The surgical skill is acquired from practices under expert supervision, and the knowledge of anatomy is acquired from books and dissection.

In 1909, Testut and Jacob recommended that “the human body should be transparent like crystal” for an excellent surgeon.

This chapter presents the most frequent approaches in foot and ankle surgery based on a summarized session of surface anatomy with landmarks, followed by an illustrated step-by-step description of ten extended incisions which include 15 different approaches.

---

A.L. Godoy-Santos, MD, PhD (✉) • A.C. Zoboli, MD  
Department of Orthopaedic Surgery, Orthopaedics and  
Traumatology Institute, São Paulo, Rua Dr. Ovídio Pires de  
Campos 333, São Paulo 05403-010, SP, Brazil  
e-mail: [alexandrelemegodoy@gmail.com](mailto:alexandrelemegodoy@gmail.com);  
[alejandrozobolli@gmail.com](mailto:alejandrozobolli@gmail.com)

S. Rammelt, MD, PhD  
University Hospital Carl Gustav Carus, Dresden,  
University Center of Orthopaedics and Traumatology,  
Fetscherstrasse 74, Dresden 01307, Germany  
e-mail: [strammelt@hotmail.com](mailto:strammelt@hotmail.com)

## Surface Anatomy

The knowledge of the surface anatomy around the ankle is important for adequate tailoring of the surgical approaches and safe placement of arthroscopy portals.

## Anteroposterior View

From the front it is possible to identify the orientation of the lateral and medial malleolus (Fig. 1.1), the anterior tibial and extensor digitorum longus tendons which cross the deep anterior neurovascular bundle and serve as references for the superficial peroneal and saphenous nerve branches (Fig. 1.2).

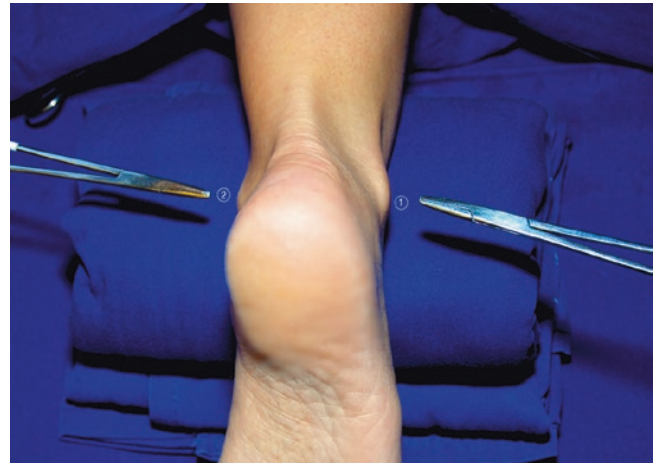
## Posteroanterior View

From the back it is possible to identify the posterior tuberosity of the calcaneus, Achilles tendon, peroneus longus tendon, and posterior tibial tendon which serve as references for the deep posterior neurovascular bundle and the sural nerve (Figs. 1.3 and 1.4).

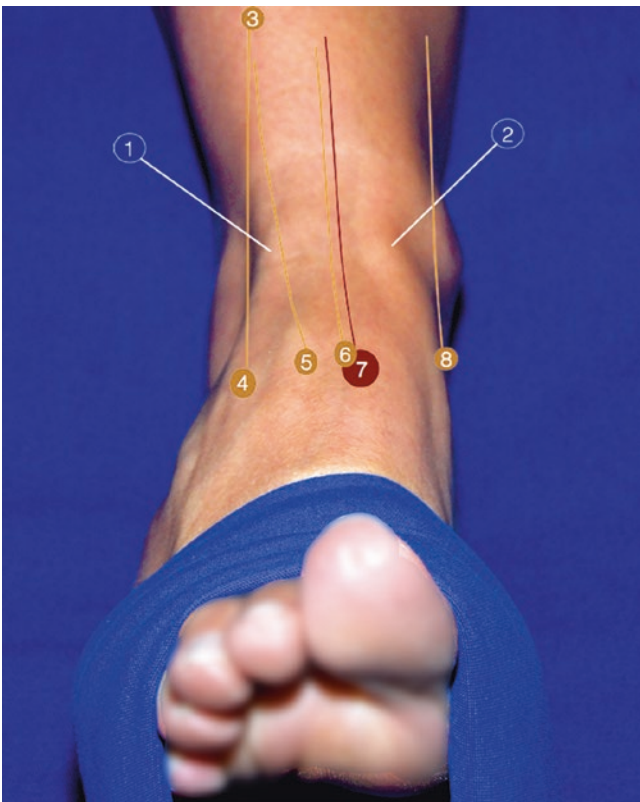




**Fig 1.1** 1. Lateral malleolus, 2. medial malleolus



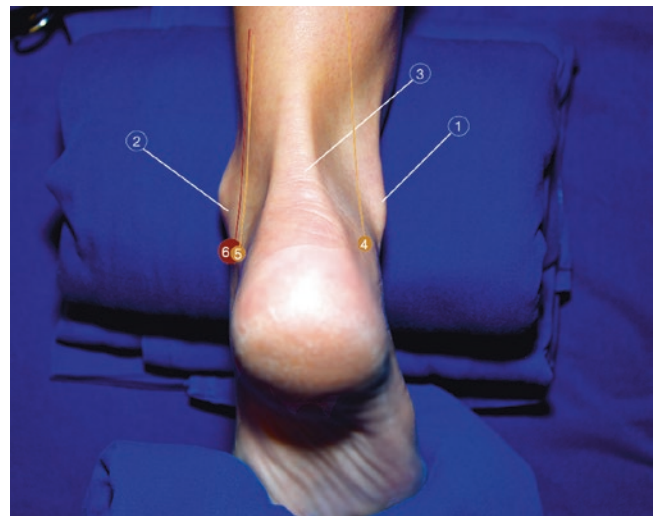
**Fig 1.3** 1. Lateral malleolus, 2. medial malleolus



**Fig 1.2** 1. Extensor digitorum longus tendon, 2. anterior tibial tendon, 3. superficial peroneal nerve, 4. intermediate branch of the dorsal cutaneous nerve, 5. medial branch of the dorsal cutaneous nerve, 6. deep peroneal nerve, 7. anterior tibial artery, 8. saphenous nerve

### Lateral View

From lateral it is possible to identify the lateral malleolus, the peroneal tendons, the base of the fifth metatarsal and the extensor brevis muscle which are references for the sural nerve and superficial peroneal nerve (Figs. 1.5 and 1.6).



**Fig 1.4** 1. Peroneus longus tendon, 2. posterior tibial tendon, 3. Achilles tendon, 4. sural nerve, 5. tibial nerve, 6. posterior tibial artery

### Medial View

From medial it is possible to identify the medial malleolus, the navicular tuberosity, the posterior tibial tendon, the deltoid ligament and the Achilles tendon which are references for the deep posterior neurovascular bundle (Figs. 1.7 and 1.8).

## Surgical Approaches

### Anterolateral Suprafibular Approach

#### Included

- Anterolateral subtalar joint approach
- Oblique lateral approach
- Sinus tarsi approach



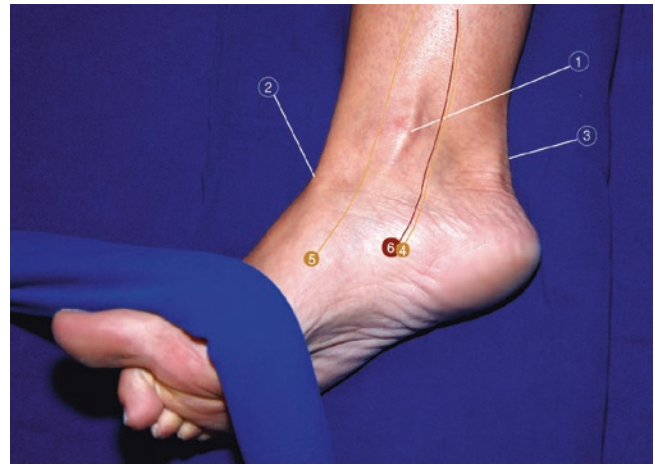
**Fig. 1.5** 1. Lateral malleolus, 2. base of the fifth metatarsal



**Fig. 1.7** 1. Medial malleolus, 2. navicular tuberosity



**Fig. 1.6** 1. Peroneus longus tendon, 2. extensor digitorum longus tendon, 3. extensor digitorum brevis muscle, 4. intermediate branch of dorsal cutaneous nerve, 5. medial branch of dorsal cutaneous nerve (both branches from the superficial peroneal nerve), 6. sural nerve (continuing as lateral branch of the dorsal cutaneous nerve)



**Fig. 1.8** 1. Posterior tibial tendon, 2. anterior tibial tendon, 3. Achilles tendon, 4. tibial nerve, 5. saphenous nerve, 6. posterior tibial artery

### Indications

- Lateral ankle ligament reconstruction
- Peroneal tendon exploration/repair
- Retinacular repair/groove deepening
- Os trigonum excision
- Anterolateral talar dome exposure
- Lateral process fractures of the talus
- Subtalar joint loose body excision
- Subtalar arthrodesis
- Calcaneal fractures (posterior facet exposure)
- Exposure of the calcaneocuboid (CC) joint
- Excision of the calcaneonavicular coalitions

### Position

Supine with a sandbag or bump underneath the ipsilateral hip or lateral oblique position

### Hazards

The sural nerve runs parallel and inferior to the incision. It has to be held away plantarly and protected. The superficial peroneal nerve runs above the incision and has to be protected with an oblique (Ollier's) approach.

### Superficial Dissection

Make a 6–8 cm incision starting at the posterior border of the fibular tip slightly curved towards the base of the fifth metatarsal (Fig. 1.9).

For dissection purposes the anterior and posterior skin flaps are elevated, taking care to protect the superficial peroneal nerve and sural nerve. Observe the peroneal sheaths, the



**Fig. 1.9** Anterolateral subfibular approach



**Fig. 1.11** Peroneus longus (1) and peroneus brevis tendons (2)



**Fig. 1.10** Sural nerve (1) and peroneal tendon sheath (2)

fat pad from the sinus tarsi and extensor digitorum brevis muscle (Fig. 1.10).

### Deep Dissection

The peroneal tendons (Fig. 1.11), the anterior talofibular ligament (ATFL, Fig. 1.12), the calcaneofibular ligament (CFL) crossing below the peroneal tendons (Fig. 1.13), the anterolateral talar dome after anterior lateral ankle capsulotomy (Fig. 1.14), the inferior extensor Retinacula (Fig. 1.15), the peroneal groove of the fibula (Fig. 1.16), the lateral portion of the subtalar joint (Fig. 1.17) and the calcaneocuboid joint below the extensor digitorum brevis muscle (Fig. 1.18) are inspected.



**Fig. 1.12** Anterior talofibular ligament (ATFL)



**Fig. 1.13** Calcaneofibular ligament (CFL)

### Medial Approach

#### Included

- Medial malleolar fracture and osteotomy approach
- Medial approach to the talus

- Sustentacular approach to the calcaneus
- Tarsal tunnel and posterior tibial tendon approach
- Medial utility approach (extending to the midfoot as needed)



**Fig. 1.14** Anterolateral talar dome (1) after anterolateral ankle capsulotomy



**Fig. 1.15** Inferior extensor retinacle



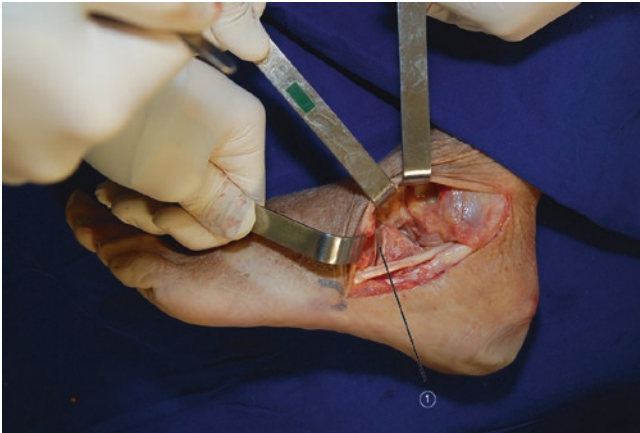
**Fig. 1.16** Peroneal groove of the distal fibula



**Fig. 1.17** Lateral exposure of the subtalar joint

### Indications

- Open reduction and internal fixation for displaced medial malleolar fractures
- Open reduction and internal fixation for displaced talar neck fractures
- Open reduction and internal fixation for talar body fractures (with medial malleolar osteotomy)
- Treatment of osteochondral lesions of the medial talar dome (with medial malleolar osteotomy)
- Posteromedial osteochondral talar lesions
- Medial ligament reconstruction (Deltoid, Spring ligament)
- Ankle loose bodies and osteochondral fragment removal
- Double/Triple arthrodesis (single approach)
- Talonavicular arthrodesis (medial approach)
- Tibialis posterior tendon exploration/reconstruction (including FDL-transfer)
- Flexor digitorum longus (FDL) tendon exploration/repair
- Flexor hallucis longus (FHL) tendon exploration/repair
- Kidner-Procedure
- Tarsal tunnel release
- Exposure of the posteromedial tibia
- Fractures of the sustentaculum tali (short direct approach)



**Fig. 1.18** The calcaneocuboid joint (1) is inspected below the peroneal brevis tendon

### Position

Supine with a sandbag or bump underneath the contralateral hip or medial oblique position

### Hazards

The saphenous nerve variably crosses the ankle at its antero-medial portion; it can be injured throughout the surgical incision.

The tibial nerve and posterior tibial artery run behind the tibialis posterior and FDL tendons. Both are endangered when dissecting around the FHL tendon.

### Superficial Dissection

Make a 12–15 cm incision along the posterior border of the medial malleolus; then curve it slightly towards the navicular tuberosity (Fig. 1.19). For dissection purposes the anterior skin flap is elevated, taking care to protect the saphenous nerve and vein and identifying the extensor retinaculum (Fig. 1.20).

### Deep Dissection

The posterior tibial and flexor digitorum longus tendon (Fig. 1.21), the neurovascular bundle (Fig. 1.22), the flexor hallucis longus tendon (Fig. 1.23), superficial deltoid ligament (Fig. 1.24), the anterior tibial tendon (Fig. 1.25), the anteromedial talar dome can be inspected after anterior medial ankle capsulotomy (Fig. 1.26), the medial talar dome only after a medial malleolar osteotomy, the tibiospring ligament is observed and the deep deltoid ligament are inspected (Fig. 1.27).



**Fig 1.19** Medial extended approach



**Fig. 1.20** The saphenous vein running over the medial aspect of the extensor retinaculum



**Fig. 1.21** Posterior tibial (1) and flexor digitorum longus tendons (2)



**Fig. 1.22** Deep posterior neurovascular bundle (1) running behind the posterior tibial (2) and flexor digitorum longus tendons (3)



**Fig 1.25** Anterior tibial tendon



**Fig 1.23** Flexor hallucis longus tendon



**Fig 1.26** View of the anteromedial talar dome after an anterior medial capsulotomy



**Fig 1.24** Tibionavicular portion of the superficial deltoid ligament (\*)



**Fig 1.27** Exposure of the talar head and neck with the spring ligament (1) and retracted tibionavicular portion of the superficial deltoid ligament (2). The sustentaculum tali of the calcaneus lies at the tip of the blunt retractor

## Anterior Ankle Approach

### Indications

- Anterior ankle impingement
- Ankle arthrodesis
- Total ankle arthroplasty
- Open reduction and internal fixation of tibial pilon and talar body fractures

### Position

Place a bump under the ipsilateral hip to maintain neutral rotation.

### Hazards

The superficial peroneal nerve and the deep anterior neurovascular bundle (anterior tibial artery, deep peroneal nerve) may be injured.

### Incision

Make a 15 cm midline incision between the malleoli over the anterior aspect of the ankle, beginning 10 cm proximal to the joint line and lateral to the palpable tibial anterior tendon (Fig. 1.28). When dissecting the subcutaneous tissue care is taken not to injure the branches of the superficial peroneal nerve that cross from lateral into the distal aspect of the incision (Fig. 1.29)

### Superficial Dissection

Open the superior extensor retinaculum between the tibialis anterior (TA) and extensor hallucis longus (EHL) tendons (Fig. 1.30). Retract the EHL laterally and observe the anterior tibial artery and deep peroneal nerve (Fig. 1.31).

### Deep Dissection

Retract TA medially and EHL with anterior tibial artery and deep peroneal nerve – protected – laterally to visualize the anterior ankle joint capsule (Fig. 1.32). Incise the capsule of the ankle joint in line with the incision. Expose the full width of the ankle joint by subperiosteal and subcapsular dissection



**Fig. 1.28** Midline anterior ankle approach

of the tibia and talus (Fig. 1.33). Retractors should be placed on the periosteal layer to avoid excessive pressure on the skin or neurovascular bundle (Fig. 1.34).

## Posterolateral Approach

### Indications

- Reconstruction and lengthening of the peroneal tendons and the Achilles tendon
- Open reduction and internal fixation of displaced posterior malleolar fractures
- Open reduction and internal fixation of displaced lateral malleolar fractures
- Open reduction and internal fixation of displaced fractures of the posterior body of the talus
- Open treatment of osteochondral defects of the posterolateral talus
- Arthrodesis of the subtalar and/or ankle joint
- Removal of a symptomatic os trigonum



**Fig. 1.29** The superficial peroneal nerve branches cross the distal part of the incision from lateral

### Position

Supine with a sandbag or bump underneath the ipsilateral hip or lateral oblique position or lateral total.

### Hazards

The sural nerve and the lesser saphenous vein cross the incision from medial and may be injured in the proximal part of the incision.

### Superficial Dissection

Define the posterolateral border of lateral malleolus and posterolateral border of the Achilles tendon. Make a 10–12 cm



**Fig. 1.30** After opening the superior extensor retinacle, TA sheath and EHL sheath

longitudinal incision at half distance between and parallel to them (Fig. 1.35). Elevate the posterior skin flap, taking care to protect the sural nerve and lesser saphenous vein (Fig. 1.36).

### Deep Dissection

Incise the deep calf fascia and superior peroneal retinaculum in line with the skin incision and identify the peroneus longus tendon which lies posterolateral and has less muscle fibers at this level (Fig. 1.37), mobilize the tendon posterolaterally to expose the peroneus brevis tendon which lies anterior and has more muscle fibers behind the fibula (Fig. 1.38). Finally, mobilize both tendons posterolaterally and expose posterior aspect of lateral malleolus (Fig. 1.39).

In the posterior plane incise the deep fascia over the flexor hallucis longus (FHL) muscle (Fig. 1.40) to expose posterior





**Fig 1.31** The anterior tibial artery and deep peroneal nerve (above the strap) become visible when retracting the EHL tendon laterally



**Fig 1.32** Anterior ankle joint capsule

talofibular ligament (Fig. 1.41), the posterior tibiofibular ligament (posterior syndesmosis), FHL tendon, posterior aspect of the subtalar joint, the posterior talar body and posterior tubercle of the distal tibia (Fig. 1.42).

## Posteromedial Approach

### Indications

- Achilles tendon rupture
- Achilles tendinopathy
- Haglund exostosis resection
- Flexor hallucis longus transfer
- Posteromedial talar body fracture fixation

### Position

Prone position

### Hazards

The saphenous nerve and vein may be injured along the incision. The deep posterior neurovascular bundle may be injured during deep dissection.

### Incision

Make a 10 cm longitudinal incision 1 cm medial and parallel to the Achilles tendon (Fig. 1.43) to improve soft tissue coverage over the tendon after repair the deep fascia over the Achilles tendon is incised medial to the tendon (Fig. 1.44)

### Superficial Dissection

The Achilles paratenon is incised at the midline so that the paratenon can be closed after the Achilles tendon suture (Fig. 1.45).



**Fig 1.33** Capsulotomy



**Fig 1.34** Ankle joint (full exposure after capsulotomy)

## Deep Dissection

After retraction of the Achilles tendon the Kager fat pad is seen (Fig. 1.46) and the extension of a Haglund exostosis is estimated (Fig. 1.47). The deep fascia over the FHL (Fig. 1.48) can be incised at the midline to expose the FHL tendon (Fig. 1.49). Finally, with the FHL tendon and deep posterior neurovascular bundle retracted, the posterior aspect of the subtalar and ankle joints are exposed (Fig. 1.50).

## Midfoot Approaches

### Dorsomedial Approach

#### Indications

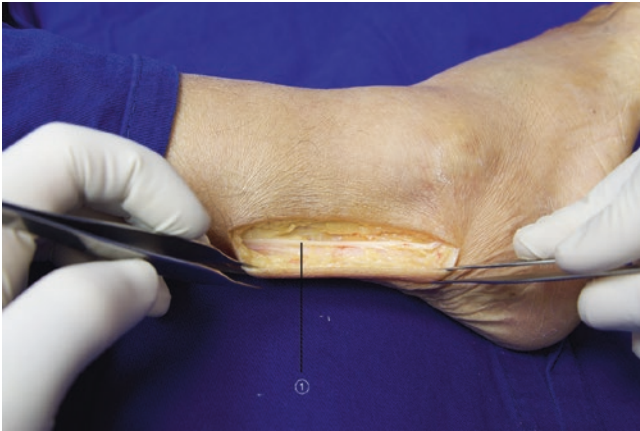
- First TMT (Lapidus) arthrodesis
- Naviculo-cuneiform arthrodesis
- Basal MT 1 osteotomy
- Cotton osteotomy
- Open reduction and fixation of navicular, cuneiform and first MT fractures



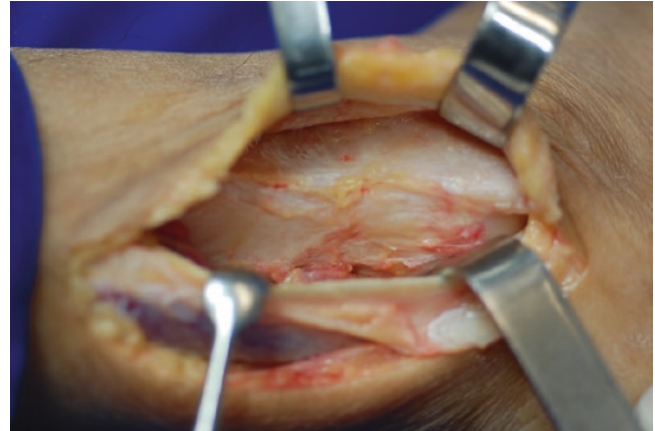
**Fig. 1.35** Full extension of the posterolateral approach

#### Position

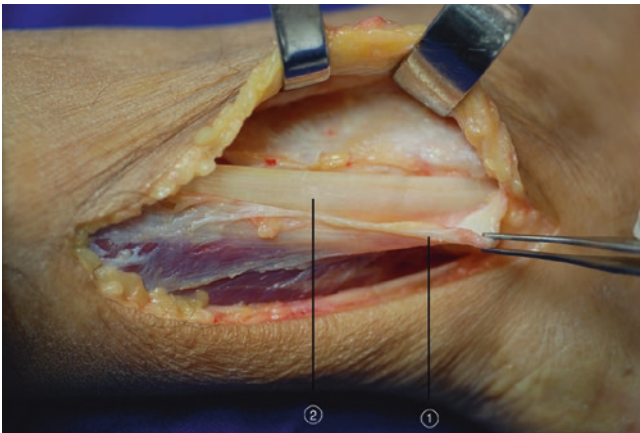
Supine with a sandbag or bump underneath the contralateral hip or medial oblique position.



**Fig. 1.36** posterolateral ankle approach and sural nerve (1) crossing from medial



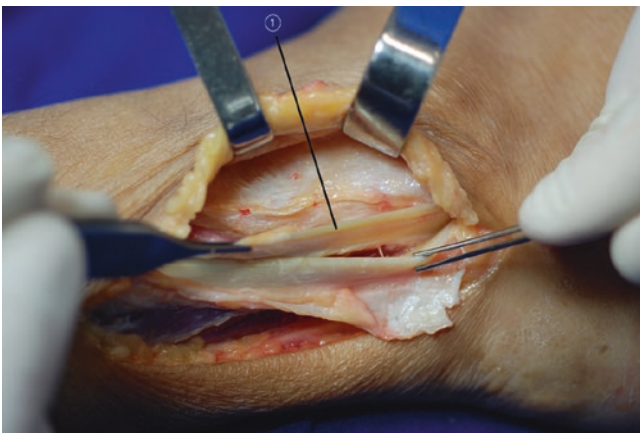
**Fig. 1.39** Posterior aspect of the lateral malleolus after retraction of the peroneal tendons



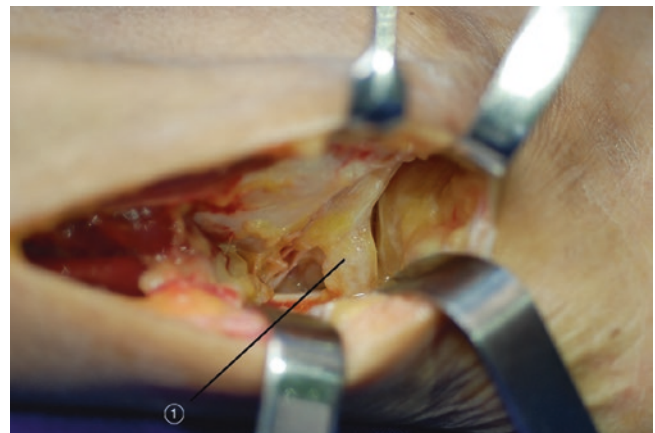
**Fig. 1.37** Superior peroneal retinaculum (1) and peroneus longus tendon (2)



**Fig. 1.40** Incision of the deep fascia over the flexor hallucis longus muscle



**Fig. 1.38** Peroneus brevis tendon after retraction of the peroneus longus tendon



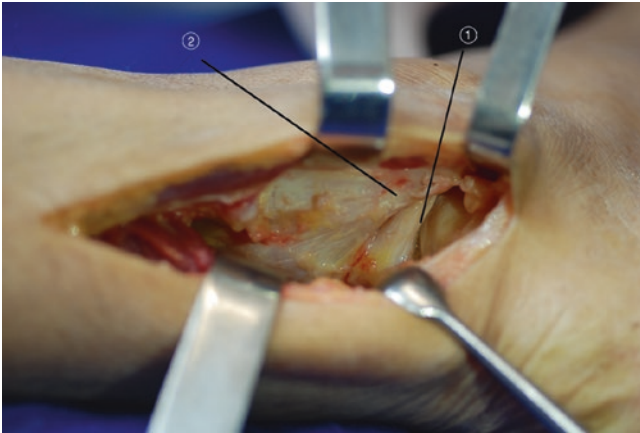
**Fig. 1.41** Posterior talofibular ligament (1)

### Hazards

The medial dorsal cutaneous nerve, deep peroneal nerve and dorsalis pedis artery may be injured.

### Incision

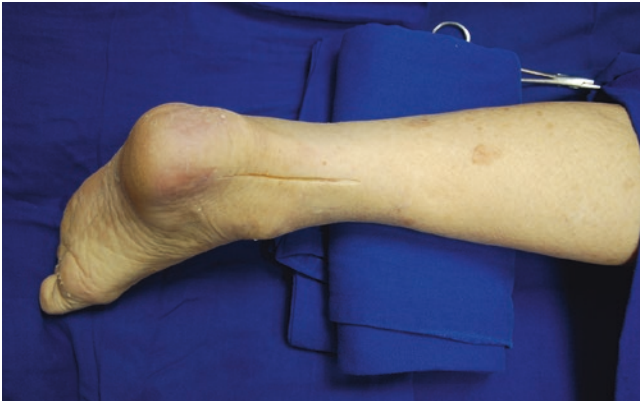
The dorsomedial longitudinal incision uses the interval between the TA and the EHL tendons centered over the first tarsometatarsal (TMT) joint extending about 4–6 cm distally (Fig. 1.51).



**Fig. 1.42** Posterior aspect of the subtalar joint (1), the talus and posterior tubercle of the distal tibia with the posterior tibiofibular (syndesmosis) ligament (2)



**Fig. 1.45** The Achilles tendon and plantaris tendon after incision of the paratenon



**Fig. 1.43** Prone position and posteromedial approach



**Fig. 1.46** The Kager fat pad



**Fig. 1.44** The Achilles tendon within its paratenon. Note the small plantaris tendon at the medial border of the Achilles tendon that may mimick a “partial rupture” of the latter



**Fig. 1.47** A prominent Haglund exostosis is exposed

### Superficial Dissection

Skin and subcutaneous tissue are incised and the medial dorsal cutaneous nerve (a branch of the superficial peroneal nerve) is identified (Fig. 1.52).

### Deep Dissection

Incise the TA and EHL-sheath, mobilize the TA tendon inferiorly and the EHL tendon dorsally to expose first metatarsocuneiform joint (Figs. 1.53 and 1.54). After capsulotomy the first TMT joint is exposed (Fig. 1.55).



**Fig. 1.48** The deep fascia overlying the FHL



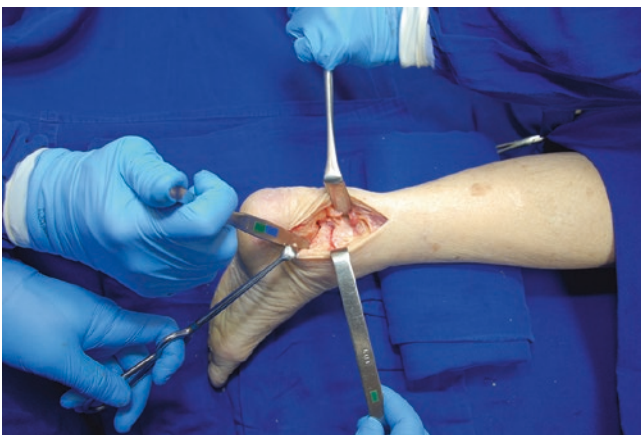
**Fig. 1.51** Dorsomedial longitudinal midfoot approach with the foot in a medial oblique position



**Fig. 1.49** The flexor hallucis longus (FHL) tendon exposed. The deep neurovascular bundle is held away medially with the blunt retractor



**Fig. 1.52** The medial dorsal cutaneous nerve



**Fig. 1.50** The posterior aspect of the subtalar and ankle joints are exposed



**Fig. 1.53** The anterior tibial tendon

### Indications

- Lisfranc (TMT) joint injuries
- Lisfranc arthrodesis

- Dermatofasciotomy for foot compartment syndrome (double incision together with a parallel dorsolateral approach)



**Fig. 1.54** The extensor hallucis longus tendon



**Fig. 1.56** Extension of the dorsal intermediate approach to the mid-foot. The patient is placed in a supine position with a bump under the ipsilateral hip to maintain neutral rotation



**Fig. 1.55** The first tarsal-metatarsal joint exposed after capsulotomy



**Fig. 1.57** Branch of the superficial peroneal nerve

### Position

Supine with a bump placed under the ipsilateral hip to maintain neutral rotation.

### Hazards

The deep peroneal nerve, the dorsalis pedis artery and its perforating branch to the plantar arch may be injured.

### Incision

The dorsal longitudinal incision is centered between the second and third tarsometatarsal joints, starting 1 cm proximally to the 2nd TMT-joint on the dorsal medial aspect of the midfoot and extending distally about 5–7 cm (Fig. 1.56).

### Superficial Dissection

Skin and subcutaneous tissue are incised and the branches of the superficial peroneal nerve are identified (Fig. 1.57).

### Deep Dissection

Incise the extensor digitorum longus sheath, mobilize the extensor hallucis brevis muscle medially and the extensor digitorum longus tendons laterally. The deep peroneal nerve and dorsalis pedis artery lie beneath the extensor hallucis brevis (EHB) tendon and muscle. They can be mobilized together and protected by the EHB. Care must be taken not to injure the perforating branch from the dorsalis pedis artery to the plantar arch that runs between the bases of the first and second metatarsals. After capsulotomy the second and third tarsometatarsal joints are fully exposed (Figs. 1.58 and 1.59).

### Dorsolateral Approach

#### Indications

- Lisfranc injuries (4th and 5th TMT joints)
- Arthritis of the lateral Lisfranc joint (4th and 5th TMT joints)



**Fig. 1.58** Extensor hallucis brevis muscle and extensor digitorum longus tendon



**Fig. 1.59** The second and the third tarsometatarsal joints

- Cuboid fractures
- Injuries to the Calcaneocuboid joint
- Dermatofasciotomy for foot compartment syndrome (double incision together with a parallel dorsal intermediate approach)
- Lateral column lengthening (with and without calcaneocuboid fusion)

#### Position

Supine with a sandbag or bump underneath the ipsilateral hip or lateral oblique position or lateral total.

#### Hazard

The sural nerve end branch (lateral dorsal cutaneous nerve) may be injured.

#### Incision

The dorsolateral longitudinal incision is centered over the 4th and 5th TMT (metatarsal-cuboid) joints, starting 1 cm proximal to the joint and extending distally about 5–7 cm (Fig. 1.60).



**Fig. 1.60** The dorsolateral approach to the midfoot. The patient is placed supine with a sandbag or bump underneath the ipsilateral hip to obtain a lateral oblique position

#### Superficial Dissection

Skin and the subcutaneous tissue are dissected while taking care not to injure the sural nerve.

#### Deep Dissection

The peroneus brevis tendon (Fig. 1.61), extensor digitorum brevis muscle and 4th and 5th extensor digitorum longus tendons. Mobilize the peroneus brevis tendon plantar and inferior and extensor digitorum brevis muscle dorsal and proximally (Fig. 1.62). After capsulotomy the cuboid-metatarsal joint and third metatarsocuneiform joint can be visualized (Fig. 1.63).

#### First Metatarsophalangeal Joint Approach

##### Indications

- Distal 1st metatarsal osteotomy or fracture
- Medial sesamoid excision

- Open treatment of sesamoid pathology, osteochondral defects/fractures
- Medial capsular imbrications
- Proximal phalangeal osteotomy
- Cheilectomy for hallux rigidus (limitus)
- Fracture-dislocation of the first MTP joint (“turf toe”)



**Fig. 1.61** Peroneus brevis tendon



**Fig. 1.62** Extensor digitorum brevis muscle and extensor digitorum longus tendons (oblique)



**Fig. 1.63** The 4th and 5th metatarso-cuboid (TMT) are exposed together with the lateral aspect of the third tarsometatarsal joint

### Position

Supine with a sandbag or bump underneath the contralateral hip.

### Harzards

The dorsomedial digital branch of the superficial peroneal nerve and medial branch of the common digital artery cross the first metatarsophalangeal joint on its dorsomedial portion and may be injured.

### Incision

Make a 5–6 cm longitudinal medial incision along the proximal two thirds of the proximal phalanx and extend it over the medial eminence (bunion) to the distal third of the metatarsal shaft (Fig. 1.64).

### Superficial Dissection

Raise the dorsal and plantar flaps, observe the dorsomedial branch of the common digital nerve and medial branch of the common digital artery (Figs. 1.65 and 1.66). Incise the deep fascia in line with the skin incision and identify the EHL tendon (Fig. 1.67).

### Deep Dissection

Using sharp dissection a capsulotomy is performed along the length of the incision and reflect the capsule surrounding the exostosis to expose the first metatarsal phalangeal joint (Fig. 1.68). The medial and lateral sesamoids are exposed (Fig. 1.69). The FHL tendon is identified between the two sesamoids. Distally and proximally the sesamoids are embedded into the flexor hallucis brevis tendon (Fig. 1.70).



**Fig. 1.64** Supine position with a sandbag or bump placed underneath the contralateral hip for the first metatarsophalangeal joint approach





**Fig. 1.65** Dorsomedial digital branch of the superficial peroneal nerve (*I*)



**Fig. 1.66** Medial branch of the common digital artery (*I*)



**Fig. 1.67** Extensor hallucis longus tendon (*I*)



**Fig. 1.68** The first metatarsal phalangeal joint is exposed

## Dorsal Intermetatarsal Space

### Indications

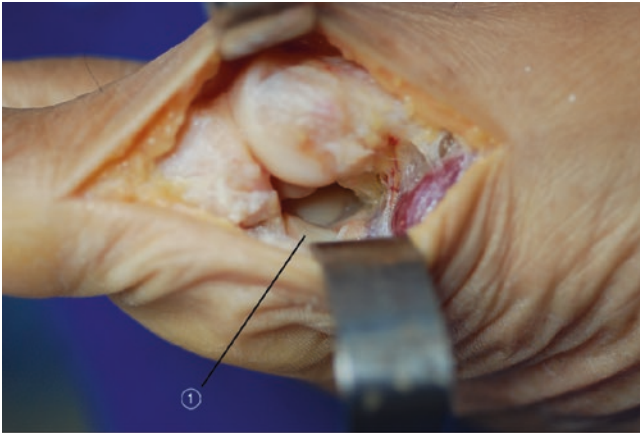
- Osteotomy or fracture of a metatarsal head/neck
- Plantar plate repair
- Excision of interdigital neuromas
- Stabilization of acute/chronic toe dislocations

### Position

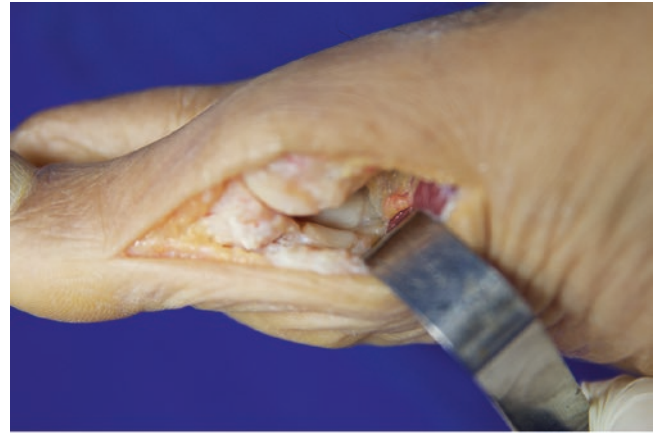
Supine with a sandbag under the ipsilateral hip (Fig. 1.71).

### Harzards

The common digital nerves and arteries branch into the proper digital nerves and arteries. Injury throughout the surgical incision may lead to numbness or even necrosis of the adjacent toe, if both the dorsal and plantar branches are injured.



**Fig. 1.69** Exposure of the medial (1) and lateral sesamoids (2)



**Fig. 1.70** The FHL tendon (1) is identified between the two sesamoids (2). The sesamoids are embedded distally and proximally into the flexor hallucis brevis tendon

### Incision

Make a 3–4 cm longitudinal incision along the intermetatarsal space (Fig. 1.72).

### Superficial Dissection

Raise the medial and lateral flaps while identifying the terminal branches of the digital nerves and arteries (Fig. 1.73). Incise the extensor digitorum longus fascia in line with the skin incision and identify the extensor digitorum longus tendon (Fig. 1.74)

### Deep Dissection

Perform a dorsolateral capsulotomy of the 2nd metatarsophalangeal joint exposing the second metatarsal head and base of the proximal phalanx of the second toe (Fig. 1.75). In the second web space the transverse metatarsal ligament is seen (Fig. 1.76) and the lumbrical tendon, which inserts at the medial aspect of the adjacent proximal phalanx.

Incise the dorsal transverse metatarsal ligament, exposing the deep intermetatarsal space and the plantar common digital nerve (Fig. 1.77).



**Fig. 1.71** Supine position with a sandbag under the ipsilateral hip for dorsal intermetatarsal space approach



**Fig. 1.73** Terminal branching of the dorsal common digital artery (I)

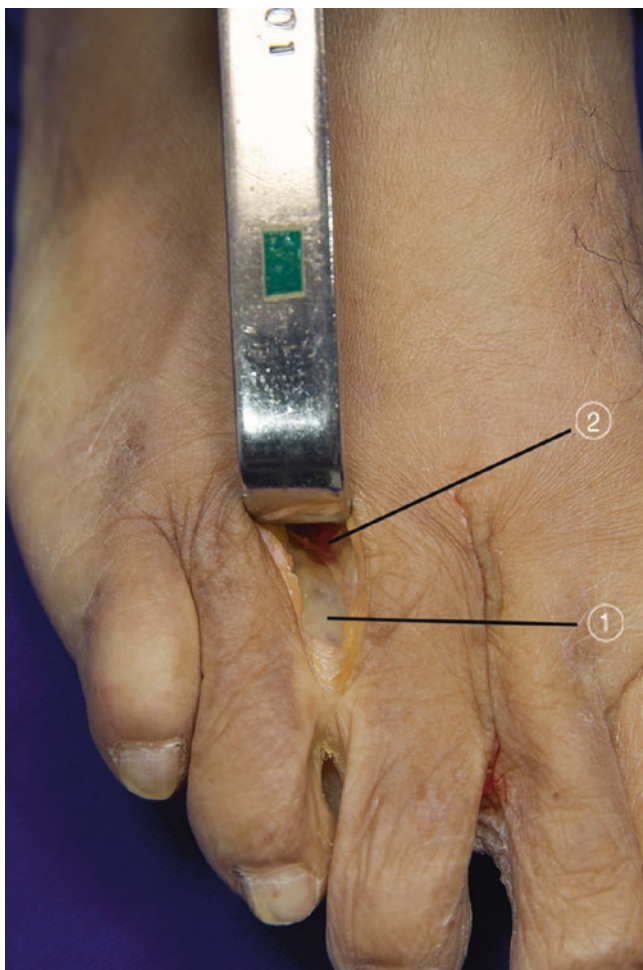
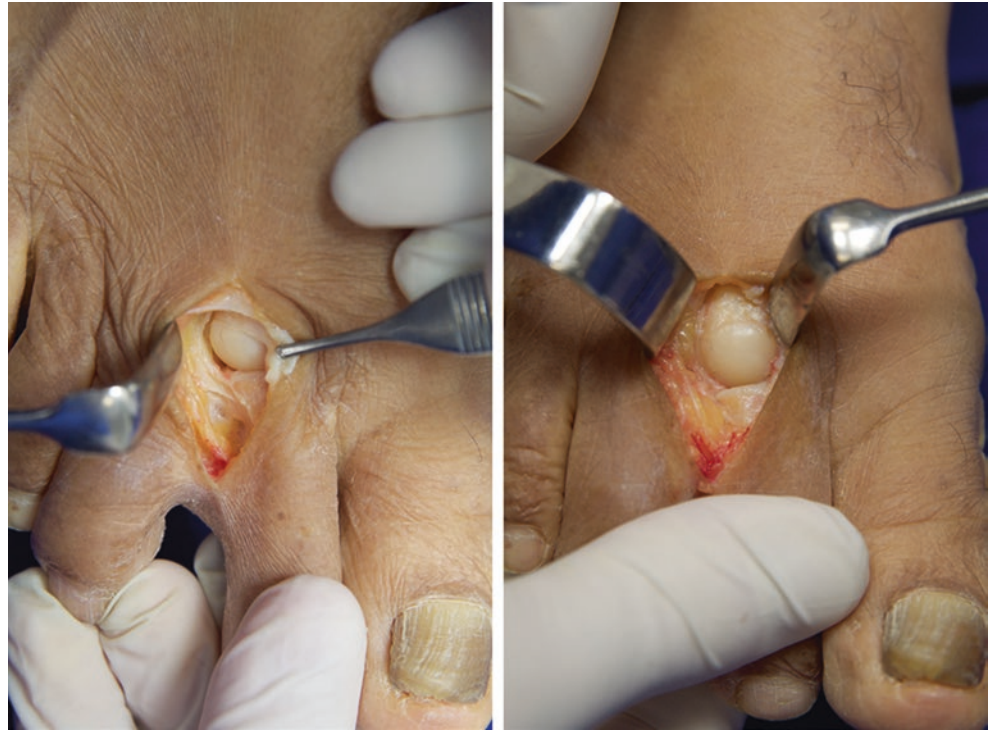


**Fig. 1.72** Terminal branching of the dorsal common digital nerve (I)

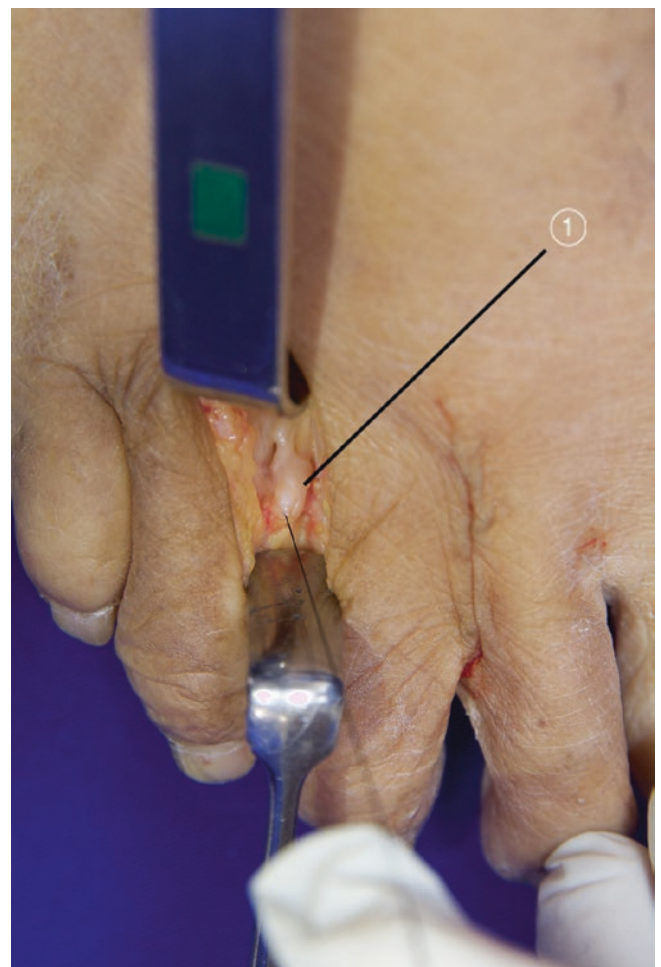


**Fig. 1.74** The extensor digitorum longus tendon (I)

**Fig. 1.75** Exposure of the second metatarsal head and the base of the proximal phalanx of the second toe



**Fig. 1.76** The dorsal transverse metatarsal ligament (1) and the lumbrical tendon (2)



**Fig. 1.77** The deep intermetatarsal space with the plantar common digital nerve (1) exposed

## References

1. Sarrafian SK, Kelikian AS. Sarrafian's anatomy of the foot and ankle: descriptive, topographic, functional. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2011. p. 1–598. ISBN 978-0781797504.
2. d'Hooghe PPRN, Kerkhoffs GMMJ. Chapter: Anatomy of the ankle. In: Golano P, et al. The ankle in football. 1st ed. Springer Paris Publisher; 2010. p. 1–24. doi:[10.1007/978-2-8178-0523-8\\_1](https://doi.org/10.1007/978-2-8178-0523-8_1).
3. Hoppenfeld S. Surgical exposures in orthopaedics: the anatomic approach. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2012. p. 245–309. ISBN: 9780781776233.
4. Zwipp H, Rammelt S. Tscherne Unfallchirurgie: Fuß. Berlin/Heidelberg/New York: Springer; 2014. p. I–812.

Matthias G. Walcher and Ryan du Sart

## Abstract

This Chapter presents biomechanics of foot and ankle with a particular focus on biomechanics in sports, and the influence of surgical procedures on biomechanics. A thorough biomechanical understanding is necessary for successful treatment strategies for foot and ankle pathologies. Any treatment should aim to restore physiological biomechanics or get biomechanics as close to normal as possible. Impaired function and range of motion of joints of the foot and ankle overloads adjacent joints, disturbs gait and increases energy consumption. Certain sports require more than a physiological performance of the foot or overuse structures of the foot even when performed correctly and with an excellent and well-balanced technique and training schedule. In sports biomechanical demands on the foot and ankle are high. This is why even small deformities and abnormalities that usually would not cause any problems can lead to overuse or make prone for specific injuries.

## Keywords

Foot and ankle Biomechanics • Human Gait • Kinematics Gait • Kinetics Gait • Surgical Biomechanics Foot and Ankle • Sports Biomechanics Foot and Ankle

## Introduction

The human foot is a very complex construction. Leonardo da Vinci (1452–1519) stated as a consequence of his anatomical research, that the human foot is a masterpiece of engineering – and a work of art. It is not a rigid lever and support for the body – it is rather a tool changing constantly throughout gait allowing for efficient forward-motion and adaption to uneven, irregular grounds.

The human foot is part of a biomechanical chain, where each part of the limb is depending on one another. There are

extensive individual variations still lying within the normal range. Therefore understanding the biomechanical reason that resulted in a foot and ankle pathology, like the biomechanical consequences of an intervention, is not always easy. Nonetheless, a thorough understanding of biomechanics is the basis of good decision making in foot and ankle orthopaedics, and essential for successful therapy.

Foot and ankle surgeons nowadays do not only have to be aware of biomechanics during stance and gait. In Western lifestyle, where activity and sport is playing an important role, a sound knowledge of biomechanics of running is crucial.

Movements of the foot and the ankle can be single-plane or multi-plane movements. The frontal (coronal) plane describes movement in the inversion/eversion direction. Sagittal plane motions take place in the dorsal extension/plantar flexion direction. Horizontal plane movements apply abduction and adduction. It needs to be noted, that nearly all the motions in foot and ankle are tri-planar, taking place around oblique axes. For example pronation consists of

---

M.G. Walcher, MD (✉)  
Section Foot and Ankle Surgery, Department of Orthopaedics,  
University of Würzburg König-Ludwig-Haus,  
Brettreichstr. 11, Würzburg 97074, Germany  
e-mail: [m-walcher.klh@uni-wuerzburg.de](mailto:m-walcher.klh@uni-wuerzburg.de)

R. du Sart, MD  
University of Western Australia, Perth, Royal Perth Hospital,  
197 Wellington Street, Perth 6000, Australia  
e-mail: [rcdusart@yahoo.com](mailto:rcdusart@yahoo.com)

components of abduction, eversion and dorsal extension, supination of adduction, inversion and plantar flexion.

There are different foot and ankle models available to facilitate understanding of biomechanics. A lot of models for gait analysis regard foot and ankle as a unity and the foot as rigid. Therefore different biomechanical models dividing foot and ankle into segments have been developed to allow an accurate understanding of biomechanics. There is no agreement in the scientific community as to the exact number and allocation of these segments, different authors describe different numbers of segments and different segments [1–3].

---

## Kinematics

Walking is involving the entire human body. Different components need to work together properly. Conditions proximal to the foot can influence the way the foot works. Foot conditions can affect patterns of motion proximally, too.

Human locomotion is very individual, allowing us recognizing familiar people already from a distance. One reason for this is that human walking is not an inborn reflex, it has to be learnt, a fact that every parent knows well. There are extensive inter-individual variations. Therefore it is more important to understand the functional relations.

Kinematics describes the movements of all body parts in 3 dimensions. In the vertical axis, there is a slight upward and downward movement during walking, with an amplitude of 4–5 cm [4, 5]. The maximum body height is reached immediately after the passage over the weight bearing limb at the moment of toe-off. The minimal height is reached at heel strike. Precise coordination of the motions of knee, pelvis and hip, ankle and foot is necessary to smoothen that vertical displacement to a clean sinusoidal curve with minimal extensions to minimize energy use.

The human body also shifts from side to side during walking, with the purpose to keep the centre of gravity over the weight bearing foot. The amplitude of this lateral displacement is about 4–5 cm with each stride. The slight physiological genu valgum allows the lower leg to remain vertical, with the feet close together and the femurs diverging.

Moreover there are rotational movements in the horizontal plane in the pelvis, shoulder girdle, femur and lower leg during walking. The rotational movements of the femur and the tibia are relatively similar. They rotate internally during the swing and the first interval of stance until the foot is positioned flat on the ground. In the later stance phase, starting with the contralateral toe-off, they rotate progressively externally. The internal rotation is mainly initiated by the valgus collapse of the subtalar joint. External Rotation is enhanced by the ankle joint axis, the alignment of the metatarsophalangeal break and the plantar aponeurosis. It is originated by

swing of the contralateral leg, which rotates the pelvis forward. This external rotation is transmitted distally, with the subtalar joint going into inversion, thereby stabilizing the foot for push-off.

It is interesting to have a look on the range of motion of different joints of the foot: Valderrabano and Co-workers found a mean of about 15° of dorsiflexion, and a mean of 28° of plantar flexion in cadaver ankles. They described a mean of 5° for eversion and roughly 14° for inversion, 8° for internal rotation, and 15° for external rotation [6]. The range of motion of the subtalar joint spans from about 40° to 60°. Usually the inversion is greater than the eversion [7].

---

## Kinetics

It has to be mentioned, that the foot is functioning in a closed kinetic chain, as it is fixed to the ground for most of the time of the gait cycle, whereas the more proximal parts of the limb are free to move. Therefore, movement of the foot and ankle causes motion of the tibia and fibula, which is transmitted to the femur and the hip, if not absorbed by the knee. That means that any movement of the foot and ankle should be also regarded in the context of its effect on the complete leg. For example pronation produces inward rotation, valgus of the knee, and a minimal forward inclination of the leg.

Assessing forces and torques acting on the lower limb during walking is important to understand human gait and associated pathologies. A variety of measurement tools has been developed to study the interaction of the foot with the ground. The walking speed has a direct effect on the plantar pressures during gait. Speed is related linearly to maximum ground reaction forces [8, 9], but is inversely related to the pressure time integral [10]. With increasing walking speed, a medialisation of the forces can be observed, with rising peak pressures on the medial side of the foot, and decreasing pressures on the fifth metatarsal [11]. Any condition reducing the range of motion of the ankle like ankle osteoarthritis or ankle fusion increases metabolic energy expenditure [12–14].

---

## Gait

Human gait involves the complete human body. It can be described with the gait cycle. The gait cycle is defined from the initial contact of one leg until the next initial contact of the same leg, e.g. from right heel-strike to right heel-strike.

In some parts of the gait cycle, there is double limb support, with both feet resting on the ground, in others there is single heel support, with one foot swinging through without contact to the ground. The step length is the distance from one heel strike to the next, the stride the length of two successive steps. It is about 1.5 m for healthy adults.

Cadence is the number of steps per minute, a value of about 110 per minute or approximately 2 steps per second are normal values for adults. The cadence is inversely proportional to the leg length, so smaller people have a higher cadence.

The walking cycle consists of a stance phase, when the foot is on the ground, representing about 62% of the gait cycle, and the swing phase, when the foot is swinging forward, making up the missing 38% of the gait cycle.

To facilitate understanding the complex patterns of motion that are taking place at the same time, the stance phase is further divided into three intervals: interval 1 extends from the moment the heel strikes the ground, the so-called initial contact, until the foot is positioned flat on the ground. Weight is transferred rapidly on the foot, an incident referred to as the loading response. During the loading response, there is double-limb support. Interval 2 lasts from the moment the foot is positioned flat on the ground until the body weight passes over the stable, single foot, the midstance. Interval 3 lasts from ankle plantar flexion to toe-off. The forward movement of the body over the foot results in weight being transferred to the forefoot.

In the pre-swing phase, the foot is unloaded as weight is transferred to the other foot. There is double limb support again. The Swing is also divided in three phases: The initial swing, when the thigh is advancing parallel to the foot being lifted off the ground. Then the mid-swing, with continuous advancement of the thigh. The knee is beginning to extend, and the foot clears the ground completely. In the terminal swing, the knee extends further and the leg prepares to touch the ground. The anterior compartment muscles, in particular the tibialis anterior muscle contracts concentrically and by this extends the ankle dorsally. The hindfoot is pulled in slight inversion, as the insertion of the tibialis anterior is located medially. This is why the heel is minimally inverted at heel strike.

In the first interval of the stance phase, the longitudinal arch of the foot flattens, the heel and the subtalar joint are everted. Because of this, the transverse tarsal articulation is unlocked, so that the foot can absorb and dissipate the forces that are generated by the heel strike. From the moment the heel touches the ground, there is quick plantar flexion of the ankle, until the foot rests flat. Usually the other heel is not on the ground any more, but the forefoot is still in contact with the floor. This interval takes about 15% of the gait cycle. The eversion of the heel causes a slight inward rotation of the tibia, transmitted by the subtalar joint. The anterior muscle compartment is essential in this interval, as it is slowing down and controlling the plantar flexion of the ankle after heel strike in eccentric contractions. The posterior compartment and the muscles supporting the longitudinal arch are not active.

In interval 2 of the stance phase, there is increasing dorsiflexion of the ankle, followed by plantar flexion as the body passes over the weight bearing foot. The foot turns from a flexible force absorber to a more rigid fulcrum being able to support the weight of the body. This is going along with a progressive subtalar inversion, locking the transverse tarsal articulation, making the midfoot rigid. At the end of this interval the heel begins to rise, and plantar flexion of the ankle begins. The other leg is swinging through, causing external rotation of the standing limb. These forces, transmitted by the subtalar joint support the progressive inversion of the hindfoot, like the metatarsophalangeal break and the tightening plantar fascia do. The maximum forces acting on the ankle joint are as high as 4.5 times the body weight. Interval 2 takes about 25% of the gait cycle. The intrinsic muscles of the foot contract to support the arch of the foot and the dorsal muscles of the calf control the forward motion.

In interval 3 of the stance phase the ankle plantar flexes quickly. The subtalar joint goes into maximal inversion, finishing the inversion movement started in interval 2. The toes are extended dorsally, resulting in the plantar fascia being tightened. At the moment of toe-off, the subtalar joint is maximally inverted. All these mechanisms are working together to stabilize the foot and get it rigid for a strong and efficient toe-off. The maximum inversion of the foot on the ground is transmitted as an external rotation to the lower leg. After toe-off there is immediate dorsal extension of the ankle, allowing the foot to swing through until the next heel-strike. Interval 3 takes about 22% of the gait cycle. The load applied on the foot reaches about 120% of the body weight. The dorsal calf muscles are mainly active in this interval, initiating plantar flexion with concentric contractions. Towards the end of the interval, weight is transferred to the other foot, and the anterior compartment muscles start dorsal extension of the ankle to prepare for the swing. The intrinsic muscles of the foot stabilize the longitudinal arch until toe off, although the plantar fascia is the main stabilizer in this section of the gait cycle.

The forces in the ankle during normal walking reach four times body weight [15]. The forces in the ankle during running are estimated to range up to 14 times body weight [16, 17]. The loads in the subtalar joint behave quite similar. Giddings found values of 5.4 times body weight there in walking and 11.4 times body weight in running [18]. In sports it is important to keep in mind that different playing surfaces significantly influence foot loading [19], like different overground surfaces or treadmills in running [20, 21]. Manoeuvres like cutting, jumping and landing, integral part of many sports, expose the foot to pressures more than doubled compared to running [22]. Cutting puts load on the medial side of the foot, sprinting on the first and second ray.



In soccer-kicks predominantly the lateral foot is stressed [23]. Fencing loads in particular the heel and the hallux region [24].

## Component Biomechanics

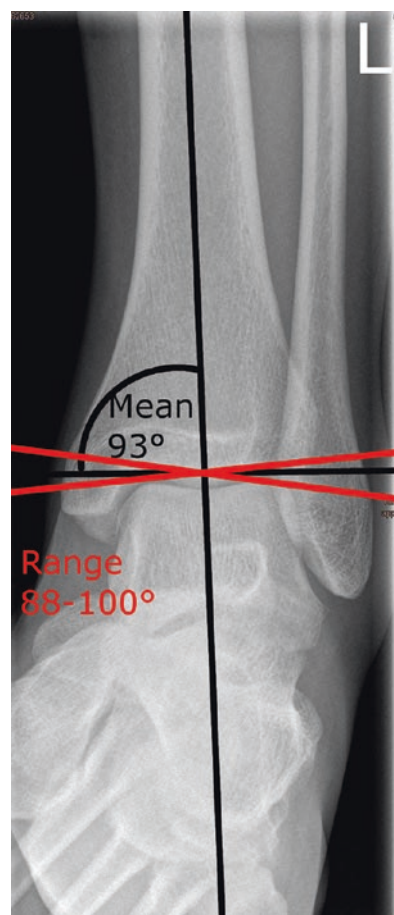
During the human gait cycle complex patterns of motion are taking place at the same time. Therefore it is worthwhile having a look at the individual components to completely understand biomechanics of human walking.

### Ankle Joint

The ankle is dorsally extended at heel strike from the swing phase. Then it plantar flexes rapidly to position the foot flat on the ground. The muscles of the anterior compartment of the lower leg slow and control this plantar flexion with eccentric contractions. The posterior compartment of the lower leg controls the progressive dorsiflexion of the ankle from foot flat with eccentric contractions and continues to contract concentrically as ankle plantar flexion is starting. The posterior compartment of the muscles works as a group. The tibialis posterior and peroneus longus start to work a little bit earlier than the rest of the compartment.

The axis of the ankle joint is oblique and changes continuously through the entire range of motion of dorsi- and plantarflexion [25]. In the coronal plane it deviates about 88–100° from the vertical axis of the leg [26]. Moreover the ankle plain is rotated in the horizontal plane [26]. Palpating the tips of the malleoli and connecting them with an imaginary line allows a clinically sufficient estimation of the ankle joint axis (Figs. 2.1, 2.2, and 2.3). A reason for this helical dynamic axis is the shape of the talus with a smaller radius medially as laterally and the strong deltoid ligament as a rotational point.

Because of the oblique joint axis, every dorsal extension and plantar flexion movement also leads to a rotation of the foot, if the foot is free, and to a rotation of the leg, if the foot is fixed. The free foot moves outward in dorsal extension and inward in plantar flexion. On the other hand if the foot is fixed dorsal extension will rotate the leg internally, and plantar flexion externally. Likewise external rotation of the lower leg leads to a movement transfer into inversion of the hind-foot, and elevation of the medial side of the foot. In internal rotation of the lower leg, it is just the other way round. This phenomenon is called coupled movement. Nonetheless the ankle joint obliquity contributes only partially to the rotational motions of the foot and lower leg. The subtalar joint is responsible for the additional rotational movement. Compared to the knee joint axis the ankle axis is about 20–30° externally rotated.

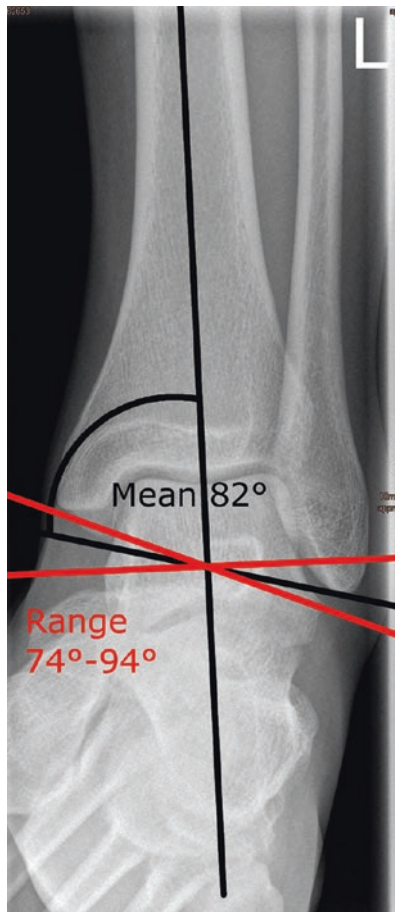


**Fig. 2.1** Axis alignment of the ankle. Variations of the alignment of the distal tibia [26]

### The Subtalar Joint

The subtalar joint is a joint with a single fixed axis. It acts as a hinge/torque converter between the talus and the calcaneus. In the transverse plane, the joint axis deviates about 23° medial to the longitudinal axis of the foot, ranging from 4° to 47° [27]. In the sagittal plane, the axis is about 41° oblique, ranging from 21° to 69° [27] (Figs. 2.4 and 2.5).

In the anatomy and the alignment of the subtalar joint there are immense individual variations. In summary the subtalar joint acts as a torque converter between the lower leg and the foot. It has a crucial function in locking and unlocking the transverse tarsal articulation, it is determinative to the distal foot joints. During the gait cycle, the subtalar joint is slightly inverted in the heel strike position, and progresses quickly into eversion with maximum eversion when the foot is flat on the ground. It then moves towards progressive inversion until toe-off. The eversion at heel strike is a passive motion, forced by the fact, that the subtalar axis is situated laterally to the weight bearing axis. The eversion is



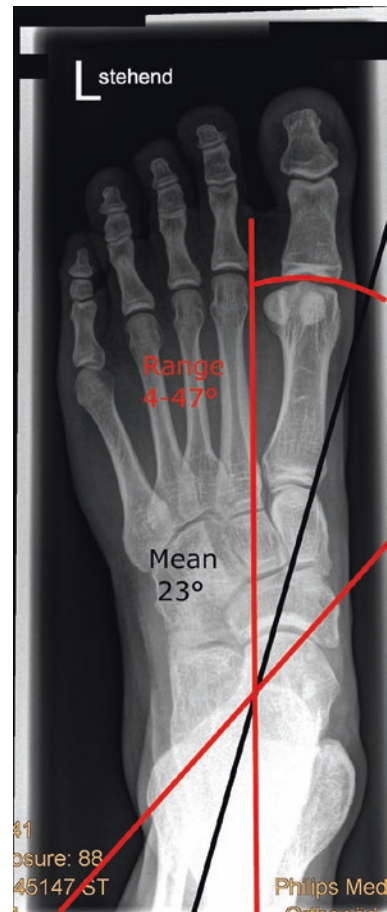
**Fig. 2.2** Axis alignment of the ankle. Empirical axis of the ankle [26]



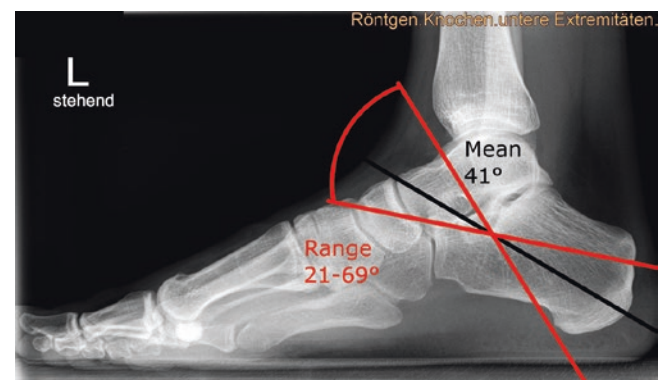
**Fig. 2.3** Empirical axis of the ankle in the clinical examination

controlled by the ligaments of the ankle and the subtalar joint, and serves to absorb the impact of the heel strike. Muscle pull of the deep posterior compartment supports hindfoot inversion.

In flat foot deformity the axis of the subtalar joint is more horizontalized. This results in the same rotation of



**Fig. 2.4** Alignment of the subtalar joint in the transverse plane. The joint axis deviates with a mean of 23° to the long axis of the foot, with wide variations [26]



**Fig. 2.5** Alignment of the subtalar joint in the sagittal plane. The joint axis deviates with a mean of 41° to the long axis of the foot, with wide variations [26]

the lower leg generating greater pronator/supinator effects on the foot. This explains why asymptomatic flat feet usually present with a greater range of motion in the hindfoot and midfoot. In cavovarus deformity, the opposite is true.

## Transverse Tarsal Articulation

The transverse tarsal articulation includes the talonavicular joint and the calcaneocuboid joint, forming one functional unit. It connects the hindfoot with the midfoot. These joints each have their independent range of motion, but they work together as a functional unit crucial for hindfoot biomechanics. In Supination the navicular glides medially and inferiorly on the talar head. The cuboid follows the navicular, also moving medially and plantarly. In pronation, the reverse takes place. There are two axes of motion in the transverse tarsal articulation. The longitudinal axis allows motion similar to the subtalar joint in the pronation/supination direction. The oblique axis is nearly parallel to the axis of the ankle joint, enabling movements in the dorsal extension and plantar flexion direction. This explains why compensatory hypermobility in patients with fused ankles is mostly taking place in the transverse tarsal joint.

If the heel is everted, the joint lines of the talonavicular and the calcaneocuboid joint are parallel to each other, making the hindfoot/midfoot transition flexible. The longitudinal arch of the foot is flattened [28] (Figs. 2.6 and 2.7).

In inversion, the joint lines are angulated to one another, and the hindfoot/midfoot transition is rigid. The transverse tarsal articulation ensures that the foot is flexible at heel strike, and rigid and stable for toe-off. The longitudinal arch of the foot is elevated and stabilized. Therefore the foot can work as an extension of the leg, and allow a sufficient stride length. The transverse tarsal joint transmits hindfoot motion to the more distal parts of the foot. It has to be noted, that the radius of the talar head is different in the anterior-posterior and the lateral direction. Because of this additional stability is provided intrinsically when force is applied through the joint at toe-off.



**Fig. 2.6** Function of the transverse tarsal articulation: in eversion the midfoot is flexible



**Fig. 2.7** Function of the transverse tarsal articulation: in inversion the transverse tarsal articulation is locked. The foot is rigid

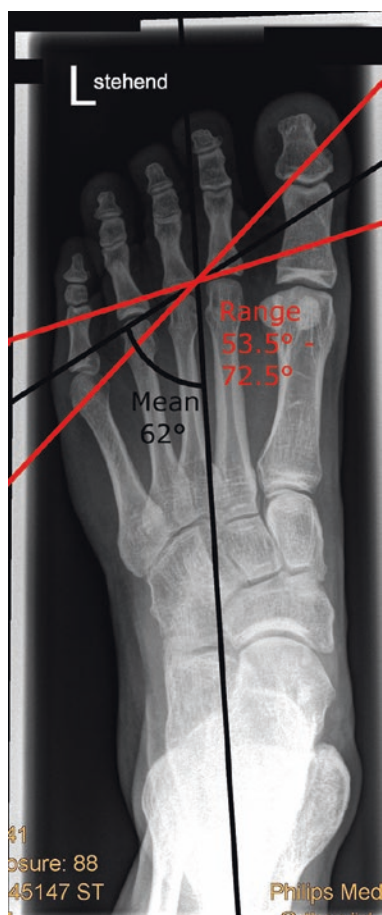
## Metatarsophalangeal Break

The metatarsophalangeal break is the angle formed by the metatarsal heads because of their different lengths. It angles 50–70° to the long axis of the foot. Usually the 2nd metatarsal head is located most distal. As the 1st metatarsal head is slightly elevated and supported by two sesamoid bones, it comes up to the length of the 2nd metatarsal head functionally. With the heel elevated immediately before toeing off, the body weight is usually distributed to all the metatarsal heads. To allow this weight distribution, slight supination and lateral deviation is necessary because of the obliquity of the metatarsophalangeal break. The subtalar joint balances this supination, so that the foot remains vertical on the ground (Fig. 2.8).

## Plantar Aponeurosis – Windlass Mechanism

The plantar aponeurosis extends from the calcaneal tubercle to the bases of the proximal phalanges. Passing the metatarsal heads it forms the plantar plate together with the joint capsule. In dorsal extension of the phalanges the plantar aponeurosis is pulled over the metatarsal heads. This leads not only to depression of the metatarsal heads, but also an elevation of the longitudinal arch of the foot. This so called “truss and windlass mechanism” is most functional on the medial side of the foot [29]. Tightening of the plantar fascia will also lead to inversion of the calcaneus and external rotation of the tibia. This happens as the heel begins to rise as the body passes over the foot until toe-off (Fig. 2.9). Pathologies of the plantar fascia like plantar fasciitis are frequent in athletes, in particular in runners. The force acting on the plantar fascia reaches up to 2.9 times body weight in running [17].

Athletes with strength and flexibility deficits are at a special risk for plantar fasciitis [30].



**Fig. 2.8** Metatarsophalangeal Break: The axis of the metatarsal heads angles a mean of  $62^\circ$  to the longitudinal axis of the foot. Variations occur [26]

## Biomechanics of Running

In running the same basic mechanisms apply. The gait cycle in contrast is altered significantly. In normal walking one foot always stays on the ground, in running a float phase is included, during which no foot is on the ground. The stance phase is relevantly shortened from about 0.6 s when walking to 0.2 s when sprinting. Increasing range of motion of the joints of the lower limb of about 50% helps absorb these higher forces. The muscles must control these larger motions at a shorter time with a larger force. This is why injuries and overuse occur mostly during sports. Moreover muscle action is necessary over a larger percentage of the gait cycle, when compared to walking. With rising speed of running, the time for the gait cycle itself is getting shorter. Also the time of the stance phase in real time as in percentage of the gait cycle is reduced. With increasing speed the gastrocnemius muscle plays a role with rising importance. In normal walking its contribution to forward motion is minimal. Forefoot runners have a relevantly higher load of about 15% in their Achilles tendon than non-forefoot runners [31].



**Fig. 2.9** (a, b) Function of the plantar aponeurosis: dorsal extending toe I tightens the plantar fascia, thereby elevating the longitudinal arch of the foot

## Surgical Biomechanics

### Biomechanics of Ankle Osteoarthritis

Ankle osteoarthritis has a significant impact on human locomotion. Patients with ankle osteoarthritis present a shorter stride length, walking speed is reduced. The stance phase is shorter. The range of motion of the ankle is diminished in all three planes. The greatest deficit usually concerns dorsi- and plantarflexion [14]. There is increased stress on the adjacent joints.

### Biomechanics of Total Ankle Replacement

Total ankle replacement is the treatment option for endstage ankle osteoarthritis mimicking physiological ankle joint range of motion and biomechanics the closest. This potentially shelters the adjacent joints from accelerated wear and leads to improved function. Still it needs to be mentioned that patients with a total ankle replacement performing sport very actively will very likely experience an earlier loosening

of the implants. Most impact sports cannot be recommended for patients with joint replacements.

### Biomechanics of Ankle Fusion

Ankle fusion eliminates motion in the ankle joint and changes ankle joint kinematics relevantly. In patients with a fused ankle joint, there is increased stress in the subtalar joint, the Chopart joint line and the knee joint. The adjacent joints develop a compensatory hypermobility, in particular the transverse tarsal articulation. If the ankle is mal-positioned in excessive internal rotation, the body passes over the foot only with difficulty. There is increased stress in the subtalar joint, the midfoot, the knee and the hip. There may be overuse problems of the hip and the knee because of compensatory external rotation of the hip.

In excessively externally rotated position the foot rolls over the medial side. Increased stress acts there with a frequent development of hallux valgus, and problems on the medial side of the knee.

As far as alignment in the varus-valgus plane is concerned, the surgeon fusing an ankle should aim for a physiological slight hindfoot valgus. It is crucial to take the alignment of the complete lower limb like the subtalar joint into account. If the subtalar joint is stiff, it is crucial to make sure that there is sufficient valgus to allow for a plantigrade position of the foot during walking. Fusing the ankle in varus position increases the stress on the lateral side of the foot. In addition this locks the transverse tarsal articulation making the transition from the hindfoot to the midfoot rigid, disturbing force dissipation during gait, and thereby overloading the small joints of the midfoot.

A plantarflexed ankle fusion leads to a functional lengthening of the limb. Patients develop a compensatory back thrust of the knee, and an uneven gait pattern. There is increased stress on the midfoot. In patients with a shorter leg or a loss of function of the knee extensors with a subsequent unstable knee fusion of the ankle joint in slight plantar flexion can therefore improve function.

Increased dorsiflexed position concentrates the ground impact on a small area of the heel, which is easily mechanically overloaded and painful.

### Biomechanics of Subtalar Joint Fusion

The very moment the subtalar joint is fused, higher rotation forces are acting on the ankle joint. The ankle needs to absorb the transverse rotational forces that are normally compensated for by the subtalar joint.

The varus-valgus position affects the forefoot position and the midfoot articulations in the way already mentioned in this chapter. Excessive varus position will put the forefoot into supination, so that the weight bearing line is shifted laterally. Moreover the concomitant locking of the transverse tarsal articulation in at least a semi-locked position makes the hindfoot-midfoot transition at least semi-rigid and this inhibits the usual compensatory hypermobility. It is advisable to fuse the subtalar joint in a physiological slight valgus position. This makes the forefoot flexible and will enable an even force distribution through the forefoot.

### Biomechanics of Fusion of the Chopart Joint

Fusion of talonavicular joint or the transverse tarsal articulation eliminates subtalar motion completely. Isolated arthrodesis of the calcaneocuboid joint reduces the range of motion in the subtalar joint about 30%. The range of motion of the subtalar joint has an important influence on gait, as it is the key joint to lock or unlock the transverse tarsal articulation, and therefore controls the forefoot.

Again it is best, to go for physiological alignment in fusion.

It can be stated as a general principle in ankle and hindfoot fusions, that if ever a non-physiological position is going to be inevitable, it is better to err into too much valgus, as this will preserve the flexibility of the foot.

Fusions of the medial three rays in the Lisfranc Joint line and intertarsal joint fusions do not affect the range of motion very much. The two lateral rays in the Lisfranc joint line are quite mobile physiologically to allow the foot to adapt on uneven ground. Fusion of these lateral rays should be avoided if possible.

### Biomechanics of Forefoot Corrections

A removal of the base of the proximal phalanx of the 1st toe like in the Keller-Resection arthroplasty disrupts the windlass mechanism of the plantar aponeurosis. There is a decreased loading of the 1st metatarsal, and an increased loading of the lesser metatarsal heads. In the lesser toes the same effect in a smaller extent can be observed. In fusions of the hallux joint, the position is absolutely critical. Most authors recommend a slightly dorsally extended and valgus position. If the metatarsophalangeal joint I is fused, an overuse of the interphalangeal joint and the tarsometatarsal joint I can be observed, in some cases also with radiological signs of osteoarthritis. Luckily this is rarely symptomatic. Hallux interphalangeal joint fusions don't seem to have a significant effect on biomechanics of human gait.

## Tendon Transfers

For the planning of a tendon transfer in foot and ankle it is crucial to have a look on the position of the muscle and its insertion in relation to the axes of the joints. It is crucial to examine the function of the individual muscles thoroughly. Thereby it is possible to determine which muscles are potentially transferrable, and where they need to be inserted, to achieve a balanced foot.

Transferring a muscle usually active in the stance phase to a swing muscles is difficult and the other way round – in-phase transfers are preferable and usually have better results.

## Ankle Ligaments

The ligaments of the ankle allow free simultaneous motion of the ankle and the subtalar joint. The deltoid ligament is the shape of a fan that stabilizes on the medial side. On the lateral side there are three individual ligament. The calcaneofibular ligament (CFL) stabilizes the ankle and the subtalar joint. The CFL runs oblique-posteriorly in neutral position of the ankle. In dorsal extension it runs in line with the fibula, acting as a true collateral ligament. The AFTL in contrast runs horizontally in dorsal extension not stabilizing in this position. In plantar flexion the CFL runs parallel to the ground with the consequence that it doesn't stabilize against inversion. The AFTL runs in line with the fibula then, thus resisting inversion in this position. So depending on the position of the ankle the AFTL or the CFL are the primary stabilizers of the ankle joint. The angle between these two ligaments ranges from 70° to 140° [26]. Throughout the range of motion of the ankle there are theoretically positions where neither of these ligaments stabilises the joint. The stability of the ankle joint should be examined in dorsal extension like in plantar flexion to be able to estimate stability of the CFL like the AFTL.

The most important stabilizers of the subtalar joint are the interosseous talocalcaneal ligaments in the sinus tarsi.

Ankle sprains and ankle ligament injuries are among the most common injuries in sports, and affect in most of the cases the lateral ligaments. It is important to keep in mind, that 10–30° of the patients with acute ligament lesions of the ankle develop a chronic instability, even if they are treated consequently. Chronic ankle instability allows a too big amount of talus translation and rotation. This creates relevant shear forces on the cartilage, which is frequently already damaged by the initial trauma. Thereby chronic ankle instability accelerates cartilage wear and causes joint degeneration [32]. Nonetheless the latency for posttraumatic ligamentous ankle osteoarthritis is according to Valderrabano more than 30 years [32].

## Summary

Biomechanics of the foot and ankle is complex. It needs to be understood in the context of the whole extremity and the whole body.

A thorough biomechanical understanding is necessary for successful treatment strategies for foot and ankle pathologies. Any treatment should aim to restore physiological biomechanics or get biomechanics as close to normal as possible.

Impaired function and range of motion of joints of the foot and ankle overloads adjacent joints, disturbs gait and increases energy consumption.

Certain sports require more than a physiological performance of the foot or overuse structures of the foot even when performed correctly and with an excellent and well-balanced training schedule.

In sports biomechanical demands on the foot and ankle are high. This is why even small deformities and abnormalities that usually would not cause any problems can lead to overuse or make prone for specific injuries.

## References

1. Kitaoka HB, Crevoisier XM, Hansen D, Katajarvi B, Harbst K, Kaufman KR. Foot and ankle kinematics and ground reaction forces during ambulation. *Foot Ankle Int.* 2006;27(10):808–13.
2. Myers KA, Wang M, Marks RM, Harris GF. Validation of a multi-segment foot and ankle kinematic model for pediatric gait. *IEEE Transact Neural Syst Rehabil Eng Publ IEEE Eng Med Biol Soc.* 2004;12(1):122–30.
3. De Ridder R, Willems T, Vanrenterghem J, Robinson M, Pataky T, Roosen P. Gait kinematics of subjects with ankle instability using a multisegmented foot model. *Med Sci Sports Exerc.* 2013;45(11):2129–36.
4. Ryker NJ. Glass walkway studies of normal subjects during normal level walking: prosthetic Devices Research Project, Institute of Engineering Research, University of California; Berkeley. 1952.
5. Saunders JB, Inman VT, Eberhart HD. The major determinants in normal and pathological gait. *J Bone Joint Surg Am.* 1953;35-A(3):543–58.
6. Valderrabano V, Hintermann B, Nigg BM, Stefanyshyn D, Stergiou P. Kinematic changes after fusion and total replacement of the ankle: part 1: range of motion. *Foot Ankle Int.* 2003;24(12):881–7.
7. Jastifer JR, Gustafson PA. The subtalar joint: biomechanics and functional representations in the literature. *Foot (Edinburgh, Scotland).* 2014;24(4):203–9.
8. Andriacchi TP, Ogle JA, Galante JO. Walking speed as a basis for normal and abnormal gait measurements. *J Biomech.* 1977;10(4):261–8.
9. Nilsson J, Thorstensson A. Ground reaction forces at different speeds of human walking and running. *Acta Physiol Scand.* 1989;136(2):217–27.
10. Zhu H, Wertsch JJ, Harris GF, Alba HM. Walking cadence effect on plantar pressures. *Arch Phys Med Rehabil.* 1995;76(11):1000–5.
11. Rosenbaum D, Westhues M, Bosch K. Effect of gait speed changes on foot loading characteristics in children. *Gait Posture.* 2013;38(4):1058–60.
12. Waters RL, Mulroy S. The energy expenditure of normal and pathologic gait. *Gait Posture.* 1999;9(3):207–31.

13. Lobet S, Hermans C, Bastien GJ, Massaad F, Detrembleur C. Impact of ankle osteoarthritis on the energetics and mechanics of gait: the case of hemophilic arthropathy. *Clin Biomechan (Bristol, Avon)*. 2012;27(6):625–31.
14. Valderrabano V, Nigg BM, von Tscharnner V, Stefanyshyn DJ, Goepfert B, Hintermann B. Gait analysis in ankle osteoarthritis and total ankle replacement. *Clin Biomechan (Bristol, Avon)*. 2007;22(8):894–904.
15. Procter P, Paul J. Ankle joint biomechanics. *J Biomech*. 1982;15(9):627–34.
16. Burdett RG. Forces predicted at the ankle during running. *Med Sci Sports Exerc*. 1982;14(4):308–16.
17. Scott SH, Winter DA. Internal forces of chronic running injury sites. *Med Sci Sports Exerc*. 1990;22(3):357–69.
18. Giddings VL, Beaupre GS, Whalen RT, Carter DR. Calcaneal loading during walking and running. *Med Sci Sports Exerc*. 2000;32(3):627–34.
19. Ford KR, Manson NA, Evans BJ, Myer GD, Gwin RC, Heidt Jr RS, et al. Comparison of in-shoe foot loading patterns on natural grass and synthetic turf. *J Sci Med Sport/Sports Med Aust*. 2006;9(6):433–40.
20. Hong Y, Wang L, Li JX, Zhou JH. Comparison of plantar loads during treadmill and overground running. *J Sci Med Sport/Sports Med Aust*. 2012;15(6):554–60.
21. Wang L, Hong Y, Li JX, Zhou JH. Comparison of plantar loads during running on different overground surfaces. *Res Sports Med (Print)*. 2012;20(2):75–85.
22. Orendurff MS, Rohr ES, Segal AD, Medley JW, Green 3rd JR, Kadel NJ. Regional foot pressure during running, cutting, jumping, and landing. *Am J Sports Med*. 2008;36(3):566–71.
23. Eils E, Streyl M, Linnenbecker S, Thorwesten L, Volker K, Rosenbaum D. Characteristic plantar pressure distribution patterns during soccer-specific movements. *Am J Sports Med*. 2004;32(1):140–5.
24. Trautmann C, Martinelli N, Rosenbaum D. Foot loading characteristics during three fencing-specific movements. *J Sports Sci*. 2011;29(15):1585–92.
25. Lundberg A, Svensson OK, Nemeth G, Selvik G. The axis of rotation of the ankle joint. *J Bone Joint Surg*. 1989;71(1):94–9.
26. Stiehl JB. Inman's joints of the ankle. Auflage: 2 sub ed. Baltimore: Lippincott Williams and Wilkins; 1991. p. 152.
27. Isman RE, Inman VT. Anthropometric studies of the human foot and ankle. Biomechanics Laboratory, University of California, San Francisco and Berkeley. Technical Report 58. The Laboratory, San Francisco; 1968 1968. Report No.
28. Elftman H. The transverse tarsal joint and its control. *Clin Orthop*. 1960;16:41–6.
29. HICKS JH. The mechanics of the foot. II. The plantar aponeurosis and the arch. *J Anat*. 1954;88(1):25–30.
30. Kibler WB, Goldberg C, Chandler TJ. Functional biomechanical deficits in running athletes with plantar fasciitis. *Am J Sports Med*. 1991;19(1):66–71.
31. Almonroeder T, Willson JD, Kernozek TW. The effect of foot strike pattern on achilles tendon load during running. *Ann Biomed Eng*. 2013;41(8):1758–66.
32. Valderrabano V, Hintermann B, Horisberger M, Fung TS. Ligamentous posttraumatic ankle osteoarthritis. *Am J Sports Med*. 2006;34(4):612–20.

John S. Lewis Jr. and Mark E. Easley

## Abstract

Injuries of the foot and ankle are common among recreational and competitive athletes. Like most sports-related injuries, management typically entails a period of rest, aggressive rehabilitation and return to early competitive activity without compromising long-term functional outcomes. Timing of intervention and return to play is often a prime consideration with respect to sports, and thus particular emphasis is often given to early diagnosis and treatment in the athlete. A combination of intrinsic and extrinsic factors may predispose athletes to certain injuries in the foot and ankle. Common acute athletic injuries to the foot and ankle include ankle sprains, ankle fractures, turf toe, Lisfranc injuries, and overuse syndromes ranging from Achilles tendonitis to stress fractures. As in all of sports medicine, the goal of treatment and surgical decision-making is to ensure safe return to play and to reduce the risk of further or recurrent injury, aligned with the goals of coaches, teams, and the athlete.

## Keywords

Ankle • Athlete • Injury • Overuse • Stress fracture • Sports

## Introduction

Sports medicine has evolved beyond the care of the athlete with a knee injury to encompass all subspecialty areas in orthopaedics, including the foot and ankle [1]. Injuries of the foot and ankle are common among recreational and competitive athletes. Like most sports-related injuries, management typically entails a period of rest, aggressive rehabilitation and return to early competitive activity without compromising long-term functional outcomes [2]. Timing of intervention and return to play is often a prime consideration with respect to sports, and thus particular emphasis is often given to early diagnosis and treatment in the athlete [1]. For athletes with evidence of structural abnormalities requiring surgical intervention, emphasis on anatomic reconstruction and repair

is paramount [1]. These objectives, however, are often influenced by the high expectations of teams, coaches, parents, agents, and the athlete [2]. Limited or percutaneous approaches, endoscopic methods, bioabsorbable implants, and tissue-engineered transplants and other novel surgical methods are becoming more accepted in an effort to meet the unique challenges a foot or ankle injury in an athlete can present [1]. Common acute athletic injuries to the foot and ankle include ankle sprains, ankle fractures, turf toe, Lisfranc injuries, and overuse syndromes ranging from Achilles tendonitis to stress fractures.

## Epidemiology

Sports participation has become a fundamental characteristic of American society. In terms of the most popular sports in this country, it is estimated that approximately 17.7 million Americans play football, 14.5 million play soccer, 15.6 million play baseball, 28.9 million play basketball, and 13.6 million play softball. [1, 3] Both foot and ankle and sports medicine surgeons should be familiar with the spectrum of athletic

J.S. Lewis Jr., MD (✉) • M.E. Easley, MD  
Department of Orthopaedic Surgery,  
Duke University Medical Center,  
4709 Creekstone Drive, Box 3000, Durham, NC 27710, USA  
e-mail: [john.lewis@duke.edu](mailto:john.lewis@duke.edu); [mark.e.easley@dm.duke.edu](mailto:mark.e.easley@dm.duke.edu)



injuries that affect the foot and ankle, as traumatic injuries of the foot and ankle occur frequently in sporting activity. In recent decades, public health surveillance programs have more accurately quantified the frequency and breadth of foot and ankle injuries in many sports and at many different levels of competition [2]. Injuries to the lower extremity constitute the majority of injuries for most sports, particularly the running, jumping, and kicking sports [1]. In a review of injuries seen in a multispecialty sports clinic, 25 % of 12,681 injuries that occurred in 19 sports involved the foot or ankle [4, 5]. In addition, an estimated 20–35 % of total time lost to injury in running and jumping sports can be attributed to ankle injuries [4, 6].

In 1982, the National Collegiate Athletic Association (NCAA) developed the Injury Surveillance System (ISS) in an effort to improve athlete safety by recording and reviewing the epidemiology of the most common injuries suffered by collegiate athletes [2, 7]. The data collected in the ISS have been instrumental in implementing rule and equipment change recommendations and ultimately reducing the number of collegiate athletic injuries [2]. For a 16 year period, from 1988–89 to 2003–4, ankle ligament sprains were reported to be the most common injury during practice and competition, comprising 14.9 % of all injuries across all three collegiate divisions [2, 8]. This represents an injury rate of 0.83 per 1000 athlete-exposures [8]. In comparison, anterior cruciate ligament injuries accounted for 2.6 % of all injuries, with 0.15 injuries per 1000 athlete exposures [2, 8]. At the high school level, 39.7 % of injuries among athletes involve the foot and ankle, with sprains being the most common [2, 9].

---

## Etiologic Factors

The ultimate goal of sports medicine is to enable athletes to perform at their maximum abilities while minimizing the risk of injury [2]. There are certain identifiable intrinsic and extrinsic risk factors that influence the type and severity of injuries that athletes may face. Factors that are intrinsic to the athlete, such as tarsal coalitions or hindfoot malalignment, or to the sport, such as high weekly mileage requirements in competitive long distance running, are often unable to be modified. Factors that are extrinsic to the athlete, such as equipment, dietary habits, playing surfaces, and rules governing play, can also directly impact the type of injuries an athlete may suffer. Perpetual scrutiny of the safety and efficacy of equipment used in sporting activity is critical in reducing the risk of injury. For example, in 1988 Janda et al. found that 71 % of all recreational softball injuries were attributed to sliding into bases, with 56 % of these injuries being isolated to the foot and ankle [10]. By substituting breakaway bases they demonstrated a reduction of injuries from 1 in 14 games to 1 in 317 games [1, 10, 11]. In addition, interventions in the

form of rule changes such as the elimination of the “crack-back” block and targeting with the helmet can effectively reduce the incidence and severity of injury [1].

In particular, there has been considerable concern that the evolution of artificial playing surfaces has increased injury rates among athletes. Although substantial attention has been given to this subject, there has been no definitive evidence of this being true despite over twenty studies investigating this issue [1]. Natural grass can also play a role in injuries, however, if surfaces are poorly maintained. Mueller and Blyth showed a 30 % reduction in injury rate by careful maintenance and upkeep of grass practice and game fields [1, 12]. In competitive and recreational running, hard surfaces and hills are often implicated in injuries to foot and ankle, although several studies [13, 14] have found no relationship between surface hardness/type and injury [1]. One study did note the only significant relationship between injuries and training surface was to be in female runners running on concrete [1, 15]

## Biomechanical Abnormalities

Certain underlying anatomic abnormalities are often implicated as a predisposing factor in athletic injuries. For example, rigid tarsal coalitions in the hind foot are often associated with recurrent ankle sprains. Many runners and coaches feel that athletes with abnormal foot biomechanics, such as the runner who “overpronates,” have an innately higher risk for sustaining a running-related injury [1]. Many authors feel that a hyperpronated cavus foot and/or forefoot varus predisposes individuals to Achilles tendonitis and injury [16]. However, this has not been conclusively borne out in the literature. The Ontario Cohort Study [14] of 1680 runners found little correlation between anthropometric measurements (femoral neck version, pelvic obliquity, patella alignment, hindfoot valgus, pes planus/cavus) and risk for injury [1].

Some studies of the incidence and epidemiology of stress fractures in military recruits have suggested that flat or pronated feet have no greater incidence of stress fracture than normal ankle and foot configurations [1, 17, 18]. A study in Israeli military recruits showed an increased incidence of metatarsal fractures in soldiers with a low arch height but fewer tibial and femoral stress fractures [1, 19]. Furthermore, a study in Navy SEAL trainees [20] demonstrated that abnormal hindfoot motion predisposed recruits to injury; specifically, those with restricted hindfoot inversion were more prone to femoral stress fractures, and those with increased hindfoot inversion had a higher incidence of tarsal and metatarsal stress fractures [1]. Others have supported the notion that both high-arched and low-arched feet can cause impairment of function in the subtalar and transverse tarsal joints, predisposing to overuse injuries with repetitive loading in sport [1, 20].

Although the published literature is not conclusive, certainly some ankle and foot conditions do seem to predispose athletes to certain injuries in sports. Careful analysis of lower extremity alignment, including hindfoot varus/valgus and arch height, should always be assessed during a focused physical exam of an injured athlete.

## Footwear

Determining the type of footwear worn by an injured athlete can be a valuable component of a thorough history. Improper fit of an athletic shoe is a common cause of foot complaints in recreational and competitive athletes. For example, shoes that are too tight can cause metatarsalgia or pain at the site of the narrow toebox (i.e., the site of bunion and bunionette pathology); shoes that are too loose can result in blisters [1]. Shoes that are too short on toe strike can result in repetitive crush injuries to the toes, resulting in subungual hematomas, nail plate/bed injury, and ultimately ulcers and osteomyelitis [1].

In most sports, athletes prefer that their footwear confer maximum traction for superior performance in cutting and running, but increasing traction with the playing surface invariably increases the torque load borne by the body or extremity [1]. In sports in which cleated footwear is employed, longer cleats [21] and traditional seven-cleated football shoes [22] have been associated with higher torque and a higher number of ankle injuries than when athletes were switched to a shorter- and more-studded soccer style shoe [1]. Similarly, often basketball players prefer a lower-profile shoe to maximize mobility during games. More traditional high-top shoes can be 50% stiffer than low-cut models and theoretically reduce the load seen by the collateral ligaments of the ankle during inversion/eversion moments [1, 23]. However, a prospective randomized study of high- versus low-top shoes showed no evidence that high-top shoes lowered the incidence of ankle injuries [24]. Basketball shoe height increases the maximum resistance to an inversion moment but may not change the athlete's ability to resist an eversion moment [1, 25]. Others have suggested that strong active ankle evertor muscles alone provide superior protection to inversion stress than three-quarter top basketball shoes [26].

---

## Sports-Related Injuries to the Foot and Ankle

Specific sports-related pathology and injuries of the foot and ankle are reviewed in later chapters. Broadly, these conditions can be separated into acute injuries, in which a particular movement or moment of contact strains or damages native tissue; and overuse-type injuries, in which normal

tissue suffers repetitive microtrauma leading to pain, decreased function/performance, and at times rupture or fracture of the affected anatomic structure.

## Acute Injuries

Intrinsic and extrinsic factors certainly may predispose individuals to a specific type and severity of acute injury to the foot and ankle. In athletic activity, acute injury most commonly occurs when a particular force vector is applied (often through contact with another participant or via the extremity being malpositioned with weightbearing, or "non-contact" type injury) which exceeds the yield strength of a particular anatomic structure, resulting in injury or rupture. Common soft tissue injuries about the foot and ankle include ankle sprains (acute and chronic), syndesmotic injuries (high ankle sprains), Lisfranc injuries and midfoot sprains, turf toe (plantar plate) injuries, and tendon ruptures (most commonly Achilles, peroneal or posterior tibial). Often, an acute rupture or injury indicates the final point on a spectrum of repetitive microtrauma or overuse, such as the athlete with chronic Achilles tendonitis who finally ruptures his or her Achilles tendon during a forceful pushoff during competition. The classification, anatomy, and surgical management of these injuries are discussed in detail in ensuing chapters.

## Overuse Injuries

The ankle and foot are prone to overuse-type injuries ranging from soft tissue inflammation such as Achilles tendonitis to stress reactions and fractures of bone. In competitive athletes, stress fractures of the tibia, foot, and ankle are common and lead to considerable delay in return to play [27]. Stress fractures are common in athletes who engage in repetitive activities, especially runners [2]. When weekly mileage surpasses 40 miles, the risk of injury to runners increases exponentially [1]. High-risk stress fractures, such as those of the anterior tibial diaphysis, navicular, proximal fifth metatarsal, and medial malleolus present particular management challenges and often require surgical intervention, particularly in high-level athletes desiring a quicker return to play [27]. Stress reactions and fractures most commonly occur in watershed areas of relatively poorly-vascularized bone, limiting the ability of the bone to respond to repetitive stress and heal [27].

In athletes, common causes of overuse injuries are training errors, such as sudden changes in terrain surface, workout intensity or schedule, or inappropriate footwear [16]. Often mild cases can be effectively treated with correction of the underlying training error, such as changing inappropriate footwear or modifying the training schedule. These injuries often have an insidious onset and may often be initially

misdiagnosed. Once appropriately recognized, these injuries can require considerable a period of rest, even when surgery is performed, before an athlete can be allowed to safely return to sport.

## Initial Evaluation of the Injured Athlete

The initial approach to evaluating the athlete with an injury about the foot or ankle involves identifying and localizing the injured bony and soft-tissue structures [2]. Careful attention to the mechanism of injury and a review of any worn protective equipment and footwear is vital. Importantly, a focused history and physical exam will narrow the differential diagnosis and determine if further imaging studies are needed. The prevailing trends are expansion of diagnostic imaging and a low threshold to obtain magnetic resonance imaging (MRI) [1]. Once a particular injury has been correctly diagnosed, often the most important issue is determining when it is safe to return to play. This decision, in addition to the severity and type of injury, often also depends on the patient's competition level and temporal issues (in-season versus off-season, approaching events such as combines or tournaments) [2]. As in all of sports medicine, the goal of treatment and surgical decision-making is to ensure safe return to play and to reduce the risk of further or recurrent injury, aligned with the goals of coaches, teams, and the athlete [2].

## References

1. Clanton TO, McGarvey M. Athletic injuries to the soft tissues of the foot and ankle. In: Coughlin MJ, editor. *Surgery of the foot and ankle*. 8th ed. St. Louis: Mosby; 2007. p. 1425–563.
2. Anderson RB, Hunt KJ, McCormick JJ. Management of common sports-related injuries about the foot and ankle. *J Am Acad Orthop Surg*. 2010;18(9):546–56.
3. National Safety Council. *Injury facts*, 2004 edition. Itasca: National Safety Council; 2004. p. 130.
4. Saluta J, Nunley JA. Managing foot and ankle injuries in athletes. *J Musculoskelet Med*. 2010; 355–63.
5. Garrick JG. The frequency of injury, mechanism of injury, and epidemiology of ankle sprains. *Am J Sports Med*. 1977;5:241–2.
6. Baxter DE. Traumatic injuries to the soft tissues of the foot and ankle. In: Mann RA, editor. *Surgery of the foot and ankle*. 5th ed. St. Louis: Mosby; 1986. p. 456–72.
7. Dick R, Agel J, Marshall SW. National Collegiate Athletic Association Injury Surveillance System commentaries: Introduction and methods. *J Athl Train*. 2007;42(2):173–82.
8. Hootman JM, Dick R, Agel J. Epidemiology of collegiate injuries for 15 sports: summary and recommendations for injury prevention initiatives. *J Athl Train*. 2007;42(2):311–9.
9. Borowski LA, Yard EE, et al. The epidemiology of US high school basketball injuries, 2005–2007. *Am J Sports Med*. 2008;36(12):2328–35.
10. Janda DH, Wild DE, Hensinger RN. Softball injuries: etiology and prevention. *Sports Med*. 1992;18:632–5.
11. Janda DH, Wojtys EM, Hankin FM, et al. A three-phase analysis of the prevention of recreational softball injuries. *Am J Sports Med*. 1990;18:632–5.
12. Mueller FO, Blyth CS. North Carolina high school football injury study: equipment and prevention. *Am J Sports Med*. 1974;2:1–10.
13. Powell KE, Kohl HW, Casperson CJ, et al. An epidemiological perspective on the causes of running injuries. *Physician Sports Med*. 1986;14:100–14.
14. Walter SD, Hart LE, McIntosh JM, et al. The Ontario cohort study of running-related injuries. *Arch Intern Med*. 1989;149:2561–4.
15. Macera CA, Pate RR, Powell KE, et al. Predicting lower-extremity injuries among habitual runners. *Arch Intern Med*. 1989;149:2565–8.
16. Heckman DS, Gluck GS, Parekh SG. Tendon disorders of the foot and ankle, part 2: achilles tendon disorders. *Am J Sports Med*. 2009;37:1223–34.
17. Bensek CK. The effects of tropical and leather combat boots on lower extremity disorders among US Marine Corp recruits. Technical report No 76-49-CEMEL, March 1976. Natick: Clothing, Equipment, and Materials Engineering Laboratory, US Army Natick Research and Development Command; 1976.
18. DeVan WT, Carlton DC. The march fracture persists: a report on 143 cases during a fifteen-month period at an infantry basic training center. *Am J Surg*. 1954;87:227–31.
19. Simkin A, Leichter I, Giladi M, et al. Combined effect of foot arch structure and an orthotic device on stress fractures. *Foot Ankle*. 1989;10:25–9.
20. Kaufman KR, Brodine SK, Shaffer RA, et al. The effect of foot structure and range of motion on musculoskeletal overuse injuries. *Am J Sports Med*. 1999;27:585–93.
21. Torg JS, Quedenfeld T. Effect of shoe type and cleat length on incidence and severity of knee injuries among high school football players. *Res Q*. 1971;42:203–11.
22. Rowe ML. Varsity football: knee and ankle injury. *N Y State J Med*. 1969;69:3000–3.
23. Johnson GR, Dowson D, Wright V. A biomechanical approach to the design of football boots. *J Biomech*. 1976;9:581–5.
24. Barrett JR, Tanji JL, Drake C, et al. High- versus low-top shoes for the prevention of ankle sprains in basketball players: a prospective randomized study. *Am J Sports Med*. 1993;21:582–5.
25. Ottaviani RA, Ashton-Miller JA, Kothari SU, et al. Basketball shoe height and the maximum muscular resistance to applied ankle inversion and eversion moments. *Am J Sports Med*. 1995;23:418–23.
26. Ashton-Miller JA, Ottaviani RA, Hutchinson C, et al. What best protects the inverted weightbearing ankle against further inversion? *Am J Sports Med*. 1996;24:800–9.
27. Schindle MK, Endo Y, et al. Stress fractures about the tibia, foot, and ankle. *J Am Acad Orthop Surg*. 2012;20:167–76.

Alexander J. Lampley, Christopher E. Gross,  
Mitchell Klement, and Mark E. Easley

#### Abstract

The foot and ankle is comprised of an array of joints, ligaments and tendons that act to propel the body forward during ambulation while providing a stable weight bearing surface. While the complex relationship between these bones and soft tissue structures can be challenging, the examiner can obtain valuable diagnostic information from a systemic examination of the foot and ankle. All foot and ankle examinations should include a thorough inspection, gait observation, range of motion and neurovascular examination. Specialized tests can further aid the examiner in making the correct clinical diagnosis.

#### Keywords

Foot exam • Ankle exam • Physical examination

## Introduction

The foot and ankle act to propel the body forward during ambulation, to absorb loading forces, and to provide a stable weight bearing surface. To accomplish these tasks, the foot and ankle is comprised of an intricate array of joints, ligaments and tendons. The complicated synergistic relationship between the bones and soft tissue structures combined with the number of joints in the foot can be challenging for an examiner. However, by following a systemic approach, the examiner can obtain an exam that aids him in making the correct diagnosis.

---

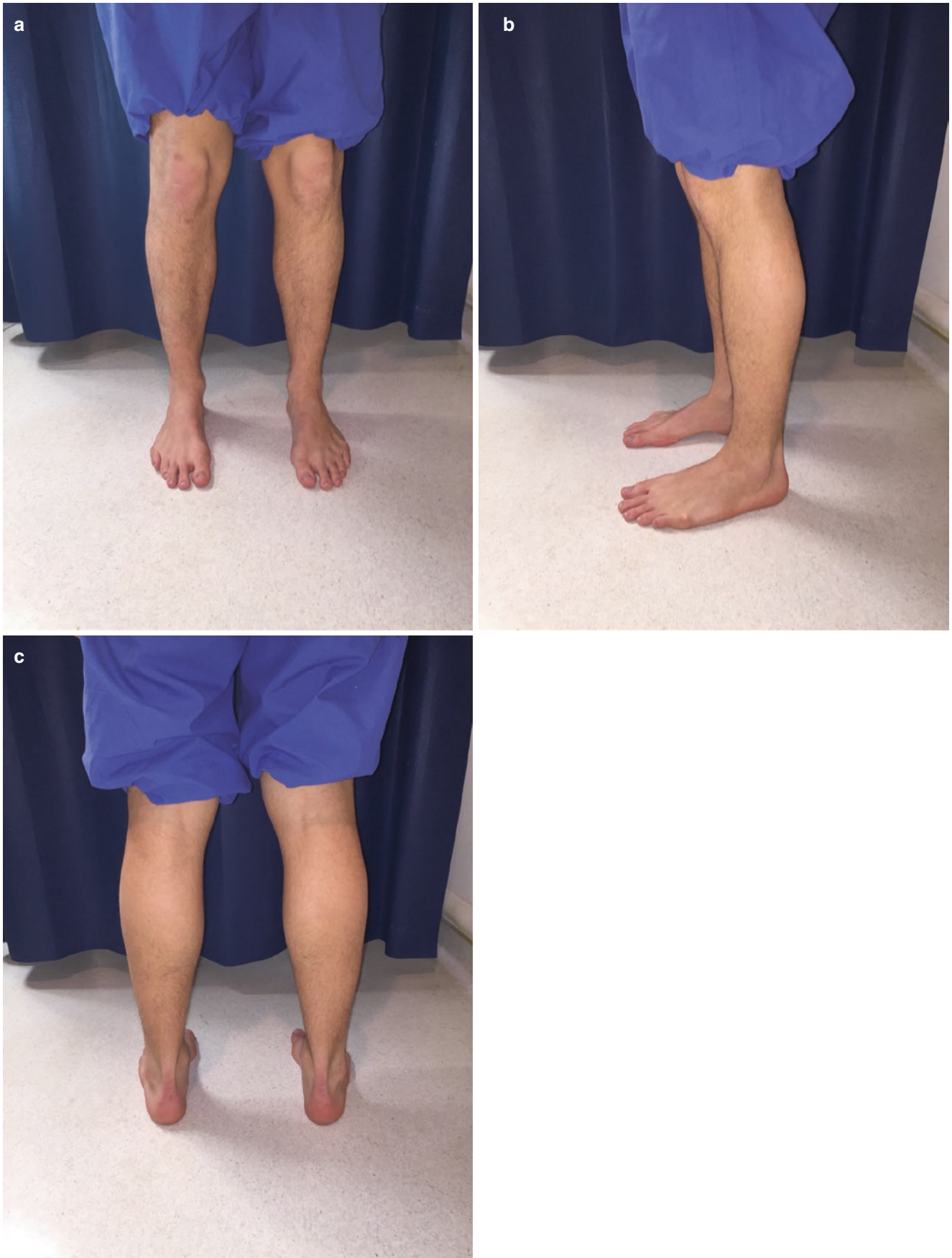
A.J. Lampley, MD • C.E. Gross, MD  
M. Klement, MD • M.E. Easley, MD (✉)  
Department of Orthopaedic Surgery, Duke University Medical  
Center, 4709 Creekstone Drive, Durham, NC 27703, USA  
e-mail: [alexander.lampley@dm.duke.edu](mailto:alexander.lampley@dm.duke.edu); [cgross144@gmail.com](mailto:cgross144@gmail.com);  
[mitchell.klement@dm.duke.edu](mailto:mitchell.klement@dm.duke.edu); [mark.e.easley@dm.duke.edu](mailto:mark.e.easley@dm.duke.edu)

## Inspection

The patient should expose both lower limbs from the knee to the foot. Shoes should be removed. Inspection of the patient's footwear can reveal wear patterns that may give clues to any gait abnormalities such as lateral overloading or if the running athlete is a heel striker. The examination of the patient begins by observing the patient from the front, side and back with the patient standing (Fig. 4.1). The overall general alignment of the limb and approximate leg lengths should be noted. With the patient's back toward the examiner, the alignment of the hindfoot is noted (Fig. 4.1c). At the same time, one can evaluate for the "too many toes" sign, in which more than the fifth toe and half of the fourth toe are seen. This sign is may be indicative of a flatfoot deformity.

The skin should be inspected for swelling, erythema, or ecchymosis which may suggest location of an injury or infectious process.

The foot is then inspected from distal to proximal noting the shape of the foot, alignment of the midfoot and toes in relation to the hindfoot, and any toe deformities (e.g. hallux



**Fig. 4.1** The examination of the patient begins by observing the patient from the front (a), side (b) and back (c) with the patient standing

valgus, claw toe, mallet toe, or hammer toe deformity). Attention should be paid to the medial arch to evaluate for pes planus or pes cavus. A subtle cavovarus foot may predispose running athletes to injury.

---

## Gait Observation

Much of the gait examination can take place as the patient is walking toward the waiting area to the examination room. The patient's gait pattern is observed with the patient walking away and towards the examiner. It is important to observe the patient walking down the hall, since examination rooms are rarely large enough to accommodate a full gait cycle. An antalgic gait is seen in any condition that causes pain in the lower extremity causing the stance phase to be shorter in the painful limb. This type of gait may be present with an acute injury or stress fracture. A foot slap or steppage gait is seen in patients with weak ankle dorsiflexors and involves pronounced hip and knee flexion to clear the affected foot and toes. Special gait tests include:

- heel walking: testing ankle dorsiflexor strength, particularly the tibialis anterior,
- toe walking: testing ankle plantar flexor strength, particularly the gastrocnemius-soleus complex
- lateral foot walking: testing inversion strength, particularly the tibialis posterior
- medial foot walking: testing eversion strength, particularly the peroneal longus and brevis

---

## Range of Motion

The range of motion (ROM) assessment should include both passive and active ROM examination. Range of motion should always be compared to the contralateral limb to evaluate any deficits and differences.

## Ankle ROM

Plantar flexion, dorsiflexion, inversion and eversion are the four main motions that occur at the ankle. The examiner must be sure to stabilize the midfoot when testing ankle ROM as motion through these distal joints can falsify the true ankle ROM. A goniometer is useful to both the novice and skilled examiner.

Normal passive ROM for ankle dorsiflexion is 10–15°, ankle plantar flexion is 50–70°, inversion is 30–40°, and eversion is 10–15°.

## Subtalar ROM

Subtalar motion is measured in eversion and inversion of the hindfoot in respect to the leg. A line is drawn that bisects the posterior heel and that bisects the leg. Subtalar ROM is then found by measuring the angle between the heel and lower leg with maximal eversion and inversion of the hindfoot.

## First Metatarsal-Phalangeal Joint ROM

The first metatarsal-phalangeal joint (MTPJ) normally extends 60–70° and flexes 40–50°. Hallux limitus presents with loss of motion at the MTPJ while hallux rigidus presents with a greater loss of motion and pain with MTPJ ROM.

---

## Palpation

Because the foot and ankle have minimal overlying soft tissue, the examiner usually is able to palpate specific bones, ligaments and tendons fairly accurately. Palpating areas of tenderness can help localize injury to specific areas or structures. Patients with medial tibial stress syndrome (pain related to traction on the periosteum by the posterior tibialis or flexor digitorum longus) will have posteromedial tibial pain. Tibial stress fractures will have pain with palpation of the tibia.

---

## Neurovascular Exam

A complete foot and ankle exam should include test of intact motor and sensory function as well as vascular status of the extremity. The motor branch of the superficial peroneal nerve is tested with eversion and inversion of the foot (peroneal muscles) (Fig. 4.2). The deep peroneal nerve is tested with dorsiflexion of the ankle (tibialis anterior), and the tibial nerve is tested with plantar flexion of the ankle (gastrocnemius-soleus complex) and inversion (tibialis posterior) (Fig. 4.3). Sensation of the medial and lateral plantar nerves, saphenous nerve, sural nerve, superficial peroneal nerve, and deep peroneal nerve must be tested as well. Specific deficiencies may be indicative of compression neuropathy. In the fourth toe flexion sign, when the fourth toe is passively plantarflexed, the skin overlying the superficial peroneal nerve is tented. A thorough vascular examination should include palpation of dorsalis pedis and posterior tibial pulses with observation of any signs of venostasis or vasculopathy. Capillary refill should be noted as well.



**Fig. 4.2** Evaluation of the eversion strength of the peroneal tendons



**Fig. 4.3** Evaluation of the inversion strength of the posterior tibialis tendon

## Specialized Tests of the Ankle

### Ankle Instability

The anterior drawer test evaluates the integrity of the anterior talofibular ligament (ATFL). The test is performed with the knee flexed  $\sim 20^\circ$ , the hindfoot in neutral, and the ankle in neutral dorsiflexion (Fig. 4.4). The examiner stabilizes the leg with one hand and pulls the heel forward with the opposite hand. Laxity with respect to the contralateral limb suggests ATFL injury. If the ankle is tested in plantarflexion, the CFL is tested (Fig. 4.5).

The talar tilt test evaluates the calcaneofibular ligament (CFL) and ATFL integrity. The test is performed by stabilizing the leg and applying a varus force to the ankle via the heel. An angulation of greater than  $23^\circ$  or more than  $10^\circ$  difference between the injured ankle and contralateral ankle suggests complete rupture of both the CFL and ATFL [1].

The squeeze test (Fig. 4.6) and external rotation test are useful in evaluating the syndesmosis, particularly the anterior and posterior inferior tibiofibular ligaments. The squeeze test is performed by compressing the tibia and fibula together in the midshaft area. The external rotation test (Fig. 4.7) is done by applying an external rotation force on the ankle with

the ankle maximally dorsiflexed. Pain with either exam suggests a high ankle sprain.

### Ankle Impingement

Anterior ankle impingement symptoms can be reproduced by quick, forceful dorsiflexion of the ankle. Reproduction of the patient's anterior joint line pain is a positive test (Fig. 4.8). Posterior impingement symptoms of posterior joint line pain may be reproduced with quick, forceful plantar flexion of the ankle (Fig. 4.9).

### Achilles Tendon Rupture

An Achilles tendon rupture can be diagnosed with the Thompson test (Fig. 4.10). With the patient lying prone with the knee flexed to  $90^\circ$ , the examiner squeezes the calf to induce passive plantar flexion of the foot. If the Achilles tendon is completely ruptured, the foot will not plantar flex. The patient will also present with significant swelling and ecchymosis over the Achilles tendon, a palpable defect



**Fig. 4.4** The anterior drawer test evaluates the integrity of the anterior talofibular ligament. The test is performed with the hindfoot in neutral, and the ankle in neutral dorsiflexion. (a) Anterior view. (b) Side view.

approximately 3 cm proximal to the Achilles tendon insertion and inability to toe walk. Matles test also aims to diagnose a complete rupture of the Achilles tendon by comparing the resting position of the affected foot to the contralateral foot. This test is performed with patient lying prone and the knee flexed to 90° with both the foot and ankle relaxed. The uninjured foot should rest in slight plantar flexion, while the injured foot with an Achilles tendon rupture will rest in more dorsiflexion (Fig. 4.11).

### Achilles Tendinopathy

Achilles tendonitis can be separated into non insertional and insertional tendinitis. Non insertional tendinitis will present with tenderness to palpation along the Achilles tendon substance, roughly 2–3 cm proximal to its insertion site (Fig. 4.12). A defect in the tendon will not be felt, and Thompson test will be negative. Insertional Achilles tendonitis, on the other hand, presents with tenderness at the insertion of the tendon onto the calcaneus (Fig. 4.13). A prominence at the superior lateral posterior aspect of the calcaneus is called Haglund's deformity.

### Achilles Tendon Contracture Verses Gastrocnemius Contracture

The Silverskiold test is used to differentiate between a tight gastrocnemius and a tight Achilles tendon. The test is performed by comparing dorsiflexion of the ankle with the patient's knee extended to dorsiflexion of the ankle with the patient's knee flexed to 90°. Because the gastrocnemius crosses the knee and ankle joint, the muscle can be effectively lengthened with knee flexion. Therefore, an increase in ankle dorsiflexion with the knee flexed localizes the contracture to the gastrocnemius. No change in ankle dorsiflexion in the extended and flexed knee localizes the contracture to the Achilles tendon.

### Peroneal Tendonitis and Subluxation

Peroneal tendonitis is a common cause of lateral ankle pain. The peroneus brevis is palpable at the posterior border of the lateral malleolus to its insertion at the base of the fifth metatarsal. Resisted eversion and plantar flexion of the foot can





**Fig. 4.5** Performing the anterior drawer test in plantarflexion will test the strength of the calcaneofibular ligament



**Fig. 4.6** The squeeze test



**Fig. 4.7** The external rotation test



**Fig. 4.8** Forced dorsiflexion of the ankle to examine for anterior osteophytes

be performed to test the strength of the peroneal tendons and also make them more visible on exam.

The examiner may be able to diagnosis a subluxing peroneal tendon by palpating the tendon at the posterior lateral malleolus and having the patient rotate the ankle clockwise and counterclockwise (Fig. 4.14). Resisted dorsiflexion and eversion may also reproduce the subluxation of the peroneal tendons [1].



**Fig. 4.9** Forced plantarflexion to test for impingement



**Fig. 4.11** Matles Test



**Fig. 4.10** Thompson test

## Specialized Tests of the Foot

### Stress Fractures

Common locations stress fractures from overuse are the distal second and third metatarsal, the proximal fifth metatarsal and calcaneus. Pain over these areas or pain with compression of the calcaneus may suggest overuse stress fracture of



**Fig. 4.12** Tenderness 3–5 cm proximal to insertion site of Achilles may indicate of tendinopathy



**Fig. 4.13** Pain at the insertion of the Achilles



**Fig. 4.15** Tenderness at the N-spot is indicative of a navicular stress fracture



**Fig. 4.14** Eversion of the peroneals may cause subluxation



**Fig. 4.16** Single limb heel rise



**Fig. 4.17** Lachman test of the 1st MTP joint

these bones in the setting of normal radiographs. Likewise, navicular stress fractures often present with tenderness to palpation and pain localized at the dorsum of the navicular bone, the appropriately named ‘N spot’ [2] (Fig. 4.15).

### Morton’s Neuroma

A Morton’s neuroma is most often found between the third and fourth metatarsal head. A squeeze test is performed by compressing the first and fifth metatarsal heads together. Pain with this maneuver is a positive test. A palpable click felt by the examiner is called a Mulder’s click.

### Posterior Tibialis Tendon Dysfunction

Posterior tibialis tendon dysfunction is a common cause of acquired flatfoot which presents with loss of the medial arch (pes planus), hindfoot valgus, and forefoot abduction. Forefoot abduction results in the “too many toes” sign. The “too many toes” sign is positive when the examiner observes the patient from behind and the more toes are seen lateral to the tibia on the affected foot when compared to the contralateral foot. The

posterior tibial edema sign is described as objective evidence of posterior tibialis tendon dysfunction when there is pitting edema along the course of the posterior tibialis tendon posterior to the medial malleolus [3]. A single limb heel raise is a crucial test in evaluating the posterior tibialis tendon (Fig. 4.16). The patient is asked to stand on the toes of the affected limb. The patient may use the wall for balance. In patients that can perform a single leg heel raise, the posterior tibialis tendon is likely intact while inability to perform a single leg heel raise suggests posterior tibialis tendon pathology.

### Tarsal Tunnel Syndrome

Physical examination of the tarsal tunnel include percussion testing, triple compression stress test, and dorsiflexion-eversion test. Percussion testing is performed by percussing over the tarsal tunnel. The triple compression stress test is performed by placing the ankle in full plantar flexion and the foot in inversion with constant digital pressure applied over the posterior tibial nerve at the tarsal tunnel. Dorsiflexion and eversion of the foot is also described as a maneuver to increase pressure on the posterior tibial nerve in the tarsal tunnel. Aggravation of paresthesias or pain with any of these maneuvers is considered a positive test.

### Turf Toe/Plantar Plate Injury

Turf toe presents with plantar swelling and ecchymosis of the injured toe. The plantar plate can be evaluated with the Lachman test of the MTPJ. This is performed by holding the base of the proximal phalanx with one hand while supporting the corresponding metatarsal neck with the other hand (Fig. 4.17). Dorsal pressure is applied to the proximal phalanx in an attempt to sublux the joint dorsally. The test is positive if there is greater laxity of the MTPJ when compared to the contralateral side.

### Evidence

Multiple studies have demonstrated the sensitivity and specificity of certain specialized foot and ankle tests. When evaluating lateral ankle sprains, the anterior drawer test was found to have sensitivity of 58% and specificity of 100%, and the talar tilt stress test had a sensitivity of 58% and specificity of 88% [4].

For the evaluation of high ankle sprain or syndesmotic ankle injury, the external rotation test was reported to have a sensitivity of 20% and specificity of 85–99% [5, 6]. The syndesmosis squeeze test was found to have a sensitivity of 30% and specificity of 94% [6].

The Thompson test was reported to have a sensitivity and specificity of 96 % and 93 % respectively in diagnosing Achilles tendon rupture [7]. A palpable gap in the Achilles tendon was reported as having a 73 % sensitivity and 89 % specificity [7]. The Matles test was found to have a sensitivity and specificity of 96 % and 93 % respectively [7]. In addition, the American Academy of Orthopaedic Surgeons clinical practice guidelines recommend that the diagnosis of acute Achilles tendon rupture can be made with two or more of the following physical exam findings: positive Thompson test, palpable defect in the Achilles tendon, decreased plantar flexion strength and increased passive ankle dorsiflexion [8].

For the diagnosis of anterior ankle impingement, the forced dorsiflexion test was found to have a sensitivity and specificity of 95 % and 88 % respectively [9].

The posterior tibial edema sign for posterior tibial tendon dysfunction was found to have a sensitivity of 86 % and a specificity of 100 % [3].

Three studies [10, 11] addressed the sensitivity and specificity of the percussion test, triple compression stress test and dorsiflexion-eversion test for the diagnosis of tarsal tunnel syndrome. The percussion test was found to have a sensitivity of 58 % while specificity was not calculated. The triple compression stress test demonstrated a sensitivity of 86 % and specificity of 100 %. The dorsiflexion-eversion test yielded a sensitivity of 25–98 % and a specificity of 100 %. Overall, the majority of special tests in the foot and ankle examination provide valuable information to aid in proper diagnosis of the patient's pathology.

---

## Summary

- A good appreciation and understanding of the biomechanics and anatomy of the foot and ankle along with a careful examination of the foot and ankle is critical in aiding in the diagnosis of foot and ankle injuries.

- An understanding of the potential injuries and pathologies that can affect the foot and ankle is needed to put the examination into proper context.
- A foot and ankle examination should include evaluation of the patient's gait.
- Specialized tests or palpation that reproduces the patient's symptoms is critical to making a correct diagnosis.
- Specialized tests demonstrate good specificity which makes them helpful tools in confirming a diagnosis.

---

## References

1. Mahaffey D, Hiltz M, Fields KB. Ankle and foot injuries in sports. *Clin Fam Pract.* 1999;1:233–50.
2. Towne LC, Blazina ME, Cozen LN. Fatigue fracture of the tarsal navicular. *J Bone Joint Surg Am.* 1970;52:376–8.
3. DeOrto JK, Shapiro SA, McNeil RB, et al. Validity of the posterior tibial edema sign in posterior tibial tendon dysfunction. *Foot Ankle Int.* 2011;32:189–92.
4. Hertel J, Denegar CR, Monroe MM. Talocrural and subtalar joint instability after lateral ankle sprain. *Med Sci Sports Exerc.* 1999;31:1501–8.
5. Beumer A, Swierstra BA, Mulder PG. Clinical diagnosis of syndesmotric ankle instability: evaluation of stress tests behind the curtains. *Acta Orthop Scand.* 2002;73:667–9.
6. de Cesar PC, Avila EM, de Abreu MR. Comparison of magnetic resonance imaging to physical examination for syndesmotric injury after lateral ankle sprain. *Foot Ankle Int.* 2011;32:1110–4.
7. Maffulli N. The clinical diagnosis of subcutaneous tear of the Achilles tendon. *Am J Sports Med.* 1998;26:266–70.
8. Kou J. AAOS Clinical Practice Guidelines: acute Achilles tendon rupture. *J Am Acad Orthop Surg.* 2010;18:511–3.
9. Molloy S, Solan MC, Bendall SP. Synovial impingement in the ankle. *J Bone Joint Surg Am.* 2003;85:330–3.
10. Abouelela AA, Zohiery AK. The triple compression stress test for diagnosis of tarsal tunnel syndrome. *Foot.* 2012;22:146–9.
11. Kinoshita M, Okuda R, Morikawa J, et al. The dorsiflexion-eversion test for diagnosis of tarsal tunnel syndrome. *J Bone Joint Surg Am.* 2001;83:1835–9.

John Wesley Latting and Charles E. Spritzer

## Abstract

As is true for orthopedics in general, the assessment of a sports related injuries of the foot and ankle begins with a good history and physical. While many maladies are readily diagnosed, often a differential remains. In these instances, imaging assessment is often useful. Additionally, as our understanding of injury to the foot and ankle evolves, a more nuanced approach to treatment has developed. Imaging is often required to optimize treatment. This brief chapter is intended to provide an overview to imaging sports related pedal injuries. This chapter is not a comprehensive review. Whole books intended to provide such information have been written. Rather, this manuscript is intended to provide information to guide the reader in deciding when and with what imaging modality to assess a patient further. By necessity and design, this manuscript is based much upon the author's opinions and experience. It is understood that different imaging approaches to a clinical problem are possible, depending upon the experience of the imager and which imaging modalities are readily available.

## Keywords

Magnetic resonance imaging • Ultrasound • Computerized tomography • Foot • Ankle • Achilles tendon • Stress Fracture • Sesamoid • Tendinosis • Ankle Ligaments • Peroneal tendon • Lisfranc Joint

## Introduction & Techniques

With society attempting to adopt a more healthy lifestyle, there is an increasing participation by the population at large in recreational sports and exercise with many individuals competitively competing in sports activities. Unfortunately, such activities are associated with injury. While a good clinical history and physical examination are the mainstays of

diagnosing injuries of the foot and ankle, imaging has assumed a critical role in confirming clinical impressions and potentially optimizing patient care [1].

Even with the advent of cross sectional imaging, conventional radiographs are the first imaging modality to be utilized in most clinical scenarios. Besides their low cost and ready availability, they often are sufficient to make the diagnosis (e.g. fracture) or provide supplemental information to more advanced (and expensive) imaging techniques. Small avulsions, air, and calcifications are better appreciated on radiographs than on MR images.

CT continues to improve in both its speed of acquisition and spatial resolution. Weight bearing CT scanners are becoming available. Subtle fractures missed on radiographs are more readily identified. Small avulsions such as seen with Lis Franc injuries may be readily diagnosed. Optimal assessment requires high resolution acquisitions often with

---

J.W. Latting, MD  
Department of Radiology, Duke University Hospital,  
2301 Erwin Road, Durham, NC 27710, USA  
e-mail: [john.latting@duke.edu](mailto:john.latting@duke.edu)

C.E. Spritzer, MD (✉)  
Department of Radiology, Duke University School of Medicine,  
Duke University Medical Center,  
Durham, NC, PO Box 3808, 27710, USA  
e-mail: [chuck.spritzer@duke.edu](mailto:chuck.spritzer@duke.edu)

orthogonal image reconstruction. Both bone and soft tissue algorithms are required for maximum diagnostic capability. Dual Energy CT will reduce metal artifacts and likely improve marrow sensitivity.

MR imaging has become tremendously important in assessing sports related injuries in the foot and ankle. Capsular, ligament, and tendon injuries are readily appreciated. Subtle but important osseous abnormalities, such as contusions, imperceptible on radiographs and CT are readily diagnosed. The presence of chondral abnormalities are routinely determined. While there are multiple approaches to acquiring optimal MR studies, it is our opinion that optimal imaging requires two if not three orthogonal planes of high resolution images. We routinely acquire T1 weighted nonfat suppressed images and either T2 weighted fat suppressed fast spin echo images or fast inversion recovery (STIR) images in multiple planes. For the major tendons about the ankle we routinely acquire fat suppressed proton density weighted fast spin echo images orthogonal to the course of the posterior tibialis tendon.

Ultrasound (US) imaging continues to assume a more important role in assessing injury of the foot. This is especially true for tendon examination. It has the advantage of low cost and lack of ionizing radiation. Dynamic assessment for tendon and ligament stability offer information only indirectly obtainable by MRI. The introduction of high frequency transducers provides resolution at least equivalent to MRI. Its principle advantage however is the potential use at the point of care. Indeed, many clinicians not formally trained are using US as an initial assessment in their offices.

Spect and Spect CT have been demonstrated to provide significant information concerning osseous injuries of the foot. Indeed, there are studies suggesting increased sensitivity over MRI for stress fractures and degenerative causes of pain [2]. However, the widespread incorporation of these modalities is limited by their cost, availability, and concern of radiation exposure. At our site, we believe that the modality is probably underutilized, and has been reserved for problem solving, pain localization, and used in cases where other imaging modalities have failed to elucidate a definitive answer.

---

## Osseous Abnormalities

### Stress Fractures

Most stress fractures in the foot occur in the metatarsals, especially the second & third. However, they can occur in most tarsal bones especially the navicular [3]. Stress fractures develop in the setting of recurrent microfracture with insufficient interval bone healing. These lesions tend to develop in watershed areas of highly stressed bone where bony remodeling is unable to keep pace with repetitive

micro-damage [4]. Within the foot, intrinsic causes such as cavus foot and forefoot varus deformities and extrinsic causes such as excessive training, improper technique, poor equipment, and improper training surfaces predispose to fracture [3, 5, 6]. Early diagnosis and treatment can lead to better outcomes, especially in athletes.

Radiographs are an easily accessible modality for primary survey and surveillance of stress fractures. Early radiographic signs can be subtle. In the metatarsals, they begin as a subtle linear lucencies, perhaps with faint periosteal reaction that progresses to focal thickening of the cortex. As the fracture continues to evolve, linear sclerosis and eventually disruption of the cortex are seen. In cancellous bone, such as the calcaneus and navicular, stress fractures appear in the relative vascular poor mid body of the bones as subtle linear sclerosis perpendicular to the trabecular bony lines [4].

There is often a significant delay between onset of symptoms and radiographic signs of stress fracture. In the setting of negative radiographs where stress fracture is clinically favored, radiographs can be repeated after 10–14 days allowing time for bone remodeling and periosteal reaction to occur. However, in athletes and highly active individuals, early treatment of stress fracture can decrease recovery time by weeks making timely diagnosis imperative. In these instances, MR is the study of choice. Subtle marrow reactive change, edema, infarctions and even cortical breaks can be seen by MRI weeks before structural abnormalities become evident on radiographs [4, 7]. Fat suppressed water sensitive sequences with decreased marrow signal are essential, allowing edema to be seen as high signal against a dark background of normal bone. Edema is a secondary finding that can be seen in early stress fracture before a fracture line is present. As such, Fredrickson and colleagues proposed a four tier grading system to categorize the spectrum of MR findings associated with stress fractures [3, 8]: Grade one injury demonstrates periosteal edema on T2-weighted fat suppressed images. Grade 2 injury shows T2-weighted periosteal and marrow edema, without corresponding T1 abnormalities. Grade 3 injury demonstrates findings of bony and periosteal edema on both T1 and T2 weighted images. A cortical fracture line is seen in addition to all of these findings in Grade 4 injury Fig. 5.1.

Technecium 99 m bone scan is another alternative following negative radiographs with a high sensitivity for the diagnosis of stress fracture. Scintigraphic evidence of fracture can present weeks before radiographic findings [4]. These manifest as linear or focal areas of increased radiotracer activity at the site of fracture. Despite its high sensitivity bone scans have a lower specificity than MR [4].

The mobility and low cost of ultrasound have made it a rapidly evolving modality for initial evaluation of musculoskeletal injuries and several studies have shown some success in sonographic diagnosis of stress fracture. However, this



**Fig. 5.1** 19 year old male athlete with worsening fore foot pain. (a) Plain film is normal. (b) Sagittal STIR image through the 3rd metatarsal shows increased T2 signal within the shaft representing a contusion with a linear area of decreased signal representing a stress fracture (*arrow*) involving the dorsal surface. (c) Sagittal T1 weighted image

showing decreased T1 signal within the marrow. There is cortical thickening and a decreased signal linear band of the stress fracture dorsally (*arrow*). (d) Sagittal CT reformation suggesting the stress fracture dorsally

requires visualization of superficial structural abnormalities, such as cortical step off that are often seen later in the evolution of the stress fracture [4, 5]. As such early abnormalities detected with MRI or radionuclide imaging may be missed delaying diagnosis.

Although CT is rarely used in the initial diagnosis of stress fractures due to its low sensitivity compared with MR or radionuclide imaging and its relative high radiation dose,

it is occasionally employed to more definitely assess a cortical break and the extent of healing [7].

### Sesamoid Pathology

The first MTP joint sesamoids form a true synovial joint with the metatarsal head and are biomechanically a continuation



of the plantar plate. They are anchored at the MTP joint by the medial and lateral metatarsal-sesamoid and sesamoid-phalangeal ligaments [9]. A variety of disease processes occur here including fracture, inflammation, infection, and arthritis [10]. These are not specific to the athlete, but some are more commonly found in the acute traumatic setting. The first metatarsophalangeal (MTP) joint is much more often affected than the lesser MTP joints because the sesamoids at this joint are always present and endure the greatest amount of stress during running [10].

At the first MTP joint, fracture occurs more commonly at the medial sesamoid, which is larger and bears more weight than the lateral sesamoid. Radiographs are the initial modality of choice for foot pain. The sesamoids can be seen on AP, oblique, and lateral views of the foot. However, for optimal evaluation an axial view of the sesamoids with the first toe in a flexed position, as well as a lateral view will provide the most information [9, 11]. Fractures can be difficult to diagnose on radiographs for several reasons. The sesamoids are small and cortical breaks can be hard to visualize. Furthermore, they can be multipartite in a variety of combinations making it difficult to distinguish a congenital variant from an acute fracture [10]. The presence of jagged edges along the contour can help make the diagnosis, especially if there are reciprocal contours on the adjacent bone fragment. MR can be used when radiographs are equivocal, or in the setting of normal radiographic findings when a stress fracture is suspected, and will demonstrate edematous reaction within the bone and adjacent soft tissues. CT is excellent for evaluating bony details and may be useful to assess the extent of fracture or healing. Nuclear scintigraphy bone scan can be used to evaluate fractures and stress fractures with much greater sensitivity than radiographs. However, it will provide little detail regarding the surrounding soft tissues and cannot differentiate between fracture, inflammation, and infection [9, 11].

Inflammatory conditions can involve the sesamoid bones, often in the setting of repetitive stress. Known as sesamoiditis, this inflammatory reaction is most common at the first MTP sesamoids, which endure the greatest stress. Again, MR is the imaging modality of choice and will demonstrate T2 hyperintensity throughout the sesamoid. A hypointense T1 line within the sesamoid or the presence of a bone fragment is indicative of fracture, and surrounding T2 hyperintense signal will likely extend into the adjacent soft tissues. MR may also be helpful to evaluate infection at the hallux sesamoids and will likely demonstrate more extensive soft tissue involvement as well as extension into the adjacent joint space [9, 10].

Osteoarthritis may affect older athletes, but in young athletes post-traumatic degenerative changes and seronegative arthropathies are more likely to be seen. Erosions and geodes can be seen on radiographs if they become large enough.

However, CT may be necessary to evaluate fine bony destruction in these conditions [9, 11]. MRI is considered the study of choice to assess erosive disease and cartilage loss.

## Osteochondral Injuries

Osteochondral lesions of the talus (OLT), also known as osteochondral defects or osteochondritis dissecans were first described by Kappis in 1922 [12]. The etiology remains unclear, but is likely some combination of repetitive micro-trauma, trauma, and vascular compromise [13]. While OLTs may occur anywhere on the talar dome, the posteromedial location is most frequent.

Imaging begins with weight-bearing AP, ankle mortise, and lateral radiographs. While radionuclide imaging, CT, dual energy CT, and Spect CT have all been shown to have utility in assessing OLTs, MRI is currently the study of choice [2, 13–17]. CT is particularly useful for assessing bony detail and subchondral cyst formation and at our site is routinely obtained for surgical planning. Preliminary data suggests that dual energy CT may assess bone marrow edema as well as MRI [17]. For MR assessment of OLTs, both high resolution T1 weighted and fat suppressed T2 weighted images in the sagittal and coronal planes are required. To improve resolution, there is a trend to image at 3 Tesla and use volume acquisitions Fig. 5.2.

Prognosis and treatment depends upon the lesion size, its stability, and the status of the overlying cartilage [13, 16]. Operative treatment of OLTs is based on likelihood of progression of necrosis and bone detachment. Lesions at low risk for progression may heal with conservative management, while high risk lesions require aggressive intervention. High risk lesions are associated with linear increased T2 signal at the periphery of the lesion, cystic fluid signal adjacent to the lesion, increased T2 signal extending to the articular surface, and a focal defect at the articular surface of the lesion [18]. A direct relationship exists between the extent to which these findings are present and the predictive value of progression to detachment. Progressive separation of the lesion on serial imaging carries a poor prognosis [19].

## Fracture Healing

As Morshed et al. [20] recently stated, “Although there are numerous methods for defining fracture-healing in clinical studies, no consensus exists regarding the most valid and reliable manner for assessing union or for determining which outcomes are most important.” As such, comparing imaging modalities in the assessment of fracture healing is problematic at best. Clearly conventional x-rays are the initial study for following fractures and are usually sufficient. If x-rays



**Fig. 5.2** A female with prior ankle injuries and persistent pain. **(a)** Ankle mortise view showing an OLT in the medial talar dome. **(b, c)** Coronal fat suppressed T2 weighted and T1 weighted images. The OLT is readily identified. There is mild depression of the cortical surface and

disruption of the overlying cartilage. Reactive changes as well as a subchondral cyst are present. **(d)** Corresponding CT, obtained prior to surgery shows the subchondral cyst to better advantage

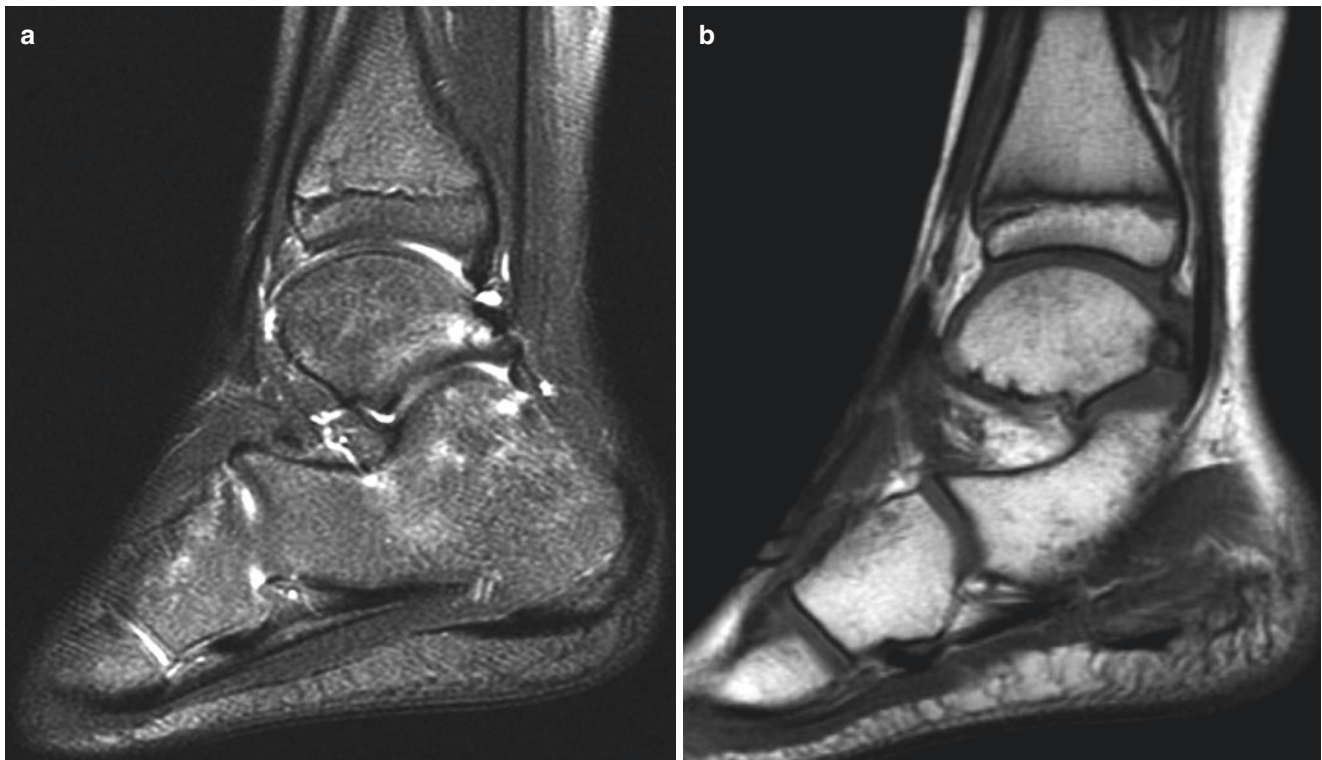
are equivocal, CT is typically employed as overlapping structures are not an issue and spatial resolution is improved. More importantly, bony bridging and even subtle callus formation are better appreciated.

The presence of hardware is a significant limitation in assessing healing. Plates and screws obscure the areas of healing when viewed on plain films and cause significant artifacts on CT. Photon starvation, beam hardening, and scatter all degrade the CT images. While high energy acquisitions and more appropriate reconstruction techniques have improved image quality, studies are often suboptimal [21]. More recently, dual energy CT scanning has become readily available. As the name suggests, data is obtained using x-ray beams of two different energies. Post processing allows display of information dependent upon specific absorption spectra. Preliminary data suggests that these techniques are more robust in assessing tissue and bone surrounding hardware [21–23].

The role of MRI in fracture healing is poorly defined and little literature exists even defining normal healing [24, 25]. At our site, MRI is employed when there are additional issues of clinical concern. Examples include: (1) Possible AVN of the talar dome with an associated talar neck fracture and (2) Assessment of navicular healing following surgical repair.

## Painful Bone Marrow Edema

Painful bone marrow edema syndrome (PBMES) of the foot and ankle is a diagnosis of exclusion [28] well appreciated on MRI. Although changes can be seen with radionuclide studies [26], the modality is nonspecific. Osteopenia may be detected on x-rays and CT [27–29]. As the name suggests, it is a painful condition lasting for months, classically seen in the lower extremity without a history of acute or repetitive trauma. The diagnosis by MRI requires well fat suppressed T2 weighted or STIR images. On these images two or more bones in the foot are affected and are characterized by diffuse irregular increased signal intensity on STIR or fat suppressed T2 weighted images. Non fat suppressed T1 images show patchy ill defined areas of subtle decreased signal intensity Fig. 5.3. According to Orr et al. [30], in descending order, the calcaneus, talus, navicular, cuneiforms and cuboid. The differential diagnosis is broad including trauma (contusion), neoplasm, neurogenic arthropathy, AVN, infection, transient osteoporosis, and complex regional pain syndrome. It is the latter three entities which are the most difficult to distinguish from PBMES by MRI alone. Indeed, many authors believe these to be a spectrum of the same disease [27, 28]. Infection should be easily excluded although a mild elevation of the sedimentation rate in PBMES has been



**Fig. 5.3** 11 year old male with several months of pain, inconclusive clinical examination and negative radiographs. (a) Fat suppressed T2 weighted sagittal image shows patchy areas of increased signal in the

tibia, talus, calcaneus and cuboid. (b) Corresponding T1 weighted image shows subtle patchy areas of decreased signal corresponding to the abnormal areas in (a)

reported. The presence of osteopenia on radiographs supports the diagnosis of transient osteoporosis. Complex regional pain syndrome is typically associated with skin and soft tissue changes which may be seen on MRI or are clinically apparent.

## Soft Tissue Abnormalities

### Tendon Tears

For assessment of the tendons of the foot, US and MRI are the imaging modalities of choice. The advantages of US include its lower cost and its ability to be utilized at the point of care [31]. MRI provides a more comprehensive examination and better spatial visualization of the pathology. Regardless of the modality, the issues for tendon assessment are the same. Is the tendon intact and in the correct location? If it is torn, is it a partial tear or a complete tear? If it is intact, is there tendinosis or tenosynovitis? By both modalities, complete tears demonstrate tendon disruption, perhaps with retraction. There will be fluid/hemorrhage, scar and/or granulation tissue in the gap depending upon the chronicity of the tear. Partial tears by US appear as linear (longitudinal split tear) or focal hypoechoic regions within the tendon substance [32]. By MRI, these same areas of abnormality will be near fluid bright on a STIR or fat suppressed T2 weighted sequence. Loss of tendon size would be an additional finding concerning for partial tear. Tendinosis is evident by tendon thickening. By US, this will be associated as decreased echogenicity and loss of the normal architecture in the longitudinal plane. With MRI, diffuse or focal areas of increased T2 signal will be present. For both MRI and US, the distinction of marked tendinosis from partial tearing can be problematic [33, 34]. Fluid and thickened synovium are the hallmarks of tenosynovitis by both modalities. With US, color flow doppler will demonstrate increased neovascularity.

Both MRI and US have been shown to be accurate in the assessment of peroneal tendon injury [35]. In the athlete, peroneal tenosynovitis is commonly associated with repetitive use, ankle injuries, and instability. Tears of the peroneus brevis are considered to occur more commonly than peroneus longus tears due to its anatomic position. However, both tendons can tear, especially in middle aged athletes. While chronic instability with associated disruption of the superior peroneal retinaculum are well demonstrated by either modality, US incorporating dynamic imaging is better able to diagnose intermittent subluxation or instability [31, 36, 37].

Posterior tibial tendon injury, classically associated with overuse in the athlete, may be well assessed by either modality, with comparable accuracy [38]. However, injury of the spring ligament is best assessed by MRI. The normal

superior medial spring ligament is similar in signal intensity and thickness to the adjacent PT tendon. As the tendon often stretches rather than discretely tears, increased T2 signal or thinning as well as a focal defect are signs of injury [39]. MRI can also assess the sinus tarsi, which in one study was abnormal in 72% of 25 patients with PT tears [40] Fig. 5.4.

Achilles tendon injuries are associated with numerous sports and are readily assessed by both US and MRI. Although acute Achilles tendon rupture is usually readily diagnosed clinically, upwards of 25% may be missed in the emergency department due to swelling and some residual plantar flexion [41]. Both US and MRI are considered accurate for the diagnosis of full thickness tears. While both modalities readily diagnose tendinosis and partial thickness tears in chronic Achilles tendinopathy, there is the suggestion in the literature that MRI may be slightly more accurate and in at least one study may be more prognostic of clinical outcome [33, 41–43]. Many favor MRI over US for injuries at the musculotendinous junction. Both modalities can image the peritenon, Kager's fat, and the retrocalcaneal bursa for inflammation Fig. 5.5.

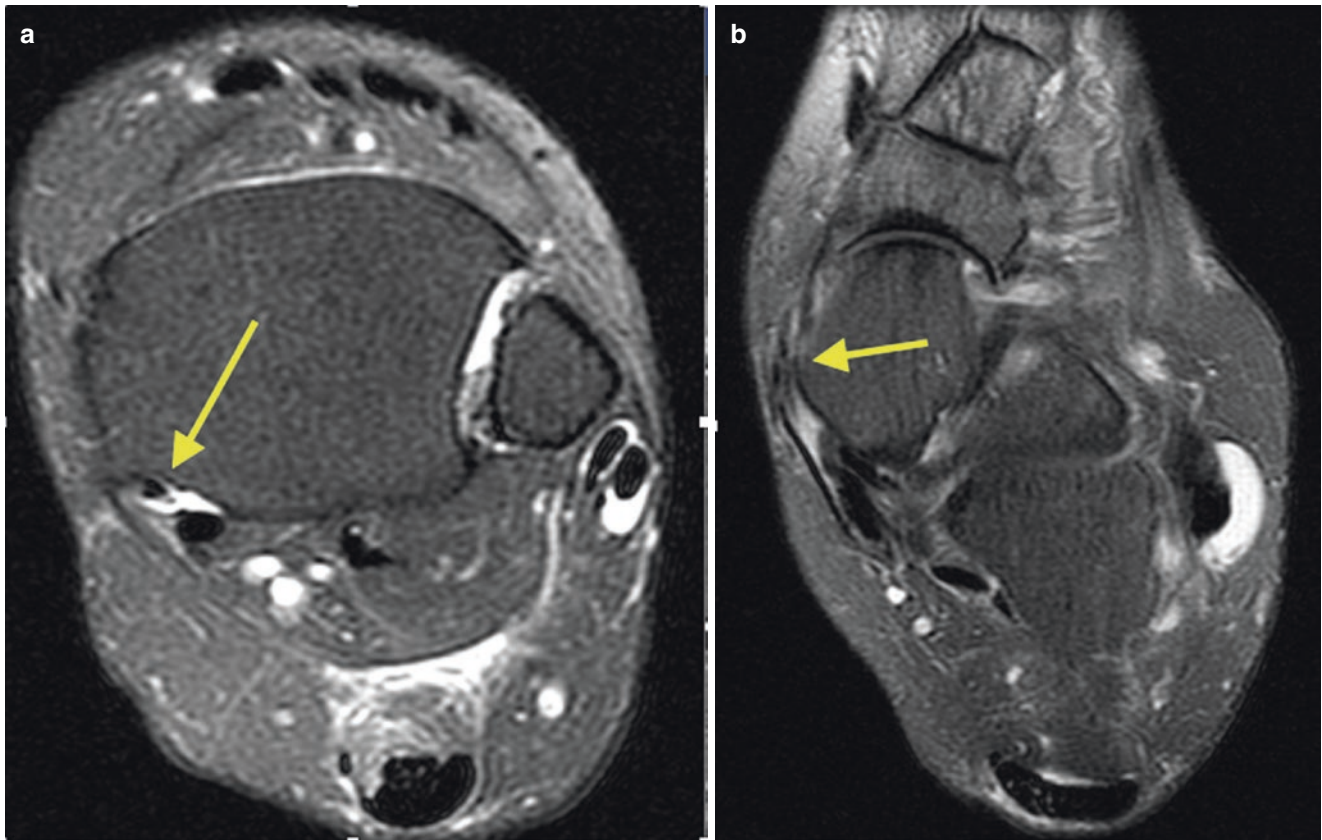
### Ankle Ligament Injuries

Ankle sprains and injuries rank among the most common sports related injuries. Imaging assessment begins with weight bearing views to assess for acute fractures, osteochondral lesions, and evidence of prior ligamentous injury. In the case of more chronic instability, stress views may be obtained. Sensitivity of up to 74% with 100% specificity have been reported [44].

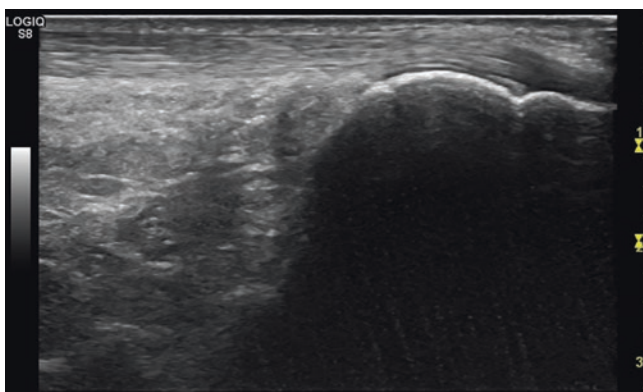
Accuracies of over 90% have been reported with both US & MRI in assessing the anterior talofibular and calcaneofibular ligaments [45]. For both modalities, lack of ligament identification and ligament discontinuity confirm a tear. Injured ligaments show variable ligament thickness and hypoechogenicity or altered T1 and T2 signal for US and MRI respectively. For tibiofibular ligament injury accuracies above 95% have been reported for MRI [46]. Both US and MRI have been used to assess anterolateral gutter impingement syndrome with some success [47].

### Lisfranc Injuries

Mid foot injuries are the second most common athletic foot injury, only second to first MTP joint injuries [48]. Classification and treatment are different from the traumatic injuries usually associated with motor vehicle accidents. Following clinical examination, imaging often plays a significant role in assessing the athlete with mid foot injury.



**Fig. 5.4** 56 year old female with polyarthralgias. (a) Fat suppressed T2 weighted image showing high grade partial tear of the posterior tibialis tendon (*arrow*). (b) Slightly more distal T2 weighted image showing tear of the spring ligament (*arrow*)



**Fig. 5.5** Sagittal US showing marked Achilles tendinopathy and thickening with focal areas of partial tearing (Curtsey of Blake Bogess, D. O)

Nearly 20% of Lisfranc injuries are missed or misdiagnosed on initial radiographs [48–50]. Nunley & Vertullo [48] report a 50% miss rate on non-weight bearing radiographs. Accordingly weight bearing AP radiographs with the opposite side for comparison and oblique and lateral views of the foot are the preferred radiographic series to be obtained [50]. Others suggest an AP abduction-pronation stress view for further assessment [50]. As it is painful and

may require an ankle block this is not routinely performed. The second metatarsal medial border should align with the medial cuneiform on the weight bearing AP while the 4th metatarsal border should align with the medial cuboid border on the oblique image. If there is 2 mm or more greater widening between the medial cuneiform and the first metatarsal when compared to the normal contralateral side, there is Lisfranc instability. Similarly, there is instability when there is tarsal-metatarsal subluxation greater than 2 mm compared to the normal side. The fleck sign, an avulsion of the second metatarsal base or medial cuneiform confirms Lisfranc injury [51].

If radiographs are equivocal, radionuclide imaging, CT, Spect CT, and MRI have all been suggested as useful for further imaging evaluation. Precisely which study or studies to employ next have not been rigorously evaluated against one another. It has been demonstrated that both CT and MRI are superior to radiographs for assessing Lisfranc injuries [52]. In the setting of significant trauma, it is our preference to obtain CT after plain films. CT has been shown to be more accurate than x-rays in this setting. In the setting of subtle or sports related injuries, our preference is to employ MRI. Lisfranc ligament tears or sprains are readily detected with sensitivities as high as 94% [53].

Stress fractures, degeneration, and other ligamentous injuries may be excluded as confounding causes of pain Fig. 5.6.

### Plantar Plate & Turf Toe Injuries

Injury to the plantar plate can be divided into injuries affecting the first metatarsal-phalangeal (MTP) joint and those affecting the lesser MTP joints. MR is the preferred imaging

modality for evaluating these ligamentous injuries. However, associated bony findings on radiographs can be seen in the setting of chronic injury.

The plantar plate is a fibrous band of tissue that inserts on the proximal phalanx and bridges the plantar aspect of the MTP joints, resisting dorsal translocation of the proximal phalanx. At the first toe, its distal aspect is continuous with the sesamoids, which provide leverage to the flexor hallucis brevis during plantar flexion of the toe. The medial and lateral sesamoids are embedded in the respective heads of the



**Fig. 5.6** Dorsiflexion injury of the left foot. (a) Weight bearing radiographs show a fleck sign consistent with Lisfranc ligament injury. (b) Axial CT showing fleck sign. (c) Axial CT demonstrating second meta-

tarsal base fracture not seen on radiographs (*arrow*). (d) Axial fat suppressed T2 weighted image showing disruption of the Lisfranc ligament (*arrow*)

flexor hallucis brevis tendon, which straddle the flexor hallucis longus as they converge on the plantar plate.

Turf toe, which technically involves an acute plantar plate injury, is most often used to describe any acute traumatic injury to the plantar plate complex. Injury to the plantar plate is the result of dorsiflexion at the first MTP, most often in the setting of axial loading, causing a spectrum from sprain to rupture of the plate that is often complicated by synovial injury to the adjacent MTP joint [54, 55]. Initial imaging begins with radiographs, which may demonstrate proximal migration of one or both of the sesamoids in more severe tears. Comparison to prior radiographs or to the contralateral side can be helpful as migration of greater than 3 mm is associated with large tears of the plantar plate [55, 56].

At the first MTP joint, the plantar plate commonly tears at its medial aspect and mild hallux valgus can sometimes be seen on radiographs. The joint is stabilized laterally by the phalangeal collateral ligaments, which are less commonly ruptured. In the case of more severe tears, disruption of the plantar plate may allow for dorsal subluxation of the proximal phalanx. In chronic injuries that are not repaired heterotopic bony formation in the plantar plate and osteophyte formation from repeated subluxation may be seen at the metacarpal heads [9].

Radiographs often appear normal in the acute setting, even if the injury is severe and MR should be performed when injury to the plantar plate is suspected. MR imaging of the plantar plate requires high resolution imaging with slice thickness no greater than 3 mm. with T1 weighted, proton density (PD) weighted, and fat suppressed fluid sensitive sequences (T2 weighted or short tau inversion recovery (STIR)) images. Sagittal images are most important. Long axis and short axis images provide supplemental information.

Normally, MR demonstrates the plantar plate as a low signal band, approximately 2.0 cm long, with a slight plantar concavity bridging the MTP joint. Fluid signal can be seen within the adjacent MTP joint space, but should not cross the plantar plate unless its integrity is compromised. In hyperextension injuries, the plantar plate is stretched and thins. Areas of thickening may also be seen at sites of injury. Indistinctness or intermediate T2 signal within the plantar plate and ligaments can be a sign of sprain or partial tear [9].

With complete tears, fluid signal will traverse the plantar plate Fig. 5.7. Several grading systems have been proposed to characterize plantar plate injuries. In one form or another, they range from low-grade minor sprains to complete tears with dislocation of the joint. However, the grading systems rely heavily on clinical exam findings as MR has not been shown to reliably differentiate rupture from high grade partial tears [9].

Injury to the plantar plate of the lesser MTP joints is a chronic injury caused by repetitive trauma. Lesser plantar

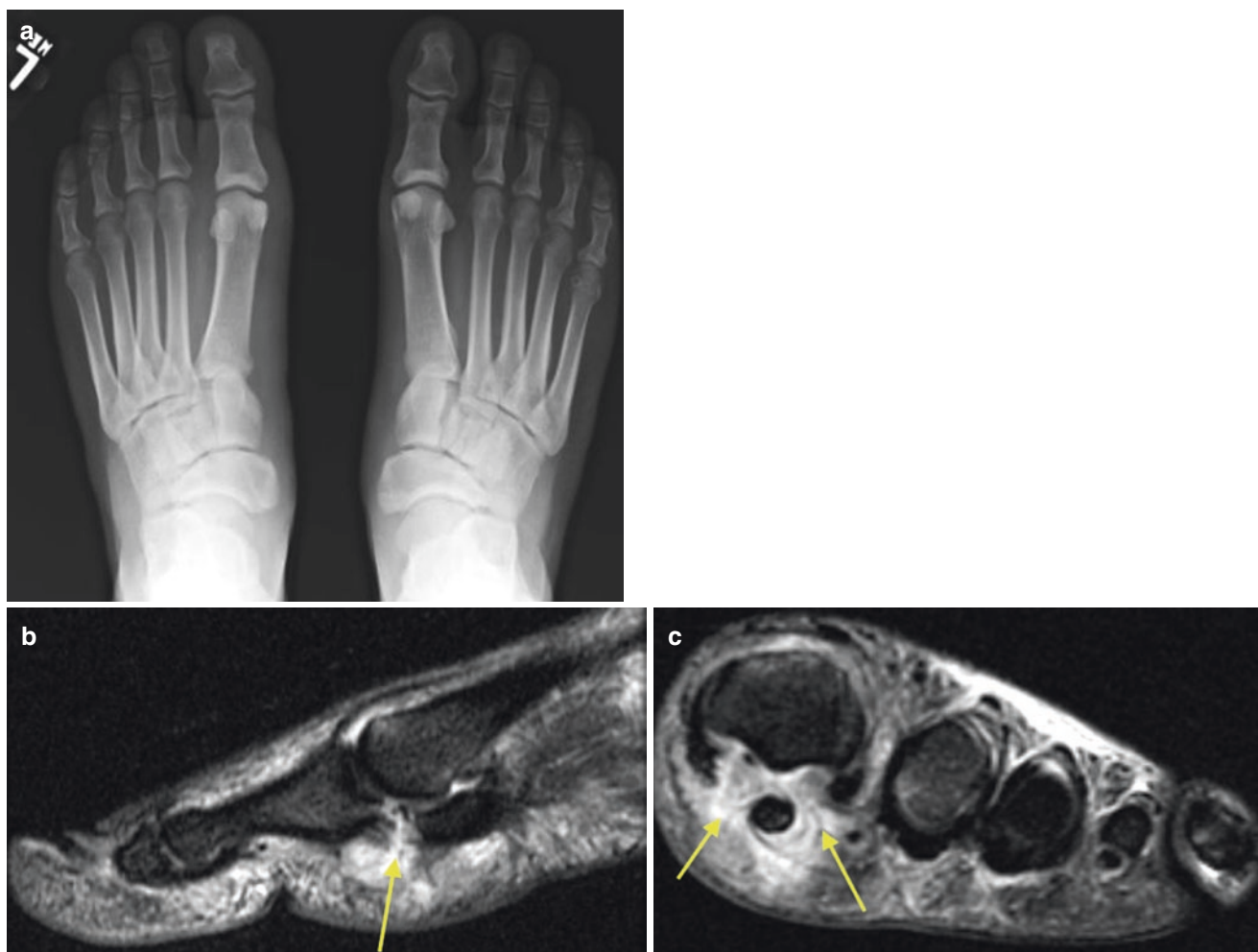
plate injuries most commonly occur at the second MTP joint, followed much less commonly by the third MTP plantar plate [57]. In contrast to first MTP injuries, tears at the lesser plantar plates occur most often at the lateral aspect of the proximal phalanx attachment. Radiographic findings of medial subluxation may be seen on radiographs. As the tear progresses, the second phalanx may sublux superiorly crossing over the first phalanx. Osteophytes can form at the metatarsal heads due to repeated subluxation. Hammer toe and claw toe deformities are also commonly seen on radiographs in later stages of injury [57].

Two studies suggest that US is more sensitive than MRI in detecting lesser plantar plate injuries, but is significantly less specific [58, 59]. The authors continue to utilize MRI following radiographs for lesser plantar plate assessment. As in first MTP plantar plate injuries, disruption of the plantar plate is best evaluated by fat suppressed T2 weighted or STIR MR images. Indistinctness or intermediate signal suggest sprain and rupture manifests with fluid signal traversing the low signal band of the plantar plate.

## Plantar Fascia

Plantar fasciitis is a common cause of heel pain. Injuries to the plantar fascia range from inflammation to rupture and are more common in athletes and active individuals. Plantar fasciitis is commonly associated with repetitive stress from extended activity or long distance running. Osteophytes or soft tissue calcifications may be seen near the calcaneal attachment on radiographs and radiographs may be helpful to evaluate for other etiologies such as infection or fracture. Both radionuclide imaging and US are used for evaluation of the plantar fascia, with one study reporting equal efficacy [60]. However, MR provides the greatest details in assessing partial tear and fascial rupture. Increased radiotracer uptake at the calcaneus can be detected suggesting the diagnosis [21]. Unfortunately, increased radiotracer activity is not specific to fasciitis. When US has been directly compared with MR, US has been shown to be less sensitive and specific [61].

The plantar fascia may be imaged in all three planes, but is best seen on sagittal and short axis images. When normal, it is seen on MR as a continuous low signal band coursing from the inferior aspect of the calcaneus and dividing into medial, middle, and lateral bands before spreading out into the soft tissues of the anterior plantar foot [62]. Increased T2 signal in or around the plantar fascia and thickening of the fascia are signs of injury/inflammation. Incomplete interruption of the normal low signal on T1 and T2 images indicates a partial tear, and traverses the fascia in complete tears.



**Fig. 5.7** 42 year old with acute great toe injury. (a) Negative radiographs. (b, c) Disruption of the plantar plate is clearly seen on these fat suppressed sagittal and coronal images (arrows)

### Conclusion

As our understanding and treatment of sports related injuries continues to evolve, imaging has improved and diversified as well. Depending upon the issue at hand, there are one or more imaging methods to supplement the history and clinical examination.

### References

1. Bearcroft PW, Guy S, Bradley M, Robinson F. MRI of the ankle: effect on diagnostic confidence and patient management. *AJR Am J Roentgenol.* 2006;187(5):1327–31. doi:10.2214/ajr.05.1071. Epub 2006/10/24.
2. Saha S, Burke C, Desai A, Vijayanathan S, Gnanasegaran G. SPECT-CT: applications in musculoskeletal radiology. *Br J Radiol.* 2013;86(1031):20120519. Epub 2013/10/08. doi:10.1259/bjr.20120519. PubMed PMID: 24096590; PubMed Central PMCID: PMC3830427.
3. Wall J, Feller JF. Imaging of stress fractures in runners. *Clin Sports Med.* 2006;25(4):781–802. doi:10.1016/j.csm.2006.06.003. Epub 2006/09/12.
4. Shindle MK, Endo Y, Warren RF, Lane JM, Helfet DL, Schwartz EN, et al. Stress fractures about the tibia, foot, and ankle. *J Am Acad Orthop Surg.* 2012;20(3):167–76. doi:10.5435/jaas-20-03-167. Epub 2012/03/03.
5. Banal F, Gandjbakhch F, Foltz V, Goldcher A, Etchepare F, Rozenberg S, et al. Sensitivity and specificity of ultrasonography in early diagnosis of metatarsal bone stress fractures: a pilot study of 37 patients. *J Rheumatol.* 2009;36(8):1715–9. doi:10.3899/jrheum.080657. Epub 2009/07/02.
6. Pepper M, Akuthota V, McCarty EC. The pathophysiology of stress fractures. *Clin Sports Med.* 2006;25(1):1–16. doi:10.1016/j.csm.2005.08.010. vii. Epub 2005/12/06.
7. Gaeta M, Minutoli F, Scribano E, Ascenti G, Vinci S, Bruschetta D, et al. CT and MR imaging findings in athletes with early tibial stress injuries: comparison with bone scintigraphy findings and emphasis on cortical abnormalities. *Radiology.* 2005;235(2):553–61. doi:10.1148/radiol.2352040406. Epub 2005/04/29.
8. Fredericson M, Bergman AG, Hoffman KL, Dillingham MS. Tibial stress reaction in runners. Correlation of clinical symptoms and



- scintigraphy with a new magnetic resonance imaging grading system. *Am J Sports Med.* 1995;23(4):472–81. Epub 1995/07/01.
9. Schein AJ, Skalski MR, Patel DB, White EA, Lundquist R, Gottsegen CJ, et al. Turf toe and sesamoiditis: what the radiologist needs to know. *Clin Imaging.* 2015;39(3):380–9. doi:10.1016/j.clinimag.2014.11.011. Epub 2014/12/09.
  10. Nwawka OK, Hayashi D, Diaz LE, Goud AR, Arndt WF, 3rd, Roemer FW, et al. Sesamoids and accessory ossicles of the foot: anatomical variability and related pathology. *Insights Imaging.* 2013;4(5):581–93. Epub 2013/09/06. doi:10.1007/s13244-013-0277-1. PubMed PMID: 24006205; PubMed Central PMCID: PMCPC3781258.
  11. Taylor JA, Sartoris DJ, Huang GS, Resnick DL. Painful conditions affecting the first metatarsal sesamoid bones. *Radiograph Rev Publ Radiol Soc N Am Inc.* 1993;13(4):817–30. doi:10.1148/radiographics.13.4.8356270. Epub 1993/07/01.
  12. Kappis M. Weitere Beiträge zur traumatisch-mechanischen Entstehung der „spontanen“ Knorpelablösungen (sogen. Osteochondritis dissecans). *Deutsche Zeitschrift für Chirurgie.* 1922;171(1):13–29. doi:10.1007/bf02812921.
  13. Talusan PG, Milewski MD, Toy JO, Wall EJ. Osteochondritis dissecans of the talus: diagnosis and treatment in athletes. *Clin Sports Med.* 2014;33(2):267–84. doi:10.1016/j.csm.2014.01.003. Epub 2014/04/05.
  14. Deol PPS, Cuttica DJ, Smith WB, Berlet GC. Osteochondral lesions of the talus: size, age, and predictors of outcomes. *Foot Ankle Clin.* 2013;18(1):13–34. <http://dx.doi.org/10.1016/j.fcl.2012.12.010>.
  15. Leumann A, Valderrabano V, Plaass C, Rasch H, Studler U, Hintermann B, et al. A novel imaging method for osteochondral lesions of the talus—comparison of SPECT-CT with MRI. *Am J Sports Med.* 2011;39(5):1095–101. doi:10.1177/0363546510392709. Epub 2011/02/09.
  16. McCollum GA, Calder JD, Longo UG, Loppini M, Romeo G, van Dijk CN, et al. Talus osteochondral bruises and defects: diagnosis and differentiation. *Foot Ankle Clin.* 2013;18(1):35–47. doi:10.1016/j.fcl.2012.12.002. Epub 2013/03/08.
  17. Pache G, Krauss B, Strohm P, Saueressig U, Blanke P, Bulla S, et al. Dual-energy CT virtual noncalcium technique: detecting post-traumatic bone marrow lesions—feasibility study. *Radiology.* 2010;256(2):617–24. doi:10.1148/radiol.10091230. Epub 2010/06/17.
  18. Kijowski R, De Smet AA. MRI findings of osteochondritis dissecans of the capitellum with surgical correlation. *AJR Am J Roentgenol.* 2005;185(6):1453–9. doi:10.2214/ajr.04.1570. Epub 2005/11/24.
  19. Hepple S, Winson IG, Glew D. Osteochondral lesions of the talus: a revised classification. *Foot Ankle Int.* 1999;20(12):789–93. Epub 1999/12/28.
  20. Morshed S, Corrales L, Genant H, Miclau T, 3rd. Outcome assessment in clinical trials of fracture-healing. *J Bone Joint Surg Am Vol.* 2008;90(Suppl 1):62–7. Epub 2008/03/20. doi:10.2106/jbjs.g.01556.
  21. Bamberg F, Dierks A, Nikolaou K, Reiser MF, Becker CR, Johnson TR. Metal artifact reduction by dual energy computed tomography using monoenergetic extrapolation. *Eur Radiol.* 2011;21(7):1424–9. doi:10.1007/s00330-011-2062-1. Epub 2011/01/21.
  22. Guggenberger R, Winklhofer S, Osterhoff G, Wanner GA, Fortunati M, Andreisek G, et al. Metallic artefact reduction with monoenergetic dual-energy CT: systematic ex vivo evaluation of posterior spinal fusion implants from various vendors and different spine levels. *Eur Radiol.* 2012;22(11):2357–64. doi:10.1007/s00330-012-2501-7. Epub 2012/05/31.
  23. Komlosi P, Grady D, Smith JS, Shaffrey CI, Goode AR, Judy PG, et al. Evaluation of monoenergetic imaging to reduce metallic instrumentation artifacts in computed tomography of the cervical spine. *J Neurosurg Spine.* 2015;22(1):34–8. doi:10.3171/2014.10.spine14463. Epub 2014/11/08.
  24. Baron K, Neumayer B, Widek T, Schick F, Scheicher S, Hassler E, et al. Quantitative MR imaging in fracture dating—Initial results. *Forensic Sci Int.* 2016;261:61–9. doi:10.1016/j.forsciint.2016.01.020. Epub 2016/02/19.
  25. Lewis M, Ebreo D, Malcolm PN, Greenwood R, Patel AD, Kasmali B, et al. Pharmacokinetic modeling of multislice dynamic contrast-enhanced MRI in normal-healing radial fractures: a pilot study. *J Magnet Reson Imaging JMRI.* 2016;43(3):611–9. doi:10.1002/jmri.25039. Epub 2015/09/04.
  26. Fernandez-Canton, et al. SKEL RAD 03: 32:273–278
  27. Starr AM, Wessely MA, Albastaki U, Pierre-Jerome C, Kettner NW. Bone marrow edema: pathophysiology, differential diagnosis, and imaging. *Acta Radiologica (Stockholm, Sweden: 1987).* 2008;49(7):771–86. Epub 2008/07/09. doi:10.1080/02841850802161023.
  28. Thiryayi WA, Thiryayi SA, Freemont AJ. Histopathological perspective on bone marrow oedema, reactive bone change and haemorrhage. *Eur J Radiol.* 2008;67(1):62–7. doi:10.1016/j.ejrad.2008.01.056. Epub 2008/03/14.
  29. Patel S. Primary bone marrow oedema syndromes. *Rheumatology.* 2014;53(5):785–92. doi:10.1093/rheumatology/ket324.
  30. Orr JD, Sabesan V, Major N, Nunley J. Painful bone marrow edema syndrome of the foot and ankle. *Foot Ankle Int.* 2010;31(11):949–53. doi:10.3113/fai.2010.0949. Epub 2010/12/30.
  31. Bianchi S, Martinoli C, Gaignot C, De Gautard R, Meyer JM. Ultrasound of the ankle: anatomy of the tendons, bursae, and ligaments. *Semin Musculoskelet Radiol.* 2005;9(3):243–59. doi:10.1055/s-2005-921943. Epub 2005/10/26.
  32. Waitches GM, Rockett M, Brage M, Sudakoff G. Ultrasonographic-surgical correlation of ankle tendon tears. *J Ultrasound Med Off J Am Instit Ultrasound Med.* 1998;17(4):249–56. Epub 1998/04/17.
  33. Astrom M, Gentz CF, Nilsson P, Rausing A, Sjöberg S, Westlin N. Imaging in chronic achilles tendinopathy: a comparison of ultrasonography, magnetic resonance imaging and surgical findings in 27 histologically verified cases. *Skeletal Radiol.* 1996;25(7):615–20. Epub 1996/10/01.
  34. Paavola M, Paakkala T, Kannus P, Jarvinen M. Ultrasonography in the differential diagnosis of Achilles tendon injuries and related disorders. A comparison between pre-operative ultrasonography and surgical findings. *Acta Radiol (Stockholm, Sweden: 1987).* 1998;39(6):612–9. Epub 1998/11/17.
  35. Grant TH, Kelikian AS, Jereb SE, McCarthy RJ. Ultrasound diagnosis of peroneal tendon tears. A surgical correlation. *J Bone Joint Surg Am.* 2005;87(8):1788–94. doi:10.2106/jbjs.d.02450. Epub 2005/08/09.
  36. Neustadter J, Raikin SM, Nazarian LN. Dynamic sonographic evaluation of peroneal tendon subluxation. *AJR Am J Roentgenol.* 2004;183(4):985–8. doi:10.2214/ajr.183.4.1830985. Epub 2004/09/24.
  37. Hyer CF, Dawson JM, Philbin TM, Berlet GC, Lee TH. The peroneal tubercle: description, classification, and relevance to peroneus longus tendon pathology. *Foot Ankle Int.* 2005;26(11):947–50. Epub 2005/11/29.
  38. Khoury V, Guillin R, Dhanju J, Cardinal E. Ultrasound of ankle and foot: overuse and sports injuries. *Semin Musculoskelet Radiol.* 2007;11(2):149–61. doi:10.1055/s-2007-1001880. Epub 2007/12/21.
  39. Toye LR, Helms CA, Hoffman BD, Easley M, Nunley JA. MRI of spring ligament tears. *AJR Am J Roentgenol.* 2005;184(5):1475–80. doi:10.2214/ajr.184.5.01841475. Epub 2005/04/28.
  40. Balen PF, Helms CA. Association of posterior tibial tendon injury with spring ligament injury, sinus tarsi abnormality, and plantar fasciitis on MR imaging. *AJR Am J Roentgenol.* 2001;176(5):1137–43. doi:10.2214/ajr.176.5.1761137. Epub 2001/04/20.
  41. Weatherall JM, Mroczek K, Tejwani N. Acute achilles tendon ruptures. *Orthopedics.* 2010;33(10):758–64. doi:10.3928/01477447-20100826-21. Epub 2010/10/20.

42. Bleakney RR, White LM. Imaging of the Achilles tendon. *Foot Ankle Clin.* 2005;10(2):239–54. doi:[10.1016/j.fcl.2005.01.006](https://doi.org/10.1016/j.fcl.2005.01.006). Epub 2005/06/01.
43. Khan KM, Forster BB, Robinson J, Cheong Y, Louis L, Maclean L, et al. Are ultrasound and magnetic resonance imaging of value in assessment of Achilles tendon disorders? A two year prospective study. *Br J Sports Med.* 2003;37(2):149–53. Epub 2003/03/29. PubMed PMID: 12663358; PubMed Central PMCID: PMCPMC1724608.
44. Tourne Y, Besse JL, Mabit C. Chronic ankle instability. Which tests to assess the lesions? Which therapeutic options? *Orthop Traumatol Surg Res OTSR.* 2010;96(4):433–46. doi:[10.1016/j.otsr.2010.04.005](https://doi.org/10.1016/j.otsr.2010.04.005). Epub 2010/05/25.
45. Nazarenko A, Beltran LS, Bencardino JT. Imaging evaluation of traumatic ligamentous injuries of the ankle and foot. *Radiol Clin North Am.* 2013;51(3):455–78. doi:[10.1016/j.rcl.2012.11.004](https://doi.org/10.1016/j.rcl.2012.11.004). Epub 2013/04/30.
46. Oae K, Takao M, Naito K, Uchio Y, Kono T, Ishida J, et al. Injury of the tibiofibular syndesmosis: value of MR imaging for diagnosis. *Radiology.* 2003;227(1):155–61. doi:[10.1148/radiol.2271011865](https://doi.org/10.1148/radiol.2271011865). Epub 2003/03/05.
47. Dimmick S, Linklater J. Ankle impingement syndromes. *Radiol Clin North Am.* 2013;51(3):479–510. doi:[10.1016/j.rcl.2012.11.005](https://doi.org/10.1016/j.rcl.2012.11.005). Epub 2013/04/30.
48. Nunley JA, Vertullo CJ. Classification, investigation, and management of midfoot sprains: Lisfranc injuries in the athlete. *Am J Sports Med.* 2002;30(6):871–8. Epub 2002/11/19.
49. DeOrio M, Erickson M, Usuelli FG, Easley M. Lisfranc injuries in sport. *Foot Ankle Clin.* 2009;14(2):169–86. doi:[10.1016/j.fcl.2009.03.008](https://doi.org/10.1016/j.fcl.2009.03.008). Epub 2009/06/09.
50. Watson TS, Shurnas PS, Denker J. Treatment of Lisfranc joint injury: current concepts. *J Am Acad Orthop Surg.* 2010;18(12):718–28. Epub 2010/12/02.
51. Myerson MS, Fisher RT, Burgess AR, Kenzora JE. Fracture dislocations of the tarsometatarsal joints: end results correlated with pathology and treatment. *Foot Ankle.* 1986;6(5):225–42. Epub 1986/04/01.
52. Peicha G, Preidler KW, Lajtai G, Seibert FJ, Grechenig W, Unfallchirurg, Springer-Verlag. 2001;104:1134–9.
53. Raikin SM, Elias I, Dheer S, Besser MP, Morrison WB, Zoga AC. Prediction of midfoot instability in the subtle Lisfranc injury. Comparison of magnetic resonance imaging with intraoperative findings. *J Bone Joint Surg Am.* 2009;91(4):892–9. doi:[10.2106/jbjs.h.01075](https://doi.org/10.2106/jbjs.h.01075). Epub 2009/04/03.
54. Haaga JR, Lanzieri CF, Gilkeson RC. CT and MR imaging of the whole body. St. Louis: Mosby; 2003.
55. McCormick JJ, Anderson RB. Turf toe: anatomy, diagnosis, and treatment. *Sports Health.* 2010;2(6):487–94. Epub 2010/11/01. doi:[10.1177/1941738110386681](https://doi.org/10.1177/1941738110386681). PubMed PMID: 23015979; PubMed Central PMCID: PMCPMC3438874.
56. Waldrop 3rd NE, Zirker CA, Wijdicks CA, Laprade RF, Clanton TO. Radiographic evaluation of plantar plate injury: an in vitro biomechanical study. *Foot Ankle Int.* 2013;34(3):403–8. doi:[10.1177/1071100712464953](https://doi.org/10.1177/1071100712464953). Epub 2013/03/23.
57. Umans HR, Elsinger E. The plantar plate of the lesser metatarsophalangeal joints: potential for injury and role of MR imaging. *Magn Reson Imaging Clin N Am.* 2001;9(3):659–69. xii. Epub 2001/11/06.
58. Gregg J, Silberstein M, Schneider T, Marks P. Sonographic and MRI evaluation of the plantar plate: a prospective study. *Eur Radiol.* 2006;16(12):2661–9. doi:[10.1007/s00330-006-0345-8](https://doi.org/10.1007/s00330-006-0345-8). Epub 2006/07/05.
59. Klein EE, Weil Jr L, Weil Sr LS, Knight J. Magnetic resonance imaging versus musculoskeletal ultrasound for identification and localization of plantar plate tears. *Foot Ankle Spec.* 2012;5(6):359–65. doi:[10.1177/1938640012463061](https://doi.org/10.1177/1938640012463061). Epub 2012/10/18.
60. Kane D, Greaney T, Shanahan M, Duffy G, Bresnihan B, Gibney R, et al. The role of ultrasonography in the diagnosis and management of idiopathic plantar fasciitis. *Rheumatology (Oxford, England).* 2001;40(9):1002–8. Epub 2001/09/19.
61. Sabir N, Demirlenk S, Yagci B, Karabulut N, Cubukcu S. Clinical utility of sonography in diagnosing plantar fasciitis. *J Ultrasound Med Off J Am Instit Ultrasound Med.* 2005;24(8):1041–8. Epub 2005/07/26.
62. Theodorou DJ, Theodorou SJ, Kakitsubata Y, Lektrakul N, Gold GE, Roger B, et al. Plantar fasciitis and fascial rupture: MR imaging findings in 26 patients supplemented with anatomic data in cadavers. *Radiograph Rev Publ Radiol Soc N Am, Inc.* 2000;20 Spec No:S181–97. Epub 2000/10/25. doi:[10.1148/radiographics.20.suppl\\_1.g00oc01s181](https://doi.org/10.1148/radiographics.20.suppl_1.g00oc01s181).

Thomas Jöllenbeck and Juliane Pietschmann

**Abstract**

Modern biomechanical motion analyses visualize sequence of (athletic) movements in their dynamics and complexity. As biomechanical functional imaging they expand the common spectrum of static orthopaedic diagnosis by the dynamical component. Detection of reasons and mechanisms of limitations of movement and injuries demands a problem-oriented analytic approach with regard to basic classes of movements as well as to sports-specific profiles of strains and sports-specific demand profiles. The instrumental gait and run analysis as standard method includes three different supplementary analytical ways of access where kinematics describes the temporal progress of a movement in space, kinetics deals with the effect of strength and moments and electromyography describes time, duration and intensity of muscular activity. Resulting intervention measures primarily should be oriented to individual normality of movement and asymmetries rather than in accordance with a normative exercise.

**Keywords**

Biomechanics • Motion analysis • Gait analysis • Kinematics • Kinetics • Dynamometry • Pedography • Electromyography

**Introduction**

Athletes usually have individual problems in different phases of their sequence of movements regardless of type of sport or performance category. Modern biomechanical analysis of movements in (sports-) medicine, (sports-) orthopedics and (sports-) traumatology are at present not widely spread but they have range of service and the capability to visualize sequence of movements in their dynamics and complexity this means in temporal and spatial process. Analyses of movement enhance static diagnostics, i.e. in state of rest which is usual for diagnostics in orthopedics and traumatology (clinical screening, taking X-ray, CT, NMRI), by

dynamic component. Analyses of movements can help to detect reasons and mechanisms of limitations of movements, help sports injury recovery and the optimizing of movements prevention and rehabilitation or to estimate the biomechanical performance or performance of sports before revival acquisition to increase a sporty activity [1].

Standard methods of analysis of movements are biomechanical gait, running and treadmill analysis. Walking and running are ordinary form of locomotion of human. In a complex interaction of musculoskeletal system according to the principles of economy and efficiency the necessary forces are produced in lower extremities for propulsion and in upper extremities for retention of balance. The force transmission takes place in foot as the distal phalanx of kinematic string both inactive in the moment of heel strike and active at the moment of the footprint. Walking and running are the foundations of skills or other derived or modified shapes of movements are the foundation of movement and performance for a wide range of sports.

---

T. Jöllenbeck (✉) • J. Pietschmann  
Klinik Lindenplatz GmbH, Institut für Biomechanik,  
Weslarner Str. 29 D, Bad Sassendorf 59505, Germany  
e-mail: [Thomas.Joellenbeck@klinik-lindenplatz.de](mailto:Thomas.Joellenbeck@klinik-lindenplatz.de); [Juliane.Pietschmann@klinik-lindenplatz.de](mailto:Juliane.Pietschmann@klinik-lindenplatz.de)

## Problem-Oriented Analysis of Movement

Analyses of movement indicate a problem-oriented reflection and they have to analyze the individual biomechanical influencing factors of the active and passive musculoskeletal system. Qualifications are knowledge and consideration of essential biomechanical classes of movement, sports specific profiles of strains, sports specific profiles of demand, dependent sports and sub-discipline resp. playing position in different participation and severity.

Basic classes of movements can roughly be defined as

- Movements in natural, phylogenetic physiological scope, movements in direction of motion (running, cross-country skiing classic) and movements especially of arms inside field of view (boxing, weightlifting) and
- Movements with elements outside of that means movements lateral of direction of motion (cross-country skiing diagonal, gymnastics) and movements outside of field of view (swimming, tennis) in terminal joint angle positions and physiological border area

Even by slight external forces the latter may lead to a considerably higher strain of all participating structures to inconvenient protection and conduction of the device of muscle, band and tendons at the same time. These include a higher risk of injuries and impingement syndrome.

Sports-specific profiles of strains:

- Static strains (sailing, shooting)
- Cyclic repetitive strains (running, swimming)
- Maximum strains (weightlifting)
- Strains in moments of jump or drop, explosive (athletics jumps and throw disciplines, ball sports)
- Strains in moments of impact (gymnastics, ball sports)
- Contraction on impact (tennis, football/soccer)
- Movements of rotation (ball sports, ballet)

and also the analysis has to consider outside parameters (interaction, subsoil, shoes, sports equipment).

Sports-specific demand profiles can roughly be divided into:

- maximum terminal velocity (jump, throw, hit/strike)
- minimal duration (punch, hit of fencing)
- maximum effectiveness of minimal start-finish-time (running disciplines, bicycle racing, swimming)
- optimal energy input and energy conversion (gymnastics)
- optimal posture during and at the end of a flight phase (somersaults, backflips, twists, high jump, long jump)

The results of a problem-oriented analysis of movement can provide very detailed information about the kinetic behaviour of an athlete. To use this purposefully and

problem-oriented a great amount of responsibility on the athlete's side is necessary especially to prevent overstrain and inappropriate physical strain, especially during rehabilitation after an injury as well as for the prevention of further injuries. Concerning up-and-coming young athletes a diagnosis of strain and stress that is given when a problem of movement turns up early should be replenished by the honest prediction of the youth's skill of sport, of his/her ability of sport at the moment and eventually after the transition from juvenile sport to high-performance sport.

To sum up, the knowledge concerning movement and the disclosure and treatment of the main deficits of movement are the prerequisite and key components of a successful and sustainable diagnosis and therapy. The optimal diagnosis and therapy integrates medical, biomechanical, movement-analytical as well as therapeutic aspects.

---

## Gait-, Run- and Treadmill Analysis

As standardized method of modern movement analysis gait and run analysis as well as treadmill analysis conduce to the systematic recording of the human gait and running and the parameters. The accompanying procedure and standard values and pathologies of the gait to assess the results are essentially based on the papers of Perry [2], Whittle [3], and Winter [4]. A uniform standard of analysis has not been reached until today. There is a distinction between observational gait analysis and instrumental gait analysis [2].

The simplest and established form as part of orthopedic and therapeutic appraisal and diagnosis is the observational gait- and treadmill analysis. The assessment of the course of movement by sheer observation does not need any instruments and the assessment is immediately available. For this one can use systematic observation forms and schemes of analysis [2, 5]. However, the value of such an analysis is subjective and limited. The subjective assessment of the results often leads to a low accordance of different observers [6].

The instrumental gait analysis essentially includes three different supplementary analytical ways of access [4]. Kinematics describes the temporal progress of a movement in space including associated variables such as distance, time, angle or speed. Kinetics deals with the causes of motion i.e. the effect of strength and moments in magnitude and direction. Electromyography (EMG) describes time, duration and intensity of muscular activity. As an objective method, the instrumental analysis of movements provides reliable and valid information.

As a special form of gait and running analysis the treadmill analysis expands the spectrum of analysis of walking and running on the treadmill by variable speed and inclination of the treadmill. The advantages of a treadmill consist of

the standardization of guidelines, modest local and time requirements including a multitude of analyzable step cycles in controlled environmental conditions and an individually selected constant velocity. For acclimatization a period of 3–5 min is necessary. However, irregularities while walking or running must be compensated at each step on the treadmill by the test person. Previously, the integration of a treadmill in clinical settings was seen critically especially with regard to the kinematic [7] and kinetic parameters [8] as being not directly comparable to gait analysis in the plane [6]. Despite the ongoing discussion of the conception recent studies are of the opinion that the use of a treadmill is acceptable due to the insignificantly small differences in diagnosis and training [9, 10] Fig. 6.1.

Regardless of the selected method it is always advisable to use one, better multiple synchronized video cameras if possible in the dorsal and/or lateral arrangement in order to

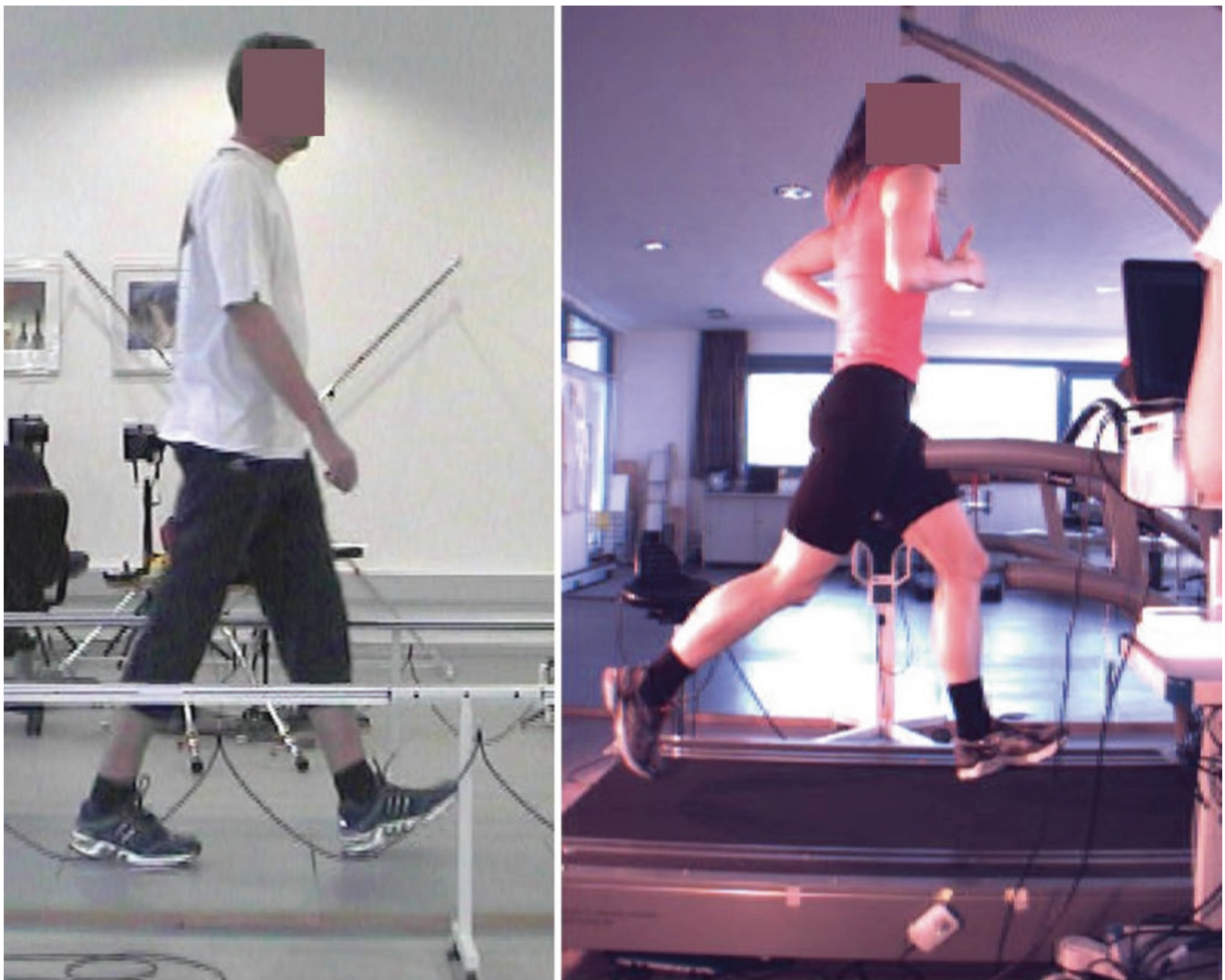
put the partially abstract results of way or force-time-curves in relation to the motion image.

The identification of the sequence of partial movements and particularly the question at which part of the body an unusual abnormality begins and the reason why needs special attention. The kinematic chain in its construction and coupling as well as the number of degrees of freedom admits ascending as well as descending directions of effects.

### Kinematic Motion Analysis

The basic method of kinematic analysis is image-based recording of a motion by video camera and the corresponding computer assisted analysis [11].

The question will determine the number, location and size of the cut of the video cameras. During fast movements it can



**Fig. 6.1** “Gait analysis – treadmill analysis”. *Left*: gait analysis on a gangway with 2 force measuring plates (Kistler). *Right*: treadmill analysis with force sensors underneath the treading surface (Zebris FDM-T)

be helpful for further details of movement to use digital video cameras that allow for higher frame rates than 50 Hz (100 Hz or more). For the calculation of kinematic parameters the test persons should previously be provided with markers. The 2D motion analysis can be realized quickly and with relatively little effort.

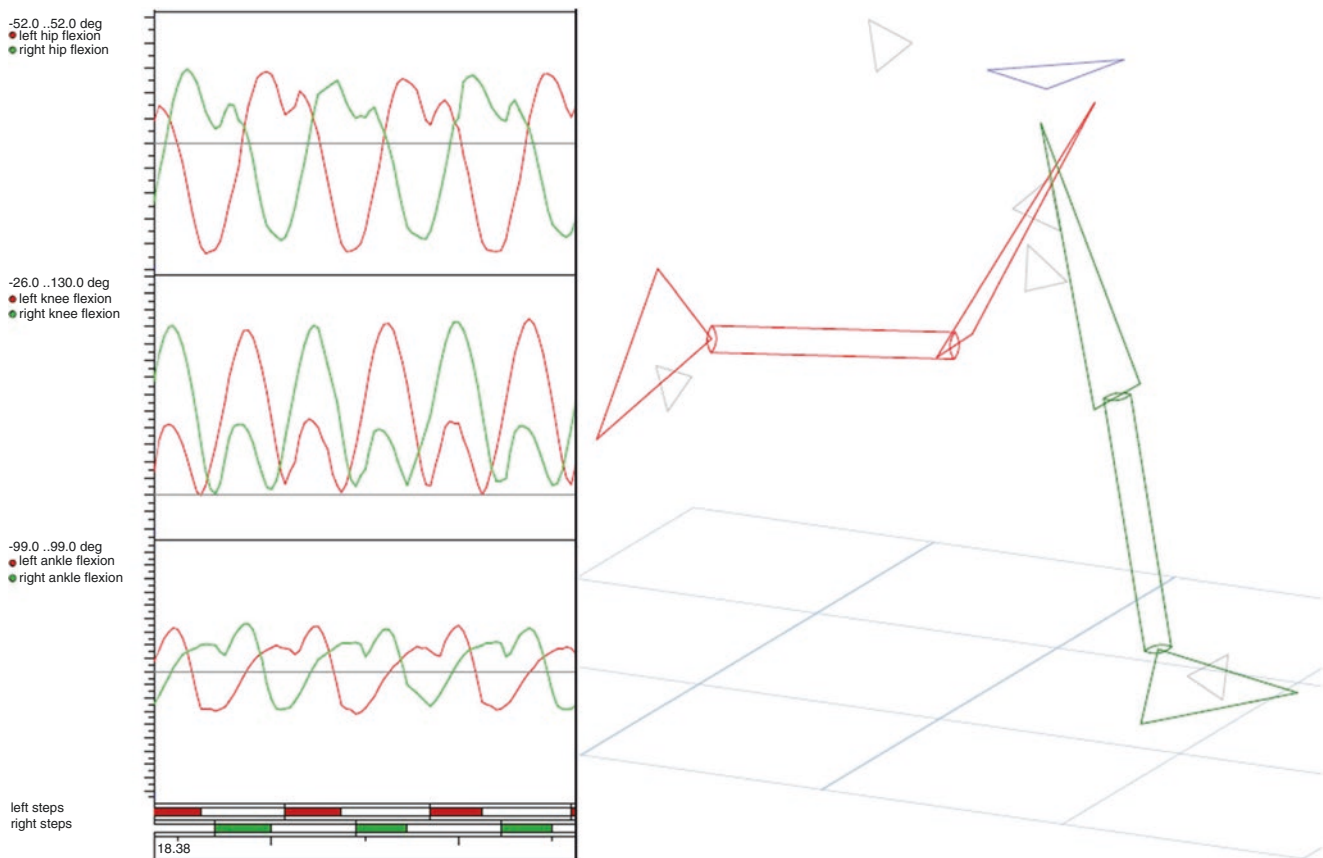
The complex way of a kinematic analysis is the 3D motion analysis. Modern infra-red cameras, ultrasonic sensors or inertial sensors can record the movements of body parts in space and time (Fig. 6.2). The use of special markers or sensors is a requirement as well as a careful calibration and often the use of appropriate body models targeted at the question. The presentation is mostly abstract in the form of distance- or angle-time-curves mostly in sagittal, frontal and transversal perspective, additionally to any inclined planes. Overall 3D motion analyses are still time-consuming, costly and require specially trained personnel.

For motion analysis, the thing to do is the consideration in the main planes of motion, i.e. in the frontal, sagittal and transverse planes. Regardless of the perspective, special consideration is required for differences in the side comparison that indicate possible unilateral deficits or improper strains.

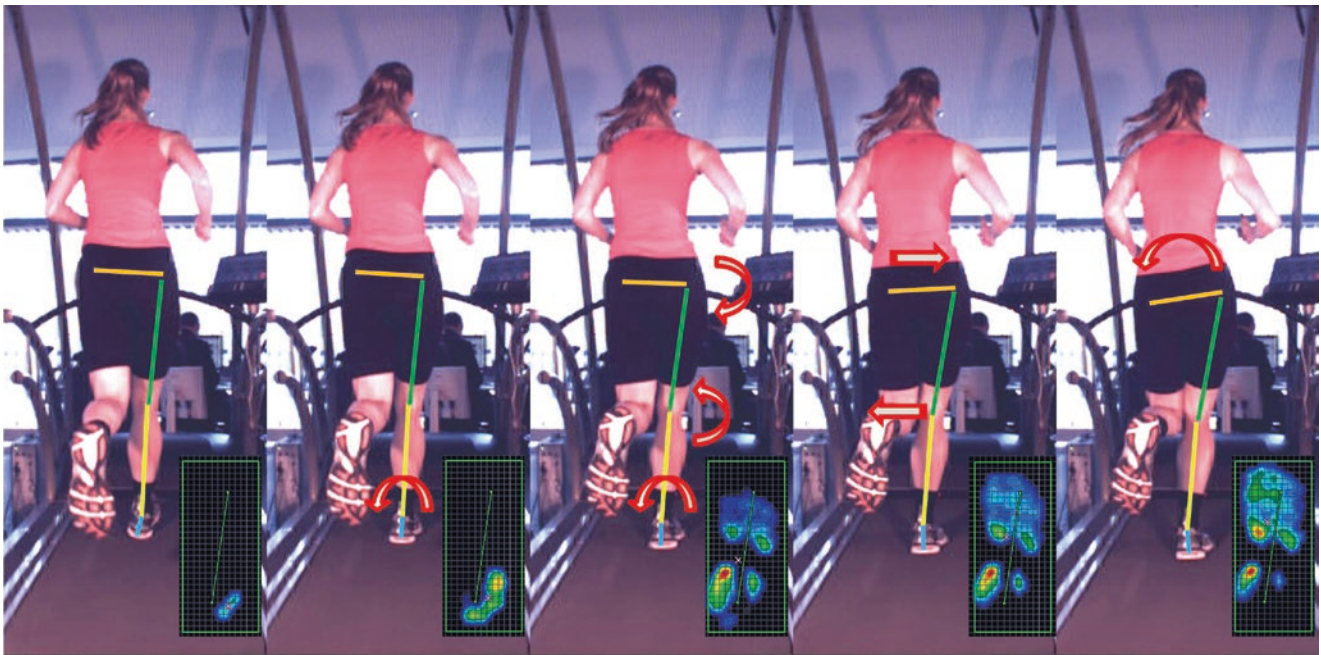
## Important Analysis Parameters

In frontal perspective the foot strike and footprint behavior is particularly of interest. The exact consideration of the initial ground contact and lowering movement of the foot is useful to detect an abnormal movement of pronation or supination (Fig. 6.3). Of particular importance is the direct comparison with the rolling motion barefoot, which will reveal whether the external structures of the shoe, the foot position or pathologies of the foot are initially responsible for any wrong movement. The effectiveness of a shoe supply can be assessed as well. Concerning the footprint the main concern is with a conspicuous inward or outward rotation of the foot as possible causes of problems in the ball and big toe area. The diagnosis should be based on normative specifications, but also take into account essential features such as the rate of pronation and the motion sequence of the entire chain of movements especially of the knee, hip and pelvis in terms of cause and effect mechanisms (Fig. 6.3).

While the test person is running, in sagittal perspective the typing of back- and metatarsal- or forefoot runner can be decided by the kind of rolling behavior. The velocity of plantar flexion after foot strike, in particular in the side compari-



**Fig. 6.2** “3D motion analysis”. 3D ultrasonic motion analysis (Zebis CMS System) on a treadmill at 12 km/h. *Left side:* Angle-time-curves of hip (above), knee (center) and ankle (bottom), *Right side:* 3D motion picture, left-hand side (red) and right-hand side (green)



**Fig. 6.3** “Treadmill analysis foot strike”. Treadmill analysis with force sensors (Zebris FDM-T) and high-speed video (Contemplas, 100 Hz) at 14 km/h. Picture sequence in 20 ms steps from initial ground contact to

mid stance. Foot strike and footprint (little pictures) behavior showing pronation followed by inwards movement of knee and hip instability

son, may give indications for muscular or neural deficits such as e.g. a drop foot syndrome. The degree of plantar flexion of the great toe in the footprint may indicate the use and the proportion of the big toe during the push off motion.

In transversal perspective, which is usually available only in the context of a 3D motion analysis, in particular different ranges of foot strike and footprint as well as the positioning of the pelvis and pelvic rotation deserve special attention. Located malpositions can be identified. Especially rotational movements, which cannot be seen in the sagittal or frontal plane, are visible in the transverse plane and give important information as to reasons of movement disorders and malpositions. The movement of the foot and foot position with an almost completely stretched knee joint before the foot strike is most important evidence of increased inward or outward rotation of the hip joint.

## Kinetic Motion Analysis

### Dynamometry

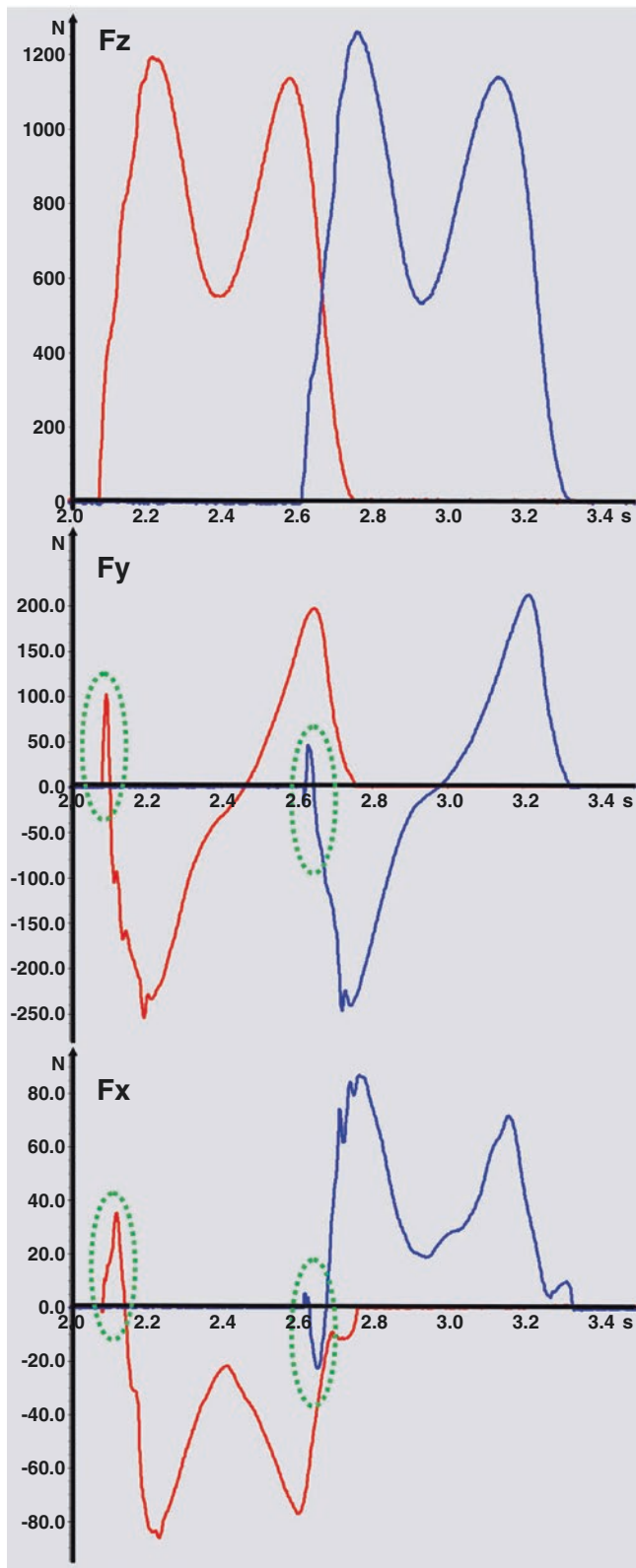
The standard method of kinetic gait and running analysis is the recording of the force-time-profiles of a movement by multi-component force plates [11]. These are usually localized in a gangway. Standard parameters are the ground-reaction-forces in all three dimensions and the force application line and the duration of the support phase. If at least two force plates are arranged one behind the other, asymmetries and isolate reasons can easily be revealed and

additionally also kinematic parameters such as stride length, duration of support and swing phase or the walking speed in the side comparison can be determined. The course of the force application lines can show the position of the foot and indicate shortcomings in the rolling motion. However, an assignment to the structure of the foot is not possible. A few treadmills with integrated one- or three-dimensional force plates already exist.

The force-time-curves (Fig. 6.4) while walking vertically show the size of the load as the response at foot strike, the pressure force at push off and the dynamics of movement represented as the differences between the typical two maxima and the minimum. In the running direction the breaking force impact during the loading response and acceleration impulse during the push-off phase in height and course can be delineated from each other. The transverse to the running direction of forces indicates in degree and course, how far the center of gravity of the perpendicular is removed from the bottom point of contact during a step. From the force-time-curves the respective momentum, figuratively the area below the curve, can easily be calculated as a key measure of the force transmission and thus the motion dynamics.

### Pedography

If there are problems in the area of the foot, the pedography has established itself as an imaging method of kinetic analysis especially in the field of orthopedic shoes and insoles. Here, the pressure-time-curves under the foot- or shoe-soles



**Fig. 6.4** “Force-time-curves”. Gait analysis: force-time-curves, ground reaction force:  $F_z$  vertical (above),  $F_y$  horizontal in movement direction (center) and  $F_x$  horizontal in lateral direction (bottom), left-hand side (red) and right-hand side (blue)

are recorded through pressure measuring plates or pressure distribution measuring systems of the foot primarily for detecting and analyzing the load characteristics [11]. Pressure measuring plates are fixedly inserted into a gangway or integrated in treadmills underneath the treading surface. Pressure distribution measuring system of foot however is used like insoles and is mobile. They examine the interaction between foot, shoe and ground.

Measurements are possible statically while standing and dynamically when walking or running. The foot can be analyzed as a whole or divided into stress-bearing areas. The parameters of measurements are the mean and maximum pressure and in addition load duration, strength and momentum course, center of pressure and rolling are available (Fig. 6.5). At treadmill also kinematic parameters such as stride length and track width, foot position and length of the gait line as well as information on gait symmetry and gait stability are available.

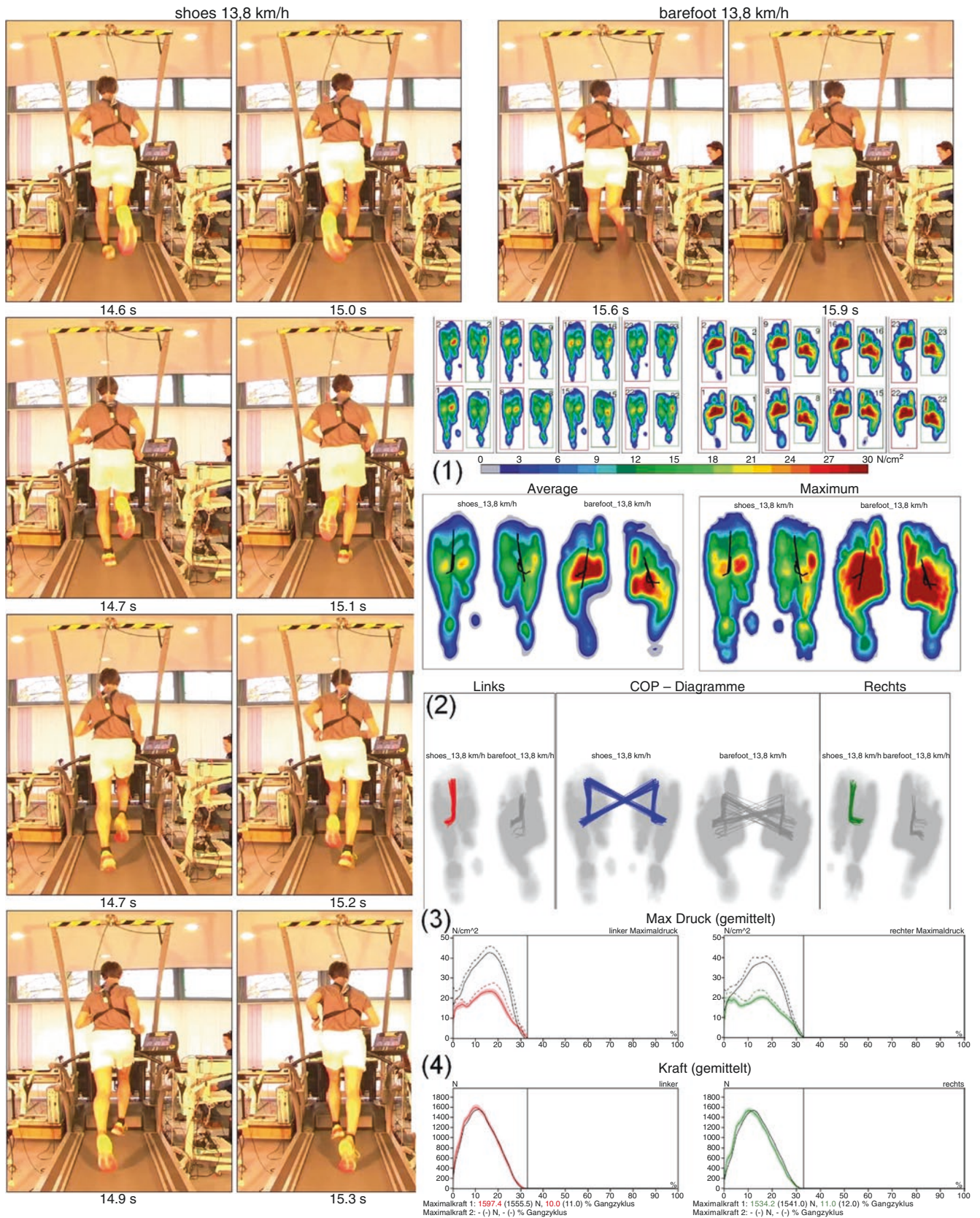
Currently there are no generally accepted guidelines for the evaluation of pedographical measurements. The side comparison in the stand can detect the relocation or dislocation of the center of gravity. Barefoot measurements are the basis for the description and limitation of problem areas and very helpful concerning pathologies like shoes and insoles. The pressure distribution measuring system of foot also allows you to check the effectiveness of supply (Fig. 6.5).

If kinetic and kinematic data are available, generally the calculation of joint moments by inverse dynamics is possible [12]. This requires a comprehensive multi-dimensional set of data and corresponding software. All in all, enormous and expensive efforts are necessary and due to the requisite assumptions regarding the position of the hinge points results will still be relatively inaccurate.

### Important Analysis Parameters

Total force-time curves are an important indicator of potential impairment. The results can identify the affected side of the body and the deficient movement phase. However, the causally affected body part cannot be determined. Vertical force-time curves show by the distinction of the maxima when walking or the left or right shift of the maximum while running, whether a pronounced foot strike, footprint or even rolling motion is present (Fig. 6.6) and facilitate the assessment of knee problems. A unilaterally reduced dynamic of the vertical ground reaction force through reduced maxima and a higher minimum indicates a deficit of this body side, a reduced 2nd maximum force in conjunction with a reduced 1 maximum force of the next step indicates a careful side transition. Unidirectionally higher momentums across the movement direction during unilateral stance phase indicate a deficit on this side of the body, meaning a shift of gravity as evasive





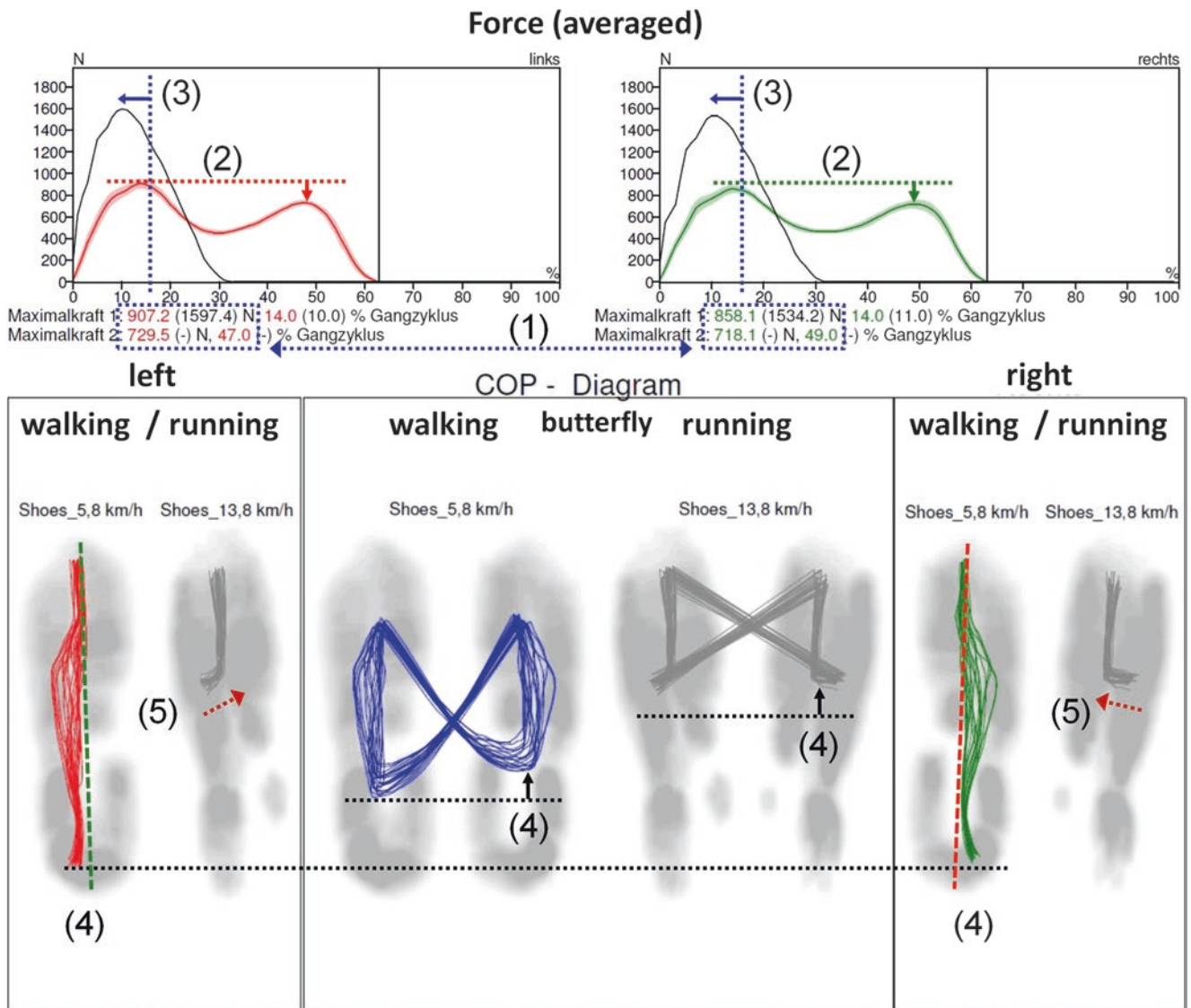
**Fig. 6.5** “Treadmill analysis widespread”. Treadmill analysis at 13.8 km/h, foot ground contact in hyper-supination. Picture sequences for side, phase and shoe vs. barefoot comparison (2 columns left: with

shoe, 2 columns right: barefoot). (1) Single, mean and maximum footprints, (2) centre of pressure course; (3) maximum pressure; (4) force-time-curve, each with shoe (left: red, right: green) and barefoot (black)

movement on the contralateral side of the body. Comparatively lower braking or acceleration impulses in the running direction also indicate deficiencies in the foot strike or foot push off. The often existing short initial peaks of forces in and/or transversely to the movement direction opposite to the normal direction of force are signs that the foot at the end of the forward swing at foot strike in the direction towards the rear and transverse to the direction inward “falls” to the ground (Fig. 6.4, green ellipses). Is this peak particularly high, the reason should be traced because of the resulting momentums for the ankle. A comparison to walking barefoot is required in order to assess the influence of the shoes.

Resulting pressure- and force-time-curves during pedography in particular on the treadmill are indeed less accurate

than those from force plates, but include other important results. The fixed-image shows beside foot deformities and stress symptoms such as fallen arches, splayfoot, pes cavus (contracted foot) or characteristics of flat foot in the barefoot viewing the individual load distributions with shoes and associated abnormalities. During walking and running the cyclogram (Fig. 6.6) provides important information of gait stability and gait symmetry. In particular, while running next to the identification of the runner type, especially type immanent features such as pronation during hindfoot runner, the stress distribution at the midfoot runner or the burden of the bale when forefoot runner can be assessed very well and in the case of improper loading counteractions can be taken. The comparison between walking and running barefoot or



**Fig. 6.6** “Force-time-curves and centre-of-pressure”. Analysis of force-time-curves and centre of pressure (COP) in walking and running: mild to moderate higher forces left (1) with distinctly accentuated foot strike in walking (2, higher first maximum) and running (3,

left shift of maximum); ground contact in distinctly supinated position with hindfoot in walking (4) and forefoot in running (5) followed by an inwards movement; in cyclogram (butterfly) distinctly reduced right-hand sided centre of pressure course in walking (6) and running (7)

with shoes provides important information on the influence of the shoes and the quality of the insole supply.

While prominent peak pressures usually can clearly be attributed to a foot problem most of the results of the kinetic analysis can be considered only in the context of the overall movement. While the power is transmitted during walking and running over by the foot, but this is only the end link of a complex kinematic chain with the goal of moving the body i.e. the center of gravity. That is why as a rule abnormalities must be checked as to whether the cause comes from the foot or another part of the body, in particular the associated leg. In addition to the mean and maximum print images (Fig. 6.5) mainly the rolling behavior is of particular importance (Fig. 6.3, foot prints).

## Electromyography

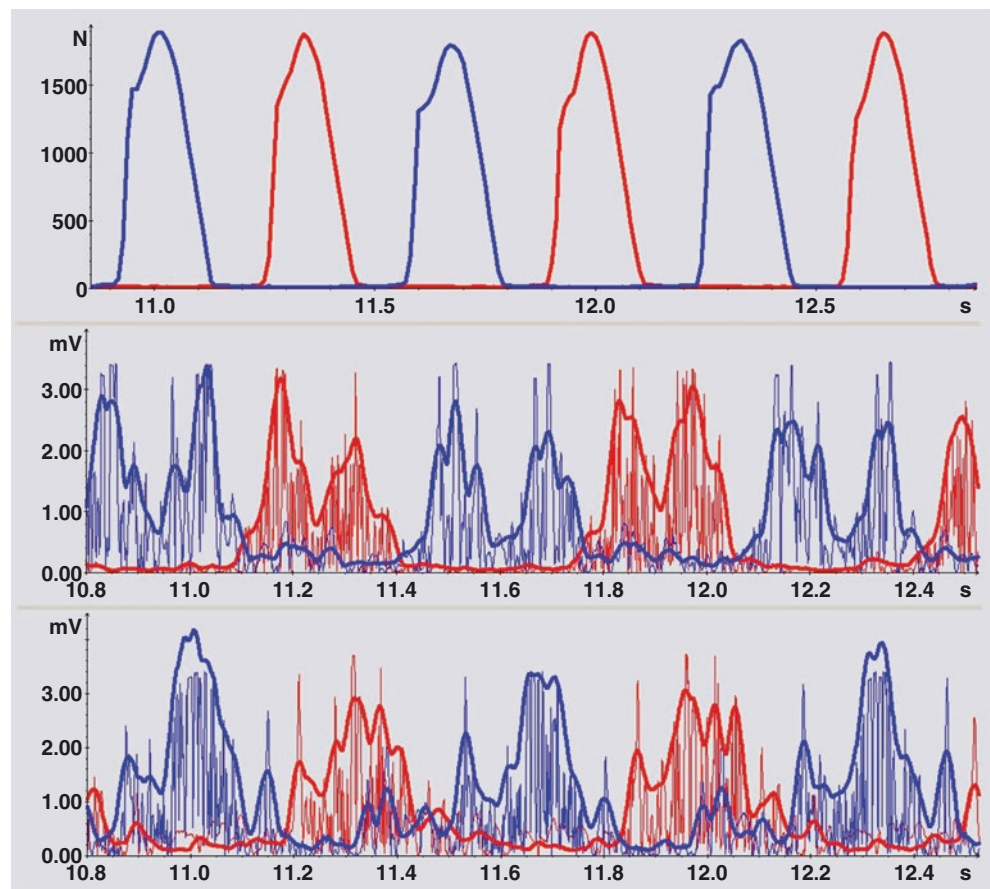
To extend the kinetic and kinematic gait or treadmill analyses a muscle function analysis by surface electromyography can be added. By electrodes that are applied to the examined muscles can be diagnosed, which muscle when and how long is what type and how intra- and inter-muscular interplay active. This information can help to detect muscular, neural or sensory-motor abnormalities and provide possible explanations

for kinetic or kinematic abnormalities. This method can only detect muscles that are located on the surface of skin. The use of needle electrodes is not appropriate for gait and treadmill analyses. In addition to various disturbing noise sources due to the low power of the EMG-signal, motion artefacts are to be expected in particular in the dynamic application. To avoid or reduce sources of interference, therefore, a careful application and evaluation is required [13, 14].

## Important Analysis Parameters

For muscle function analysis of foot flexion and extension for example the m. tibialis anterior, m. peroneus longus and brevis, m. gastrocnemius medialis and lateralis and m. soleus can be derived. As measuring parameters time and duration as well as the amplitude of the signal are available.

The unilateral analysis reveals especially the extent to which a muscle is activated according to time and amplitude of its function (Fig. 6.7). Reduced or absent activity of m. peroneus can e.g. reveal or prove a weak dorsiflexion. The comparison of sides also allows the evaluation of the activation of symmetry. Differences in activation patterns in time and in amplitude indicate functional deficits the cause of



**Fig. 6.7** “Treadmill analysis EMG”. Treadmill analysis at 16 km/h, above: ground reaction force (*vertical*), center: EMG of m. biceps femoris, bottom: EMG of m. gastrocnemius medialis; EMGs rectified (*thin*) and smoothed (*thick*) with power spectrum envelope; left-hand side (*red*) and right-hand side (*blue*)

which must be clarified [14]. In order to allow a quantitative side comparison of the amplitudes, the determination of the maximum EMGs of the muscles to be compared is required.

## Summary and Take Home Message

The gait and treadmill analysis in instrumental form is a reliable instrument for expanding and securing medical diagnostics. Thanks to modern instruments, the effort of instrumental gait and treadmill analysis is significantly reduced and the results are available promptly. By use of modern methods of analysis abnormalities in the dynamics of movement can be recognized that remain hidden to the human eye. Early preventative measures can preclude possible damage to the musculo-skeletal system or at least postpone it. Concerning rehabilitation changes can be identified, remaining deficiencies assessed and therapeutic measures supported and monitored.

### Take Home Message

*The understanding of movement, and the detection and treatment of primary deficits of movement is the key component of successful and sustainable diagnosis and treatment.*

Kinetic force-time-curves show problematic movement phases. Pedography helps to assess the rolling performance and the identification of faults or overload in the area of the foot. Kinematic 3D motion analysis as well as electromyography support the search for the reasons of aberration and help with the choice of the treatment approach. However, in addition to the opportunities, especially the limits of the methods used in biomechanical measurement must be observed. Kinematics can describe the motion of the body exactly in space and time, but does not provide any information about the ensuing forces or muscle activity. Kinetics provides precise data for the force-time-curve of the whole body movement, but the contributions of single body parts to these movements or of individual muscles are not visible.

### Take Home Message

*For the diagnosis of a limited problem the use of a single biomechanical measurement method may be sufficient.*

*To diagnose a complex cause-effect relationship the simultaneous use of different biomechanical measurement methods is essential because the significance of each individual measurement method is limited.*

Normal values and standard curves are a great aid in understanding complex interrelationships and the classification of current results. In this sense any deviation from the norm requires special attention. The desire for fast, automated results and simple causal relationships is tempting to use normative data as representative for the evaluation based on motion analysis [15]. Human individuality is expressed mainly in the way of deviation and not by conformity to a standard. The high complexity of motion contains a multitude of degrees of freedom and allows for a corresponding number of individual solutions. Human movement is not only a subject of principle of economy and efficiency, but is also characterized by high variability and compensation ability. This can be expressed by a lack of exercise, stress or fatigue effects, as well as by a natural range of movement and exploiting the degrees of freedom. Pathologies are only one possible reason of a standard deviation.

### Take Home Message

*The assessment of a movement with respect to its individual normality is more important than the accordance with a normative exercise. Symmetric but non-standard movements primarily have to be changed only when pathological misalignments or afflictions are present. Asymmetric movements deserve special attention in any case, even if the individual one still remains within the standard norm.*

## References

1. Jöllenbeck T. Biomechanische Bewegungsanalyse – Unverzichtbarer Bestandteil moderner sportmedizinischer Diagnostik. Deut Z Sportmed. 2012;63(3):59–60.
2. Perry J. Gait analysis – normal und pathological function. Thorofare: Slack; 1992.
3. Whittle MW. Gait analysis – an introduction. 3rd ed. Oxford: Butterworth-Heinemann; 2002.
4. Winter DA. The biomechanics and motor control of human gait. 2nd ed. Waterloo: Waterloo Biomechanics; 1991.
5. Götz-Neumann K. Gehen verstehen – Ganganalyse in der Physiotherapie. Stuttgart/New York: Thieme; 2003.
6. Vogt L, Banzer W. Instrumentelle Ganganalyse. Deut Z Sportmed. 2005;56(4):108–9.
7. Alton F, Baldey L, Caplan S, Morissey MC. A kinematic comparison of overground and treadmill walking. Clin Biomech. 1998;13:434–40.
8. White SC, Yack HJ, Tucker CA, Lin HY. Comparison of vertical ground reaction forces during overground and treadmill walking. Med Sci Sports Exerc. 1998;30:1537–42.
9. Riley P, Paolini G, Croce U, Paylo K, Kerrigan D. A kinematic and kinetic comparison of overground and treadmill walking in healthy subjects. Gait Posture. 2007;26:17–24.

10. Terrier P, Dériaz O. Kinematic variability, fractal dynamics and local dynamic stability of treadmill walking. *J Neuroeng Rehabil.* 2011;8(12):1–13.
11. Rosenbaum D. Klinische Ganganalyse in der Orthopädie und Traumatologie. In: Jerosch J, Nicol K, Peikenkamp K, editors. *Rechnergestützte Verfahren in Orthopädie und Unfallchirurgie.* Darmstadt: Steinkopff; 1999.
12. Winter DA. *Biomechanics and motor control of human movement.* 3rd ed. Waterloo: Wiley; 2005.
13. Hermens HJ, Freriks B. *The Seniam CD-rom – European Recommendations for Surface ElectroMyoGraphy.* Enschede: Roessingh Research and Development; 1999.
14. Freiwald J, Baumgart C, Konrad P. *Einführung in die Elektromyographie, Sport – Prävention – Rehabilitation.* Balingen: Spitta; 2007.
15. Freiwald J, Engelhard M. Stand des motorischen Lernens und der Koordination in der orthopädisch-traumatologischen Rehabilitation. *Sport Orthop Traumatol.* 2002;18:5–10.

# Arthroscopic Supplementation of Imaging Findings: Using Arthroscopy to Detect Abnormalities Missed on Imaging

Eric Ferkel and Bruce E. Cohen

## Abstract

Arthroscopic surgery has given the foot and ankle surgeon the ability to identify pathology that can be missed on advanced imaging studies. Ankle fractures are a common injury that often only receives a plain radiograph prior to surgery. Arthroscopy, as part of the surgical fixation of ankle fractures, can demonstrate syndesmotic widening and osteochondral defects that otherwise may have been missed. Arthroscopic evaluation during surgical management of ankle instability can show significant osteochondral lesions or loose bodies. Arthroscopy is essential in the management of osteochondral defects of the tibia or talus, specifically in assessing the size and depth of the lesion, as well as the quality of the surrounding cartilage. Subtalar arthroscopic evaluation has been shown to be effective in confirming the diagnosis and assisting with treatment. When assessing peroneal tendon pathology, tenoscopy has shown promise in localizing and sometimes even treating peroneal tendon tenosynovitis and small tears. The advancements in the use of arthroscopy have given the orthopaedic surgeon the capability to diagnose and treat injuries that otherwise may have been missed with standard imaging studies.

## Keywords

Imaging • Osteochondral lesion of the talus • Peroneal Tendon • Ankle Fractures • Ankle Instability • Subtalar synovitis • Interosseous talocalcaneal ligament tears • Arthroscopic evaluation of the foot and ankle

The development of foot and ankle arthroscopy over the past 30–40 years has greatly improved the orthopaedic surgeon's ability to treat and diagnose pathology previously missed on imaging or explored with an open surgery. The first ankle arthroscopy was described by Burman in 1931 [1]. Watanabe was the first to describe a case study, reporting on a series of ankle arthroscopies in 1972 [2]. Foot and ankle arthroscopy can be used for both diagnostic and therapeutic purposes to treat a variety of joint-related conditions, including the anterior and posterior ankle joints, as well as the first metatarsophalangeal (MTP) and subtalar joints. Recently arthroscopy

of tendons, known as tenoscopy, has emerged as a tool to address pathology of the posterior tibial tendon, flexor hallucis longus (FHL) tendon and the peroneal tendon in a minimally invasive fashion. Arthroscopy has the distinct advantage of giving the surgeon the intra-articular or intra-tendinous view that traditional imaging cannot, while limiting surgical exposure and soft tissue trauma.

Foot and ankle arthroscopy is typically indicated for anterior soft tissue and bony impingement syndrome, osteochondral defects of the talus or tibia, ankle synovitis, arthrofibrosis, loose body removal, debridement and irrigation of septic arthritis, as well as in the treatment of ankle fractures and arthrodesis. Posterior hindfoot arthroscopy can be used for removal of the os trigonum and FHL tenosynovitis, which both combine to form the diagnosis of posterior impingement [3]. Subtalar and posterior tibiotalar joint arthritis as

E. Ferkel, MD (✉) • B.E. Cohen, MD  
OrthoCarolina Foot and Ankle Institute,  
2001 Vail Avenue #200B, Charlotte, NC 28207, USA  
e-mail: [eferkel@scoi.com](mailto:eferkel@scoi.com); [Bruce.Cohen@orthocarolina.com](mailto:Bruce.Cohen@orthocarolina.com)

well as Haglund's deformity are further indications for posterior or hindfoot arthroscopy.

In this chapter the role of ankle arthroscopy to diagnose and treat conditions that may be missed on imaging will be explored. Specifically, we will review the use of arthroscopy in treating ankle fractures, assisting in the treatment of ankle instability, evaluating osteochondral lesions, subtalar debridement and peroneal tendoscopy.

Before performing any ankle arthroscopy it is vital to perform an initial diagnostic arthroscopy in order to ensure a complete evaluation of the ankle and the joint. The 21 point ankle exam has been advocated as a thorough diagnostic tool in ankle arthroscopy [4]. Performing the evaluation in a systematic fashion ensures that no areas associated with the patient's underlying pathology will be missed.

The 21 point exam for a diagnostic ankle arthroscopy, as described by Ferkel [4], requires one to pause at each point of the exam to study the patient's anatomy. The order in which one evaluates each area is irrelevant as long as the evaluation is thorough and efficient. The structures visualized include:

With the scope in the antero-medial portal:

1. Deltoid ligament
2. Medial gutter
3. Medial talus
4. Central talus and central overhang
5. Lateral talus
6. Trifurcation with the talus, tibia and fibula, including anterior inferior tibiofibular ligament
7. Lateral gutter, including anterior talofibular ligament
8. Anterior gutter

The central and posterior structures of the ankle are then visualized from the antero-medial or antero-lateral portal. These include:

1. Medial tibia/talus
2. Central tibia/talus
3. Lateral tibia/talar-fibular articulation

The arthroscope is then maneuvered more posteriorly from either of the anterior portals to visualize the posterior structures. These include:

1. Posterior inferior tibiofibular ligament
2. Transverse tibiofibular ligament
3. Reflection of the flexor hallucis longus

The arthroscope is then changed to the postero-lateral portal to complete the exam, visualizing the following:

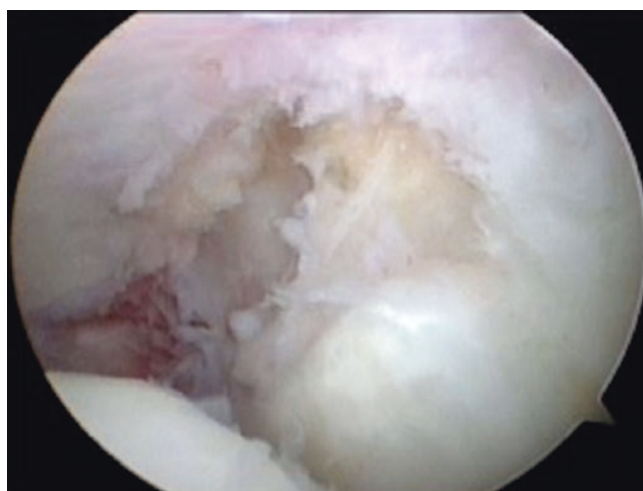
1. Posteromedial talus

2. Posteromedial talus
3. Postero-central talus
4. Posterolateral talus
5. Posterior talofibular articulation
6. Posterolateral gutter, including the posterior talofibular ligament
7. Posterior gutter

After completely visualizing the ankle from the antero-medial portal, it is necessary to visualize again from the anterolateral portal. Notice the difference in accessibility of the medial and lateral gutters depending on the location of the arthroscope. Visualize the anterior inferior tibiofibular ligament and anterior talofibular ligament inferiorly. After the surgeon completes the diagnostic ankle arthroscopy, he/she should be well informed to make a decision regarding therapeutic intervention.

## Ankle Fractures and Arthroscopy

The use of arthroscopy with surgical treatment of ankle fractures can greatly improve the surgeon's overall ankle evaluation and assist in the creation of a treatment plan. Arthroscopy allows the surgeon to assess intra-articular pathology, identifying loose bodies, osteochondral lesions or torn ligamentous structures. Additionally arthroscopy can aid the surgeon in reduction and internal fixation and help with post reduction evaluation of the joint and syndesmosis. Several studies have shown that initial evaluation of the articular surface after ankle fracture revealed a significant amount of intra-articular injury (Fig. 7.1). Ferkel et al. showed 63% traumatic articular surface lesions (TASL) in 48 patients [5]. The investigators found that of the 12 fractures with syndesmotic disruptions, nine sustained full-thickness damage to the talar



**Fig. 7.1** Image of an ankle fracture through the arthroscope

chondral surface. Ono et al. found that 20% of ankle fractures requiring open reduction internal fixation (ORIF) had a cartilage injury and 51.4% had a ligamentous injury with the anterior inferior tibiofibular ligament (AITFL) being the most commonly injured ligament [6]. Leontaritis et al. published a retrospective study of 84 ankle fractures in which ankle arthroscopy was utilized during the index procedure [7]. Their study included supination external rotation (SER) type II and pronation external rotation type I (PER) ankle fractures, however, the majority (66%) were PER and SER IV. They found that 73% of ankle fractures had concomitant chondral lesions with the majority involving the talar dome (61%). PER and SER IV ankle fractures were eight to nine times more likely than PER and SER type I or II ankle fractures to be associated with two or more chondral lesions respectively.

Lui et al. looked at 53 patients without radiographic evidence of frank syndesmotic diastasis in Weber B or C ankle fractures, then performed intraoperative radiography combined with ankle arthroscopy. Sixteen patients had positive intraoperative stress x-rays (30%) and 35 cases had positive arthroscopic findings of syndesmosis diastasis (66%), demonstrating that ankle arthroscopy is a better tool than intraoperative stress x-rays in distinguishing syndesmotic diastasis [8] (Fig. 7.2). Takao et al. evaluated 38 patients in which arthroscopy was used to supplement plain x-ray diagnosis of syndesmotic injuries in operatively treated ankle fractures [9]. They found that syndesmotic widening was confirmed in all cases that were seen on preoperative radiologic studies and 12 frank syndesmotic disruptions were identified exclusively on arthroscopy that were otherwise unrecognized on preoperative plain x-rays, again reaffirming the importance of evaluating the ankle joints after an ankle fracture.



**Fig. 7.2** Image of a widened syndesmosis from looking through the anteromedial portal during a stress test

Arthroscopy is an effective tool in assisting in diagnosis and therapeutic treatment of ankle fractures. The amount of functional improvement or the difference in outcomes in patients who have pathology identified on arthroscopy at the time of surgical fixation as compared to those treated without arthroscopy is still undetermined. It is important, however, to be able to identify intra-articular lesions which may affect the long-term prognosis after these injuries.

## Ankle Instability and Arthroscopy

Ankle sprains are one of the most common injuries in athletes and can typically be treated non-operatively with functional rehabilitation. However, approximately 29–42% of patients have continued symptoms of chronic ankle instability, including the feeling of “giving out” with persistent pain and decrease in function.[10] MRI and CT are not typically indicated for an acute ankle sprain unless there are other suspected injuries, however imaging can be useful for the evaluation of chronic ankle instability. MRI has been shown to be anywhere from 20 to 80% accurate in detecting osteochondral lesions, loose bodies or peroneal tendon injuries, specifically peroneus brevis, in patients with chronic ankle instability [11–13]. Therefore the role of arthroscopy is essential in prevention of long term sequela that would otherwise be missed if one were to rely upon imaging alone prior to surgery.

Hintermann et al. found significant cartilage damage in 66% of patients with lateral ligament pathology and 98% of patients with deltoid ligament injury [14] (Fig. 7.3). Taka et al. found osteochondral lesions in 95% of chronically unstable ankle and 89% of acute ankle injuries. Ferkel showed in two separate studies a similar rate of intra-articular pathology of 93 and 95% [15, 16]. It is clear from these studies that there is a high prevalence of intra-articular pathology with chronic ankle instability and that arthroscopy can be beneficial to assist in the procedure and obtain a good to excellent result. For these reasons, it is our routine practice to perform diagnostic arthroscopy in all patients undergoing surgical reconstruction of ankle instability. Concerns of fluid extravasation, which could compromise concomitant ligament reconstruction, are unfounded. An efficient diagnostic arthroscopic examination can be a very useful tool in treating ankle instability.

## Osteochondral Lesion Evaluation

Cartilage evaluation of osteochondral lesions is essential prior to making surgical decisions. It is imperative that the surgeon accurately evaluates the articular cartilage to make a correct diagnosis and assess the stability of the lesion in order to





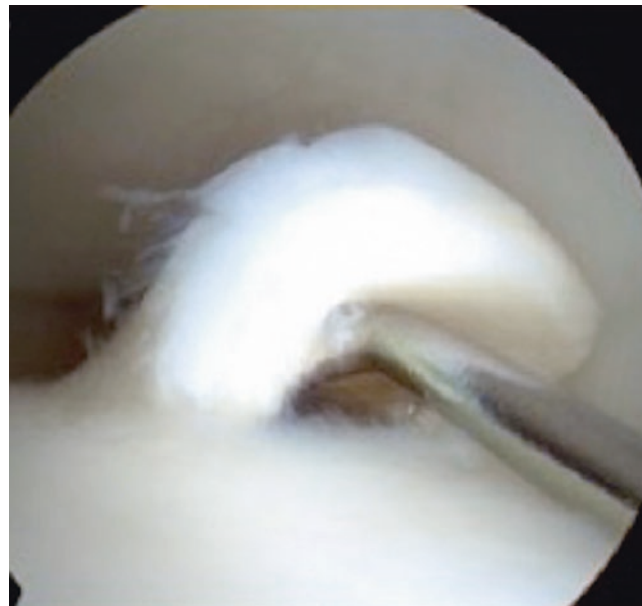
**Fig. 7.3** Evidence of ankle instability on arthroscopic examination showing increased widening at the lateral gutter

preserve function and avoid long-term pain. Understanding the correct stage of the lesion and the extent of the defect is essential to surgical planning. MRI has been shown to accurately stage osteochondral lesions in 65–92% of cases, although there are concerns that MRI is unable to identify isolated cartilage surface lesions, possibly due to the relatively thin talar articular cartilage [17–20]. CT is useful for evaluating osteochondral lesions and providing information on the condition of the underlying subchondral bone related to the cartilage damage (Fig. 7.4). Unfortunately CT cannot adequately assess the presence of an intact cartilage surface. This may influence treatment options, i.e. transtalar drilling techniques [21, 22].

The size of an osteochondral lesion can be critical in determining surgical treatment options such as OATS procedures or debridement and microfracture. MRI has been shown in some cases to overestimate the size of the lesion compared to CT; most often the most accurate diagnosis of size will be made by arthroscopy.

### Subtalar Evaluation by Arthroscopy

Arthroscopy of the subtalar joint is an excellent tool to evaluate pathology that may not be readily apparent on MRI or CT. Subtalar arthroscopy is typically indicated in patients who are experiencing hindfoot pain or pain along their sinus tarsi. Indications include osteochondral injury with loose body, synovitis, and interosseous talocalcaneal ligament



**Fig. 7.4** Image of an osteochondral lesion being elevated by a probe

(ITCL) tears. MRI was found to be nonspecific and insensitive when correlated with arthroscopic findings in regards to tears of the ITCL [23]. Goldberg and Conti found that preoperative MRI underestimated the degree of articular cartilage damage in patients who then underwent an arthroscopic subtalar debridement [24]. Frey et al., found that patients who had been given the preoperative diagnosis of “sinus tarsi syndrome”, were all found to have alternative diagnoses on subtalar arthroscopy, with the most common being tears of the interosseous ligament [25].

### Peroneal Pathology

Peroneal tendon tears, most often of the brevis, are frequently associated with chronic ankle instability, ankle sprains, peroneal tendon subluxation, and the varus hindfoot. Bare and Ferkel reported finding 60 intra-articular lesions in 30 patients undergoing peroneal tendon repair, with 78% of these lesions undetected on preoperative imaging. This suggests that ankle arthroscopy should be regularly completed in the setting of peroneal tendon repair, especially an acute peroneal tendon repair [26].

Peroneal tendoscopy is emerging as excellent tool to assist in diagnosis, accurate localization and treatment of peroneal tendon tenosynovitis and small tears [27, 28]. Chronic lateral retromalleolar pain, often seen in runners and dancers, can be attributed to peroneal tendon pathology. However given the various extrinsic and intrinsic causes of the pain in the region, MRI may not be reliable as a diagnostic tool, and may often be inconclusive [29, 30]. In these cases peroneal tendoscopy

can be useful to diagnose and treat peroneal tenosynovitis or minor tears of the tendons. Van Dijk et al., retrospectively reviewed their series of patients who had either synovitis with or without tearing of the peroneus brevis and there were no complications or recurrence of the preoperative pathology [28]. Peroneal tendoscopy is gaining in popularity, however at this time, its role is mostly limited to diagnostic assistance, allowing for a localized minimal incision repair or debridement.

## Summary

In conclusion, arthroscopy is an essential tool in diagnosis and therapeutic management of foot and ankle injuries, giving one a more complete picture of the patient's pathology than imaging alone. In particular, the use of diagnostic ankle arthroscopy in ankle fractures is essential to identifying intra-articular pathology that might otherwise be missed. It can also be helpful in visualizing fracture or syndesmotic reduction. In ankle instability, arthroscopy has proven to be superior to imaging alone, giving the surgeon a more comprehensive view of the concomitant injuries. Finally, in peroneal tendon injuries, tendoscopy may become a valuable aid in localization and treatment of tendon pathology. Arthroscopy of the foot and ankle is a vital instrument in the surgeon's toolkit to ensure the patient's best outcome.

## References

- Burman MS. Arthroscopy or the direct visualization of joints: an experimental cadaver study. *J Bone Joint Surg.* 1931;13:669–95.
- Watanabe M. Selfoc-Arthroscope. Watanabe no 24 arthro-scope. Teishin Hospital, Toyko; 1972.
- Abramowicz S, Simon LE, Susarla HK, Lee EY, Cheon JE, Kim S, et al. Are panoramic radiographs predictive of temporomandibular joint synovitis in children with juvenile idiopathic arthritis? *J Oral Maxillofac Surg Off J Am Assoc Oral Maxillofac Surg.* 2014;72(6):1063–9.
- Ferkel RD. *Arthroscopic surgery: the foot and ankle.* 1st ed. Philadelphia: Lippincott-Raven; 1996.
- Loren GJ, Ferkel RD. Arthroscopic assessment of occult intra-articular injury in acute ankle fractures. *Arthrosc J Arthrosc Related Surg Off Publ Arthrosc Assoc N Am Int Arthrosc Assoc.* 2002;18(4):412–21.
- Ono A, Nishikawa S, Nagao A, Irie T, Sasaki M, Kouno T. Arthroscopically assisted treatment of ankle fractures: arthroscopic findings and surgical outcomes. *Arthrosc J Arthrosc Related Surg Off Publ Arthrosc Assoc N Am Int Arthrosc Assoc.* 2004;20(6):627–31.
- Leontaritis N, Hinojosa L, Panchbhavi VK. Arthroscopically detected intra-articular lesions associated with acute ankle fractures. *J Bone Joint Surg Am.* 2009;91(2):333–9.
- Lui TH, Ip K, Chow HT. Comparison of radiologic and arthroscopic diagnoses of distal tibiofibular syndesmosis disruption in acute ankle fracture. *Arthrosc J Arthrosc Related Surg Off Publ Arthrosc Assoc N Am Int Arthrosc Assoc.* 2005;21(11):1370.
- Takao M, Uchio Y, Naito K, Fukazawa I, Kakimaru T, Ochi M. Diagnosis and treatment of combined intra-articular disorders in acute distal fibular fractures. *J Trauma.* 2004;57(6):1303–7.
- Berlet GC, Anderson RB, Davis WH. Chronic lateral ankle instability. *Foot Ankle Clin.* 1999;4(4):713–28.
- DIGiovanni BF, Fraga CJ, Cohen BE, Shereff MJ. Associated injuries found in chronic lateral ankle instability. *Foot Ankle Int.* 2000;21(10):809–15.
- Rolf CG, Barclay C, Riyami M, George J. The importance of early arthroscopy in athletes with painful cartilage lesions of the ankle: a prospective study of 61 consecutive cases. *J Orthop Surg Res.* 2006;1:4.
- O'Neill PJ, Van Aman SE, Guyton GP. Is MRI adequate to detect lesions in patients with ankle instability? *Clin Orthop Relat Res.* 2010;468(4):1115–9.
- Hintermann B, Boss A, Schafer D. Arthroscopic findings in patients with chronic ankle instability. *Am J Sports Med.* 2002;30(3):402–9.
- Komenda GA, Ferkel RD. Arthroscopic findings associated with the unstable ankle. *Foot Ankle Int.* 1999;20(11):708–13.
- Ferkel RD, Chams RN. Chronic lateral instability: arthroscopic findings and long-term results. *Foot Ankle Int.* 2007;28(1):24–31.
- Mintz DN, Tashjian GS, Connell DA, Deland JT, O'Malley M, Potter HG. Osteochondral lesions of the talus: a new magnetic resonance grading system with arthroscopic correlation. *Arthrosc J Arthrosc Related Surg Off Publ Arthrosc Assoc N Am Int Arthrosc Assoc.* 2003;19(4):353–9.
- Lee KB, Bai LB, Park JG, Yoon TR. A comparison of arthroscopic and MRI findings in staging of osteochondral lesions of the talus. *Knee Surg Sports Traumatol Arthrosc Off J ESSKA.* 2008;16(11):1047–51.
- Schimmer RC, Dick W, Hintermann B. The role of ankle arthroscopy in the treatment strategies of osteochondritis dissecans lesions of the talus. *Foot Ankle Int.* 2001;22(11):895–900.
- Bae S, Lee HK, Lee K, Lim S, Rim NJ, Kim JS, et al. Comparison of arthroscopic and magnetic resonance imaging findings in osteochondral lesions of the talus. *Foot Ankle Int.* 2012;33(12):1058–62.
- Nakasa T, Adachi N, Kato T, Ochi M. Appearance of subchondral bone in computed tomography is related to cartilage damage in osteochondral lesions of the Talar Dome. *Foot Ankle Int.* 2014;35(6):600–6.
- Ferkel RD, Zanotti RM, Komenda GA, Sgaglione NA, Cheng MS, Applegate GR, et al. Arthroscopic treatment of chronic osteochondral lesions of the talus: long-term results. *Am J Sports Med.* 2008;36(9):1750–62.
- Lee KB, Bai LB, Park JG, Song EK, Lee JJ. Efficacy of MRI versus arthroscopy for evaluation of sinus tarsi syndrome. *Foot Ankle Int.* 2008;29(11):1111–6.
- Goldberger MI, Conti SF. Clinical outcome after subtalar arthroscopy. *Foot Ankle Int.* 1998;19(7):462–5.
- Frey C, Feder KS, DiGiovanni C. Arthroscopic evaluation of the subtalar joint: does sinus tarsi syndrome exist? *Foot Ankle Int.* 1999;20(3):185–91.
- Bare A, Ferkel RD. Peroneal tendon tears: associated arthroscopic findings and results after repair. *Arthrosc J Arthrosc Related Surg Off Publ Arthrosc Assoc N Am Int Arthrosc Assoc.* 2009;25(11):1288–97.
- van Dijk CN, Kort N. Tendoscopy of the peroneal tendons. *Arthrosc J Arthrosc Related Surg Off Publ Arthrosc Assoc N Am Int Arthrosc Assoc.* 1998;14(5):471–8.
- Scholten PE, van Dijk CN. Tendoscopy of the peroneal tendons. *Foot Ankle Clin.* 2006;11(2):415–20. vii.
- Bassett 3rd FH, Speer KP. Longitudinal rupture of the peroneal tendons. *Am J Sports Med.* 1993;21(3):354–7.
- Sammarco VJ. Peroneal tendoscopy: indications and techniques. *Sports Med Arthrosc Rev.* 2009;17(2):94–9.

# Management of Injured Athletes at the Field

8

Thilo Hotfiel, Hans-Dieter Carl, Casper Grim,  
and Martin Engelhardt

## Abstract

Traumatic injuries of the foot and ankle occur frequently during sports competitions and represent a wide spectrum of injuries. The on-field and side-line management to injured athletes imposes high demands on the medical practitioner. A high level of knowledge about sports and discipline specify requirements regarding biomechanic principles and the resulting injuries and overuses are absolutely essential. The on-field management should follow a standardized algorithm of assessing the injured mechanism, history and physical examination. Under high time pressure, serious decisions about the following treatment referring to the individual athlete have to be taken very quickly and have to be adapted to the aggregate of the athletes' individual disorder, the physical examinations and the specific requirements of the sport.

## Keywords

On-field Management • Sport Injuries • Injured Athlete • RICE Principle

## Challenges and Requirements of On-Field Management

Acute traumatic injuries of the foot and ankle occur frequently during sports competitions, and ankle sprains are one of the most common injuries. A wide spectrum of acute and overuse injuries include tendon, muscle or ligament sprains, soft-tissue damages, skin lacerations, cartilage lesions or fractures up to

T. Hotfiel, MD (✉)

Division of Orthopedic Rheumatology, Department of Orthopedic Surgery, University of Erlangen-Nuremberg, Rathsberger Str. 57, Erlangen D-91054, Germany  
e-mail: [Thilo.Hotfiel@fau.de](mailto:Thilo.Hotfiel@fau.de)

H.-D. Carl, MD

Department of Orthopedic Surgery and Trauma Surgery, Martha-Maria Hospital Nuremberg, Stadenstrasse 58, Nürnberg 90491, Germany  
e-mail: [hans-dieter.carl@martha-maria.de](mailto:hans-dieter.carl@martha-maria.de)

C. Grim, MD • M. Engelhardt, MD

Department for Orthopaedics, Trauma Surgery and Hand Surgery, Klinikum Osnabrück, Am Finkenhügel 1, Osnabrück 49076, Germany  
e-mail: [Casper.Grim@klinikum-os.de](mailto:Casper.Grim@klinikum-os.de); [Martin.Engelhardt@klinikum-os.de](mailto:Martin.Engelhardt@klinikum-os.de)

joint dislocation including injuries of neurovascular structures. The on-field and side-line management of injured athletes, imposes high demands on the medical coverage practitioner. A high level of knowledge about sports and discipline specify requirements as well as biomechanical principles and the resulting injuries are absolutely essential. This knowledge refers to the contest phase in which peak performance is required and is just as important for the excessive training period dominated by high weekly training hours. Furthermore the athletes' specific conditions regarding individual risk factors or medical history including prior injuries or fitness level have to be known and complied. In general, a wide variety of medical coverage practitioners care for the athletes' health. Among these practitioners orthopaedic surgeons, sports medicine specialists, physical therapists or emergency medical services with a different level of experience and skills in treatment of sports related injuries can be found. In order to ensure a high medical standard, it is the medical coverage practitioners' duty to acquire the sports specific knowledge and skills. Under high pressure of time serious decisions referring to the individual athlete have to be taken very quickly and have to be adapted to the aggregate of the athletes' individual disorder, the physical findings and the sports specific requirements (Fig. 8.1). The goals of on-field



**Fig. 8.1** The picture represents the on-field treatment of an acute ankle injury and demonstrates the restricted medical supplies for initial assessment and examination

treatment are defined as the following: Prevention of further injury, minimizing the zone of injury, decreasing pain, promoting healing and allowing a safe return to athletic competition [1]. Subject to the adopted decision within its initial treatment in case of a present injury, the immediate and long-term sporting success and moreover the athletes' health depends on this initial correct management of an injured athlete on the field.

## Epidemiology

The incidence, mechanisms and circumstances of injuries differ among the different sports. Injuries can be described as non-contact trauma events, as contact-events (with another athlete, a moving object (e.g. ball, puck) or a static object (e.g. hurdles, net, goalpost), as recurrent injury or overuse injury [2]. During the Summer Olympic Games in London 2012, Beijing 2008 and IAAF World Championships in Daegu 2011, most of the injuries occurred in competition (55% Competition vs. 45% Training, London; 74% vs. 26%, Beijing; 56% vs. 44% Daegu) [3–5]. The majority of injuries occurring in international sports events are reported to be of lower severity, but up to 35–49% of injuries in the last two Olympic Games [3, 4] and in mean half of all injuries in the last three IAAF World Championships [5–7] were estimated in time absence from training or competition. Regarding the anatomical localisation of all injuries and overuses in London 2012, the highest incidence was detected for the foot and ankle, especially in Volleyball (30%), Handball (25%), Taekwondo (24%) Athletics (24%) and Soccer (22%) [3].

## Preparation of Medical Coverage

In advance to an upcoming competition or training camp the medical coverage has to be planned and arranged conscientiously. In order to ensure the treatment of higher injuries up to severe injuries, the local conditions including the accessibility of a medical centre or the next hospital and its medical supplies have to be observed. In this regard it is advisable to contact the local rescue services previously in order to guarantee a good level of cooperation. In addition to the medical supplies, local requirements considering injury related risk factors like the playing or running surface [8] as well as weather conditions have to be assessed. An early on-field response of acute injuries assumes the presence of a well-resourced emergency bag [9], which is especially in youth sport unfortunately often insufficient provided [10]. In view of a universal emergency medical bag which has to be available at any time and is standardised in football for example as the “FIFA medical emergency bag” [11], following material is needed to ensure an early treatment especially of injuries of foot and ankle [12]:

- Cool box implying ice water sponges
- Underarm Crutches
- Cooling spray
- Tapes in different sizes
- Elastic bandages
- Splints
- Pain killers (NSAIDs)

## Initial Assessment

In the ideal case, a constant observation of the sports field should be achieved in order to assess the mechanism of injury (e.g. anatomical movement of the extremity, contact with a stagnant object, contact with another athlete, non-contact, initial loading capacity after injury). Participating in multi-sports events (track and field) the medical practitioner possibly meets later and a short history of the injury mechanism is needed to acquire information in order to evaluate the injury. In some cases an examination in performance area or side-line has to be permitted by the referee or security guard. After assessing the injury mechanism, the athlete has to describe his complaints and severity of pain. Subsequently the initial examination should follow a standardised triage:

## Inspection

- Severity and anatomic localisation of the pain
- Existing swellings or redness?
- Presence of deformities?
- Skin conditions (Haematoma, Abrasions)
- Ability to full weight bearing or to complete gait cycle?

## Palpation

- Provocation of pain or crepitations occurring on anatomic relevant structures (Fig. 8.2, Table 8.1)

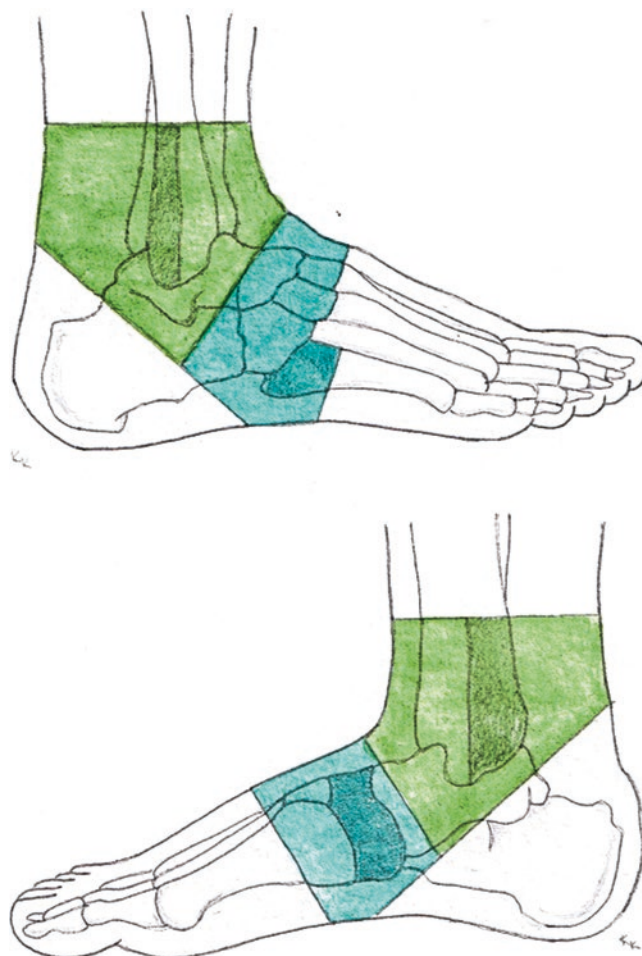
## Functional Examination

- Active and passive range of motion of the affected joint
- Achilles tendon: Ease of rising onto the toes, Thompson-Test, tendon-identifiability
- Ankle joint: anterior talus shift, medial or lateral stress test, Squeeze test
- Hindfoot and Midfoot: sideways and axial compression, calcaneal inversion/eversion, ability to torsion
- Forefoot: lateral and medial stress test of MTP-joints, axial compression, Lachman-Test, Squeeze-test
- Evaluation of neurologic and vascular compromise should always occur in the field if possible, taking care not to make the injury worse [13].

After a short review of the initial and expeditiously assessment, the responsible medical practitioner has to decide if an emergent transfer to a medical centre is needed, if the athlete has (temporary) to interrupt the sports activity or if a return to play is permitted. A misjudgment of

this initial evaluation can be linked with an aggravation of the existing damage that can be associated with severe sequels [13]. In most cases of injuries, an emergent transportation to a medical center is not necessary. But this is required in consideration of suspected vascular injuries, long bone fractures, open fractures, open joint injuries and joint dislocations [14, 15]. Considering the serious decision about the ability to play or not, standardized evidence based guidelines are lacking. Every athlete with their injuries has to be treated on an individual basis. However, published by German-Austrian-Swiss Society for Orthopaedic Traumatologic Sports Medicine (GOTS), in view of ankle sprain, the following criteria should be helpful to decide a cancellation of play (Table 8.2):

In case of an aspired return to play, a final functional exercise test can be implemented to assess the sports aptitude: Single leg stands test, jump test or sprint test. Orientated to the special athletic demands of the athletes discipline, the practicability of these functions helps to assess the functional performance.



**Fig. 8.2** Illustration of the Ottawa foot and ankle rules criteria – anatomic relevant areas

**Table 8.1** Ottawa Ankle rules [30]

The Ottawa foot and ankle rules criteria	
<b>Ankle: Pain in either malleolus plus 1 of the following:</b>	
1. Bone tenderness at tip of lateral malleolus or along posterior edge within 6 cm proximal to tip of malleolus	
2. Bone tenderness at tip of medial malleolus or along posterior edge within 6 cm proximal to tip of malleolus	
3. Inability to bear weight both immediately after the injury and in the emergency department	
<b>Foot: Pain in the midfoot plus 1 of the following:</b>	
1. Bone tenderness over the base of the fifth metatarsal	
2. Bone tenderness over the navicular bone	
3. Inability to bear weight both immediately after the injury and in the emergency department	

Stiell et al. [30]

**Table 8.2** Return to sports criteria (GOTS Expert Meeting Ankle Instability 2012 [12])

Criteria	Return to sports	Interruption
Restriction of mobility	Unchanged	New
Instability, "Giving way"	Unchanged	New
Pain	Initial pain, then painless	Under motion or weight bearing
Weight bearing	Full weight bearing	Limited weight bearing
Athletes' subjective Evaluation	Possible	Not possible

## Initial Treatment

The primary treatment of acute injuries can be performed using the acronym "RICE" and is common and recommended for initial treatment for most sports injuries [16–19]. This common and recommended therapy method requires rest, ice, compression, and elevation (Fig. 8.3). The primary therapy target is to reduce pain, swelling and bleeding and in this way to limit the initial inflammatory process and to prevent further damage. Despite the common application of the "RICE" principle, there is insufficient evidence that determines the effectiveness and improved clinical outcome of the "RICE"-principle in treatment of soft tissue injuries (Table 8.3), [20–23].

- **Rest:** Restricting activity and immobilisation should prevent worsening of the injury and leading to pain relief. The affected area should be immobilized using different sizes of splints, pads or crutches or applying bandages or tapes especially on toes (Fig. 8.4). Using crutches is helpful if a limit of weight bearing is aspired.
- **Ice:** Cryotherapy is often applied for soft tissue sports injuries. The application can be performed in an ideal way with an ice soaked sponge or an ice pack wrapped into a towel. The application of ice, leads to a vasoconstriction and reduces local muscular blood-flow by approximately 50% after 10 min [24] and therewith a decrease of swelling and initial bleeding. A cold application has also been found to decrease the inflammatory reaction in an experimental situation [25]. Another effect can be seen as reducing pain by increasing threshold levels in the free nerve endings and

**Fig. 8.3** Applied "RICE"-principle

at synapses and by increasing nerve conduction latency to promote analgesia [20, 26]. Additionally ice spray can be applied especially for pain relief. The direct contact between the applied ice and the skin has to be avoided in order to beware of blistering or necrosis of the skin.

- **Compression.** Elastic compression bandages should limit edema and swelling. Swelling may develop slowly hours after the injury event, so in view of an imminent compartment syndrom that can occur in context of fractures or soft tissue damages, the applied pressure has to be checked regularly.
- **Elevation:** Raising the lower limb upside should reduce perfusion and the accumulation of interstitial fluid by reduction of intravascular hydrostatic pressure.

**Table 8.3** Reviews – evidence for RICE principle

Author	Journal	Clinical question	Literature search	Main outcome measures	Conclusions
Van den Bekerom et al. (2012), Netherlands [20]	<i>Journal of Athletic Training</i>	Effectiveness of applying rest, ice, compression, and elevation (RICE) therapy start within 72 h after trauma for patients in the initial period after ankle sprain	Data Sources: MEDLINE (1966–2010), Cochrane Clinical Trial Register (1988–2010), CINAHL (1988–2010) and EMBASE (1988–2010). Inclusion of <b>11 trials</b> involving <b>868 patients</b>	Pain, swelling, ankle mobility or range of motion, return to sports, return to work, complications and patient satisfaction	Insufficient evidence is available from randomized controlled trials to determine the relative effectiveness of RICE therapy for acute ankle sprains in adults
Collins et al. (2007), Ireland [21]	<i>Emergency Medicine Journal</i>	Evidence to support an improvement in clinical outcome following the use of ice or cryotherapy	Data Sources: Medline 1966–2006 (using the MeSH system), EMBASE 1988–2006, the Cochrane Library, Google Scholar and citation tracking. Including <b>six human and four animal trials</b>	Reduction in pain and swelling or edema, improved function, return to participation in normal activity	Cryotherapy may have a possible benefit in the treatment of acute soft tissue injury if it is instituted soon after the injury The volume and quality of data are inadequate
Hubbard et al. (2004), USA [22]	<i>Journal of Athletic Training</i>	What is the clinical evidence base for cryotherapy use?	Data Sources: MEDLINE, Proquest, ISI Web of Science, Cumulative Index to Nursing and Allied Health (CINAHL) on Ovid, Allied and Complementary Medicine Database (AMED) on Ovid, Cochrane Database <b>Inclusion of 22 trials</b>	Function (subjective or objective), pain, swelling, or range of motion	Cryotherapy seems to be effective in decreasing pain. The low methodologic quality of the available evidence is of concern. More high-quality studies are required
Bleakley et al. (2004) Ireland [23]	<i>American Journal of Sports Medicine</i>	To explore the clinical evidence base for cryotherapy	Human studies up to 2002 Multiple databases, hand searches and citation tracking. <b>22 studies found</b>	Pain, swelling, range of motion and function	Many more high quality studies are needed to ensure adequate evidence-based practice

In addition to the physical treatment nonsteroidal anti-inflammatory drugs (NSAIDs) are attributed with reducing pain and decreasing the initial inflammatory process. The indication of NSAIDs has to be decided seriously and adverse drug reactions have to be respected.

Even if most common injuries can be treated with RICE protocol, severe injuries demand a special treatment that should be applied immediately:

**Dislocations** A dislocated ankle joint has to be relocated in order to reduce the risk of neurovascular or articular cartilage damages. In general, side-line treatment of dislocated joints should only be attempted by experienced practitioners. The neurovascular status has to be checked both before and after relocation. A referral to the medical centre, as soon as possible, is needed in case of present or impending neurovascular injuries [15]. Referring to irreducible dislocations, repetitive frustrating attempts should be

refrained. Instead of that, transportation to a medical centre is necessary for open relocation by surgical treatment.

**Fractures** Bone fractures have to be treated conscientiously in order to minimize the risk of soft tissue damages as well as surrounding neurovascular structures by splintered bone ends and to prevent becoming an open fracture [27]. As a result, major displaced bone fractures should be relocated in advance by application of in line longitudinal traction in order to improve realignment. The neurovascular status has to be checked also. Directly after, the concerned fractured bone and its adjacent joints have to be immobilized with a splint or vacuum beanbag in order to ensure the reached relocated position [27, 28]. Any change in neurovascular status requires an urgent transfer to hospital and an early surgical treatment [14, 27].

In view of the majority of the existing injury, an additional diagnosis algorithm (radiographic examination using



**Fig. 8.4** Taping of a first metatarsophalangeal joint in case of plantar plate injury

ultrasound, X-ray, CT or MRI) has to be attempted directly in a medical centre/local hospital or can be planned for the next days after further orthopaedic re-examination. The medical coverage practitioner is often confronted with the question of obtaining radiographs following an injury. The decision for further radiographs has to be made based on physical examination. Especially for foot and ankle injuries

the Ottawa Foot and Ankle Rules have been validated and implemented and help to decide if a further radiography is needed [29, 30] (Table 8.1).

## Judicial Situation

Although the medical care is outsourced on sports fields, legal obligations have to be respected. The duty of diligence towards the athletes is the same as in daily clinical practice. As a consequence, the initial assessment and treatment of an injured athlete and the decision whether a return to sports is possible or not, is insufficient -it is the physician's duty to care for the thorough medical support. If he is not able to complete the medical treatment by himself, he has to send the injured athlete to hospital or a medical centre. Just as in medical practice, the medical treatment has to be documented [31]. This concerns the history, the examination findings and the treatment including applied drugs and if the athlete doesn't want to keep to the physicians advice. Moreover, the athlete has to be provided with clear information about the diagnosis, possible complications and aftercare requirements. Attention should also be paid to the media attention. In this way it is the physician's duty to ensure that medical matters are confidentially upheld and only employees with the appropriated authorization in respect to the athletes' interests should be included.

## Evidence

Author	Clinical question	Level of Evidence
Van den Bekerom et al. (2012), Netherlands [20]	Effectiveness of RICE principle	Level I
Collins et al. (2007), Ireland [21]	Evidence of ice or cryotherapy	Level I
Hubbard et al. (2004), USA [22]	Evidence base for cryotherapy	Level I
Bleakley et al. (2004) Ireland [23]	Evidence base for cryotherapy	Level I
Ekstrand et al. (2006), Sweden [8]	Risk of Injury in elite football	Level II

## Summary

- Medical coverage has to be planned and arranged conscientiously in advance to an upcoming competition.
- The sports physician has to acquire the sports specific knowledge and skills.
- A standardized algorithm of assessing the injured mechanism, history and physical examination should be followed.
- The Primary therapy target is to reduce pain, swelling and bleeding and in this way to limit the initial inflammatory process and to prevent further damage.
- Every athlete with their injuries has to be treated on an individual basis.
- Legal obligations have to be respected also on sports field.



## References

- Wascher DC, Bulthuis L. Extremity trauma: field management of sports injuries. *Curr Rev Musculoskelet Med*. 2014;7(4):387–93.
- Junge A, Engebretsen L, Alonso JM, Renström P, Mountjoy M, Aubry M, Dvorak J. Injury surveillance in multi-sport events: the International Olympic Committee approach. *Br J Sports Med*. 2008;42(6):413–21.
- Engebretsen L, Soligard T, Steffen K, Alonso JM, Aubry M, Budgett R, Dvorak J, Jegathesan M, Meeuwisse WH, Mountjoy M, Palmer-Green D, Vanhegan I, Renström PA. Sports injuries and illnesses during the London Summer Olympic Games 2012. *Br J Sports Med*. 2013;47(7):407–14.
- Junge A, Engebretsen L, Mountjoy ML, Alonso JM, Renström PA, Aubry MJ, Dvorak J. Sports injuries during the Summer Olympic Games 2008. *Am J Sports Med*. 2009;37(11):2165–72. doi:10.1177/0363546509339357. Epub 2009 Sep 25.
- Alonso JM, Edouard P, Fischetto G, Adams B, Depiesse F, Mountjoy M. Determination of future prevention strategies in elite track and field: analysis of Daegu 2011 IAAF Championships injuries and illnesses surveillance. *Br J Sports Med*. 2012;46(7):505–14.
- Alonso JM, Tscholl PM, Engebretsen L, et al. Occurrence of injuries and illnesses during the 2009 IAAF World Athletics Championships. *Br J Sports Med*. 2010;44:1100–5.
- Alonso JM, Junge A, Renstrom P, et al. Sports injuries surveillance during the 2007 IAAF World Athletics Championships. *Clin J Sport Med*. 2009;19:26–32.
- Ekstrand J, Timpka T, Hägglund M. Risk of Injury in elite football played on artificial turf versus natural grass: a prospective two cohort study. *Br J Sports Med*. 2006;40:975–80.
- Buettner CM. The team physician's bag. *Clin Sports Med*. 1998;17(2):365–73.
- Krutsch W, Voss A, Gerling S, Grechenig S, Nerlich M, Angele P. First aid on field management in youth football. *Arch Orthop Trauma Surg*. 2014;134(9):1301–9.
- Dvorak J, Kramer EB, Schmied CM, Drezner JA, Zideman D, Patricios J, Correia L, Pedrinelli A, Mandelbaum B. The FIFA medical emergency bag and FIFA 11 steps to prevent sudden cardiac death: setting a global standard and promoting consistent football field emergency care. *Br J Sports Med*. 2013;47(18):1199–202.
- Miltner O, Glaser C, Greitemann B, Grim C, Leumann A, Weisskopf L, Krüger-Franke M. Ankle joint Instabilit. On-field management. GOTS Expert Meeting Ankle Instability 2012. [www.gots.org](http://www.gots.org).
- Flinn SD. On-field management of emergent and urgent extremity conditions. *Curr Sports Med Rep*. 2006;5(5):227–32.
- Schupp CM. Sideline evaluation and treatment of bone and joint injury. *Curr Sports Med Rep*. 2009;8:119–24.
- Skelley NW, McCormick JJ, Smith MV. In-game management of common joint dislocations. *Sports Health*. 2014;6:246–55. A review of criteria and methods for common joint dislocation reduction.
- Perryman JR, Hershman EB. The acute management of soft tissue injuries of the knee. *Orthop Clin North Am*. 2002;33(3):575–85.
- Bleakley CM, O'Connor S, Tully MA, Roche LG, Macauley DC, McDonough SM. The PRICE study (Protection Rest Ice Compression Elevation): design of a randomised controlled trial comparing standard versus cryokinetic ice applications in the management of acute ankle sprain [ISRCTN13903946]. *BMC Musculoskelet Disord*. 2007;8:125.
- Järvinen TA, Järvinen M, Kalimo H. Regeneration of injured skeletal muscle after the injury. *Muscles Ligaments Tendons J*. 2014;3(4):337–45. eCollection 2013 Oct.
- Hotfiel T, Carl HD, Jendrissek A, Swoboda B, Barg A, Engelhardt M. Turf toe injury—extension sprain of the first metatarsophalangeal joint. *Sportverletz Sportschaden*. 2014;28(3):139–45.
- van den Bekerom MP, Struijs PA, Blankevoort L, Welling L, van Dijk CN, Kerkhoffs GM. What is the evidence for rest, ice, compression, and elevation therapy in the treatment of ankle sprains in adults? *J Athl Train*. 2012;47(4):435–43. doi:10.4085/1062-6050-47.4.14.
- Collins NC. Is ice right? Does cryotherapy improve outcome for acute soft tissue injury? *Emerg Med J*. 2008;25(2):65–8. doi:10.1136/emj.2007.051664.
- Hubbard TJ, Denegar CR. Does cryotherapy improve outcomes with soft tissue injury? *J Athl Train*. 2004;39(3):278–9.
- Bleakley C, McDonough S, MacAuley D. The use of ice in the treatment of acute soft tissue injury: a systematic review of randomized controlled trials. *Am J Sports Med*. 2004;34:251–61.
- Thorsson O. Cold therapy of athletic injuries. *Current literature review*. *Lakartidningen*. 2001;98(13):1512–3.
- Swenson C, Swärd L, Karlsson J. Cryotherapy in sports medicine. *Scand J Med Sci Sports*. 1996;6(4):193–200.
- Kerr KM, Daley L, Booth L, Stark J. PRICE guidelines: guidelines for the management of soft tissue (musculoskeletal) injury with protection, rest, ice, compression, elevation (PRICE) during the first 72 hours (ACPSM). *ACPOM*. 1998;6:10–1.
- Hutchinson M, Tansey J. Sideline management of fractures. *Curr Sports Med Rep*. 2003;2:125–35.
- Honsik K, Boyd A, Rubin AL. Sideline splinting, bracing, and casting of extremity injuries. *Curr Sports Med Rep*. 2003;2:147–54.
- Gould SJ, Cardone DA, Muniyak J, Underwood PJ, Gould SA. Sideline coverage: when to get radiographs? A review of clinical decision tools. *Sports Health*. 2014;6(3):274–8. doi:10.1177/1941738114529701.
- Stiell IG, Greenberg GH, McKnight RD, Nair RC, McDowell I, Worthington JR. A study to develop clinical decision rules for the use of radiography in acute ankle injuries. *Ann Emerg Med*. 1992;21:384–90.
- Miltner O. Notfallbehandlung, juristische Aspekte. In: Siebert CH, Breuer CH, Krüger ST, Miltner O, editors. *Tipps & Tricks für den Sportmediziner*. Springer; 2004. p. 223–4.

Thomas Schlemmer, Thomas Hügler,  
Victor Valderrabano, and Jochen Paul

### Abstract

Recent literature proves a distinct correlation between sporting activity and the development of osteoarthritis (OA). In a disease affecting millions of people worldwide this is an important and ever growing field of interest. Sports has the potential to be a positive but also negative impact factor in OA development, depending on the intensity and the level of professionalism of sporting activity. Due to the importance of muscular stability in the development of OA this is also a reasonable approach for the treatment of already established OA in the elderly.

In many different types of sports, professional athletes show higher rates of radiographic OA signs, however, clinically these patients complain of not more, sometimes even less pain than the average population with similar signs of OA. Concerning foot and ankle some sports lead to premature OA like soccer, rock climbing and beach volleyball. Joint involving trauma is an important risk factor, especially in professional athletes.

In contrast to the development of OA through sports, in the elderly with established OA some kind of sports seem to have at least short time positive effects on pain and function.

In conclusion, sports can either influence the development of OA (depending on the intensity and the kind of sports) or show positive clinical effects in cases with present OA. Sports must consequently be subdivided into different categories and carefully analyzed to determine if positive or negative effects exist regarding OA.

### Keywords

Osteoarthritis • Sports • Treatment • Prevention • Risk factors • Pathomechanism of OA

## Introduction

Osteoarthritis (OA) is a disease involving joint degeneration with severe socioeconomic consequences. There is a correlation between age and the development of OA [1].

T. Schlemmer (✉) • T. Hügler  
Orthopaedic Department, University Hospital Basel,  
Basel, Switzerland  
e-mail: [thomas.schlemmer@usb.ch](mailto:thomas.schlemmer@usb.ch); [Thomas.huegle@unibas.ch](mailto:Thomas.huegle@unibas.ch)

V. Valderrabano, MD, PhD  
Orthopaedic Department, SWISS ORTHO CENTER,  
Schmerzklinik Basel, Swiss Medical Network, Basel, Switzerland  
e-mail: [vvalderrabano@gsmn.ch](mailto:vvalderrabano@gsmn.ch)

J. Paul, MD  
Rennbahnklinik, Kriegackerstrasse 100 CH-4132 Muttenz,  
Basel, Switzerland  
e-mail: [jochen.paul@rennbahnklinik.ch](mailto:jochen.paul@rennbahnklinik.ch)

Demographic changes in our society lead to a higher percentage of elderly people and so an increasing number of patients with OA. Because up to now no cure for OA is known, the prophylaxis of the disease is of particular importance. Sports could be a very easy and cost effective way to prevent the onset and/or modify the already existing clinical symptoms of OA, depending on the intensity and kind of sports.

But the question whether sports has an impact on OA, negative or positive, is an issue that has yet to be resolved.

Already in 1989 Buckwalter and Lane discussed whether there is a connection between sports and OA [2]. They found that moderate exercise by middle aged and older people does not increase their risk of developing osteoarthritis [2].

In this overview we show which kind of sports increase the risk for the development of OA and which lower the risk. We also give an advice regarding the proper kinds of sports to decrease pain and increase mobility.

## Epidemiology and the Influence of Sports on Osteoarthritis

In 1987 Felson et al. found in the population of the “framingham osteoarthritis study” a prevalence of OA increasing with age [1]. 27 % of the patients younger than 70 years had radiological signs of OA (Kellgren Lawrence stage 2 or higher), 44 % of the patients older than 80 years. A higher prevalence was seen in women (34 %) than in men (31 %) [1]. In a systematic review in 2011 Pereira et al. showed that in knee OA more women are affected than men, in hip and hand OA no significant gender specific differences were found [3]. Horvát et al. found in the Hungarian population hip OA in 16.49 % and knee OA in 16.54 % [4]. In Austria prevalence of OA is, including all joints, 11.9 % in men and 18.6 % in women [5].

In France the prevalence of hip and knee OA was 1.9 % and 4.7 % for men and 2.5 % and 6.6 % for women, respectively [6]. The overall prevalence of knee OA in rural china is 13.8 %, more women are affected than men [7]. In the northeastern population of china symptomatic knee OA was found in 16.05 % [8].

In conclusion there is quite a big difference in the literature concerning the prevalence of OA in different countries, even between Central European countries with similar living and working conditions. This makes a comparison of sporting people and the general population difficult due to the lack of reliable consistent data. This might be due to different recruiting methods, different age of participants or different methods of analysis and definition of OA.

Lawrence et al. reported an increase of OA prevalence of hand, knee and hip joints from 21 million patients in 1995 to 27 million in 2005 in the United States of America [9]. This increase might have been caused by the higher mean age of our society and increasing rate of obesity [9]. In the future a further increase of OA is likely. Turkiewicz et al. predict a raise of OA prevalence from 26.6 to 29.5 % (any location), from 13.8 to 15.7 % at the knee and 5.8 to 6.9 % at the hip until the year 2032 in Swedish population [10]. Concerning foot and ankle various sports have the potential to cause OA, especially soccer, beach volleyball and rock climbing [11–14]. Nevertheless there are not many studies available dealing with this topic and exact numbers are frequently missing. The different kind of sports are described in detail in the paragraph “Sports as a risk factor of Osteoarthritis” of this chapter.

## Pathomechanism of Osteoarthritis

Different risk factors independent from sports or trauma are well known for the development of OA including obesity, gender, genetics, hypermobility, crystal deposition or joint specific factors like axis deviation and of course age [15, 16]. The pathogenesis of OA is complex and usually multifactorial. Both biomechanical and biological factors can trigger OA with the result of cartilage loss, and subchondral bone remodelling. Conventional radiography detects osteosclerosis, joint space narrowing and osteophytes which usually occurs at a later stage of OA. Earlier, MRI shows cartilage loss and bone marrow edema.

In the animal knee model, subchondral bone remodeling occurs as early as four weeks after meniscectomy [17]. Synovitis is regularly encountered in osteoarthritic joints. Both synovial fluid and intraarticular adipose tissue develop a proinflammatory cytokine environment [18]. In patients with knee OA, synovial proliferation correlates with radiological and clinical progression [19]. The pathogenesis of posttraumatic OA depends on the type of sport. High impact sports injuries often lead to direct cartilage damage with subsequent bone bruise. Global gene expression in chondrocytes typically is activated after trauma, which leads to an increased expression of inflammatory cytokines cartilage-degrading proteinases reactive oxygen species (ROS) [20]. More frequently than high impact injuries, sport injuries lead to ligament injuries such as ankle instability or also ACL rupture with subsequent joint instability. Joint instability per se is associated with cartilage damage [21]. Joint instability is of notable importance in the pathogenesis of ankle OA as the ankle is a highly congruent and exposed to high mechanical load (Table 9.1) [23].

In the model of ACL injury, inflammatory biomarkers along with increased collagen turnover are observed at higher levels in synovial fluid compared to the non-affected knee [24]. This indicates that low grade inflammation is substantially involved in the pathogenesis of posttraumatic OA. Subchondral bone remodelling includes an increased porosity of the underlying bone which leads to interaction between subchondral trabeculae, bone marrow cells and cartilage [25]. The contact with inflammatory synovial fluid and bone marrow also occurs in subchondral bone lesions. The result of this is bone resorption with bone marrow edema, angiogenesis and increased osteoblast activity shown by SPECT-CT imaging [26]. Uncoupled subchondral bone formation is typically observed in OA. This is characterized by an initial reduction of bone density [17] due to increased osteoclast activity. Subsequently neoangiogenesis along with woven bone formation occurs, resulting in a thickening of the subchondral plate. TGF-beta expression seems to be of paramount importance in OA-induced bone formation [27]. The strong involvement of osteoclasts might explain why

**Table 9.1** Etiology of ankle osteoarthritis

Etiology group	Distribution		
	Number of ankles (number of patients)	Percentage	Number of male/females <sup>a</sup>
Posttraumatic osteoarthritis	318 (313)	78	157/161
Malleolar fracture (AO fracture type 44)	157 (157)	39	63/94
Ankle ligament lesions	65 (60)	16	44/21
Tibial plafond fracture (AO fracture type 43)	58 (58)	14	29/29
Tibial shaft fracture (AO fracture type 42)	20 (20)	5	10/10
Talus fracture	9 (9)	2	5/4
Severe combined fracture	9 (9)	2	6/3
Secondary osteoarthritis	52 (46)	13	27/25
Rheumatoid	22 (19)	5	5/17
Hemochromatosis	11 (9)	3	8/3
Hemophilia	6 (5)	1	6/10
Clubfoot	4 (4)	1	2/2
Avascular talus necrosis	3 (3)	1	3/0
Osteochondrosis dissecans	3 (3)	1	1/2
Postinfectious arthritis	3 (3)	1	2/1
Primary osteoarthritis	36 (31)	9	25/11
Total	406 (390)	100	209/197

From Valderrabano et al. [22], with kind permission from Springer Science and Business Media

<sup>a</sup>Association between gender and etiology: chi square (13)=38.979,  $p < 0.001$

bisphosphonates or strontium ralenate have shown success in the treatment of OA [28]. Inflammatory cells, notably in form of macrophages are observed in the synovial tissue but also in the subchondral bone (unpublished data). Thus, the induction of inflammation as early osteoarthritic event e.g. by overuse or mechanical instability seems to be actively involved in the pathogenesis of OA. Likewise, in endurance sports such as marathon runners, MRI before and directly after the race shows in the knee model joint effusion and meniscal impairment but no cartilage damage or subchondral bone edema [29]. Unlike in long distance runners, subchondral bone remodeling rather occurs after continuous mechanical impairment or overuse and is associated with meniscal damage [30]. Similarly, cartilage damage is not necessarily the first sign of OA. In early knee OA, delayed gadolinium-enhanced MRI of cartilage (dGEMRIC) shows that after 12 months a decrease of proteoglycan content actually is associated with an increase in cartilage thickness in the medial compartment. This shows that OA is a bi- or multiphasic rather than a linear disease with a plethora of biomechanical and biological factors leading to inflammation, cartilage damage and subchondral bone remodeling.

## Muscles and Osteoarthritis

Muscles play an important role in joint biomechanics and impaired joint biomechanics may lead to OA [31]. Several studies have investigated the effect of muscular weakness on

joint degeneration [32–36]. Most of the studies deal with OA at the knee and strength of the quadriceps muscle. Herzog et al. showed that even a short period of muscular weakness for 4 weeks may be a risk factor for developing OA in a rabbit model [37]. Valderrabano et al. showed that reduced calf circumference and reduced mean electromyography frequencies in lower leg muscles are directly related to OA at the ankle joint, which is hypothesized by the pathomechanisms of arthrogenic muscle inhibition and disuse muscle atrophy [38]. A follow up study by Wiewiorski et al. with magnetic resonance imaging (MRI) analysis of lower leg muscle cross sectional size in patients with unilateral OA at the ankle showed that the atrophy was mainly found in the soleus muscle but all muscles of the lower leg showed fatty degeneration compared to the healthy side [39].

The connection between muscular weakness and even muscular atrophy and OA is often reported in current literature. This might be due to different reasons, like pain and accompanied physical immobilisation with decrease of activities, but might also be age related sarcopenia. Genetics and gender might also play a role [1, 16, 40, 41].

Muscular weakness and atrophy have been reported to occur even before OA becomes symptomatic [33]. Regardless of the cause, muscle weakness and atrophy is related to OA and the question is if muscular exercise can prevent the onset, and muscular reconditioning can stop the progression of OA. A further question of interest is to determine which kinds of sports have a negative and which kinds have a positive influence on OA.

## Sports as a Risk Factor of Osteoarthritis

Sports can have a positive or negative effect on the development or progression of OA at the joints of the lower extremity. Concerning the ankle joint not much literature can be found. Most of the studies deal with OA at the knee and hip joint. Especially endurance running, soccer and track and field have been investigated concerning the risk of development of premature OA at the lower extremity joints. It is important to point out that there are significant differences between performing sports at an amateur or professional level.

In soccer several studies showed that the risk of OA at the knee and hip is up to three times higher in professional athletes than in amateurs [42, 43]. Comparing amateur soccer players to non-sporting control groups there can be seen a higher prevalence of OA in amateur soccer players, but the difference is not significant [42, 43]. Injury plays an important role in the risk of development of OA as mentioned above. Soccer ankle with anterior osteophytes can be seen as a pre-osteoarthritic condition, similar to hip impingement. Iosifidis et al. found that former elite soccer player have a higher risk of ankle OA compared to the general population, clinically as well as radiologically [12]. Similar result are reported by Gouttebauge et al. who found ankle OA in 12–17 % in former elite soccer players [13]. Armenis et al. also found a higher prevalence of ankle OA in former soccer players in X-rays, but they did not complain about more pain than the persons in the matched control groups [44].

In former Volleyball Players a high prevalence of ankle OA can be found due to recurrent ligamentous injuries. Gross et al. found a prevalence of 86.4 % in former elite volleyball players, compared to 10.5 % in the control group [11].

In long distance running no signs of premature OA at the ankle, as well as at the hip and knee joints, was found by Konradsen et al. [45].

Roos et al. showed that the prevalence of knee OA in not injured non elite players was 3 % compared to a rate of 13 % of OA in the non-elite player with an injured knee, mostly meniscal lesions and ACL ruptures [43]. Nevertheless in professional athletes without a severe knee injury in their career radiological signs of OA were more frequent (11 %). In conclusion in non-elite players with no reported trauma the prevalence of knee OA is not significantly higher than in the control groups, but elite players show a higher rate of knee OA despite no trauma [43]. The reason might be a high incidence of unrecognized microtraumata in the more competitive elite than in amateur athletes.

Elleuch et al. and Armenis et al. showed that despite of more radiological degeneration at the joints of the lower extremity in professional soccer players they do not suffer from more pain [44, 46]. The reason is not proven yet, a possible explanation could be the better muscular

stabilization in professional athletes because of more frequent sporting activity even after finishing their professional career.

Despite no negative long term effects are proven yet, there seems to be the need of joints to adapt to the stress of endurance running. In an MRI study Hohmann et al. showed the effect of marathon running on hips of runners who are not used to endurance sports [47]. After the race 6 of the 7 unexperienced runners had a joint effusion, compared to none of experienced runners. So there seems to be an adaptation to the strain which occurs at the hip joints in long distance running. The exact mechanism of adaptation is unclear [47]. Krampfl et al. also showed that there are no negative effects in experienced recreational sportsmen in MRI scans of the knee before and after competing in a marathon race [48].

In athletes taking part in track and field sports a big variation of OA prevalence is found due to the different movement patterns and different amount of stress on the lower extremity joints [49–51].

Overall there is a higher prevalence of OA at the hip and knee joints compared to non-sporting control groups, but as in soccer the former athletes do not suffer from more pain or impaired function in the activities of daily life [49, 51]. Concerning the ankle limited data is available. Schmitt et al. found an equal risk of ankle OA in former elite high jumpers compared to a non-sporting, matched control group [50].

In rock climbing Schöffl et al. found a high incidence of hallux rigidus and valgus [14], assumably due to the increased stress load to the forefoot. Hallux valgus can be found in 34 % of all climbers, compared to 4.5 % in the age matched general population, concerning Hallux rigidus no exact numbers are available.

In general, sports as a recreational activity does not seem to be a risk factor for the development of premature OA, but professional sports and trauma have a negative influence. The affected joints are hip, knee, ankle, shoulder and elbow. With trauma as an acknowledged risk factor for the development of OA this might be a possible explanation due to unrecognized microtraumata which are likely to occur more often in elite athletes.

Nevertheless, despite of a higher rate of radiological OA in joints elite athletes over all do not suffer from more limitations in the daily life than the average population. This supports the theory that there is a positive influence of joint stabilization by muscular strength on pain, stiffness and function on arthritic joints.

Compared to elite athletes with higher rates of radiological OA, in the amateur level with assumable less competition und less injuries the rates of OA are similar to the general community but the athletes report from less symptoms. In conclusion sports does not seem to be a risk factor when performed at an amateur level.

## Sports as Prevention and Treatment for Osteoarthritis

The idea of treating OA by sporting activity relies on the theory of muscular weakness in the elderly (sarcopenia) and the influence on the joint with decreased stability and as a consequence pain and rapid progression of OA. Due to limited physical potential caused by higher age and possible comorbidities in the elderly not every kind of sports is suitable.

In conclusion of this, and the assumption that so called “low impact sports” like swimming, cycling and walking have a positive effect on established OA in most studies that with these kinds of sports (Table 9.2).

In stationary cycling a benefit can be found in gait speed, pain, stiffness and quality of life [52]. The intensity of the cycling exercises does not seem to have an influence on the amount of the positive effect on OA [53].

Training in water has the benefit that a longer duration of exercise sessions is possible due to less pain caused by less weight on the joints, especially in obese patients. Pain relieve was reported up to 100 % compared to land based exercises [54], highest pain relieve was found directly after the training sessions [55]. In a Cochrane review Bartels et al. shows that pain relieve is the most important and significant factor in water based over land based exercise [56]. In functional results there is a discrepancy between different studies, so no clear evidence can be found [56]. Because of longer training sessions an additional effect on the cardiovascular fitness level might be a positive side effect.

Furthermore it is easier to keep balance in the water which might be an important factor in the elderly.

Yoga and Tai Chi are sports suitable for patients of every age due to a high variation of exercise types. Compared to conventional physiotherapy Ebnezar et al. found significant better results in yoga concerning resting pain and morning stiffness in patients with OA at the knees [57]. Similar results were found by Kolasinski et al. [58] and Cheung et al. [59]. Cheung et al. points out the importance of guided sessions to provide frequent exercising [59].

In Tai Chi same results were found concerning the effect at the knee [60–62]. No studies dealing with OA at the ankle are available.

In general, no long term effects of sports as a treatment of OA are known, maximum follow up periods in the mentioned literature was 20 weeks. No adverse effects were seen in these therapies.

### Level of Evidence

Most of the studies dealing with different kind of sports are prognostic evidence Level II studies. Some important statements of this chapter are mentioned here:

- In soccer players a higher rate of osteophytes are seen compared to the general population but not more pain or restriction in daily life [44]. (Prognostic Level II)
- In beach volleyball frequent ligament trauma can lead to higher rates of OA at the ankle joint [11]. (Prognostic Level II)
- Rock climbing can lead to OA at the forefoot due to increased stress load [14]. (Prognostic Level II)
- Long distance running does not lead to premature OA at the lower limb, even when participated over decades [45]. (Prognostic Level II)

### Summary

- Despite of higher rates of radiologically diagnosed OA, athletes do not suffer from more impairment due to OA in daily life compared to non-sporting control groups; this might be due to a higher muscular stability, which seems to be an important factor in the development, prevention and cure of OA.
- Risk factors for the development of OA due to sports is a professional career and trauma involving joints.
- Moderate sports at an amateur level can have the potential to prevent and treat OA in a cost effective and easily accessible way with little negative side effects.
- Some sports like for example soccer, beach volleyball and rock climbing who involve either a high risk of injury or a high stress level to the ankle and forefoot have a high risk for development of OA.

**Table 9.2** Treatment of OA by sports with effect on pain, function and their evidence level

Sports	Pain	Function	Evidence level
Cycling	→	↑	II
Water exercise	↓	→	II-III
Yoga	↓	↑	II
Tai Chi	↓	↑	II

### References

1. Felson DT, Naimark A, Anderson J, Kazis L, Castelli W, Meenan RF. The prevalence of knee osteoarthritis in the elderly. The Framingham Osteoarthritis Study. *Arthritis Rheum.* 1987;30(8): 914–8.
2. Buckwalter JA, Lane NE. Does participation in sports cause osteoarthritis. *Iowa Orthop J.* 1997;17:80–9.

3. Pereira D, Peleteiro B, Araújo J, Branco J, Santos RA, Ramos E. The effect of osteoarthritis definition on prevalence and incidence estimates: a systematic review. *Osteoarthritis Cartilage*. 2011;19(11):1270–85.
4. Horváth G, Koroknai G, Ács B, Than P, Bellei Á, Illés T. Prevalence of radiographic primary hip and knee osteoarthritis in a representative Central European population. *Int Orthop*. 2011;35(7):971–5.
5. Dorner TE, Stein KV. Prevalence and status quo of osteoarthritis in Austria. Analysis of epidemiological and social determinants of health in a representative cross-sectional survey. *Wien Med Wochenschr*. 2013;163(9–10):206–11.
6. Guillemin F, Rat AC, Mazieres B, Pouchot J, Fautrel B, Euller-Ziegler L, Fardellone P, Morvan J, Roux CH, Verrouil E, Saraux A, Coste J; 3000 Osteoarthritis group. Prevalence of symptomatic hip and knee osteoarthritis: a two-phase population-based survey. *Osteoarthritis Cartilage*. 2011;19(11):1314–22.
7. Zhang J, Song L, Liu G, Zhang A, Dong H, Liu Z, Li X, Luo J. Risk factors for and prevalence of knee osteoarthritis in the rural areas of Shanxi Province, North China: a COPCORD study. *Rheumatol Int*. 2013;33(11):2783–8.
8. Jiang L, Rong J, Zhang Q, Hu F, Zhang S, Li X, Zhao Y, Tao T. Prevalence and associated factors of knee osteoarthritis in a community-based population in Heilongjiang, Northeast China. *Rheumatol Int*. 2012;32(5):1189–95.
9. Lawrence RC, Felson DT, Helmick CG, Arnold LM, Choi H, Deyo RA, Gabriel S, Hirsch R, Hochberg MC, Hunder GG, Jordan JM, Katz JN, Kremers HM, Wolfe F; National Arthritis Data Workgroup. Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. Part II. *Arthritis Rheum*. 2008;58(1):26–35.
10. Turkiewicz A, Petersson IF, Björk J, Hawker G, Dahlberg LE, Lohmander LS, Englund M. Current and future impact of osteoarthritis on health care: a population-based study with projections to year 2032. *Osteoarthritis Cartilage*. 2014. pii: S1063-4584(14)01188-1.
11. Gross P, Marti B. Risk of degenerative ankle joint disease in volleyball players: study of former elite athletes. *Int J Sports Med*. 1999;20(1):58–63.
12. Iosifidis MI, Tsarouhas A, Fylaktou A. Lower limb clinical and radiographic osteoarthritis in former elite male athletes. *Knee Surg Sports Traumatol Arthrosc*. 2015;23(9):2528–35.
13. Gouttebauge V, Inklaar H, Frings-Dresen MH. Risk and consequences of osteoarthritis after a professional football career: a systematic review of the recent literature. *J Sports Med Phys Fitness*. 2014;54(4):494–504.
14. Schöffl V, Küpper T. Feet injuries in rock climbers. *World J Orthop*. 2013;18, 4(4):218–28.
15. Neogi T, Zhang Y. Epidemiology of osteoarthritis. *Rheum Dis Clin North Am*. 2013;39(1):1–19.
16. Srikanth VK, Fryer JL, Zhai G, Winzenberg TM, Hosmer D, Jones G. A meta-analysis of sex differences prevalence, incidence and severity of osteoarthritis. *Osteoarthritis Cartilage*. 2005;13(9):769–81.v.
17. Anetzberger H, Mayer A, Glaser C, Lorenz S, Birkenmaier C, Müller-Gerbl M. Meniscectomy leads to early changes in the mineralization distribution of subchondral bone plate. *Knee Surg Sports Traumatol Arthrosc*. 2014;22(1):112–9.
18. Klein-Wieringa IR, Kloppenburg M, Bastiaansen-Jenniskens YM, Yusuf E, Kwekkeboom JC, El-Bannoudi H, Nelissen RG, Zuurmond A, Stojanovic-Susic V, Van Osch GJ, Toes RE, Ioan-Facsinay A. The infrapatellar fat pad of patients with osteoarthritis has an inflammatory phenotype. *Ann Rheum Dis*. 2011;70(5):851–7.
19. Krasnokutsky S, Belitskaya-Lévy I, Bencardino J, Samuels J, Attur M, Regatte R, Rosenthal P, Greenberg J, Schweitzer M, Abramson SB, Rybak L. Quantitative magnetic resonance imaging evidence of synovial proliferation is associated with radiographic severity of knee osteoarthritis. *Arthritis Rheum*. 2011;63(10):2983–91.
20. Ding L, Heying E, Nicholson N, Stroud NJ, Homandberg GA, Buckwalter JA, Guo D, Martin JA. Mechanical impact induces cartilage degradation via mitogen activated protein kinases. *Osteoarthritis Cartilage*. 2010;18(11):1509–17.
21. Golditz T, Steib S, Pfeifer K, Uder M, Gelse K, Janka R, Hennig FF, Welsch GH. Functional ankle instability as a risk factor for osteoarthritis: using T2-mapping to analyze early cartilage degeneration in the ankle joint of young athletes. *Osteoarthritis Cartilage*. 2014. pii: S1063-4584(14)01067-X.
22. Valderrabano V, Horisberger M, Russell I, Dougall H, Hintermann B. Etiology of ankle osteoarthritis. *Clin Orthop Relat Res*. 2009;467(7):1800–6.
23. Valderrabano V, Hintermann B, Horisberger M, Fung TS. Ligamentous posttraumatic ankle osteoarthritis. *Am J Sports Med*. 2006;34(4):612–20.
24. Catterall JB, Stabler TV, Flannery CR, Kraus VB. Changes in serum and synovial fluid biomarkers after acute injury. *Arthritis Res Ther*. 2010;12(6):R229.
25. Botter SM, van Osch GJ, Clockaerts S, Waarsing JH, Weinans H, van Leeuwen JP. Osteoarthritis induction leads to early and temporal subchondral plate porosity in the tibial plateau of mice: an in vivo microfocal computed tomography study. *Arthritis Rheum*. 2011;63(9):2690–9.
26. Paul J. et al. Increased osseous (99m)Tc-DPD uptake in end-stage ankle osteoarthritis: correlation between SPECT-CT imaging and histologic findings. *Foot Ankle Int*. 2015;36(12):1438–47.
27. Zhen G, Wen C, Jia X, Li Y, Crane JL, Mears SC, Askin FB, Frassica FJ, Chang W, Yao J, Carrino JA, Cosgarea A, Artemov D, Chen Q, Zhao Z, Zhou X, Riley L, Sponseller P, Wan M, Lu WW, Cao X. Inhibition of TGF- $\beta$  signaling in mesenchymal stem cells of subchondral bone attenuates osteoarthritis. *Nat Med*. 2013;19(6):704–12.
28. Reginster JY, Badurski J, Bellamy N, Bensen W, Chapurlat R, Chevalier X, Christiansen C, Genant H, Navarro F, Nasonov E, Sambrook PN, Spector TD, Cooper C. Efficacy and safety of strontium ranelate in the treatment of knee osteoarthritis: results of a double-blind, randomised placebo-controlled trial. *Ann Rheum Dis*. 2013;72(2):179–86.
29. Schueller-Weidekamm C, Schueller G, Uffmann M, Bader TR. Does marathon running cause acute lesions of the knee? Evaluation with magnetic resonance imaging. *Eur Radiol*. 2006;16(10):2179–85. Epub 2006 Mar 10.
30. Englund M, Guermazi A, Roemer FW, Yang M, Zhang Y, Nevitt MC, Lynch JA, Lewis CE, Torner J, Felson DT. Meniscal pathology on MRI increases the risk for both incident and enlarging subchondral bone marrow lesions of the knee: the MOST Study. *Ann Rheum Dis*. 2010;69(10):1796–802.
31. Egloff C, Hügle T, Valderrabano V. Biomechanics and pathomechanisms of osteoarthritis. *Swiss Med Wkly*. 2012;142:w13583.
32. Ikeda S, Tsumura H, Torisu T. Age-related quadriceps-dominant muscle atrophy and incident radiographic knee osteoarthritis. *J Orthop Sci*. 2005;10(2):121–6.
33. Slemenda C, Brandt KD, Heilman DK, Mazucca S, Braunstein EM, Katz BP, Wolinsky FD. Quadriceps weakness and osteoarthritis of the knee. *Ann Intern Med*. 1997;127(2):97–104.
34. Slemenda C, Heilman DK, Brandt KD, Katz BP, Mazucca SA, Braunstein EM, Byrd D. Reduced quadriceps strength relative to body weight: a risk factor for knee osteoarthritis in women? *Arthritis Rheum*. 1998;41(11):1951–9.
35. Amaro A, Amado F, Duarte JA, Appell HJ. Gluteus medius muscle atrophy is related to contralateral and ipsilateral hip joint osteoarthritis. *Int J Sports Med*. 2007;28(12):1035–9. Epub 2007 May 29.
36. O'Reilly SC, Jones A, Muir KR, Doherty M. Quadriceps weakness in knee osteoarthritis: the effect on pain and disability. *Ann Rheum Dis*. 1998;57(10):588–94.

37. Herzog W, Longino D. The role of muscles in joint degeneration and osteoarthritis. *J Biomech.* 2007;(40 Suppl 1):S54–63. Epub 2007 Apr 16.
38. Valderrabano V, von Tschanner V, Nigg BM, Hintermann B, Goepfert B, Fung TS, Frank CB, Herzog W. Lower leg muscle atrophy in ankle osteoarthritis. *J Orthop Res.* 2006;24(12):2159–69.
39. Wiewiorski M, Dopke K, Steiger C, Valderrabano V. Muscular atrophy of the lower leg in unilateral post traumatic osteoarthritis of the ankle joint. *Int Orthop.* 2012;36(10):2079–85.
40. Rodriguez-Fontenla C, Gonzalez A. Genetics of osteoarthritis. *Reumatol Clin.* 2014. pii: S1699-258X(14)00124-7.
41. Panoutoupoulou K, Zeggini E. Advances in osteoarthritis genetics. *J Med Genet.* 2013;50(11):715–24.
42. Lindberg H, Roos H, Gärdsell P. Prevalence of coxarthrosis in former soccer players. 286 players compared with matched controls. *Acta Orthop Scand.* 1993;64(2):165–7.
43. Roos H, Lindberg H, Gärdsell P, Lohmander LS, Wingstrand H. The prevalence of gonarthrosis and its relation to meniscectomy in former soccer players. *Am J Sports Med.* 1994;22(2):219–22.
44. Armenis E, Pefanis N, Tsiganos G, Karagounis P, Baltopoulos P. Osteoarthritis of the ankle and foot complex in former Greek soccer players. *Foot Ankle Spec.* 2011;4(6):338–43.
45. Konradsen L, Hansen EM, Søndergaard L. Long distance running and osteoarthrosis. *Am J Sports Med.* 1990;18(4):379–81.
46. Elleuch MH, Guerhazi M, Mezghanni M, Ghroubi S, Fki H, Mefteh S, Baklouti S, Sellami S. Knee osteoarthritis in 50 former top-level soccer players: a comparative study. *Ann Readapt Med Phys.* 2008;51(3):174–8.
47. Hohmann E, Wörtler K, Imhoff A. Osteoarthritis from long-distance running? *Sportverletz Sportschaden.* 2005;19(2):89–93.
48. Krampla W, Mayrhofer R, Malcher J, Kristen KH, Urban M, Hruby W. MR imaging of the knee in marathon runners before and after competition. *Skeletal Radiol.* 2001;30(2):72–6.
49. Schmitt H, Brocai DR, Lukoschek M. High prevalence of hip arthrosis in former elite javelin throwers and high jumpers: 41 athletes examined more than 10 years after retirement from competitive sports. *Acta Orthop Scand.* 2004;75(1):34–9.
50. Schmitt H, Lemke JM, Brocai DR, Parsch D. Degenerative changes in the ankle in former elite high jumpers. *Clin J Sport Med.* 2003;13(1):6–10.
51. Vingård E, Sandmark H, Alfredsson L. Musculoskeletal disorders in former athletes. A cohort study in 114 track and field champions. *Acta Orthop Scand.* 1995;66(3):289–91.
52. Salacinski AJ, Krohn K, Lewis SF, Holland ML, Ireland K, Marchetti G. The effects of group cycling on gait and pain-related disability in individuals with mild-to-moderate knee osteoarthritis: a randomized controlled trial. *J Orthop Sports Phys Ther.* 2012;42(12):985–95.
53. Mangione KK, McCully K, Gloviak A, Lefebvre I, Hofmann M, Craik R. The effects of high-intensity and low-intensity cycle ergometry in older adults with knee osteoarthritis. *J Gerontol A Biol Sci Med Sci.* 1999;54(4):M184–90.
54. Roper JA, Bressel E, Tillman MD. Acute aquatic treadmill exercise improves gait and pain in people with knee osteoarthritis. *Arch Phys Med Rehabil.* 2013;94(3):419–25.
55. Gill SD, McBurney H, Schulz DL. Land-based versus pool-based exercise for people awaiting joint replacement surgery of the hip or knee: results of a randomized controlled trial. *Arch Phys Med Rehabil.* 2009;90(3):388–94.
56. Bartels EM, Lund H, Hagen KB, Dagfinrud H, Christensen R, Danneskiold-Samsøe B. Aquatic exercise for the treatment of knee and hip osteoarthritis. *Cochrane Database Syst Rev.* 2007;(4):CD005523.
57. Ebnezar J, Nagarathna R, Yogitha B, Nagendra HR. Effect of integrated yoga therapy on pain, morning stiffness and anxiety in osteoarthritis of the knee joint: a randomized control study. *Int J Yoga.* 2012;5(1):28–36.
58. Kolasinski SL, Garfinkel M, Tsai AG, Matz W, Van Dyke A, Schumacher HR. Iyengar yoga for treating symptoms of osteoarthritis of the knees: a pilot study. *J Altern Complement Med.* 2005;11(4):689–93.
59. Cheung C, Wyman JF, Resnick B, Savik K. Yoga for managing knee osteoarthritis in older women: a pilot randomized controlled trial. *BMC Complement Altern Med.* 2014;14:160.
60. Wang C, Schmid CH, Hibberd PL, Kalish R, Roubenoff R, Rones R, McAlindon T. Tai Chi is effective in treating knee osteoarthritis: a randomized controlled trial. *Arthritis Rheum.* 2009;61(11):1545–53.
61. Yan JH, Gu WJ, Sun J, Zhang WX, Li BW, Pan L. Efficacy of Tai Chi on pain, stiffness and function in patients with osteoarthritis: a meta-analysis. *PLoS One.* 2013;8(4):e61672.
62. Song R, Lee EO, Lam P, Bae SC. Effects of tai chi exercise on pain, balance, muscle strength, and perceived difficulties in physical functioning in older women with osteoarthritis: a randomized clinical trial. *J Rheumatol.* 2003;30(9):2039–44.



Bernhard Speth and Carlo Camathias

**Abstract**

Children and adolescents are becoming more involved in sports at earlier ages and with higher levels of intensity. Sports-related foot and ankle problems are the second most common musculoskeletal problem in children and adolescents next to acute injury. Due to the changing biomechanic properties of muscles, tendons, and bones during growth and maturation the injuries and their treatment in children are different from those in adults. This chapter gives an overview of common sports-related foot injuries in this age group. It illustrates common acute injuries, overuse injuries, and growth-related problems as well as their prevention.

**Keywords**

Foot • Injury • Children • Pediatric • Overuse Injuries • Ankle Sprains • Transitional Fractures • Osteonecrosis • Apophysitis • Prevention

**Introduction/Epidemiology**

Physical exercise is a crucial factor towards a healthy development at young age. Children and adolescents are becoming more involved in sports at earlier ages and with higher levels of intensity. Trainers, athletes, and parents place increasing demands on the growing body. Sports-related foot and ankle problems are the second most common musculoskeletal problem in children and adolescents next to acute injury [1, 2]. At the age 11–18 sports is the most common cause of an injury. In a systematic review, the ankle was the most injured site in 24 of 70 included sports [2]. The incidence of ankle and foot injuries is highest in court games such as Football, Soccer, Volleyball, Handball, Basketball and racket sports.

Many of these injuries originate from an isolated trauma whereas overuse injuries are seen as a consequence of repetitive microtraumas. The developing body passes through different phases of growth and maturation. During that time there is a higher susceptibility to injury due to stress on the physes, the apophyses, and the growing cartilage.

B. Speth (✉) • C. Camathias  
Department of Pediatric Orthopedic Surgery, University Children's Hospital Basel (UKBB), Spitalstrasse 33, Basel 4031, Switzerland  
e-mail: [Bernhard.Speth@ukbb.ch](mailto:Bernhard.Speth@ukbb.ch); [Carlo.Camathias@ukbb.ch](mailto:Carlo.Camathias@ukbb.ch)

Growth and maturation also alter the biomechanic properties of muscles, tendons, and bones. Therefore, they have a potential influence on the dynamic interplay between muscle and bones. This interplay also affects the musculoskeletal balance needed for complex motor skills in certain sports and potentially leads to injury and overuse.

The health-promoting benefit of sports depends on a proper balance between stress and regeneration which should be considered in training and competition.

The treatment of many sports-related injuries of the pediatric foot such as ankle sprains, anterior ankle impingement, tendinitis and plantar fasciitis correspond to that of adults. These issues are dealt with in the corresponding chapters of this book. The problems that are specific to the pediatric foot are covered in this chapter.

**Injury Patterns in the Young Athlete**

Injuries of the foot and ankle can be subdivided in acute injuries, growth-related problems and overuse injuries. Within this differentiation these injuries may be assigned to a particular anatomical location in the ankle, hindfoot, midfoot or forefoot (Table 10.1).

**Table 10.1** Common Ankle and Foot Injuries of the pediatric athlete

	Ankle	Hindfoot	Midfoot	Forefoot
Acute injuries	Sprains Fractures Tillaux fractures Triplane fractures Osteochondritis dissecans	Calcaneal fractures Talus fractures	Lisfranc Injury	Fifth metatarsal avulsion fracture Jones fracture
Growth-related problems	Medial malleolus ossification center	Talocalcaneal coalition Os trigonum	Calcaneonavicular coalition Accessory navicular	
Overuse injuries	Anterior ankle impingement	Calcaneal apophysitis Plantar fasciitis Tendinitis	Navicular stress fracture Osteonecrosis of the navicular	Apophysitis of the fifth metatarsal Metatarsal stress fractures

Intrinsic and extrinsic factors need to be known when dealing with these injuries and its prevention. Intrinsic factors include anatomical considerations, gender, development and growth [2]. The growing skeleton is susceptible to injury due to growth cartilage at 3 locations: the epiphyseal plate, the joint surface, and the apophysis [3]. Growing cartilage and open physes are vulnerable to stress, children may not have the complex motor skills needed for certain sports until the puberty.

The extrinsic factors include training errors, shoe wear and equipment, running surface, long distance, and speed running [4, 5].

## Acute Injuries

### Ankle Sprains

Ankle sprains are the most common injury in a pediatric-orthopedic emergency department. Compared to adults less often the ankle sprain results in a ligamentous injury whereas bony avulsions and injuries to the physes are more common in children. When the Ottawa Ankle Rules are applied unnecessary exposure to x-rays can be avoided [6]. According to these guidelines, an x-ray of the ankle is indicated for the patient that (1) feels bone tenderness in the malleolar zone and the posterior edge of the tibia/fibula, or at the tip of the malleoli, or (2) could not weight bear four steps after the injury, and in the emergency department. According to these rules bone tenderness at the navicular or the base of the fifth metatarsal indicates an x-ray of the foot.

The radiographic interpretation of the growing skeleton requires specialized knowledge which allows differentiating between physiologic and pathologic anatomy.

After exclusion of a fracture, conventional treatment is the preferable treatment strategy [7, 8]. The initial treatment includes the early use of RICE (rest, ice, compression, elevation). In addition to early motion and mobility, maintaining

the range of motion, and using an ankle support are recommended. In cases of complete ligamentous tears, chronic instability, and syndesmotic injury a surgical intervention may be required.

## Fractures

There is a multitude of anatomic variations in the growing foot that make it difficult to distinguish between a pathologic and a physiologic finding. A thorough understanding of the relevant anatomy is essential towards the diagnosis and treatment of these fractures. Accessory sesamoids/ossicles (Fig. 10.1) and the physes may be mistaken for a fracture [9]. Common ossicles mistaken for avulsion fractures are: Os tibiale externum (the proximal medial aspect of the navicular), Os peroneum (lateral border of the cuboid), Os trigonum (posterior aspect of the talus), Os vesalium (base of the fifth metatarsal. Accessory Navicular/Os tibiale externum. The accessory navicular or Os tibiale externum is one of the most common causes for medial foot pain in the growing athlete. The incidence is reported in a range of 5–10%.

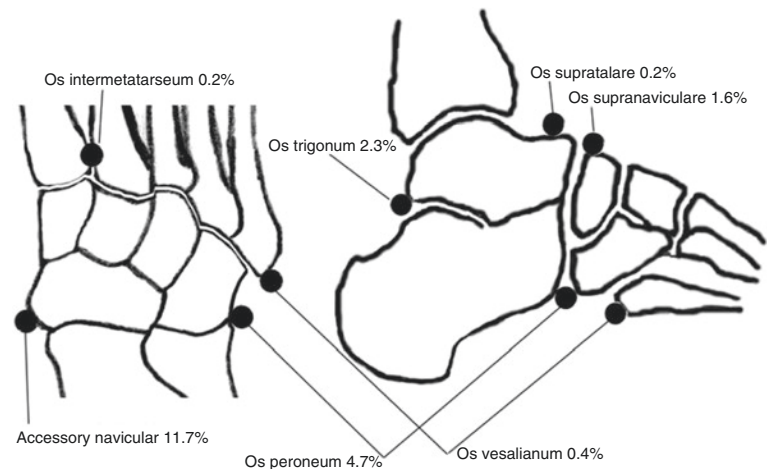
The growing bone shows different biomechanic properties than adult bone. These differences help to explain the differences between adult and pediatric fractures. The immature bone has a higher healing potential and a greater remodeling capacity. This potential is the basis for the larger number of conservative treatments in children compared to adults.

The strength of ligaments and tendons compared to the strength of bone is relatively higher than in adults. This explains why in children tendons and ligaments tend to show avulsions of their osseous, chondral, or periosteal insertions rather than ligament tears.

An apparent difference in the developing foot is the presence of growth zones. The epiphyseal plates show a diminished resistance towards shear, and bending moments.

Most pediatric fractures heal without permanent deformity. Potential complications include those seen with adult

**Fig. 10.1** Schematic illustration showing the accessory ossicles of the ankle and the foot region and their incidence [9] (Reprinted with kind permission from Springer Science and Business Media: Coskun et al. [9])



fractures. However, a small percentage is complicated by growth arrest and subsequent deformity that result from physal damage.

In children, the mechanism of trauma is often unknown. Therefore, using classifications based on the mechanism of trauma such as the Lauge-Hanson classification can not be applied to children. In fractures with physal involvement the Salter-Harris and Aitken classifications are the most common. At the level of the ankle, avulsion fractures can be distinguished from physal fractures.

## Distal Tibial Transitional Fractures

### Two/Triplane Fracture

In the transition to skeletal maturity, there are short periods during which children are susceptible to fractures involving the closing distal tibial physis. An external rotating force may lead to Triplane and Tillaux fractures. These fractures are too complex to be classified with the abovementioned classification systems. The triplane fracture is composed of two, three, or four parts, and with fracture lines in three different planes (Fig. 10.2).

- Two planes: one plane in the epiphysis, the other in the physis
- Triplane I: three planes, like a Two-plane with an additional third plane in the metaphysis
- Triplane II: three planes, like Triplane I with additionally continuation of the metaphyseal fracture through the physis, and epiphysis (intraarticular fracture)

### Tillaux Fracture

Tillaux fractures appear as Salter-Harris III fractures of the distal tibia involving the tibial epiphysis. These fractures may occur in adolescents (age 12–14 years) in the period during the closure of the distal tibial epiphysis. The closing

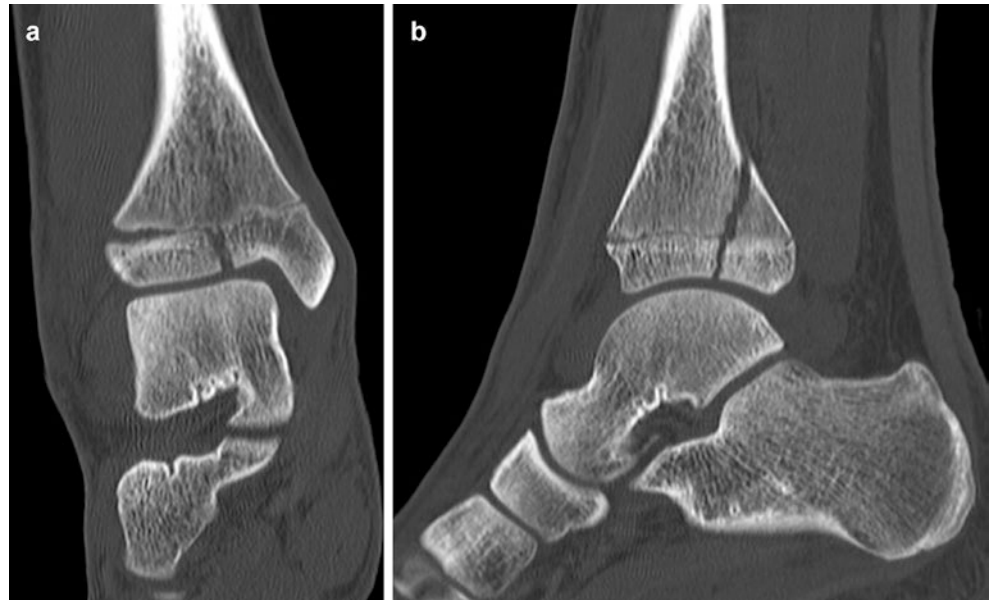
of the tibial epiphysis follows a predictable pattern with the anterolateral physis closing last. The trauma mechanism usually is a forced external rotation of the foot or an internal rotation of the leg on the fixed foot. This maneuver leads to a bony avulsion of the anterior inferior tibiofibular ligament.

Recognizing transitional fractures is of great importance because they involve a major weight-bearing articular surface. A residual deformity in the articular surface can lead to premature degenerative arthritis. In addition to standard a.p. and lateral radiographs internal oblique projection (Mortise view) and CT aid in the optimal assessment of these fractures and to determine the need for closed vs. open treatment. The aim of the therapy is to establish normal joint congruity. A residual deformity a potentially causes early degenerative arthritis. In most cases closed reduction is sufficient to restore congruity of the joint. In those cases where a displacement of >2 mm of the articular surface remains, open reduction is usually required to adequately restore the articular surface [10].

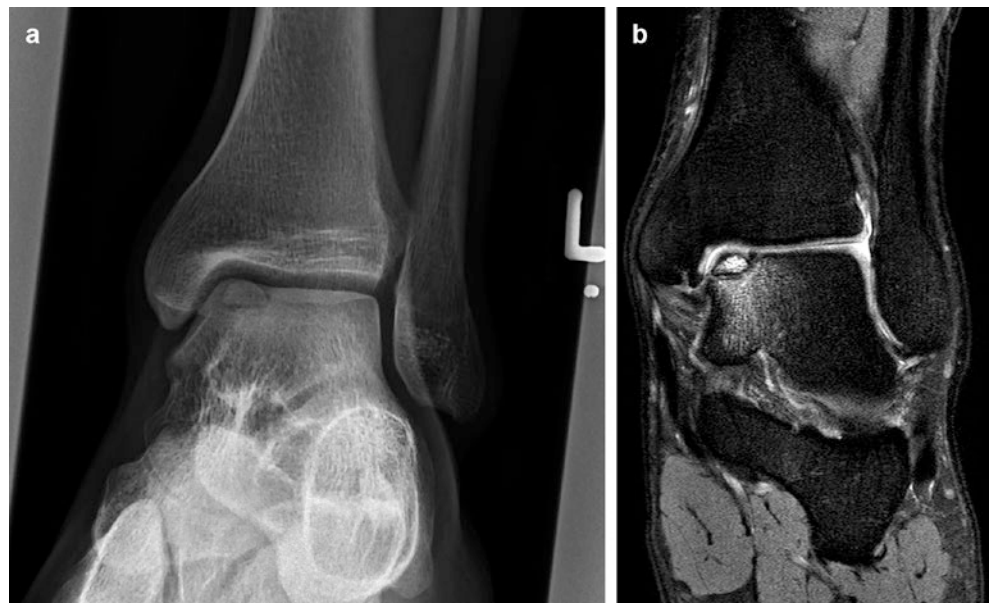
## Osteochondritis Dissecans of the Talus

Osteochondritis dissecans of the talus (OCD), i.e. a osteochondral lesion of the talus, is analogous to that found in other anatomic locations. It is characterized by necrotic bone underlying articular cartilage. OCD may be the sequelae of a traumatic incident (eversion/inversion) or be associated with chronic pain, hind-/midfoot malalignment, and ankle instability. Pain, swelling, instability and a history of repetitive strains are signs and symptoms related to OCD. Blocking of the joint as well as limitation of the range of motions can also occur. Sometimes these lesions not easy to see on conventional x-rays, and can only be visualized in additional MRIs. This modality sometimes aids to judge the

**Fig. 10.2** Triplane II-Fracture in a 14-year old patient who sustained a contusion of the ankle during football. The coronal plane of the CT-scan (a) shows two and the sagittal plane (b) the third plane of the complex fracture



**Fig. 10.3** Osteochondritis dissecans in a 16-year old patient after repetitive ankle trauma in skateboarding. (a) Conventional x-rays clearly show the osseous part of the lesion. (b) MRI offers additional information on the integrity of the cartilage and vitality of underlying bone



integrity of the articular cartilage, the extent, and possible displacement of the lesion (Fig. 10.3).

An OCD can be classified based on the system described by Berndt and Harty [11]: Type I lesions are non-displaced. Type II lesions are partially detached. Type III lesions are detached but not displaced. Type IV lesions are detached and displaced or rotated.

The treatment of an OCD is discussed controversially. Non-operative treatment has been recommended as the treatment of choice in children. For Type I lesions are immobilized for 4–8 weeks followed by modified activity and protected

weight bearing for 2–3 months; corrective insoles may follow. In OCD > Type II, or if there is no symptomatic and radiographic improvement surgery may be indicated. Because of the potential instability of the osteochondral fragment drilling alone in most cases seems not be sufficient. Additionally, the fragment should be stabilized arthroscopically or by an arthrotomy with screw fixation, sometimes even through a malleolar osteotomy. The treatment of OCD can be very tedious. During the treatment, sports are prohibited. The patient may benefit from temporary casting.

## Metatarsal Fractures

The incidence of metatarsal physeal fractures is about 2% [12]. The mechanism of injury may be either direct or indirect. Single metatarsal fractures are usually minimally displaced, so that conservative treatment can be applied. Multiple metatarsal fractures potentially lead to instability which requires temporary K-wire fixation. Before their treatment compartment syndrome of the foot should be ruled out. In children older than ten years fractures of the base of the fifth metatarsal are very common. In contrast to apophyseal fractures the fracture in these cases is perpendicular to the metatarsal bone, and only requires operative treatment in cases with a high degree of displacement.

## Phalangeal Fractures

In the majority of phalangeal fractures, the hallux is affected. Transphyseal fractures of the base of the first phalanx and compression fractures are rare. Closed fractures rarely require reduction. Diaphyseal fractures typically can be treated conservatively with buddy-taping and weight bearing as tolerated. Fractures involving the articular surface (gap >2 mm) or transphyseal fractures with marked displacement are associated with a higher risk of growth arrest, angular deformity, and early arthritis. In these cases closed reduction and pinning should be considered.

---

## Overuse Injuries/Growth-Related Problems

Most overuse injuries in the young athlete are caused by the overuse of the muscle-tendon unit. During the growth spurt, the long bones outgrow the muscle-tendon units, which is thought to place excess stress on the tendon–bone junction [4, 13–15]. Open physes and muscle inflexibility predispose for overuse injuries. Longitudinal bone growth is faster than muscle and soft tissue growth. This may lead to an increased pull of tendons on the apophyses. Tendinitis, bursitis, apophysitis, plantar fasciitis and stress fractures are typical overuse injuries in the foot of young athletes.

Most of these injuries can be managed conservatively. Their treatment is similar to those in adults and is covered in the corresponding chapters.

## Apophysitis of the Calcaneus (Sever's Disease)

Sever's Disease is one of the most common causes of heel pain in adolescent athletes. The condition has been reported with an incidence of 3.7 per 1000 patients, affecting children between 8 and 12 years of age [16]. Overuse with repeti-

tive microtraumas of the apophysis, the growth plate, and the calcaneus is held responsible for this overuse, condition. Furthermore, muscle inflexibility, heel cord tightness, and overpronation have been associated with calcaneal apophysitis. This growth-related injury shows bilateral involvement in 60% of the cases and usually limits itself at the end of growth [17].

Although the term “apophysitis” suggests an inflammatory process underlying the problem, an MRI study found bone bruising and structural changes within the trabecular bone of the metaphyseal region adjacent to the calcaneal apophysis. This change suggests that the apophysitis may be a metaphyseal trabecular stress fracture of the developing bone rather than an inflammatory process [18].

Patients usually present with activity-dependent heel pain at the posterior aspect of the calcaneus. The medial and lateral calcaneus may be painful on compression. Plain radiographs play a limited role in diagnosing a calcaneal apophysitis since they do not have a direct consequence on the treatment regimen [19].

Treatment is always conservative. Temporary immobilization, analgesics, restricted weight bearing, reduction of sports activity, and physiotherapy with emphasis on heel cord stretching are therapeutic measures that might be incorporated in the treatment. Viscoelastic heel pads for shock absorption and reduction of stress have been shown to be helpful [17].

## Stress Fractures

Stress fractures in athletes occur as a result mechanic overload. In these cases, a healthy bone is unable to withstand constant, submaximal, repetitive loads. A rapid increase in the duration and intensity of training predisposes the metatarsals to overload. The typical location for stress fractures in adults are the tibia (49%), followed by the hindfoot (25%), the metatarsals (9%), Femur (7%) and fibula (6%) [20]. In children stress fractures mainly occur in the proximal tibia or the metatarsals. The second metatarsal is most prone to overload followed by the first, third, fourth and fifth. Stress fractures are mainly located in the diaphysis or the metaphysis

Patients with stress fractures present with activity-related pain 2–3 weeks after a rapid increase of training. Conventional radiographs may not show any alterations in diaphyseal stress fractures up to 24 weeks. In many cases 2–3 weeks after the onset of symptoms a periosteal reaction may be seen radiographically. It is important to emphasize that a stress fracture in a child may resemble an infective or malignant condition [21]. In those cases where stress fractures cannot be safely distinguished from an infective or malignant condition, MRI or CT is recommended.

Treatment is conservatively in most cases and consists in the restriction from sports activities, anti-inflammatory medication, and immobilization with a stiff sole or cast. Since compliance may be restricted in very active young patients, treatment may be facilitated by immobilization in a cast for 4–6 weeks.

Re-injury is related to the development of chronic pain. Therefore, prevention plays a crucial role in the avoidance of these injuries. Chronic overuse injuries in young athletes may be related to limited recovery time from competitive sports and intense training [22]. Sports-specific motion sequences should be systematically trained in age-adapted exercises. At early age, basic training in varying sports activities should be propagated instead of specializing too early for a sport which might lead to an early selection towards high-level sports activity.

### Tarsal Coalition

A tarsal coalition is a fibrous, cartilaginous or osseous fusion between two tarsal bones (Fig. 10.4). In the majority of cases, the tarsal coalition is a congenital anomaly. However, tarsal coalition can also be acquired secondary to trauma. The overall incidence is about 1% [23]. The calcaneonavicular (53%) and the talocalcaneal coalition (37%) are the most common types followed by the talonavicular, calcaneocuboidal and naviculocuneiform types [24]. Besides these isolated forms, there are multiple forms which may be associated with complex syndromes such as Apert syndrome or longitudinal deformities for example. Not everybody with a coalition is symptomatic. However, it often becomes symptomatic in adolescent athletes. Recurrent ankle sprains not responding to conventional therapy should trigger the search for a tarsal coalition [4].

### Prevention

The prevention of pediatric sports injuries is as important as its treatment. Prevention means avoiding not only acute and overuse injuries but also avoiding adverse long-term effects of sports. The development of fundamental physical skills like running, jumping and landing depends both on maturation and practice [2, 25].

Athletes at a young age have different risk factors for injury than adults. Training should be seen in the context of the individual, age-dependent stages of physical and psychological development. Parents and coaches can reduce the risks for injuries and overuse by a critical selection of appropriate sporting events, by applying training patterns which are constructed to allow for regeneration and repair, by the supply of suitable sports gear and by promoting of fair play.



**Fig. 10.4** Talocalcaneal coalition in a 14-year old patient

### Evidence

The composition of this chapter is based on the best level of evidence available. As an entity its level of evidence is grade IV.

### Summary

- Growth and maturation alter the biomechanic properties of muscles, tendons, and bones. During that time there is a higher susceptibility to injury due to stress on the physes, the apophyses, and the growing cartilage.
- The radiographic interpretation of the growing skeleton requires special knowledge which allows to differentiate between physiologic and pathologic anatomy.
- In the young athlete, the strength of ligaments and tendons compared to the strength of bone is relatively higher than in adults. Tendons and ligaments tend to show avulsions

of their osseous, chondral, or periosteal insertions rather than ligament tears.

- In the transition to skeletal maturity, there are short periods during which children are susceptible to fractures involving the closing distal tibial physis. Recognizing transitional fractures is of great importance because they affect a major weight-bearing articular surface.
- The prevention of pediatric sports injuries is as important as its treatment. The health-promoting benefit of sports depends on a proper balance between stress and regeneration. This should be considered in training and competition.

## References

1. Omev ML, Micheli LJ. Foot and ankle problems in the young athlete. *Med Sci Sports Exerc.* 1999;31(7 Suppl):S470–86.
2. Fong DT-P, Hong Y, Chan L-K, Yung PS-H, Chan K-M. A systematic review on ankle injury and ankle sprain in sports. *Sports Med.* 2007;37(1):73–94.
3. Dalton SE. Overuse injuries in adolescent athletes. *Sports Med.* 1992;13(1):58–70.
4. Kennedy JG, Knowles B, Dolan M, Bohne W. Foot and ankle injuries in the adolescent runner. *Curr Opin Pediatr.* 2005;17(1):34–42.
5. Krivickas LS. Anatomical factors associated with overuse sports injuries. *Sports Med.* 1997;24(2):132–46.
6. Stiell IG, McKnight RD, Greenberg GH, McDowell I, Nair RC, Wells GA, et al. Implementation of the Ottawa ankle rules. *JAMA.* 1994;271(11):827–32.
7. Lynch SA, Renström PA. Treatment of acute lateral ankle ligament rupture in the athlete. *Sports Med.* 1999;27(1):61–71.
8. Herb CC, Hertel J. Current concepts on the pathophysiology and management of recurrent ankle sprains and chronic ankle instability. *Curr Phys Med Rehabil Rep.* 2014;2(1):25–34.
9. Coskun N, Yuksel M, Cevener M, Arican RY, Ozdemir H, Bircan O, et al. Incidence of accessory ossicles and sesamoid bones in the feet: a radiographic study of the Turkish subjects. *Surg Radiol Anat.* 2009;31(1):19–24.
10. Koury SI, Stone CK, Harrell G, La Charité DD. Recognition and management of Tillaux fractures in adolescents. *Pediatr Emerg Care.* 1999;15(1):37–9.
11. Berndt AL, Harty M. Transchondral fractures (osteochondritis dissecans) of the talus. *J Bone Joint Surg.* 1959;41(7):1363.
12. Mizuta T, Benson W, Foster B, Morris L. Statistical analysis of the incidence of physeal injuries. *J Pediatr Orthop.* 1987;7(5):518–23.
13. Adirim TA, Cheng TL. Overview of injuries in the young athlete. *Sports Med.* 2003;33(1):75–81.
14. Hogan KA, Gross RH. Overuse injuries in pediatric athletes. *Orthop Clin North Am.* 2003;34(3):405–15.
15. Chambers HG. Ankle and foot disorders in skeletally immature athletes. *Orthop Clin North Am.* 2003;34(3):445–59.
16. Wiegerinck JI, Yntema C, Brouwer HJ, Struijs PA. Incidence of calcaneal apophysitis in the general population. *Eur J Pediatr.* 2014;173(5):677–9.
17. Micheli LJ, Ireland ML. Prevention and management of calcaneal apophysitis in children: an overuse syndrome. *J Pediatr Orthop.* 1987;7(1):34–8.
18. Ogden JA, Ganey TM, Hill JD, Jaakkola JI. Sever's injury: a stress fracture of the immature calcaneal metaphysis. *J Pediatr Orthop.* 2004;24(5):488–92.
19. Rachel JN, Williams JB, Sawyer JR, Warner WC, Kelly DM. Is radiographic evaluation necessary in children with a clinical diagnosis of calcaneal apophysitis (sever disease)? *J Pediatr Orthop.* 2011;31(5):548–50.
20. Matheson GO, Clement DB, McKenzie DC, Taunton JE, Lloyd-Smith DR, MacIntyre JG. Stress fractures in athletes. A study of 320 cases. *Am J Sports Med.* 1987;15(1):46–58.
21. Devas M. Stress fractures in children. *J Bone Joint Surg Br.* 1963;45:528–41.
22. Emery CA. Risk factors for injury in child and adolescent sport: a systematic review of the literature. *Clin J Sport Med.* 2003;13(4):256–68.
23. Vaughan W, Segal G. Tarsal Coalition, with Special Reference to Roentgenographic Interpretation I. *Radiology.* 1953;60(6):855–63.
24. Stormont DM, Peterson HA. The relative incidence of tarsal coalition. *Clin Orthop Relat Res.* 1983;181:28–36.
25. Griffin LY. Common sports injuries of the foot and ankle seen in children and adolescents. *Orthop Clin North Am.* 1994;25(1):83–93.

Krit Prugsawan and Monika Horisberger

**Abstract**

Nowadays there is an increase in the number of people, including middle and elderly ages, who are interested in sport games. This is especially those who expect to maintain their active life. However a concern is whether aging inherently causes structural capacity and functional alteration in the human body. One assumption is aging probably make sport seniors more prone to certain types of injuries. Understanding the anatomical and functional change among seniors can make treatment and prevention of injury more effective.

The most common injuries in senior athletes are chronic overuse injuries that can reduce the flexibility and overall physical performance. Therefore, early diagnostics, conservative treatment, avoidance prolong immobilization and compensatory training programs will minimize disuse-associated loss of general fitness during treatment periods.

**Keywords**

Senior sports • Elderly athletes • Structural and functional alterations • Sport injuries • Chronic overuse injuries • Foot and ankle problems

**Introduction**

Due to higher life expectancy and improved health care the number of sports active elderly people in senior sports is increasing in developed countries and our society. This increasing activity of a large number of people is e.g. reflected in an ever increasing number of international tournaments in various sports disciplines which adapt to this new tendency and offer special competitions for elderly people, as e.g. the “National Senior Games Association” ([www.nsga.com](http://www.nsga.com)) in the USA. However, despite more and more elderly people in good health able to be active in many disciplines, aging inherently causes structural and functional alterations [1–6] in the human

body that might make elderly sports people more prone to certain types of injuries [5, 6]. Understanding the anatomical and functional change among seniors can support the effectiveness of treatment and prevention of injuries in order to maintain function and increase health and longevity in this population.

**Musculoskeletal Changes in Geriatrics****Skeletal Muscle**

Aging leads to morphological degeneration and declined function of skeletal muscle. After 30 years of age, the peak muscle strength is decreasing 15 % per decade, and 30 % per decade after 70. Decreasing numbers of motor neurons, increasing collagen content and fat infiltration and progressive muscle denervation leads to a decrease in muscle strength [7–9]. The precise mechanism of decreased muscle strength has been argued. Kaplan et al. [7] found equal loss of type I and type II fibers, but Green [10] and Larsson [11]

K. Prugsawan, MD (✉)  
Department of Orthopaedics, Vejthani Hospital,  
1 Ladprao Road 111, Bangkok, Bangkok 10240, Thailand  
e-mail: [krit.prugsawan@gmail.com](mailto:krit.prugsawan@gmail.com)

M. Horisberger, MD  
Orthopaedic Department, University of Basel, Basel, Switzerland  
e-mail: [monika.horisberger@usb.ch](mailto:monika.horisberger@usb.ch)



found mainly a selective loss of type II fibers. On the micro scale, sarcopenia, decreased mitochondrial volume, decreased fiber size, and loss of muscle flexibility has been shown [1].

It is unclear to what extent such age related alterations can be prevented, however, it has been shown that resistance training exercise can in part reverse the loss of function by increasing connective tissue production, capillary blood supply, biochemical, oxidative capacities of muscle fibres and decreasing shrinkage of type II muscle fibers [12–16].

### **Tendon and Ligaments**

Also for tendons and ligaments, significant age related alterations have been found. Increasing age led to a decrease in blood supply, glycosaminoglycan composition, fiber compliance and collagen fibre bundle morphology. Furthermore age-related decreasing in water content and increasing elastin fibril thickness contribute to connective-tissue stiffness [1]. This leads to abnormal stress and strain characteristics and might increase the vulnerability of tendons and ligaments in elderly people [17]. The flexibility and range of motion of joints can decrease, a situation that can be even aggravated by inactivity in elderly people [2, 18].

### **Bone Structure and Osteoporosis**

Typical morphological alterations of bone are associated with aging. They impose specific risks for sports active elderly people. Over the years, mineral density is decreasing. However, this process is highly dependent on hormonal changes and is gender-specific. After age 50, men lose approximately 0.4–0.75% of bone mass per year. In women bone loss already starts in their early 30s and accelerated in postmenopause to 2–3% per year [19]. This hormonally influenced bone loss in aging athletes might be even more pronounced in former elite athletes which often have a history of activity-related irregular menstruation, sometimes even secondary amenorrhoea and thus hormonal induced bone loss at a far earlier age than normal [1].

According to several studies, regular moderate sports activities in elderly people help to minimize age related bone loss and might thus help to prevent osteoporosis. Sports activities, in particular endurance sports have shown to increase thickness, strength and calcium concentration as well as nitrogen and hydroxyl proline levels in bone [3, 17, 20, 21] On the other hand, if osteoporotic elderly get engaged in sports, they might be at increased risk of osteoporotic fractures. Appropriate diagnostics and treatment of osteoporosis in elderly who wish to be active in sports is therefore recommended.

### **Articular Cartilage**

Aging is related to a decrease in chondroitin sulfate and less stability of cross-linkages collagen [7]. In case of lack of regular weight bearing, cartilage is subject of disuse atrophy and at last this might accelerate cartilage deterioration [22]. Since regular moderate mechanical loading is necessary to maintain cartilage health, sports activities in elderly might be beneficial for cartilage and even joint function. In contrast, repetitive high impact loading can result in cartilage micro-trauma and degeneration of the whole joint [1]. Furthermore repetitive mechanical loading can cause fatigue failure in chondrocyte and the cartilage matrix [23]. Horisberger et al. [24] shown that chondrocyte death was significantly increased in the loaded joint of the rabbit knee model. This leads to be one etiology for the onset of osteoarthritis.

### **Physical Performance in the Elderly**

Several studies in geriatric athletes demonstrated age-related decrease in physical performance [4, 25, 26]. Wright et al. [26] shown that physical performance decreased around 3.4% per years over 35 years of competition. Performance was decreased slowly from age 50–75 years and significantly after age 75 years. No difference in the decrease of the sprint and endurance was detected in men. Contrary to women, the decreasing in the sprint was greater than in endurance, especially after the age of 75. However, if excluding the bias of diseases, genetic variants, disuse and destructive lifestyle such as smoking, elderly people can keep their physical performance for a long time.

### **Sports Related Musculoskeletal Injury in the Elderly**

Around 36% of musculoskeletal problems in elderly athletes are related to the re-occurrence of orthopaedic problems that the athletes had suffered already previously. Mostly, these injuries happened during training and were related to fast movements. In walking and jogging between 14 and 57% of these injuries occurred and were all located in the lower extremities [27–29]. Moreover Kannus et al. [30] shown the incidence of the other sports injuries were cross-country skiing 19%, track and field sport 5%, gymnastics 5%, and tennis 5%. These study shown that time taken to visit clinic in elderly athletes were 67% in chronic phase (over 2 months), 21% in subacute phase (1 week-2 months) and 12% in acute phase (within 7 days).

### **Acute Traumatic Injuries**

In elderly athletes, acute traumatic injuries were less common than chronic overuse injuries [5, 30, 31].

The most common injuries were muscle strains [32–35] which are the typical result of strength exercises [36]. Due to loss of tissue flexibility and age related degeneration, the ability of muscles to absorb energy in response to eccentric contractions declines. Treatment should be initialised as early as possible. As first, it follows the general rules of treatment in soft tissue lesions (RICE: Rest, Ice Compression and Elevation). Then, it has to focus on avoiding prolonged immobilization in order to prevent further muscle atrophy. Physical therapy is helpful but generally has to be prescribed for a longer time period than in younger individuals [37]. Traumatic bony lesions, in particular fractures, are not common in elderly athletes despite age-related decrease in bone mass and strength. This might be because elderly athletes tend to be involved in less risky sports [30, 38].

### Chronic Overuse Injuries

The clinical symptoms of overuse injuries are non-specific. Kalimen et al. [32] found that more than on third of elderly male athletes with overuse injuries could not indicate a specific location of their symptoms. Females seem to be more prone to overuse injuries than male [27].

### Osteoarthritis

It is still controversial whether sports activity is detrimental to weightbearing joints. To date, it seems that moderate exercise such as walking, biking, skiing, rowing does not increase the risk for osteoarthritis. In contrast, moderate activity is believed to improve joint hemostasis and health in general [7, 22, 37–39]. This is true not just in healthy elderly people but also in patients with moderate osteoarthritis of load bearing joints [37]. More rigorous sports activities including heavy impact and torsional loading such as soccer, basketball, baseball and rugby might increase the risk for osteoarthritis on the longterm view [39–43]. However, it is not totally clear whether it really is the heavier load that leads to degeneration or the increased risk of joint injury. Apart from joint loading during sports activities, of course many other factors such as genetics [43, 44], previous joint surgery, joint instability [45, 46], abnormal joint alignment, neurological deficit, obesity, poor muscle quality [43, 47], shoes, playing surface, the level of competition and rate, intensity, frequency of impact loading joint can modify the individual risk for osteoarthritis in athletes [37].

### Stress Fracture

Stress fractures are typical overuse injuries of the foot. However, according to literature, their occurrence in elderly athletes seems to be little. Hulkko et al. [48] report on 368 stress fractures of which 5 cases were seen in athletes over 50 years of age. Due to osteoporosis, bone size, hormones, nutrition and biochemistry, women have a 1.5 to 12 times

increased risk for stress fractures compared to men [49, 50]. Given the much higher rate of osteoporosis in elderly women compared to men one would expect the majority of stress fractures in this age group to occur in women. However, no literature could be found on this. Vitamin D deficiency correlates with the incidence of stress fractures and should therefore be substituted in elderly athletes [51]. A careful training schedule with slow increase in intensity as well as vitamin D and Calcium supplementation in elderly sports active individuals help to prevent stress fractures. If symptoms occur that could be related to an incipient stress fracture, training should be discontinued and adequate imaging diagnostics should be initiated.

## Foot and Ankle Problems in Sport Active People

### Achilles Tendon Problems

Achilles tendon problems occur most often in sport active people and involved mostly in running sports. Some studies show that the annual incidence of Achilles tendon disorder is 7–9% in high-level runners [52–54]. And reported the most common problem in competitive athletes are paratenonitis, around two-thirds and insertional problems, one-fifth. The remaining problems are tendinopathies.

### Posterior Tibial Tendon Dysfunction (PTTD)

The epidemiological studies [55, 56] show that there is a higher prevalence of posterior tibial tendon dysfunction in women than men and it might increase with age. Gatzoulis et al. [57] reports that the prevalence of posterior tibial tendon dysfunction in women over the age of 40 was 3.3%.

Posterior tibial tendon dysfunction can cause medial hindfoot pain and swelling. Some patients may have lateral hindfoot pain, secondary to impingement of the calcaneus on the fibula [58]. Posterior tibial tendon dysfunction can decrease walking distance and impact on sport activity.

### Mortons Neuroma

Mortons neuroma is a disease involving the forefoot, which causes localized pain with tingling and numbness sensation in the intermetatarsal space. Most of the patients reported are middle-aged females. It can affect walking distance and sport activity.

### Stress Fracture

The prevalence of stress fracture from all sports injuries was 0.7–20% [59] and the highest incidence is in track and field athletes because of the repetitive loading [60]. The tibia was the most common site followed by the metatarsals and fibula. Stress fracture sites vary from types of sports. Tibial stress fractures mostly occur in distance runners, navicular stress fractures in track athletes and metatarsal stress fracture in

dancers [61]. The calcaneal stress fracture was found around 21 % and the most common site is posterior calcaneal tuberosity [62]. Stress fractures of cuneiform and cuboid bones are rare.

There is no exact data on sports active geriatric patients, however, the incidence is expected to be in such a geriatric cohort even higher than in young endurance sports people.

---

## Diagnosis and Treatment

Early diagnosis is very important for preventing long-lasting disability and prevent the injury turn chronic. But it tends to be that the aging athletes wait for the symptoms to resolve by and search for medical help late [6]. For physicians, a high index of suspicion helps to diagnose acute and chronic conditions related to the sports activities of their patients in a timely fashion and install appropriate therapy. The treatment of choice – if possible – is the conservative treatment. Important is to have a good plantigrade foot and ankle (treated e.g. by insoles treatment, good sports shoes), avoid muscular dysbalances (treated e.g. by physiotherapy), have a good bone quality status (treated e.g. by Vitamin D3 Ca<sup>2+</sup>), and a good nutritional condition. In the treatment of elderly people, prolonged immobilization should be avoided. Compensatory training programmes – sometimes under the guidance of specialised physical therapists can help to minimize disease-associated loss of general fitness during treatment periods. Treatment and counseling of elderly athletes also has to take into account non-orthopaedic comorbidities such as underlying cardiovascular and pulmonary disease.

---

## Prevention

Apart from medical prevention strategies such as Vitamin D3 and Calcium supplementation, a careful training schedule helps to prevent injuries. However, there is little evidence-based strategies. Training plans in elderly people should focus on proper warm up and cool down as well as a gradual moderate increase in training intensity paying attention to the increased fragility of aging tissue and a good gentle stretching program. Moderate exercises in elderly can help to improve tissue elasticity, cardiovascular fitness, muscle strength, proprioceptive capabilities and therefore prevent injuries.

---

## Evidence

As senior sports and sports injuries in the elderly are a young area of sports medicine, there is no great evidence in the literature so far.

## Summary

- Senior sports is an increasing sports activity in our society.
- Elderly athletes are different from young athletes, as their physiological aging process declines functional musculo-skeletal parameters, such as muscle, tendons, ligaments and bone strength.
- On one hand, these limitations have to be taken into account when setting up training schedules for elderly athletes and treating sports injuries in elderly people.
- On the other hand, moderate sports activity helps to maintain and to some extent restore musculoskeletal, cardio-pulmonary and many more body functions.
- Related to the foot and ankle there is few specific injuries and literature data is sparse. General diagnostic and treatment rules for foot and ankle injuries in elderly people are early diagnostics and if possible conservative treatment, avoiding if possible immobilization.

---

## References

1. Chen AL, Mears SC, Hawkins RJ. Orthopaedic care of the aging athlete. *J Am Acad Orthop Surg.* 2005;13(6):409–10.
2. Stauss RH. Sports medicine and physiology. Philadelphia: W.B. Saunders Co; 1991.
3. Sineft P. Aging and disease. *Aust Family Physician.* 1986;15(2): 123–7.
4. Donato AJ, Tench K, Glueck DH, Seals DR, Eskurza I, Tanaka H. Declines in physiological functional capacity with age: a longitudinal study in peak swimming performance. *J Appl Physiol.* 2003;94:764–9.
5. Mauri K, Alen M. Aging physical activity and sports injuries: an overview of common sports injuries in the elderly. *Sports Med.* 1995;20(1):41–52.
6. Menard D, Stanish WD. The aging athlete. *Am J Sports Med.* 1989;17:187–96.
7. Kaplan FS, Hayes WC, Keaveny TM, Boskey A, Einhorn TA, Iannotti JP. Form and function of bone. In: Simon SR, editor. *Orthopaedic basic science.* Rosemont: American Academy of Orthopaedic Surgeons; 1994. p. 127–84.
8. Frontera WR, Hughes VA, Lutz KJ. A cross sectional study of muscle strength and mass in 45–78 year old men and women. *J Appl Physiol.* 1991;71:644–50.
9. Overend TJ, Cunningham DA, Paterson DH. Thigh composition in young and elderly men determined by computed tomography. *Clin Physiol.* 1992;12:629–40.
10. Green HJ. Characteristics of aging human skeletal muscles. In: Sutton JR, Brock RM, editors. *Sports medicine for the mature athlete.* Indianapolis: Benchmark Press Inc; 1986. p. 17–26.
11. Larsson L, Sjodin B, Karlsson J. Histochemical and biochemical changes in human skeletal muscle with age in sedentary males, age 22–65 years. *Acta Physiol Scand.* 1978;103:31–9.
12. Beyer RE. Regulation of connective tissue metabolism in aging and exercise. A review frontiers in exercise biology. Champaign: Human Kinetics Publishers Inc; 1983. p. 85–99.
13. Fiatarone M, Marx E, Ryan N. High intensity strength training in nonagenarians: effects on skeletal muscle. *JAMA.* 1990;263:3029–39.

14. Ryan AS, Nicklas BJ. Age-related changes in fat deposition in mid thigh muscle in women: relationships with metabolic cardiovascular disease risk factors. *Int J Obes Relat Metab Disord*. 1999;23:126–32.
15. Coggan A, Spina RJ, King DS, et al. Skeletal muscle adaptations to endurance training in 60-70-yr-old men and women. *J Appl Physiol*. 1992;72:1780–6.
16. Orlander J, Ainansson A. Effects of physical training on skeletal muscle metabolism and ultrastructure in 70-75-year-old men. *Acta Physiol Scand*. 1971;109:149–54.
17. Booth FW, Gould EW. Effects of training and disuse on connective tissue. *Exer Sport Sci Rev*. 1975;3:83–112.
18. Nicholas JA, Friedman MJ. Orthopaedic problems in middle aged athletes. *Phys Sportsmed*. 1979;7:39–46.
19. Wilmore JH. The aging of bone and muscle. *Clin Sports Med*. 1991;10:231–44.
20. Beyer RE, Huang FC, Wilshire EB. The effect of endurance exercise on bone dimensions, collagen and calcium in the aged male rat. *Exp Gerontol*. 1985;20(6):315–23.
21. Twomey L, Taylor J. Age changes in lumbar intervertebral discs. *ACTA Orthop Scand*. 1985;56:496–9.
22. Hall MD. Cartilage changes after experimental relief of contact in the knee joint of the mature rat. *Clin Orthop*. 1969;64:64–76.
23. Clements K, Bee Z, Crossingham G, Adams M, Sharif M. How severe must repetitive loading be to kill chondrocytes in articular cartilage? *Osteoarthritis Cartil*. 2001;9:499–507.
24. Horisberger M, Fortuna R, Valderrabano V, Herzog W. Long-term repetitive mechanical loading of the knee joint by in vivo muscle stimulation accelerates cartilage degeneration and increases chondrocyte death in a rabbit model. *Clin Biomech*. 2013;28(5):536–43.
25. Tanaka H, Seals DR. Age and gender interactions in physiological functional capacity: insight from swimming performance. *J Appl Physiol*. 1997;82:846–51.
26. Wrigh VJ, Perricelli BC. Age-related rates of decline in performance among elite senior athletes. *Am J Sports Med*. 2008;36(3):447–8.
27. Carroll JF, Pollock ML, Graves JE, et al. Incidence of injury during moderate- and high-intensity walking training in the elderly. *J Gerontol*. 1992;47:M61–6.
28. Kilborn A, Hartley LH, Saltin B, et al. Physical training in sedentary middle-aged and older men. *Scand J Clin Lab Invest*. 1969;24:315–22.
29. Pollock ML, Carroll JF, Graves JE, et al. Injuries and adherence to walk/jog and resistance training programs in the elderly. *Med Sci Sports Exerc*. 1991;23:1194–200.
30. Kannus P, Niittymäki S, Järvinen M, et al. Sports injuries in elderly athletes: a three year prospective, controlled study. *Age Ageing*. 1989;4:263–70.
31. Marti B, Vader IP, Minder CE, et al. On the epidemiology of running injuries: the 1984 Bern Grand-Prix study. *Am J Sports Med*. 1988;16:285–94.
32. Kallinen M, Alen M. Sports-related injuries in elderly men still active in sports. *Br J Sports Med*. 1994;28:52–5.
33. Kavanagh T, Shephard RJ. The effects of continued training on the aging process. *Ann N Y Acad Sci*. 1977;301:656–70.
34. Korpi J, Haapanen A, Svahn T. Frequency, location, and types of orienteering injuries. *Scand J Sports Sci*. 1987;9:53–6.
35. Peterson M, Renström P. Sports medicine. *Läkärtidningen*. 1977;77:3613–30.
36. Jackson RW. The masters knee – past, present, and future. In: Sutton IR, Brock RM, editors. *Sports medicine for the mature athlete*. Indianapolis: Benchmark Press Inc; 1986. p. 319–28.
37. Pollock ML, Graves JE, Swart DL, et al. Exercise training and prescription for the elderly. *South Med J*. 1994;87:S88–95.
38. Dehaven KE, Littner DM. Athletic injuries: comparison by age, sport, and gender. *Am J Sports Med*. 1986;14:218–24.
39. Buckwalter JA, Lane NE. Athletics and osteoarthritis. *Am J Sports Med*. 1997;25:873–81.
40. Buckwalter JA, Lane NE. Aging, sports, and osteoarthritis. *Sports Med Arth Rev*. 1996;4:276–87.
41. Buckwalter JA, Lane NE, Gordon SL. Exercise as a cause of osteoarthritis. In: Kuettner KE, Goldberg VM, editors. *Osteoarthritic disorders*. Rosemont: American Academy of Orthopaedic Surgeons; 1995. p. 405–17.
42. Lequesne MG, Dang N, Lane NE. Sport practice and osteoarthritis of the limbs. *Osteoarthritis Cartilage*. 1997;5:75–86.
43. Felson DT. The epidemiology of osteoarthritis, prevalence and risk factors. In: Kuettner KE, Goldberg VM, editors. *Osteoarthritic disorders*. Rosemont: American Academy of Orthopaedic Surgeons; 1995. p. 13–24.
44. Williams C, Jimenez SA. Molecular biology of heritable cartilage disorders. In: Kuettner KE, Goldberg VM, editors. *Osteoarthritic disorders*. Rosemont: American Academy of Orthopaedic Surgeons; 1995. p. 35–50.
45. Ranger C, Kathrein A, Klestil T, et al. Partial meniscectomy and osteoarthritis Implications for treatment of athletes. *Sports Med*. 1997;23:61–8.
46. Roos H, Adalberth T, Dahlberg L, et al. Osteoarthritis of the knee after injury to the anterior cruciate ligament or meniscus: the influence of time and age. *Osteoarthritis Cartilage*. 1995;3:261–67.
47. Kujala UM, Kettunen J, Paananen H, et al. Knee osteoarthritis in former runners, soccer players, weight lifters and shooters. *Arthritis Rheum*. 1995;38:539–46.
48. Hulkko A, Orava S. Stress fractures in athletes. *Int J Sports Med*. 1987;8:221–26.
49. Callahan L. Stress fractures in women. *Clin Sports Med*. 2000;19:303–14.
50. Sanderlin BW, Raspa FR. Common stress fractures. *Am Fam Physician*. 2003;68:1527–32.
51. Lappe J, Cullen D, Haynatzki G, et al. Calcium and vitamin D supplementation decreases incidence of stress fractures in female navy recruits. *J Bone Miner Res*. 2008;23:741–9.
52. Lysholm J, Wiklander J. Injuries in runners. *Am J Sports Med*. 1987;15(2):168–71.
53. Johansson C. Injuries in elite orienteers. *Am J Sports Med*. 1986;14(5):410–5.
54. Kvist M. Achilles tendon injuries in athletes. *Sports Med*. 1994;18(3):173–201.
55. Pomeroy GC, Pike RH, Beals TC, Manoli II A. Current concepts review. Acquired flatfoot in adults due to dysfunction of the posterior tibial tendon. *J Bone Joint Surg*. 1992;81A:1173–82.
56. Holmes II GB, Mann RA. Possible epidemiological factors associated with rupture of the posterior tibial tendon. *Foot Ankle*. 1992;13:70–9.
57. Kohls-Gatzoulis J, Woods B, Angel JC, Singh D. The prevalence of symptomatic posterior tibialis tendon dysfunction in women over the age of 40 in England. *Foot Ankle Surg*. 2009;15(2):75–81.
58. Kohls-Gatzoulis J, Haddad F, Livingston J, Berry G, Singh D, Angel JC. Posterior tibial tendon dysfunction: a frequent and treatable cause of adult acquired flatfoot. *Br Med J*. 2004;329:1328–33.
59. Bergman AG, Fredericson M. MR imaging of stress reactions, muscle injuries, and other overuse injuries in runners. *Magn Reson Imaging Clin N Am*. 1999;7(1):151–74.
60. Johnson AW, Weiss Jr CB, Wheeler DL. Stress fractures of the femoral shaft in athletes more common than expected. A new clinical test [comment]. *Am J Sports Med*. 1994;22(2):248–56.
61. Brukner P, Bennell K, Matheson G. *Stress fractures*. Carlton: Blackwell Science; 1999.
62. Greaney RB, Gerber FH, Laughlin RL, Kmet JP, Metz CD, Kilcheski TS, et al. Distribution and natural history of stress fractures in US marine recruits. *Radiology*. 1983;146:339–46.

Michèle Kläusler and Erich Rutz

---

## Abstract

Over the last decades, sports for disabled and handicapped people have gained in importance. The Paralympics, especially has helped to advance sports for people with impairments. They were not only important to promote health and wellness among people with disabilities, but also made society aware of what these people are capable of.

Physical activity is not only very important for health prevention in able-bodied people, but it is crucial in impaired individuals, because they are at a higher risk for obesity, have a higher burden of cardiovascular disease and more often suffer from osteoporosis.

Due to the higher prevalence of disability sports these days, the topic of sports injuries in people with impairments has gained more significance over the last couple of years. There have been a few studies published, but there is a demand for further research.

As shown in the example of in cerebral palsy (CP), sport already plays an important role in many therapy programs.

For individuals with impairments, sport often becomes a very important part of their lives. It is important for their physical health but also for their mental health. It helps to establish their own body image and to gain self-esteem. And last but not least, it is significant to develop social interaction skills and establish friendships.

---

## Keywords

Disability sports • Paralympic sports • Sport injuries • Children with cerebral palsy • Hippotherapy • Aquatic therapy

---

## Definition

Disability is defined as follows: “In the context of health experience, a disability is any restriction or lack (resulting from an impairment) of ability to perform an activity in the manner or within the range considered normal for a human being.” (WHO, 1980, p. 28, 1. 1–5) Disability can be congenital, but it can also occur at any time in life as consequence of an accident or a disease.

On the other hand, handicap is defined as follows: “In the context of health experience, a handicap is a disadvantage

for a given individual, resulting from an impairment or a disability, that limits or prevents the fulfillment of a role that is normal (depending on age, sex and social and cultural factors) for that individual.” (WHO, 1980, p. 29, 1. 2–5)

Sports for disabled and handicapped people are defined as sports in people with longstanding physical or mental impairment.

---

## Introduction

The beginning of sports in disabled and handicapped people goes back to the Stoke Mandeville Hospital in Great Britain, where toward the end of 2nd World War Ludwig Guttman, a British neurosurgeon and Director of the National Spinal

---

M. Kläusler, MD • E. Rutz, MD (✉)  
Pediatric Orthopaedic Department, University Children’s Hospital  
Basel UKBB, Basel, Switzerland  
e-mail: [michele.klaeusler@gmail.com](mailto:michele.klaeusler@gmail.com); [erich\\_rutz@hotmail.com](mailto:erich_rutz@hotmail.com)

Injuries Center at Stoke Mandeville, began to integrate recreational and organized sport into the common rehabilitation therapies [1]. He believed that sports could improve the patients' physical and psychological wellbeing and this theory was confirmed by his observation of the patients. Encouraged by his clinical successes with sports therapy in 1948, Dr. Guttman organized the first Stoke Mandeville Games for people with impairments on the same day as the Opening Ceremony of the London 1948 Olympic Games was held [2]. In 1960, the first Paralympic Games took place in Rome. The first Winter Paralympic Games were held in Sweden in 1976.

Over the last 50 years the Paralympic Games have grown considerably and become more popular. The Paralympic Movement itself is not just about sports competition, it has also helped to advance many related areas in sports medicine and sports science. Most importantly it helped to promote health, wellness, and self-esteem amongst people with impairment and made our society aware of the capabilities of these individuals [2].

It is well known that sport is very important for all children, with or without disabilities. Sport does not only help to develop skills and exercise, it is also important for the evolution of interpersonal skills [1].

---

## Sports for the Disabled: Its Role in Prevention

Studies have shown that people with disabilities are more likely to be overweight or obese, have high blood pressure, use tobacco, and receive less social-emotional support, amongst other health disadvantages [3]. Regardless of the type of disability, all of these individuals are at a higher risk of becoming overweight or obese [4]. Especially adults with moderate to severe lower extremity impairments are at risk for obesity and physicians tend to not give enough counseling on the importance of exercise in this patient group [5]. Furthermore, intellectual disabled adults, when compared to the general population, have a higher prevalence of obesity and morbid obesity [6]. When it comes to cardiovascular health (hypertension, hyperlipidemia, coronary artery disease), there is a disproportionately high burden of these diseases amongst individuals with disabilities [7]. After spinal cord injury cardiovascular disease is one of the most common causes of morbidity and mortality [8]. Adults with functional impairments are also less likely to engage in leisure time physical activity when compared to the general population [4]. Children with disabilities spend as much time in daily activities as their healthy peers up to the 8th grade. After that time the rate of participation decreases [9].

For these reasons, an active lifestyle is especially important in people with functional impairments. It is very likely

that children with an active physical lifestyle will also become active adults and because of that, it is important to introduce exercise for individuals with disabilities already at a very young age [1].

---

## Paralympics

### Types of Sports

There are Paralympic sports that were developed as an adaptation to able-bodied sports, but there are also Paralympic sports with no able-bodied equivalent to be used by a certain impairment type [10]. For example, athletes with visual impairment play goalball whereas boccia is for athletes with severe hypertonia, ataxia, or athetosis. There are still new sports being added. For example, in Beijing 2008 adaptive rowing, in Rio de Janeiro 2016 para-canoe and para-triathlon events were introduced for the first time. Table 12.1 provides an overview of Paralympic sports [10].

### Classification

In 2007, the International Paralympic Committee Classification Code, based on the International Classification of Functioning, Disability, and Health terminology and taxonomy to categorize impairment groups, was introduced for the classification of Paralympic sports [11]. To compete in Paralympic sports an athlete has to have a objectively diagnosed primary permanent impairment. These impairments can be grouped in the ten following types: hypertonia, ataxia, athetosis, loss of muscle strength, loss of range of movement, loss of limb, limb deficiency, short stature, low vision, or intellectual impairment.

There are some sports designed for certain types of impairment only, as well as others, which allow athletes with different impairments to compete against one another.

In Olympic sports, athletes are classified by sex, age and weight whereas in Paralympic sports there are minimum disability criteria for participation as well as a stratification of the athletes by the severity of the impairment [12]. There are different numbers of classifications and inclusion criteria for each sport.

### Wheelchairs

Since the early days of wheelchair racing at the "Stoke Mandeville" games in England there has been an evolution of wheelchair racing technology, which can be compared to the development of cycling gear [1]. Currently there are approximately 20 manufacturers developing wheelchairs

**Table 12.1** Paralympic sports [10]

	Impairment groups	Participation dates
<b>Summer games</b>		
Archery	Amputees, LA, CP, SCR D	1960-present
Athletics	Amputees, LA, CP, II, VI, SCR D	1960-present
Boccia	CP	1988-present
Cycling	Amputees, LA, CP, VI, SCR D	1988-present
Equestrian	Amputees, LA, CP, VI, SCR D	1996-present
Football (5-a-side)	VI	2004-present
Football (7-a-side)	CP	1984-present
Goalball	VI	1980-present
Judo	VI	1988-present
Powerlifting	Amputees, LA, CP, SCR D	1964-present
Rowing	Amputees, LA, CP, VI, SCR D	2008-present
Sailing	Amputees, LA, CP, VI, SCR D	2000-present
Shooting	Amputees, LA, CP, VI, SCR D	1976-present
Swimming	Amputees, LA, CP, II, VI, SCR D	1960-present
Table tennis	Amputees, LA, CP, SCR D	1960-present
Volleyball	Amputees, LA	1976-present
Wheelchair basketball	SCR D, amputees, LA, II	1960-present
Wheelchair fencing	SCR D	1960-present
Wheelchair rugby	SCR D	2000-present
Wheelchair tennis	SCR D	1992-present
<b>Winter games</b>		
Alpine skiing	Amputees, LA, CP, VI, SCR D	1976-present
Biathlon	Amputees, LA, CP, VI, SCR D	1988-present
Ice sledge hockey	Amputees, LA, CP, SCR D	1984-present
Nordic skiing	Amputees, LA, CP, VI, SCR D	1976-present
Wheelchair curling	Amputees, LA, CP, SCR D	2006-present

LA les autres, CP cerebral palsy, SCR D spinal cord-related disability, II intellectual impairment, VI visually impaired



**Fig. 12.1** Wheelchair basketball (With permission from Basler Orthopädie, René Ruepp AG, Switzerland)

which are unique for each sport. For example, tennis chairs must be able to quickly turn and have to be very agile on the court whereas wheelchairs for basketball (Fig. 12.1) and rugby have to be agile and also steady to bear collisions, which are characteristic for these sports. The “hand cycle” is one of the newest developments. It is a 3-wheeled cycle propelled by the use of the arms and they have the advantage of gearing similar to cycling. Hand cycles are not only used for competition, hobby athletes can also use them if they want to go for a ride with friends and family.

### Prosthetics

Also in limb prosthetics there have been many technological accomplishments. While previously the design of prosthetics lent more towards light weight and concealing the missing limb, the prosthetics of today are designed to improve user function with advanced materials and designs (Fig. 12.2). There have been many advancements in biomechanics and material engineering, which provides the athlete with advantages specific for his or her sport [1].

**Fig. 12.2** Runner with above-knee prosthesis (With permission from Basler Orthopädie in cooperation with Otto Bock Switzerland)



In 2012, double amputee Oscar Pistorius competed as the first amputee athlete in the able-bodied Olympics in 400-m race. Initially he was not allowed to compete, because the International Association of Athletic Foundation, the worldwide body that governs international competition, stated in 2007 that his artificial leg would give him an unfair advantage over able-bodied athletes in the competition.

## Sports Related Injuries in Disabled and Handicapped People

### Overview

There is still a big necessity for research about sports related injuries in disabled and handicapped people, but recently there have been new studies published to address this issue. For disabled athletes, an injury can have a higher impact on their training and competing as well as their functioning in daily life compared to able-bodied athletes [13].

During the Summer Paralympic games 2012 17.8/100 athletes [14] compared to 12.9/100 athletes during the Summer Olympics [15] got injured. There was a high variability between the different sports, very high injury rates were seen in football (5-a-side), goalball, powerlifting, wheelchair fencing and wheelchair rugby. Rowing and shooting were the sport disciplines with the lowest incidence of injuries [14].

There is a high prevalence of lower extremity injuries in walking athletes, especially those with visual impairment and in those participating in ball sports and track and field,

whereas most upper extremity injuries, especially in the shoulder, occur in wheelchair athletes [13].

Fagher and Lexell [13] stated in their review article that there is a high prevalence of sports-related injuries in Paralympic athletes caused by overuse and that this number might be underestimated, because some studies only include events related to trauma and competition in their injury definition. It seems likely that athletes who participate in ball sports more often suffer from acute injuries.

Certain risk factors could be identified for disability sports. First of all the disability itself can be seen as a risk factor [16], for example athletes with impaired vision are more likely to suffer from a lower extremity injury [17]. Also, there are different risk factors for different types of impairments. The low bone mineral density in wheelchair athletes can be considered as a risk factor for injury [13]. Almost every person with spinal cord injury (SCI) has osteoporosis [18], which as a result ends in an increased number of lower extremity fractures. In wheelchair athletes there is also a high frequency of upper extremity injuries [19], because these athletes depend on their upper extremities in both their daily life and in sports. These injuries can put high stress on its tissues. In amputee athletes there is a high prevalence of injury and pain [20]. The cause of this could be the changed biomechanics in the lower extremity [21]. Problems can also occur in the intact lower limb of athletes with unilateral amputation caused by the asymmetrically higher forces while running [20].

After any sports injury it is important to keep the rehabilitation period as short as possible, at best there should not be any immobilization at all, to prevent any loss of muscle mass.



## Lower Leg and Ankle Injuries

Lower leg injuries are particularly frequent in blind athletes [13], for example during the Paralympic Games 1988 78% of all lower extremity injuries that were reported in the Canadian team occurred in blind athletes [17]. We can find an explanation for this by a study of Athanasopoulos et al. [22], which claimed that the high incidence of ankle injuries in this athletes could be caused by poor proprioception, and Aydog et al. [23] which suggested that postural stability may be affected by vision.

Individuals with CP frequently have orthopedic deformities in the foot (e.g. equinus) and knee region. Hinged ankle-foot-orthosis is the most common used type of orthosis in these patients. Patatoukas et al. [24] found out that there was a higher percentage of soft tissue injuries compared with other athletes in CP athletes. The authors explained this by citing the patients limited range of motion, spasticity and discoordination, which may cause additional stress to muscles, joints and tendons. Athanasopoulos et al. [22] suggested that this higher number of lower extremity injuries in CP athletes could be caused by an increased tensile force in the lower extremity which is a result of spasticity and deformities.

As mentioned earlier there is a higher prevalence of injury and pain in amputee athletes [20], which may be caused by the altered biomechanics in the lower extremity [21]. In Athletes with a unilateral amputation injuries also occur in the intact lower limb because of the asymmetrically higher forces while running [20].

There is a higher incidence of lower extremity fractures in wheelchair athletes, which can be explained by osteoporosis, which occurs in almost all people with Spinal cord injury [18]. For this reason it is very important in wheelchair sports to have a good regulation for the protective equipment to prevent these injuries, especially in winter sports [25].

---

## Sports for Disabled and Handicapped People Using the Example of Children with CP

### Introduction

Cerebral palsy (CP) is a permanent movement disorder based on a progressive defect or damage to the brain in its early development (generally in the first two years of life). It is a complex of symptoms and no entity itself [26]. The incidence of the infantile CP ranges between 2 and 6 per 1000 live births. It is the most common cause of disability in childhood.

There are a variety of clinical presentations, which depend on the size and localization of the lesions in the brain. Roughly they can be divided into quadriplegia, diplegia and hemiplegia. The main problems are the increased muscle

tone, the spasticity and muscular weakness. As a consequence patients can develop torsion defects in tibia and femur, joint contractures and foot deformities [27]. Often there is also a change of sensibility on the trunk and the extremities.

Sport is an important part of the therapy program in children with CP. It can enhance muscle strength and coordination. Certain sports improve sequences of motion and motion coordination. It is well known that hippotherapy can improve trunk control as well as decrease the muscle tone in lower extremities [28]. Physical activity is not only a cardiopulmonary training; it also helps to strengthen the bones, knowing that many CP patients suffer from inactivity osteoporosis.

The Gross Motor Function Classification System (GMFCS) has been established for the classification of the impairment of children with cerebral palsy (CP) into five levels, in which functional walkers are classified within level I-III, with I being the most able [29, 30]. It is a clinical classification system, independent of any consideration of instrumented gait analysis.

### Types of Therapy

**Physical and occupational therapy:** Their goal is to improve movement sequences, the prevention of joint contractures and consequently preserve the patient autonomy

**Strength and fitness training** should improve muscular strength and fitness in general. It is important to respect the biomechanical principles (especially muscular weakness and limitation of motion) during training. It may be beneficial to establish a targeted program to improve muscular weakness. Studies from Damiano [31, 32] have shown, that targeted muscle training can improve the muscle strength in children with CP.

**Hippotherapy** has become very popular over the last years, especially with children. The patients appreciate the contact to the animals and several studies showed that hippotherapy (Fig. 12.3) has a positive impact on children with CP. A recently published review study by Whalen and Case-Smith [29] identified that hippotherapy and therapeutic horse riding (THR) can amend gross motor function and mobility in children with spastic CP who were 4 years old or above. Moreover, another meta-analysis issued by Zadnikar and Kastrin [28] showed that hippotherapy and THR improves postural control and balance in children with CP, even though the intervention and comparison sample sizes of the reviewed studies were too small and the CP population was extremely diverse. Particularly the study of Snider et al. [33] showed an improvement in trunk control and a reduction of muscular tone in the lower extremity. The study of Casady and Nichols-Larsen [34] also reports an improvement of the gross motor function measure.

**Fig. 12.3** Hippotherapy (In permission with Hippotherapie-K © Ruth Obrist-Angehrn, Kaiseraugst, Switzerland)



**Aquatic therapy:** A meta-analysis by Gorter and Currie [35] found clinically significant improvements in muscle strength, energy expenditure, gross motor function scores and mobility performance in children and adolescents in several studies. However, the authors of this review article symphonize that most studies only examined CP patients with Gross motor functioning score (GMFCS) levels between I and III. A study by Fragala-Pinkham et al. [36] showed an improvement of gross motor function and walking endurance after a 14-week aquatic exercise program in children with CP, there were no changes seen in functional strength, aerobic capacity and balance.

## Sports

The aims and use of physical activity are:

- the training of strength and coordination
- to cultivate social contacts and finally also
- to have fun and joy being physically active.

For ambulatory and moderate disabled children it is basically possible to carry out all sports in consideration of some preventive measures. When there is evidence of osteoporosis there is a need for attention because of the higher risk of fracture.

The training also has a positive impact on the cardiopulmonary system.

With some appropriate adjustments (for example the breaks, tricycle) especially cycling is very popular in children with CP. This is a good training for endurance and strength. It is important for the children to wear a helmet and other protection gear, such as gloves. Running also trains strength

and endurance. There is little influence on the tone and it is often even interfering with repetitive movements. Even soccer and basketball are possible. But for these sports there is a need for a good body control as well as coordinative skills. Coordination can be trained and improved.

Also winter sports such as alpine and cross-country skiing are thinkable. However it is important to consider muscular weaknesses. It is possible to do active and passive (for example ski bob) skiing.

For children with severe impairments there is the possibility of tricycling, swimming and skiing in winter. There are also offers for wheelchair rugby or basketball and many more.

## Evidence

To our knowledge regarding the topic of our book chapter there is no level-I published data.

## Summary

Sports for disabled and handicapped people have become an important health and psychological development mechanism for the disabled. The Paralympics were relevant for the advancement and promotion of sports in people with impairments. It is necessary to always keep in mind, that physical activity is not only important for health prevention in able-bodied people, it is also crucial for disabled and handicapped people, because these individuals are at higher risk for obesity, have a higher burden of cardiovascular disease and more often suffer from osteoporosis. Based on the higher prevalence of sports for individuals with impairments there is

also a higher incidence of sports injuries in this group. So far only a few studies about this topic have been published and there is a necessity for further research. Some of these studies also describe lower leg and ankle injuries. The example of CP shows that sports already play an important part in many therapy programs. For individuals with impairments, sport often is a major part of their lives. It is not only important for their physical health but also for their mental health. It is also significant for the development of social interaction skills and to establish friendships.

## References

- Wilson PE, Clayton GH. Sports and disability. *PMR*. 2010;2(3):S46–54; quiz S5–6. Epub 2010/04/03.
- Willick SE, Lexell J. Paralympic sports medicine and sports science-introduction. *PMR*. 2014;6(8 Suppl):S1–3. Epub 2014/08/20.
- Blauwet CA, Iezzoni LI. From the paralympics to public health: increasing physical activity through legislative and policy initiatives. *PMR*. 2014;6(8 Suppl):S4–10. Epub 2014/08/20.
- Wood E, Rosenbaum P. The gross motor function classification system for cerebral palsy: a study of reliability and stability over time. *Dev Med Child Neurol*. 2000;42(5):292–6. Epub 2000/06/16.
- Palisano RJ, Hanna SE, Rosenbaum PL, Russell DJ, Walter SD, Wood EP, et al. Validation of a model of gross motor function for children with cerebral palsy. *Phys Ther*. 2000;80(10):974–85. Epub 2000/09/26.
- Whalen CN, Case-Smith J. Therapeutic effects of horseback riding therapy on gross motor function in children with cerebral palsy: a systematic review. *Phys Occup Ther Pediatr*. 2012;32(3):229–42. Epub 2011/11/30.
- Froehlich-Grobe K, Lee J, Washburn RA. Disparities in obesity and related conditions among Americans with disabilities. *Am J Prev Med*. 2013;45(1):83–90. Epub 2013/06/26.
- Garshick E, Kelley A, Cohen SA, Garrison A, Tun CG, Gagnon D, et al. A prospective assessment of mortality in chronic spinal cord injury. *Spinal Cord*. 2005;43(7):408–16. Epub 2005/02/16.
- Sabo D, Veliz P. Go out and play: youth sports in America. East Meadow, NY: Women's Sports Foundation; 2008.
- Webborn N, Van de Vliet P. Paralympic medicine. *Lancet*. 2012;380(9836):65–71. Epub 2012/07/10.
- Tweedy SM, Vanlandewijck YC. International Paralympic Committee position stand—background and scientific principles of classification in Paralympic sport. *Br J Sports Med*. 2011;45(4):259–69. Epub 2009/10/24.
- Comitee IP. Disabled sports classification. [cited 2014 September 16]. Available from: <http://www.paralympic.org/classification>.
- Fagher K, Lexell J. Sports-related injuries in athletes with disabilities. *Scand J Med Sci Sports*. 2014. Epub 2014/01/16.
- Webborn N, Emery C. Descriptive epidemiology of paralympic sports injuries. *PMR*. 2014;6(8 Suppl):S18–22. Epub 2014/08/20.
- Engebretsen L, Soligard T, Steffen K, Alonso JM, Aubry M, Budgett R, et al. Sports injuries and illnesses during the London Summer Olympic Games 2012. *Br J Sports Med*. 2013;47(7):407–14. Epub 2013/03/22.
- Magno e Silva M, Bilzon J, Duarte E, Gorla J, Vital R. Sport injuries in elite paralympic swimmers with visual impairment. *J Athl Train*. 2013;48(4):493–8. Epub 2013/06/19.
- Burnham R, Newell E, Steadward R. Sports medicine for the physically disabled: the Canadian team experience at the 1988 Seoul Paralympic Games. *Clin J Sport Med*. 1991;1(3):193–6.
- Jiang SD, Dai LY, Jiang LS. Osteoporosis after spinal cord injury. *Osteoporos Int*. 2006;17(2):180–92. Epub 2005/10/12.
- Derman W, Schwellnus M, Jordaan E, Blauwet CA, Emery C, Pit-Grosheide P, et al. Illness and injury in athletes during the competition period at the London 2012 Paralympic Games: development and implementation of a web-based surveillance system (WEB-IISS) for team medical staff. *Br J Sports Med*. 2013;47(7):420–5. Epub 2013/03/30.
- Nyland J, Snouse SL, Anderson M, Kelly T, Sterling JC. Soft tissue injuries to USA paralympians at the 1996 summer games. *Arch Phys Med Rehabil*. 2000;81(3):368–73. Epub 2000/03/21.
- Bernardi M, Castellano V, Ferrara MS, Sbriccoli P, Sera F, Marchetti M. Muscle pain in athletes with locomotor disability. *Med Sci Sports Exerc*. 2003;35(2):199–206. Epub 2003/02/06.
- Athanasopoulos S, Mandalidis D, Tsakoniti A, Athanasopoulos I, Strimpakos N, Papadopoulos E, et al. The 2004 Paralympic Games: physiotherapy services in the Paralympic Village Polyclinic. *Open Sports Med J*. 2009;3:1–8.
- Aydog E, Aydog ST, Cacki A, Doral MN. Dynamic postural stability in blind athletes using the biodex stability system. *Int J Sports Med*. 2006;27(5):415–8. Epub 2006/05/27.
- Patatoukas D, Farmakides A, Aggeli V, Fotaki S, Tsididakis H, Mavrogenis AF, et al. Disability-related injuries in athletes with disabilities. *Folia Med*. 2011;53(1):40–6. Epub 2011/06/08.
- Webborn N, Willick S, Emery CA. The injury experience at the 2010 winter paralympic games. *Clin J Sport Med*. 2012;22(1):3–9. Epub 2012/01/09.
- Bax MC, Flodmark O, Tydeman C. Definition and classification of cerebral palsy. From syndrome toward disease. *Dev Med Child Neurol Suppl*. 2007;109:39–41. Epub 2007/03/21.
- Rutz E, Baker R, Tirosh O, Brunner R. Are results after single-event multilevel surgery in cerebral palsy durable? *Clin Orthop Relat Res*. 2013;471(3):1028–38. Epub 2013/01/04.
- Zadnikar M, Kastrin A. Effects of hippotherapy and therapeutic horseback riding on postural control or balance in children with cerebral palsy: a meta-analysis. *Dev Med Child Neurol*. 2011;53(8):684–91. Epub 2011/07/07.
- Rosenbaum PL, Walter SD, Hanna SE, Palisano RJ, Russell DJ, Raina P, et al. Prognosis for gross motor function in cerebral palsy: creation of motor development curves. *JAMA*. 2002;288(11):1357–63. Epub 2002/09/18.
- Palisano R, Rosenbaum P, Walter S, Russell D, Wood E, Galuppi B. Development and reliability of a system to classify gross motor function in children with cerebral palsy. *Dev Med Child Neurol*. 1997;39(4):214–23. Epub 1997/04/01.
- Damiano DL. Rehabilitative therapies in cerebral palsy: the good, the not as good, and the possible. *J Child Neurol*. 2009;24(9):1200–4. Epub 2009/06/16.
- Damiano DL. Activity, activity, activity: rethinking our physical therapy approach to cerebral palsy. *Phys Ther*. 2006;86(11):1534–40. Epub 2006/11/10.
- Snider L, Korner-Bitensky N, Kammann C, Warner S, Saleh M. Horseback riding as therapy for children with cerebral palsy: is there evidence of its effectiveness? *Phys Occup Ther Pediatr*. 2007;27(2):5–23. Epub 2007/04/20.
- Casady RL, Nichols-Larsen DS. The effect of hippotherapy on ten children with cerebral palsy. *Pediatr Phys Ther*. 2004;16(3):165–72. Epub 2006/10/24.
- Gorter JW, Currie SJ. Aquatic exercise programs for children and adolescents with cerebral palsy: what do we know and where do we go? *Int J Pediatr*. 2011;2011:712165. Epub 2011/12/14.
- Fragala-Pinkham MA, Smith HJ, Lombard KA, Barlow C, O'Neil ME. Aquatic aerobic exercise for children with cerebral palsy: a pilot intervention study. *Physiother Theory Pract*. 2014;30(2):69–78. Epub 2013/12/18.

Ethan S. Krell, Nicholas J. Montemurro, Kristen P. Pacific,  
Lionel Emele, and Sheldon S. Lin

#### Abstract

Bone, cartilage, and tendon healing are clinically challenging processes. In order to address these concerns, orthobiologic research aims to enhance key steps involved in these processes using autologous and recombinant compounds, with the goal of achieving maximal clinical results. The potential for a quicker return to sport is appealing for athletes at all levels, and many high profile cases have driven continued orthobiologic research. In this chapter, we review the current clinical evidence regarding the use of biologic agents and scaffolds to improve healing following injury to the foot and ankle.

#### Keywords

Orthobiologics • Bone regeneration • Cartilage repair • Tendon/ligament regeneration

## Introduction

Quick return to high performance activity is of particular interest following sports injuries sustained by professional and recreational athletes. Significant research has focused on the exploration of biologic agents, both autologous and recombinant, that have the potential to maximize the natural healing process. By optimizing the local healing environment, athletes may be able to regain competitive activity levels more quickly. Collectively known as orthobiologics, these adjunct therapies fall into many broad categories including:

- Autologous, heterologous, and synthetic bone/tendon grafts
- Cell culturing
- Bone marrow and whole blood aspirates/concentrates
- Autologous and recombinant growth factors and proteins

E.S. Krell, BS • N.J. Montemurro, BS • K.P. Pacific, BA  
L. Emele, BA • S.S. Lin, MD (✉)  
Department of Orthopaedics, Rutgers New Jersey Medical School,  
90 Bergen Street, Suite 7300, Newark, NJ 07101, USA  
e-mail: [ethanskrell@gmail.com](mailto:ethanskrell@gmail.com); [nicholas.montemurro@gmail.com](mailto:nicholas.montemurro@gmail.com);  
[kristenppacific@gmail.com](mailto:kristenppacific@gmail.com); [le75@cornell.edu](mailto:le75@cornell.edu); [linss@njms.rutgers.edu](mailto:linss@njms.rutgers.edu)

While many biologic agents and scaffold materials are already widely accepted in the field of orthopaedic surgery, limited evidence exists to date that indicates their use for foot and ankle applications. The goal of this chapter is to highlight the efficacy and function of a wide range of orthobiologics used to enhance bone, cartilage, and tendon/ligament healing in the foot and ankle.

## Bone Orthobiologics

Foot and ankle surgeons originally focused on mechanical stabilization to achieve successful arthrodesis when treating the various causes of joint destruction. However, numerous risk factors are now known to impair osseous healing [1] and nonunion rates range from 10% to as high as 40% within high-risk populations [2, 3]. To address this concern, bone grafting techniques and adjunct therapies were developed to improve the local biologic environment.

## Bone Graft Properties

Three important factors describe the biologic activity of bone grafts: osteoconduction (material serves as scaffold for

existing bone to grow into), osteoinduction (material stimulates differentiation of existing osteoprogenitor cells), and osteogenesis (material contains osteoblasts that contribute to growth). Autologous bone graft (ABG) is the only graft possessing all three properties, and is considered the gold standard for managing bone defects and non-unions [4]. However, ABG is associated with chronic donor site pain and is limited in quantity [5].

## Allografts

Often, bulk allograft is needed for large defects to supplement the limited amounts of autograft. Heterologous bone grafts from cadavers can be fresh-frozen, freeze-dried, or demineralized bone matrix. Generally, these grafts must be irradiated to reduce immunogenic reactions, which in turn dramatically reduces their osteogenic and osteoinductive potential [6]. Alternatively, synthetic grafts such as tricalcium phosphate and calcium phosphate cement can be utilized, but have only osteoconductive capacity. Recent research has focused on orthobiologics (bone marrow aspirate, whole blood concentrates, recombinant proteins and growth factors) with osteoinductive properties, which can be either used alone or combined with osteoconductive grafting scaffolds. Specifically, recombinant human platelet-derived growth factor (rhPDGF) and bone morphogenetic proteins (BMPs) are common orthobiologics currently under investigation for use in foot and ankle surgery.

## Recombinant Growth Factors and Proteins

Platelet-derived growth factor (PDGF) refers to a family of growth factors that are released from platelets and macrophages following tissue damage. The PDGF-BB isoform has been shown to recruit inflammatory cells to the injury site, increase collagen deposition, and promote angiogenesis [7]. DiGiovanni et al. [8] conducted a multicenter randomized controlled trial comparing the efficacy of rhPDGF-BB in a  $\beta$ -tricalcium phosphate scaffold (Augment Bone Graft; BioMimetic Therapeutics, Inc., Franklin, TN) to ABG in achieving arthrodesis. A total of 66.5% of PDGF-treated joints and 62.6% of ABG-treated joints demonstrated 50% or greater bone bridging on CT scan ( $s < 0.001$ ; statistically significant for equivalence). PDGF has been approved for ankle and hindfoot arthrodesis in Canada, Australia, and New Zealand and is currently under review in the United States and Europe.

Recombinant human bone morphogenetic proteins (rhBMPs) stimulate mesenchymal stem cells to develop into osteogenic and chondrogenic cells during bone healing [9]. Currently, rhBMP-2 (INFUSE; Medtronic, Minneapolis, MN) and rhBMP-7

(OP-1; Olympus Biotech Corp., Hopkinton, MA) have demonstrated utility in foot and ankle surgery (Table 13.1) [10–14]. However, the literature is limited to level III and IV case series at this time. Technically, BMPs are used off-label for foot and ankle applications and are not covered by many insurance policies.

## Cartilage Orthobiologics

Osteochondral talar lesions (OTLs) occur to the anterolateral or to a lesser extent posteromedial aspect of the talus, usually following trauma that results in subsequent damage to the talar articular cartilage and often the underlying bone. A considerable percent of patients experience poor clinical outcomes following cast immobilization, surgical excision, or bone marrow stimulation techniques (drilling or microfracture), especially when the defect is large and unstable [15]. Several biologic therapies have been developed to improve healing of the affected cartilage.

## Injections

Pain and functional deficit associated with mechanically stable OTLs could potentially be improved through conservative treatment with hyaluronic acid (HA) or platelet-rich plasma (PRP). HA is present in synovial fluid and reduces pain by cushioning joints and by inhibiting inflammatory cytokines, preventing further breakdown of cartilage [16]. In a prospective study [17], fifteen patients with osteochondritis dissecans (OCD) of the talus were treated with 3 intra-articular injections (1 week intervals) of HA and were followed for 26 weeks. Pain and global functioning scores significantly improved within 12 weeks of treatment, and no adverse events were observed throughout the 6 month follow-up period. While HA may prevent further cartilage breakdown, PRP aids in cartilage repair (mechanistic action described below). A recent study [18] showed that intra-articular injection of PRP significantly improved pain and global functioning scores relative to HA treatment. Both HA and PRP may be reasonable future treatment options for conservatively treating pain caused by OTLs.

## Autograft & Allograft Techniques

Several osteochondral autograft transplantations have recently been investigated for treating OTLs. Autologous chondrocyte implantation (ACI) involves transplanting viable chondrocytes to the defect space. Cartilaginous material is harvested from the knee or ankle, cultured for 3–4 weeks, and subsequently packed into a periosteal graft and delivered

**Table 13.1** BMP-2 and BMP-7 clinical trials in the foot and ankle

Agent/Brand name	Investigator	Level of evidence	Procedure/disease	Application	Combined treatments	Comparison group	Number of patients	Follow-up	Outcome
BMP-2 (INFUSE)	Bibbo et al. [10]	Comparative study	Ankle or hindfoot arthrodesis	Surgical	No	Yes (historical controls)	69	3 months	Higher union rate (96%) than historical controls (48%)
	DeVries et al. [11]	Comparative study	Revision tibiocalcaneal arthrodesis	Surgical	Yes (procedures were highly variable)	Yes (no BMP)	7 BMP vs. 16 no BMP	24 months	Higher union rate (71.4%) than control (56.3%), but slower time to radiographic union (184 vs. 115 days)
BMP-7 (OP-1)	Fourman et al. [12]	Comparative study	Ankle arthrodesis	Surgical	Ilizarov type frame	Yes (no BMP)	42 BMP vs. 40 control	3 months	Significantly higher union rate (92%) than control (53%)
	Kanakaris et al. [13]	Case series	Ankle, subtalar, talonavicular, pubic or sacroiliac failed arthrodesis	Surgical	Bone graft (11 cases)	No	19 (7 in foot/ankle)	30 months	Clinical and radiographic improvement in 17 cases
	Schubert et al. [14]	Case series	Forefoot procedures, tibiotalar or tibiotalcaneal arthrodesis, or distal tibial osteotomy	Surgical	Yes (procedures were highly variable)	No	38	10 months	Significant difference in union between patients <50 years of age (100%) and patients >50 (73%)

to the osteochondral defect. A similar procedure, matrix-induced autologous chondrocyte implantation (MACI) avoids periosteal graft harvesting by using a porcine type-I/III collagen membrane, which also more evenly distributes the viable cells [19]. Additionally, a number of collagen membranes now on the market, for example Chondro-Gide (Geistlich Biomaterials, Switzerland), may further improve ACI techniques [20]. Currently, the literature suggests positive clinical outcomes following ACI/MACI treatment [21, 22]; however, higher level clinical studies are necessary to more clearly define the indications for autograft transplantation versus traditional excision, curettage, and microfracture techniques.

In two different studies, Giannini et al. [23, 24] investigated a novel arthroscopic technique in which autologous mesenchymal stem cells (MSCs) and platelet rich plasma (PRP) were combined with either an HA membrane or a porcine collagen scaffold and implanted into an osteochondral lesion. The first clinical trial [23] found that AOFAS scores were significantly higher than baseline scores 6 months following surgery. In two cases, biopsies were performed at one year post-surgery and histological analysis showed the presence of new cartilage formation.

The second trial [24] compared the efficacy of this new technique to both open and arthroscopic ACI. Of the 81 total patients, 25 were treated with MSC + PRP + scaffold, 46 with arthroscopic ACI, and ten with open ACI. Over a 3 year follow-up period, no intergroup differences were observed using AOFAS, radiographic, and MRI evaluations. The investigators' novel composite produced equivalent results compared to the ACI treatment, while avoiding the long surgical time and additional cost associated with ACI. MSCs derived from bone marrow have been well characterized for their multipotent capabilities and their proliferative capacity [25]; however, the results of these two trials cannot be solely attributed to either MSCs or PRP. There may instead be a synergistic value to combining multiple orthobiologics.

---

## Tendon & Ligament Orthobiologics

### Platelet-Rich Plasma

At this time, platelet-rich plasma (PRP) supplementation is permitted by the World Anti-Doping Agency and within the NFL, NBA, MLB, and NCAA communities for treating a variety of conditions [26]. PRP is derived from autologous whole blood and contains a concentrated volume of platelets. The platelets within the concentrate release multiple growth factors that contribute to healing, such as PDGF, transforming growth factor, insulin-like growth factor, and vascular endothelial growth factor, among many others. The specific actions of these growth factors vary. PDGF, the predominant

growth factor, has a known chemotactic and mitogenic action, which leads to the proliferation and expansion of mesenchymal stem cells as well as the enhancement of angiogenesis [27].

Although several elite athletes attribute their successful rehabilitations to PRP, a tremendous paucity in the literature exists to effectively support these claims. The biologic properties of PRP are ideal for promoting soft tissue regeneration; however, the variable formulation of PRP presents a challenge when comparing clinical studies. PRP can differ in platelet concentration, centrifugation method, and by the presence of activating agents, white blood cells, and anticoagulant. Further, no set dosages or treatment protocols are currently widely accepted. Until high level clinical evidence fully elucidates the therapeutic potential of PRP, we must, as physicians, critically evaluate the available research. Refer to Table 13.2 for a summary of the results of clinical trials in which PRP is utilized to treat Achilles tendinopathy [28–33], Achilles tendon rupture [34–36], and lateral ligament sprain [37].

### Autograft & Allograft Techniques

Tendon autografts and allografts are useful as adjuncts in foot and ankle repair. Tendon transfers have been used to treat: (1) chronic ankle instability [38, 39], (2) chronic Achilles tendon rupture [40, 41], (3) peroneal tendon tears [42], and (4) posterior tibial tendon dysfunction [43].

1. In a study of 68 patients with chronic ankle instability, lateral ankle ligament reconstruction using semitendinous autograft required a longer average operation time but yielded a significantly shorter healing time ( $11.2 \pm 4.1$  months vs  $13.5 \pm 5.2$  months,  $P = .0458$ ), compared to the tendon allograft group. No significant difference in AOFAS scores was found between the two groups, though the improvements from pre-operative to post-operative stability were significant within both groups [38].
2. An initial evaluation of Achilles tendon allograft with associated calcaneus bone graft showed promise for treating patients presenting with severe chronic Achilles tendon rupture. The technique allowed for better preservation of surrounding structural tissue and normal rotational ability of the Achilles tendon compared to current standard-of-care approaches [41].
3. Reconstruction of peroneal tendon tears using peroneal tendon or semitendinosus allograft were found to be effective and possess several benefits over standard tendon transfer techniques, including shorter surgical time, better preservation of range of motion, and decreased opportunity for post-operative fracture [42].

**Table 13.2** PRP clinical trial results for treating common foot and ankle sports injuries

Condition	Investigator	Study type	Treatment protocol	Combined treatments	Comparison group	Number of patients	Follow-up	Key results
Achilles tendinopathy	de Vos et al. [28] and de Jonge et al. [29]	Randomized trial	1 injection of 4 ml PRP	Local anesthesia	Yes	27 PRP vs. 27 saline	12 months	No significant difference in improvement between PRP vs. saline groups
	Monto et al. [30]	Case series	1 injection of 4 ml PRP	Local anesthesia	No	30	24 months	Clinical improvement in 27 patients
	Gaweda et al. [31]	Case series	1 injection of 3 ml PRP	No	No	14	18 months	Significant clinical and functional improvement; positive imaging results
	Filardo et al. [32]	Case series	3 injections of 5 ml PRP (2-weeks intervals)	No	No	27	54 months	Significant long-term improvement in function and activity level; duration of symptoms correlated to time to return to sport
	Owens et al. [33]	Case series	1 injection of 6 ml PRP	No	No	10	24 months	Clinical but not MRI improvement
Achilles tendon rupture	Schepull et al. [34]	Randomized trial	Intra-op injection of 10 ml PRP	Tendon suture	Yes (no PRP)	16 PRP vs. 14 control	12 months	No significant difference in biomechanical tests in PRP vs. control groups; clinical outcome significantly better in control group
	Sanchez et al. [35]	Case series	Coated sutures and intra-op injection of 4 ml PRP	Tendon suture	Yes (historical controls)	6	6 months	Faster return to training activities in PRP group (14 weeks) vs. control group (21 weeks)
	Filardo et al. [36]	Case report	3 injections of 5 ml PRP (1-week intervals)	No	No	1	18 months	Full return to pre-injury sport activity level in 75 days; no long-term recurrence
Lateral ligament sprain	Stanton et al. [37]	Randomized trial	1 injection of 5 ml PRP	No	Yes (saline)	26 PRP vs. 11 saline	12 months	Faster return to sports in PRP group (16.5 days) vs. saline group (26.3 days)

4. A technique utilizing interference screw fixation in conjunction with flexor digitorum longus transfer to successfully treat posterior tibial tendon dysfunction allowed for a less extensive harvest of donor tendon [43].

## Membranes

Acellular human dermal tissue matrix, GraftJacket Matrix (Wright Medical Technology, Inc., Arlington, TN) has been



utilized to augment suture repair of neglected [44] and acute [45] Achilles tendon ruptures. In these clinical studies, desirable return-to-activity time was achieved and no adverse events were observed. The matrix is composed of freeze-dried human dermal tissue, and is sutured circumferentially around the Achilles tendon repair site. Histological analysis demonstrated that the matrix is infiltrated by host fibroblasts and blood vessels and incorporates as tendon-like tissue [46].

Several companies have developed amniotic tissue membranes derived from the innermost lining of the human placenta. A pre-clinical study has demonstrated improved Achilles tendon healing and increased mechanical strength [47]. Although human trials are underway, no clinical results have been peer-reviewed at this time.

## Summary

- According to level I clinical evidence [8], rhPDGF (Augment) is statistically equivalent to autologous bone graft for achieving arthrodesis in the foot and ankle. This product is currently under review for FDA approval.
- Several clinical studies [10–12] have reported higher union rates for hindfoot arthrodesis procedures augmented with rhBMP-2 (INFUSE), compared to non-supplemented controls.
- Both HA and PRP injections may reduce pain and improve function in patients with mechanically stable osteochondral talar lesions [17, 18].
- Although the literature regarding PRP injection for Achilles tendinopathy remains controversial, PRP may be effective for cases of chronic Achilles tendinopathy that have not responded to standard conservative treatment [30].
- Novel membranes made from acellular human dermal tissue or amniotic tissue may have future applications for enhancing tendon and ligament regeneration and adding mechanical support following rupture.

## References

1. Kagel EM, Einhorn TA. Alterations of fracture healing in the diabetic condition. *Iowa Orthop J*. 1996;16:147–52.
2. Easley ME, Trnka HJ, Schon LC, Myerson MS. Isolated subtalar arthrodesis. *J Bone Joint Surg Am*. 2000;82(5):613–24.
3. Frey C, Halikus NM, Vu-Rose T, Ebrahmdadeh E. A review of ankle arthrodesis: predisposing factors to nonunion. *Foot Ankle Int*. 1994;15(11):581–4.
4. Pape HC, Evans A, Kobbe P. Autologous bone graft: properties and techniques. *J Orthop Trauma*. 2010;24 Suppl 1:S36–40.
5. DeOrto JK, Farber DC. Morbidity associated with anterior iliac crest bone grafting in foot and ankle surgery. *Foot Ankle Int*. 2005;26(2):147–51.
6. Keating JF, McQueen MM. Substitutes for autologous bone graft in orthopaedic trauma. *J Bone Joint Surg*. 2001;83(1):3–8.
7. Hollinger JO, Hart CE, Hirsch SN, Lynch S, Friedlaender GE. Recombinant human platelet-derived growth factor: biology and clinical applications. *J Bone Joint Surg Am*. 2008;90 Suppl 1:48–54.
8. DiGiovanni CW, Lin SS, Baumhauer JF, Daniels T, Younger A, Glazebrook M, et al. Recombinant human platelet-derived growth factor-BB and beta-tricalcium phosphate (rhPDGF-BB/beta-TCP): an alternative to autogenous bone graft. *J Bone Joint Surg Am*. 2013;95(13):1184–92.
9. Reddi AH. Bone and cartilage differentiation. *Curr Opin Genet Dev*. 1994;4(5):737–44.
10. Bibbo C, Patel DV, Haskell MD. Recombinant bone morphogenetic protein-2 (rhBMP-2) in high-risk ankle and hindfoot fusions. *Foot Ankle Int*. 2009;30(7):597–603.
11. DeVries JG, Nguyen M, Berlet GC, Hyer CF. The effect of recombinant bone morphogenetic protein-2 in revision tibiotalar arthrodesis: utilization of the Retrograde Arthrodesis Intramedullary Nail database. *J Foot Ankle Surg Off Publ Am Coll Foot Ankle Surg*. 2012;51(4):426–32.
12. Fourman MS, Borst EW, Bogner E, Rozbruch SR, Fragomen AT. Recombinant human BMP-2 increases the incidence and rate of healing in complex ankle arthrodesis. *Clin Orthop Relat Res*. 2014;472(2):732–9.
13. Kanakaris NK, Mallina R, Calori GM, Kontakis G, Giannoudis PV. Use of bone morphogenetic proteins in arthrodesis: clinical results. *Injury*. 2009;40 Suppl 3:S62–6.
14. Schubert JM, DiDomenico LA, Mendicino RW. The utility and effectiveness of bone morphogenetic protein in foot and ankle surgery. *J Foot Ankle Surg Off Publ Am Coll Foot Ankle Surg*. 2009;48(3):309–14.
15. Baums MH, Schultz W, Kostuj T, Klinger HM. Cartilage repair techniques of the talus: an update. *World J Orthop*. 2014;5(3):171–9.
16. Wang CT, Lin YT, Chiang BL, Lin YH, Hou SM. High molecular weight hyaluronic acid down-regulates the gene expression of osteoarthritis-associated cytokines and enzymes in fibroblast-like synoviocytes from patients with early osteoarthritis. *Osteoarthritis Cartil/OARS Osteoarthritis Res Soc*. 2006;14(12):1237–47.
17. Mei-Dan O, Maoz G, Swartzon M, Onel E, Kish B, Nyska M, et al. Treatment of osteochondritis dissecans of the ankle with hyaluronic acid injections: a prospective study. *Foot Ankle Int*. 2008;29(12):1171–8.
18. Mei-Dan O, Carmont MR, Laver L, Mann G, Maffulli N, Nyska M. Platelet-rich plasma or hyaluronate in the management of osteochondral lesions of the talus. *Am J Sports Med*. 2012;40(3):534–41.
19. Mitchell ME, Giza E, Sullivan MR. Cartilage transplantation techniques for talar cartilage lesions. *J Am Acad Orthop Surg*. 2009;17(7):407–14.
20. Gavenis K, Schmidt-Rohlfing B, Mueller-Rath R, Andereya S, Schneider U. In vitro comparison of six different matrix systems for the cultivation of human chondrocytes. *In Vitro Cell Dev Biol Anim*. 2006;42(5–6):159–67.
21. Baums MH, Heidrich G, Schultz W, Steckel H, Kahl E, Klinger HM. Autologous chondrocyte transplantation for treating cartilage defects of the talus. *J Bone Joint Surg Am*. 2006;88(2):303–8.
22. Aurich M, Bedi HS, Smith PJ, Rolauuffs B, Muckley T, Clayton J, et al. Arthroscopic treatment of osteochondral lesions of the ankle with matrix-associated chondrocyte implantation: early clinical and magnetic resonance imaging results. *Am J Sports Med*. 2011;39(2):311–9.
23. Giannini S, Buda R, Vannini F, Cavallo M, Grigolo B. One-step bone marrow-derived cell transplantation in talar osteochondral lesions. *Clin Orthop Relat Res*. 2009;467(12):3307–20.
24. Giannini S, Buda R, Cavallo M, Ruffilli A, Cenacchi A, Cavallo C, et al. Cartilage repair evolution in post-traumatic osteochondral

- lesions of the talus: from open field autologous chondrocyte to bone-marrow-derived cells transplantation. *Injury*. 2010;41(11):1196–203.
25. Pittenger MF, Mackay AM, Beck SC, Jaiswal RK, Douglas R, Mosca JD, et al. Multilineage potential of adult human mesenchymal stem cells. *Science*. 1999;284(5411):143–7.
  26. Mishra A, Harmon K, Woodall J, Vieira A. Sports medicine applications of platelet rich plasma. *Curr Pharm Biotechnol*. 2012;13(7):1185–95.
  27. Alsousou J, Thompson M, Hulley P, Noble A, Willett K. The biology of platelet-rich plasma and its application in trauma and orthopaedic surgery: a review of the literature. *J Bone Joint Surg*. 2009;91(8):987–96.
  28. de Vos RJ, Weir A, van Schie HT, Bierma-Zeinstra SM, Verhaar JA, Weinans H, et al. Platelet-rich plasma injection for chronic Achilles tendinopathy: a randomized controlled trial. *JAMA*. 2010;303(2):144–9.
  29. de Jonge S, de Vos RJ, Weir A, van Schie HT, Bierma-Zeinstra SM, Verhaar JA, et al. One-year follow-up of platelet-rich plasma treatment in chronic Achilles tendinopathy: a double-blind randomized placebo-controlled trial. *Am J Sports Med*. 2011;39(8):1623–9.
  30. Monto RR. Platelet rich plasma treatment for chronic Achilles tendinosis. *Foot Ankle Int*. 2012;33(5):379–85.
  31. Gaweda K, Tarczyska M, Krzyzanowski W. Treatment of Achilles tendinopathy with platelet-rich plasma. *Int J Sports Med*. 2010;31(8):577–83.
  32. Filardo G, Kon E, Di Matteo B, Di Martino A, Tesei G, Pelotti P, et al. Platelet-rich plasma injections for the treatment of refractory Achilles tendinopathy: results at 4 years. *Blood Transfus*. 2014;12(4):533–40.
  33. Owens Jr RF, Ginnetti J, Conti SF, Latona C. Clinical and magnetic resonance imaging outcomes following platelet rich plasma injection for chronic midsubstance Achilles tendinopathy. *Foot Ankle Int*. 2011;32(11):1032–9.
  34. Schepull T, Kvist J, Norrman H, Trinks M, Berlin G, Aspenberg P. Autologous platelets have no effect on the healing of human achilles tendon ruptures: a randomized single-blind study. *Am J Sports Med*. 2011;39(1):38–47.
  35. Sanchez M, Anitua E, Azofra J, Andia I, Padilla S, Mujika I. Comparison of surgically repaired Achilles tendon tears using platelet-rich fibrin matrices. *Am J Sports Med*. 2007;35(2):245–51.
  36. Filardo G, Presti ML, Kon E, Maccacci M. Nonoperative biological treatment approach for partial Achilles tendon lesion. *Orthopedics*. 2010;33(2):120–3.
  37. Leahy M. PRP effective for acute lateral ankle ligament injuries. AAOS Now [Internet]. 2014 Apr [cited 2014 Oct 15]. Available from: <http://www.aaos.org/news/aaosnow/apr14/clinical5.asp>
  38. Xu X, Hu M, Liu J, Zhu Y, Wang B. Minimally invasive reconstruction of the lateral ankle ligaments using semitendinosus autograft or tendon allograft. *Foot Ankle Int*. 2014;35(10):1015–21.
  39. Miller AG, Raikin SM, Ahmad J. Near-anatomic allograft tenodesis of chronic lateral ankle instability. *Foot Ankle Int*. 2013;34(11):1501–7.
  40. Park YS, Sung KS. Surgical reconstruction of chronic achilles tendon ruptures using various methods. *Orthopedics*. 2012;35(2):e213–8.
  41. Hanna T, Dripchak P, Childress T. Chronic achilles rupture repair by allograft with bone block fixation: technique tip. *Foot Ankle Int*. 2014;35(2):168–74.
  42. Mook WR, Parekh SG, Nunley JA. Allograft reconstruction of peroneal tendons: operative technique and clinical outcomes. *Foot Ankle Int*. 2013;34(9):1212–20.
  43. Bussewitz BW, Hyer CF. Interference screw fixation and short harvest using flexor digitorum longus (FDL) transfer for posterior tibial tendon dysfunction: a technique. *J Foot Ankle Surg Off Publ Am Coll Foot Ankle Surg*. 2010;49(5):501–3.
  44. Lee DK. Achilles tendon repair with acellular tissue graft augmentation in neglected ruptures. *J Foot Ankle Surg Off Publ Am Coll Foot Ankle Surg*. 2007;46(6):451–5.
  45. Lee DK. A preliminary study on the effects of acellular tissue graft augmentation in acute Achilles tendon ruptures. *J Foot Ankle Surg Off Publ Am Coll Foot Ankle Surg*. 2008;47(1):8–12.
  46. Liden BA, Simmons M. Histologic evaluation of a 6-month GraftJacket matrix biopsy used for Achilles tendon augmentation. *J Am Podiatr Med Assoc*. 2009;99(2):104–7.
  47. Barboni B, Russo V, Curini V, Mauro A, Martelli A, Muttini A, et al. Achilles tendon regeneration can be improved by amniotic epithelial cell allotransplantation. *Cell Transplant*. 2012;21(11):2377–95.

Abdulhameed Alattar and Shaju Kareem

## Abstract

Ankle injuries are the most common among the sports injuries, of which ankle sprains and recurrent ankle sprains are more frequent. This chapter discusses various aetiology and the pathomechanical factors responsible for the ankle sprain, grading of ankle sprains and their management at all the stages of recovery till the return to sports or competition. Long term follow up studies have demonstrated, functional treatment superior to surgical repair. The criteria in progressing to each stage is very crucial and is tabulated at each phases of tissue healing. Functional progression of early mobilisation with external ankle support is emphasised throughout the chapter. As chronic ankle instability is important sequelae of acute ankle injury, the preventive program is described to the end of the chapter.

## Keywords

Ankle injuries • Ankle sprain • Chronic ankle instability • Functional treatment • Early mobilisation • Proprioceptive Exercises • Prophylactic ankle support • Functional progression • Return to sports • Preventive ankle programme

## Introduction

This chapter addresses the rehabilitation and return to sports and competition on the common example of ankle sprains. Ankle was found to be the most common site of injury in sports and other physically active individuals. Of all the injuries presented to the emergency department 22 % were ankle injuries. Of that the ankle sprains were more common than the fractures with a ratio of sprain to the fractures as 8:1 [2]. The highest rates of ankle sprains were found in girls aged

10–14 years. In USA it is estimated to have one ankle sprain in every 10,000 people daily [1]. Of all the ankle sprains 80 % accounts for lateral sprains 16 % syndesmosis sprain and 4 % medial ankle sprains [2].

After a sprain, 30 % will have persistent residual symptom and decreased sporting performance and high risk of recurrence. Similarly after an ankle fracture symptoms like pain, swelling and stiffness persist into long-term [3]. The most common predisposing factor to lateral ankle sprain is history of at least one previous ankle sprain. Individual who suffer repetitive ankle sprain develop chronic ankle instabilities which can be either a mechanical instability or functional instability or a combination of both [4].

For acute ankle sprains no benefit of surgical repair has been shown over functional treatment with respect to repeat injury or return to function [5]. Functional treatment includes the use of removable and variable immobility devices with early exercises and early weight bearing activities as tolerated. An effective rehabilitation after a sprain or a fracture managed

A. Alattar (✉)  
Consultant Sports Physician, Physical Medicine and Rehabilitation  
Department, Rashid Hospital, Dubai Health Authority,  
Dubai 4545, United Arab Emirates  
e-mail: [amalattar@dha.gov.ae](mailto:amalattar@dha.gov.ae)

S. Kareem  
Sports Physiotherapist, NAS Sports Medicine & Rehabilitation Centre,  
NAS Sports Complex, Nad Al Sheba, Dubai, United Arab Emirates  
e-mail: [shajukhassan@gmail.com](mailto:shajukhassan@gmail.com)

conservatively or surgically often determines the success. The goal is to return the athlete to participate in sports as early as possible while allowing the injured tissue to heal without compromising it by further injury [2]. During the functional rehabilitation, the type of injury, severity, healing time, type and level of activity, foot type, biomechanics of lowerlimbs, and external support as bracing or taping is to be considered.

## Etiology and Pathomechanics

Important for the decision making of the treatment and rehabilitation of an ankle injury is the knowledge of the etiology, history, and pathomechanism. Lateral ankle sprains most commonly occurs due to excessive inversion and plantar-flexion of the hindfoot with external rotation of the lower leg. Anterior talofibular ligament (ATFL) is the first ligament to be damaged followed by the Calcaneofibular ligament (CFL). Injury to Posterior Talofibular ligament (PTFL) is seen only in severe ankle injury and often accompanied by fracture, dislocation or both [1].

Medial ligament Injuries are occasional as it requires more force to sprain this strong ligament. Eversion injuries can sprain this ligament and It takes more time to rehabilitate the medial ligament injuries when compared to the lateral ligament. Occasionally both ligaments may be injured in an ankle sprain or medial ligament injury can accompany fractures of medial malleolus, talar dome and the articular surfaces. Syndesmosis injury is less common, occurs with external rotation and or hyper dorsiflexion of the ankle, leading to the widening of the mortise resulting in the disruption of syndesmosis and talar instability.

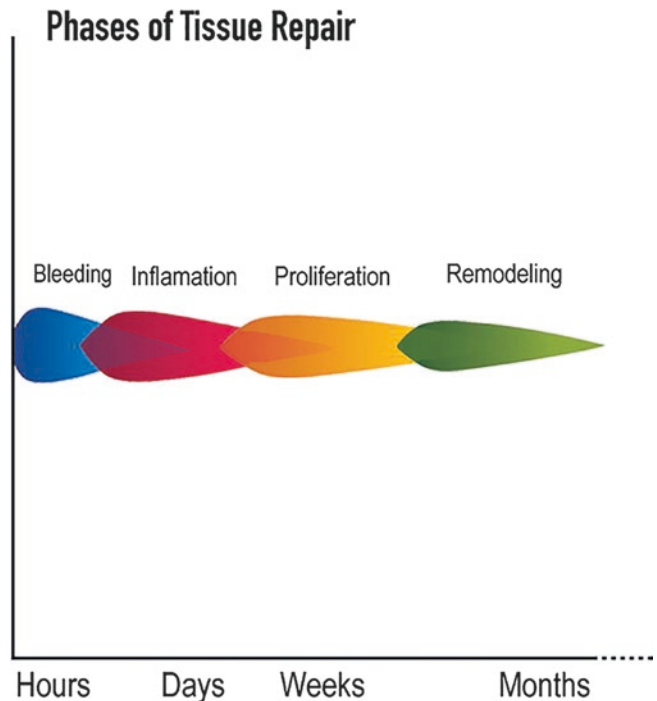
A fracture affecting one or more malleoli can be difficult to be indentified from the sever ligament injuries as the mechanism of injury is the same. Displaced fracture requires surgery and a rehabilitation program should follow a surgical procedure or upon the cast removal. The rehabilitation guidelines are the same as for the ankle sprain.

## Management

An understanding of the body's response to injury is important to design a rehabilitation program. The ligament injuries are graded or classified based on the severity of the injury, number of ligaments involved, or based on clinical presentation. The American Medical Association (AMA) classifies the ligament injuries in Grade 1–3 (Table 14.1).

**Table 14.1** American Medical Association (AMA) ligament injuries classification

Grade 1	Ligaments stretched
Grade 2	Ligaments partially torn
Grade 3	Ligaments completely torn



**Fig. 14.1** Tissue repair phases and timescale

Principles of rehabilitation remains the same for all the grades and follow the tissue repair phases and time scales (Fig. 14.1). After an initial insult, there is bleeding and tissue damage. The inflammatory response is initiated followed by proliferative and maturation phase. The phases of rehabilitation coincide with the tissue healing phases. The time frame for each phase varies depending on the grades of injury (Table 14.2). The goals of each phase vary and overlap with the next phases.

## Acute Stage

The main goals for treatment will be to protect the ankle from further injury, provide an environment to heal, and to reduce pain and control swelling. The ankle may be rested in non-weight bearing or with controlled weight bearing as tolerated (Table 14.3).

PRICE (Protection, Rest, Ice, Compression, and Elevation) is used at this phase to reduce pain and control swelling and to prevent further damages. Based on level of pain, severity of injury and level of muscle activation the ankle is protected either by strapping, ankle braces, or removable boots (Fig. 14.2). Crutches may be used to rest the ankle with appropriate weight bearing. Progress from non-weight bearing to partial weight bearing as tolerated. Patient is taught crutch walking including ascending and descending stairs. The early controlled weight bearing as tolerated (WBAT) limits muscle atrophy, limits proprioceptive loss, limits circulatory stasis and chronic pain. Protected motion facilitates collagen alignment and stronger healing.

Studies have shown that wet ice is more effective in producing analgesia [6]. It is more beneficial when used along with compression using wet bandages to reduce pain and swelling. Patient is advised to apply ice for 10 min every four waking hours. Ice and compression can be provided simultaneously and conveniently using simple modalities like Cryo-Cuff, Game Ready, or an Icebag with compression (Fig. 14.3).

Compression can be provided by elastic straps or wraps, patient needs to be taught the application as re-application may be necessary to maintain the compression. Horseshoe felt is used behind the malleolus to prevent further swelling. Elevating the injured ankle above the heart level is advised

initially to prevent swelling or further bleeding. Lymphatic drainage done proximal to distal with the legs elevated initiated at this stage is highly useful in dispersing the fluid. Toe movements are encouraged many times a day. TED stocking extending from the toes to mid thigh may be advised to prevent venous stasis and to encourage venous return.

**Precaution** No active or passive inversion and eversion past neutral, for a lateral and medial sprain respectively and no end range plantar or dorsiflexion is encouraged. For syndesmosis injury, avoid active/passive end-range dorsiflexion and eversion, Night Splits are advised to be worn to further protect the injured ligament.

**Table 14.2** Time frame of tissue healing phases

Phases	Stages	Duration
Phase 1	Acute	1–3 days
Phase 2	Sub Acute	2 days–1 week
Phase 3	Rehabilitative	1 week–6 weeks
Phase 4	Functional/Return to play	2 weeks–months

**Table 14.3** Goals of acute stage in ankle sprain

Goals: acute stage (Phase 1)
• Protection of injured joint
• Pain management
• Control inflammation and swelling
• Progress weight bearing as tolerated

### Sub-acute Stage

**Criteria to Progress** Once the pain and swelling is controlled with a near normal gait pattern treatment can be progressed to the subacute stage (Table 14.4).

The duration of this sub-acute stage varies with the severity of injury and can be from 2 days till a week (Table 14.5). Treatment at this stage is aimed to reduce the pain and swelling, and to increase the painfree range of motion of the injured ankle (Table 14.6). Strengthening exercises can be initiated at this stage along with the proprioceptive exercises, Progress the weight bearing on the injured limb as tolerated



**Fig. 14.2** Protecting orthotics. (a) Ankle Brace, as the Aircast Ankle Brace (DJO Global); (b) removable boot, as the Aircast Walker (DJO Global)



**Fig. 14.3** Cryotherapy with compression. (a) Cryo-Cuff, (b) Game Ready

**Table 14.4** Criteria to progress to phase 2 (Sub-Acute Stage)

Criteria to progress to Phase 2 (Sub-Acute Stage) (Table 14.2)
Controlled swelling and pain
Near normal gait pattern with crutches

**Table 14.5** Duration of the Sub-Acute Stage. The duration depends on the grade of ligament injury

Sub-Acute Stage duration	
Grade 1	2–4 days
Grade 2	3–5 days
Grade 3	4–8 days

**Table 14.6** Goals of Sub-Acute Stage

Goals: Sub-Acute Stage
• Decrease pain and swelling
• Initiate or increase painfree ROM
• Begin Strengthening exercises
• Begin proprioceptive exercises
• Provide protective support as needed

with protective support as needed. Depending on severity, the weight bearing on crutches can progressed from two crutches to single crutch to weaning of crutches or continue PWB throughout in the phase. Emphasis on gait training with normal gait pattern without limbing, Cryotherapy with compression is continued to control pain and swelling. Electrotherapy modalities like Pulsed Ultrasound, LASER and Interferential or TENS Therapy may be useful at this stage. Contrast bath can be used with the clearance from the physician. Gentle cross friction massage to the injured ligament can reduce the pain and help in the orientation of col-

lagen fibers during the healing stage. Lymphatic drainage and use of TED stockings is continued to prevent circulatory complications.

Neuro-muscular stimulation to the muscles with the foot on the floor to avoid any movement at the ankle is found to be advantageous in the early stage to prevent muscle atrophy (Fig. 14.4). Painfree isometric exercises to all the muscle group of the injured ankle is started and progressed to eccentric and concentric exercises with elastic resistance bands or cords or using ankle weights. Closed kinetic chain exercises in sitting like the heel raise exercise progressing to standing is also advised later in the stage. The Tibial and Peroneal group muscles are emphasized.

Aquatic exercises has profound effect at this stage to train a near normal gait and muscle activation. The buoyancy reduces the compressive force and supports injured tissues. The underwater cameras in the hydrotherapy pool can enable the athlete to have a visual feed back of his gait in sagittal and frontal plane.

Neuromuscular deficits are evident following the ankle injuries and will predispose to chronic functional instability [7, 8]. Proprioceptive training can be initiated respecting the weight bearing status, progression is made from stable surface to unstable surface. These activities are done in sitting, progressing to standing (Fig. 14.5). Rocker board activities for plantar flexion/Dorsiflexion is done initially at a painfree range, It may be required to block certain range of movement to prevent stressing the injured ligament. See the precaution section for each stage.

Painfree self stretching of Achilles tendon using a towel with no eversion/inversion in sitting can be taught to the patient. Low load long duration stretching that gently promotes tissue creep is effective. Grade 1 and 2, mobilization



**Fig. 14.4** Neuromuscular stimulation and Isometric Exercises in the Sub-Acute Stage. (a) Neuro-Muscular Stimulation, (b) toes exercises, (c) sitting heel raise, (d) standing heel raise

techniques and Manual therapy like Movement with mobilization techniques may be useful to gain painfree dorsiflexion and plantar-flexion [9].

Cardiovascular fitness of the athlete is maintained using an upper extremity ergometric exercises. The parameters of the exercises are prescribed based on the American College Of Sports Medicine guidelines. The injured ankle is protected from unwanted stress on the ligament. As ankle is a part of the lower limb kinetic chain initial level core stability exercises are started and progressed at this stage. Activities while sitting on Swiss ball can be a good initial core exercises as well as proprioceptive exercises for the injured ankle. Exercises and gait training in water improves the functional stability.

**Precautions** Precaution at this stage include no active or passive inversion past neutral and no active or passive plantar-flexion past resting position for lateral sprains. No active/passive end range dorsiflexion and no active/passive external rotation/eversion for syndesmosis sprains and medial sprains

## Rehabilitation Stage

### Criteria to Progress to Rehabilitation Stage

The next stage in treatment , the rehabilitation stage is started if the ankle has minimal swelling and pain with a reasonable gait pattern (Table 14.7).

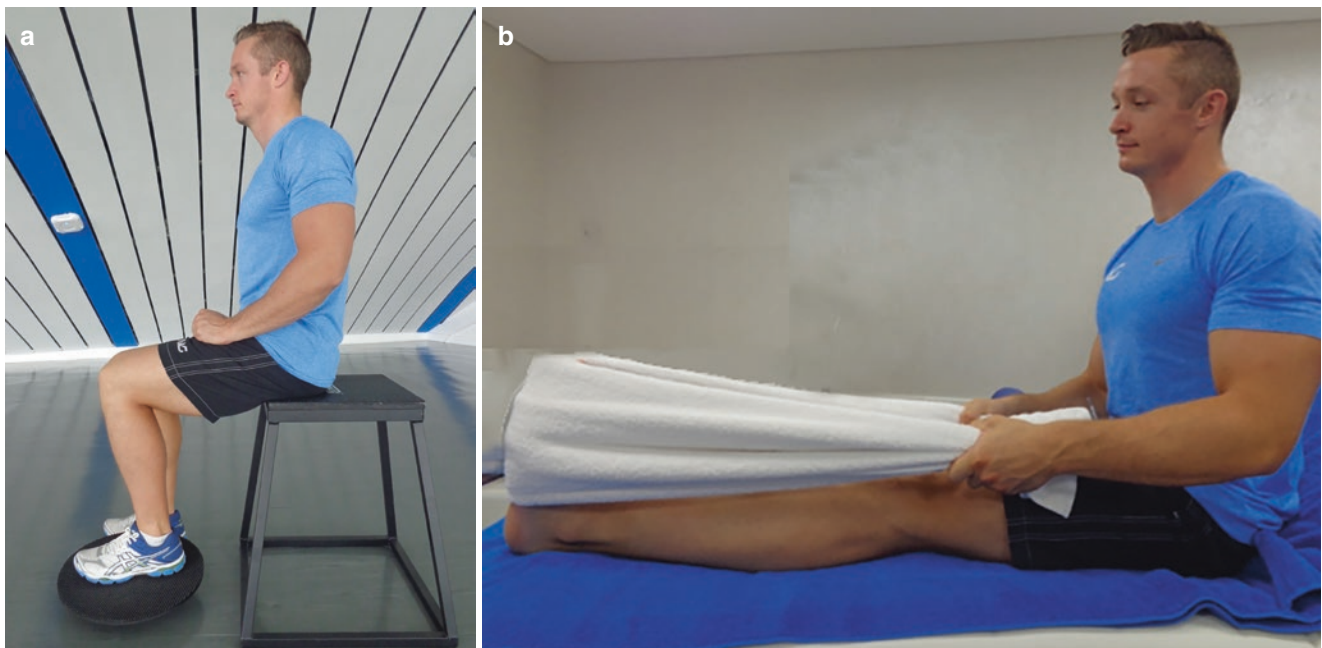
At this stage, which might go from week 1 to week 6 depending on the grade of injury (Table 14.8), we aim for a normal gait pattern with full weight bearing, full pain free range of motion with reasonable muscular strength around the injured ankle. Proprioceptive and core stability exercises are progressed to intermediate and advanced level maintaining the cardiovascular fitness. Any residual pain and swelling at the end of the exercise is managed (Table 14.9).

Early mobilization facilitated by external ankle support appears to be preferred treatment for a lateral ankle ruptures in elite athlete [10]. The weight bearing can be progressed to full weight bearing at this stage. The use of orthosis such as customized shoe inserts provides somatosensory benefits, may correct alignment of rear and fore foot for proper muscle activation and reduce unnecessary strain on the injured ligament [11]. The injured ankle should be supported by a semi rigid orthosis while walking and exercising to enable functional activities while protecting the injured ligament [12–14].

Progress with the range of motion exercises at the ankle in all direction, limited to prevent the stretching of the injured ligament (see the precaution at this phase). Achilles tendon stretching intensity increased from bilateral to unilateral

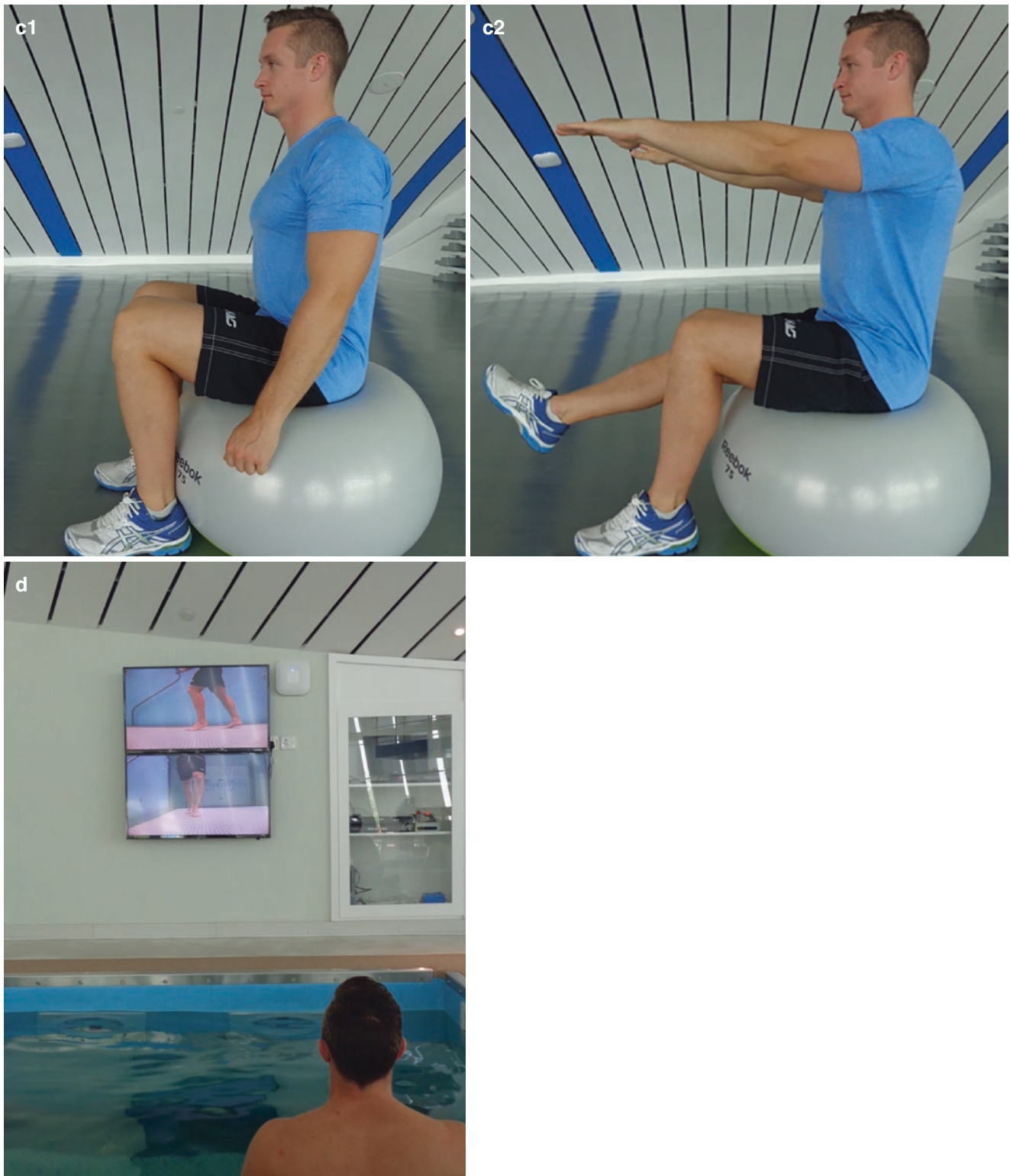
**Table 14.7** Criteria to progress to Phase 3 (Rehabilitation Stage)

Criteria to progress to Phase 3 (Rehabilitation Stage)
• Minimal or controlled swelling and pain
• Normal gait pattern



**Fig. 14.5** Functional stability training. (a) Wobble board sitting, (b) self-stretching, (c) Core stability with the Swiss Ball, (d) Aquatic Exercises and gait training in Hydro-Works





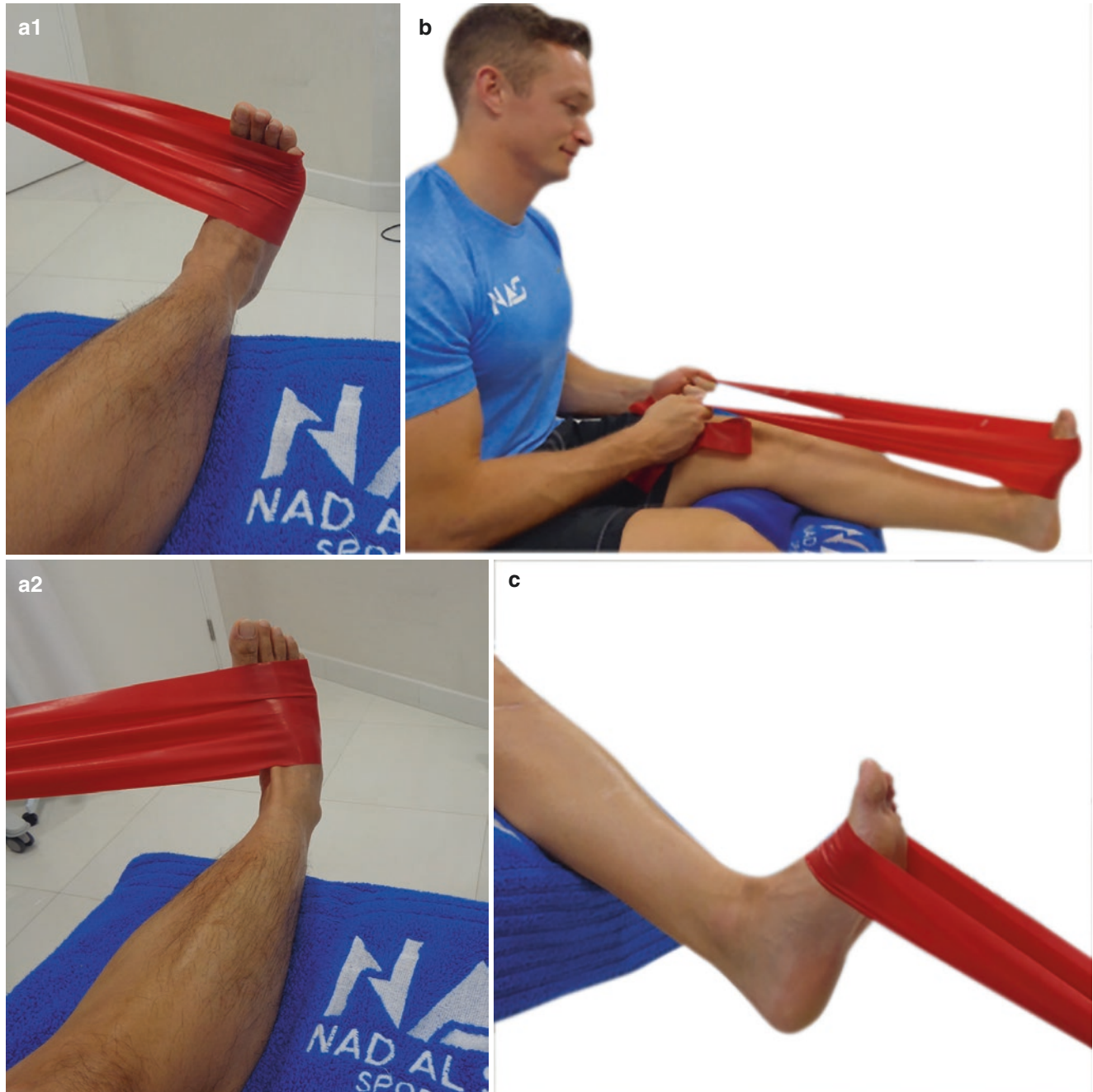
**Fig. 14.5** (continued)

**Table 14.8** Duration of the Rehabilitation Stage. The duration depends on the grade of ligament injury

Rehabilitation Stage Duration	
Grade 1	1 week
Grade 2	2 week
Grade 3	3 week–6 weeks

**Table 14.9** Goals of Rehabilitation Stage

Goals: Rehabilitation stage
• Increase Pain Free ROM
• Progress strengthening exercises
• Progress proprioceptive training
• Pain free full weight bearing and decompensated gait
• Increase pain free ROM
• To manage any residual pain and swelling



**Fig. 14.6** Elastic band exercises. The pictures (a–c) show the different options for elastic band exercises

stretching in standing. Manual mobilization techniques can aid in improving dorsiflexion and plantar flexion. Strengthening of weak muscles aids rapid recovery and prevent recurrence of ankle injuries [15]. Progress the intensity of eccentric and concentric exercises to all the muscle groups, emphasizing the tibial and the peroneal group of muscles. Avoid the compensatory rotation at the hip and knee to target the ankle muscles. Exercises using manual resistance by the therapist or by elastic resistive bands and ankle cuffs weights are suggested (Fig. 14.6). Daily Adjustable Progressive Resistant Exercises (DAPRE) program can be used for strength training in ankle [16]. PNF techniques using slow reversals and rhythmic stabilization can help to regain the agonistic and antagonistic function and more stabilization of the ankle. Isokinetic testing and training can be initiated at this stage [17]. Continuous monitoring of strength of the ankle muscles are done by a simple hand held dynamometer. Hydrotherapy can be progressed with the resistance and functional exercise can be introduced in water. The turbulence used by water jets can add resistance to the exercises in a hydrotherapy pool (Fig. 14.7).

Proprioceptive exercises can be progressed from the exercises done in sitting to standing, bilateral to single leg activities, exercising on stable surface to unstable surfaces, activities with eyes open to eyes closed, with and without distraction (Fig. 14.7). The single leg balance and postural sway can be tested and trained on a Balance master (Fig. 14.7). Core stability can be progressed from Intermediate to advanced level, and can be incorporated into functional activities. At this stage it is also important to check hindfoot correcting and supporting orthosis, as insoles or semi-rigid braces (Fig. 14.8).

**Precaution** Include no passive end range Inversion and plantar flexion for lateral ligament sprains and no passive end range dorsiflexion & passive end range eversion for syndesmotom & medial injuries

## Functional Stage

### Criteria to Progress to Functional Stage (Phase 4)

Once full range of motion is achieved with 80% strength of the muscles controlling the ankle compared to unaffected side and if a single leg balance can be maintained for 30 seconds with the eyes closed, the patient is ready for the next stage of rehabilitation, the functional stage or return to sports phase (Table 14.10).

The duration of this phase can be extended to few months depending on the severity of the injury (Table 14.11). The goal is to prepare the athlete for return to full activity in sports. The individual goals are summarized in the Table 14.12.

A functional progression is a succession of activities that simulate actual motor and sports skills, enabling the athlete

to acquire the skills needed to perform athletic endeavors safely and effectively. It must involve gradual progression of functional activities and includes plyometrics and speed agility training specific to the athletes specific sports activity (Fig. 14.9). The activities are designed so that it slowly increases the stress on injured structures. Exercises include walking with progression to jogging and then to running in straight line. That is progressed to running forward and backward and sprinting. Equipments such as cones, ladder and hurdles are used to advance the difficulty. Plyometrics enable a muscle to reach maximum force in the shortest possible time. The muscle is loaded with an eccentric (lengthening) action, followed immediately by a concentric (shortening) action. This includes activities like box jumping of different heights, hopping forward and backward and sideways etc. Speed and agility training is the logical sequence of progressive drills for pre-sports conditioning and provide objective criteria for safe return to sports. This includes activities like running in figure of eight, box running, cutting and hopping on command and carioca progressing to quick, intense and dynamic activities (Fig. 14.10).

Prophylactic ankle support with a lace up ankle brace or athletic taping can be done prior to the activities to aid an extra protection. Lace up ankle brace is more beneficial to provide proprioceptive stimulation with no skin complication as the athletic tapes and provides mechanical stability in a cost effective way comparing to the tapes. Braces are easy to use and require no professional support. The core stability and proprioceptive training is progressed to the advanced level and should be incorporated to the functional activities. Frequent assessment of lower limb biomechanics is mandatory and necessary orthotic support is modified.

**Precaution** The precaution includes careful progression of dynamic activities in lateral planes for medial and lateral sprains.

## Criteria to Return to Play

Criteria to return to play includes, being pain free with 85–90% strength compared to normal side and gradual progression of functional activities with sports specific drills done painfree at game speed with a good quality of movement and stability. The athlete can start with practice, half time progressing to full time and then into competition (Table 14.13).

## Rehabilitation Following Ankle Fracture

Ankle sprain can go together with an ankle fracture. Stable ankle fracture is managed by conservative fracture reduction and the unstable fractures may need surgical intervention, in



**Fig. 14.7** Possible exercises in rehabilitation phase. (a) Exercises against the resistance of water turbulence using water jets in hydrotherapy pool, (b) single leg ankle stretches in standing, (c) upper extremity ergometry to maintain and improve cardiovascular fitness, (d) isokinetic testing and training, (e) ankle range of motion, (f) bilat-

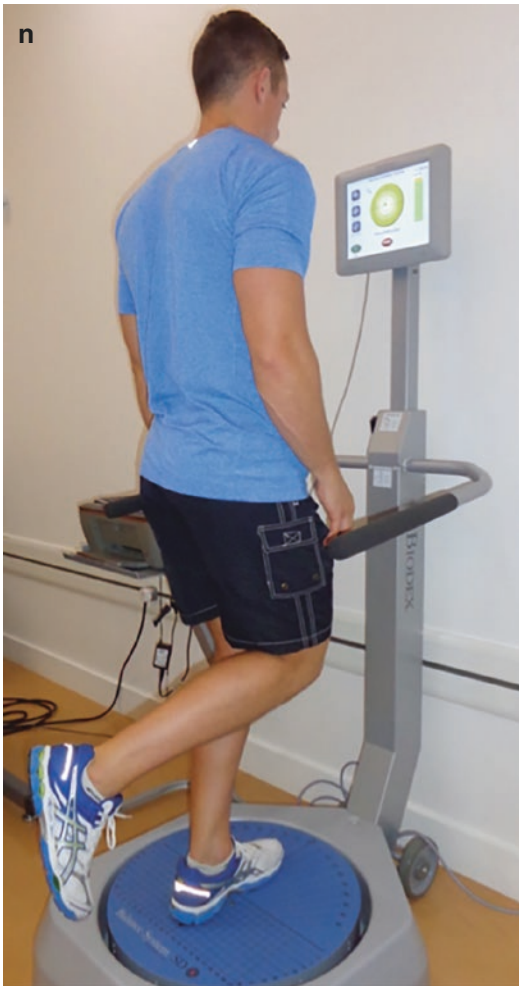
eral heel raise, (g) ankle push ups, (h) lunges with weight, (i) single heel raise, (j) proprioceptive exercises (bilateral), (k) proprioceptive exercises (single leg), (l) proprioceptive exercises (advanced), (m) core stability exercises, (n) balance testing & training using balance master



**Fig. 14.7** (continued)



**Fig. 14.7** (continued)



**Fig. 14.7** (continued)



**Fig. 14.8** Check and choice of orthosis. (a, b) Checking the shoe inserts. (c) Protection by a semi-rigid orthosis

**Table 14.10** Criteria to progress to Phase 4 (Functional Stage)

Criteria to progress to Phase 4 (Functional Stage)
• Full range of motion
• 80% strength of the muscles controlling the ankle compared to unaffected side
• Single leg balance maintained for 30 s with the eyes closed

**Table 14.11** Duration of the Functional Stage or Return to Sports Phase. The duration depends on the grade of ligament injury

Grades	Duration
Grade 1	1–2 weeks
Grade 2	2–3 weeks
Grade 3	3–6 weeks up to 6–12 months

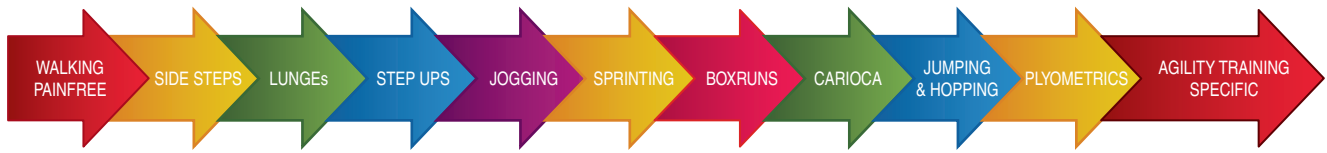
both cases there will be a period of immobilization followed by rehabilitation. During the immobilization period following the surgery, an early introduction of activity may be beneficial. This includes using a removable type of orthosis or brace gentle ankle exercises and early weight bearing. This

**Table 14.12** Goals of Functional Stage

Goals of Functional Stage
• Regain full strength and range of motion in all the planes
• Normalizing the lower limb biomechanics
• Protection and strengthening of any mild joint instability
• Improving fitness, Advanced proprioceptive & Core Stability incorporated into functional activities
• Progress to running, plyometric and agility training
• Sports specific/work specific functional training
• Return to competition/training with the team

has shown possible improvement in function, reduction of pain and swelling and improvement of ankle range of motion. This treatment needs to be applied judiciously in clinical practice to avoid any adverse effects. This is beneficial than complete immobilization in a cast [18]. Following the immobilization period, the treatment includes gradual increase in activities, progression of exercises to improve weight bearing, ankle range of motion, strength, proprioception, and functional training and modalities to manage pain and swelling. The rehabilitation will follow the same guideline as for the ankle sprain mentioned earlier.

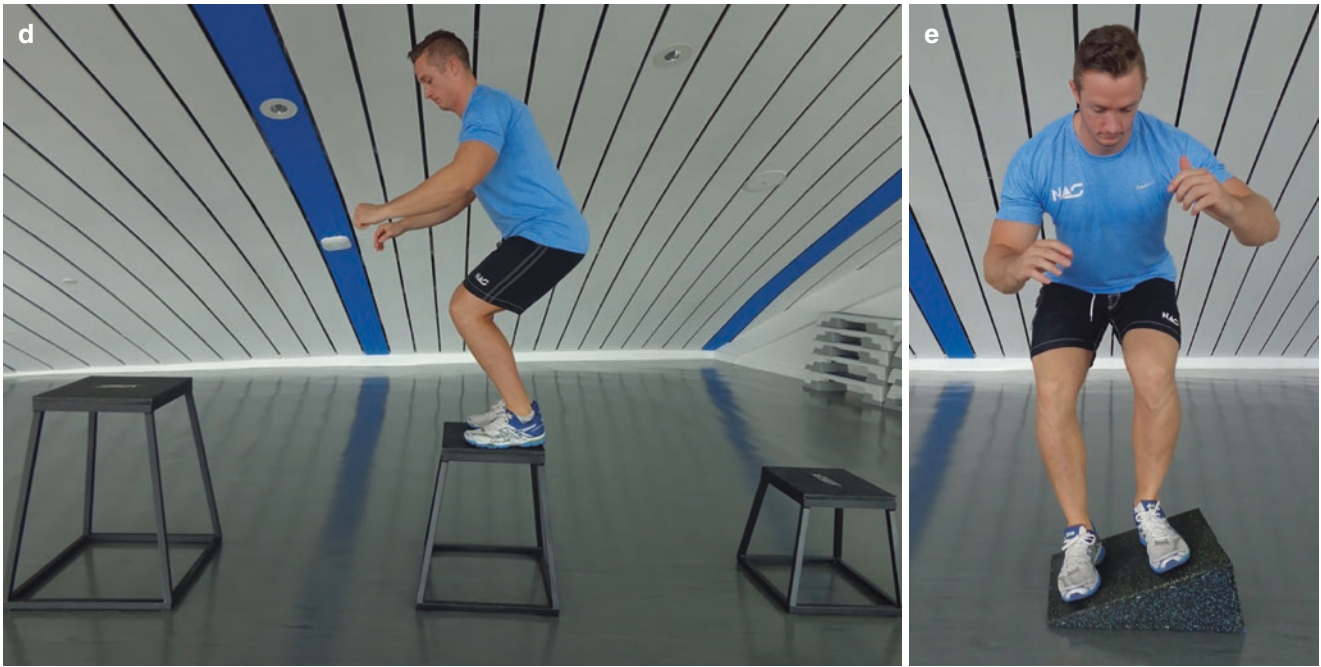




**Fig. 14.9** Agility training stages (sports-specific)



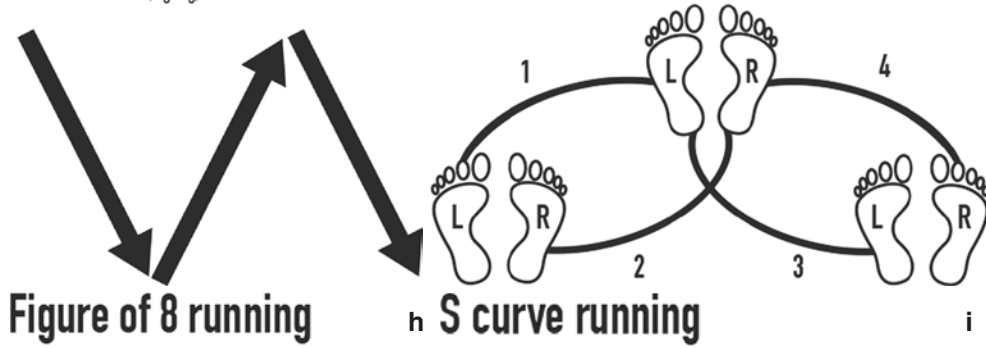
**Fig. 14.10** Exercises in functional stage or return to play phase. (a) Exercises in trampoline, (b) shuttle exercises, (c) jumping in squares, (d) box jumping, (e) jumping & landing on slanding surface, (f) zig zag running, (g) carioca, (h) figure of 8 running, (i) S curve running, (j) combined drilled, (k) cone drills, (l) ladder drills, (m) hopping on command



**Zig Zag Running**  
"Z" course (zigzag)

**f Carioca**

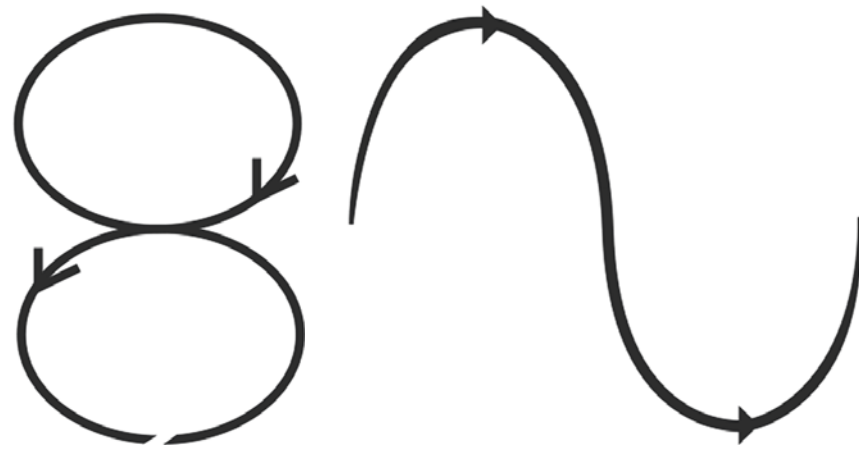
**g**



**Figure of 8 running**

**h S curve running**

**i**



**Fig. 14.10** (continued)

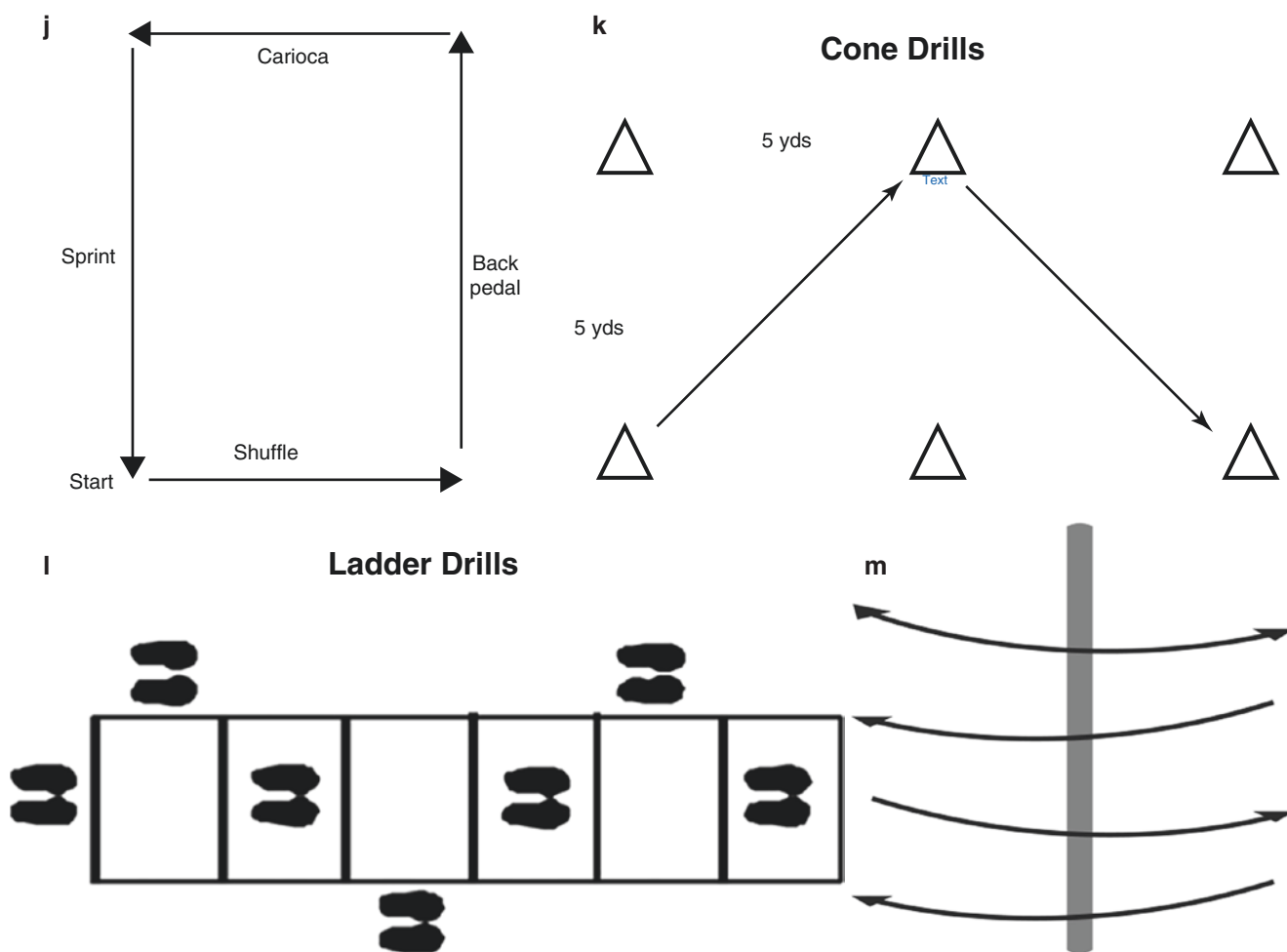


Fig. 14.10 (continued)

**Table 14.13** Criteria to return to play

Criteria to return to play
• Pain free activities
• 85–90% strength compared to normal side
• Gradual progression of functional activities with stability
• Completed sports specific drills at full speed and good quality
• Completed specific testing by the physician

**Prevention**

75% of those sustained ankle injury had a history of ankle sprains not well rehabilitated [19]. 20–40% suffer from chronic ankle instability (CAI) which is the sequelae of acute ankle injury and 50% of the CAI are due to functional instability. Deficits are seen in ankle proprioception, cutaneous sensation, nerve conduction velocity, neuromuscular response time, postural control and strength [20]. Continuing the functional drills with multi-directional balance board activities, strengthening emphasizing the tibial and the pero-

neal muscles and use of appropriate protective support to the ankle with a lace-up ankle brace while playing can prevent the recurrence of ankle injury as well as chronic ankle instability.

**Summary**

- Functional treatment is the treatment of choice for acute ankle sprains.
- Functional rehabilitation provides better results than immobilization.
- The rehabilitation process of an ankle sprain goes through these four stages/phases: acute stage, sub-acute stage, rehabilitation stage, functional stage or return to sports phase.
- Recurrence can be prevented to an extent with ongoing functional rehabilitation with strengthening and proprioceptive exercises and prophylactic ankle support.

- Criteria to return to play includes being pain free with 85–90% strength compared to normal side and gradual progression of functional activities with sports specific drills done painfree at game speed with a good quality of movement and stability. The athlete can start with practice, half time progressing to full time and then into competition.

## References

1. Kannus P, Renström P. Treatment for acute tears of the lateral ligaments of the ankle. Operation, cast, or early controlled mobilization. *J Bone Joint Surg Am.* 1991;73(2):305–12.
2. Ferran NA, Maffulli N. Epidemiology of sprains of the lateral ankle ligament complex. *Foot Ankle Clin.* 2006;11(3):659–62.
3. Lash N, Home G, Fielden J, Devane P, Lash N, Home G, Fielden J, Devane P. Ankle fractures: functional and lifestyle outcomes at 2 years. *ANZ J Sur.* 2002;72:724–30.
4. Tropp H, Odenrick P, Gillquist J. Stabilometry recordings in functional and mechanical instability of the ankle joint. *Int J Sports Med.* 1985;6:180–2.
5. Krips R, van Dijk N, Hakasi T, et al. Long-term outcome of anatomical reconstruction versus tenodesis for the treatment of chronic anterolateral instability of the ankle joint: a multicenter study. *Foot Ankle Int.* 2001;22:415–21.
6. Bleakley CM, McDonough SM, MacAuley DC, Bjordal J. Cryotherapy for acute ankle sprains: a randomised controlled study of two different icing protocols. *Br J Sports Med.* 2006;40(8):700–5.
7. Arnold BL, De La Motte S, Linens S, Ross SE, Arnold BL, De La Motte S, Linens S, Ross SE. Ankle instability is associated with balance impairments: a meta-analysis. *Med Sci Sports Exerc.* 2009;41:1048–62.
8. McKeon PO, Hertel J. Systematic review of postural control and lateral ankle instability, part II: is balance training clinically effective? *J Athl Train.* 2008;43(3):305–15.
9. Collins N, Teys P, Vicenzino B. The initial effects of a Mulligan's mobilization with movement technique on dorsiflexion and pain in subacute ankle sprains. *Man Ther.* 2004;9(2):77–82.
10. Kerkhoffs GM, Rowe BH, Assendelft WJ, Kelly K, Struijs PA, van Dijk CN. Immobilisation and functional treatment for acute lateral ankle ligament injuries in adults. *Cochrane Database Syst Rev.* 2002(3):CD003762.
11. Nigg BM, Nurse MA, Stefanyshyn DJ. Shoe inserts and orthotics for sport and physical activities. *Med Sci Sports Exerc.* 1999;31(7 Suppl):S421–8. Review.
12. Beynon BD, Renström PA, Haugh L, Uh BS, Barker H. A prospective, randomized clinical investigation of the treatment of first-time ankle sprains. *Am J Sports Med.* 2006;34(9):1401–12.
13. Cooke MW, Marsh JL, Clark M, Nakash R, Jarvis RM, Hutton JL, Szczepura A, Wilson S, Lamb SE; CAST trial group. Health Technol Assess. Treatment of severe ankle sprain: a pragmatic randomised controlled trial comparing the clinical effectiveness and cost-effectiveness of three types of mechanical ankle support with tubular bandage. *CAST Trial.* 2009;13(13):iii, ix–x, 1–121.
14. Lamb SE, Marsh JL, Hutton JL, Nakash R, Cooke MW, Collaborative Ankle Support Trial (CAST Group). Mechanical supports for acute, severe ankle sprain: a pragmatic, multicentre, randomised controlled trial. *Lancet.* 2009;373(9663):575–81. doi:10.1016/S0140-6736(09).
15. Thacker SB, Stroup DF, Branche CM, Gilchrist J, Goodman RA, Weitman EA. The prevention of ankle sprains in sports. A systematic review of the literature. *Am J Sports Med.* 1999;27(6):753–60.
16. Mattacola CG, Dwyer MK. Rehabilitation of the ankle after acute sprain or chronic instability. *J Athl Train.* 2002;37(4):413–29.
17. Knight KL. Knee rehabilitation by the daily adjustable progressive resistive exercise technique. *Am J Sports Med.* 1979;7(6):336–7. No abstract available.
18. Perrin DH. Isokinetic exercise and assessment. Champaign: Human kinetics; 1993. p. 59.
19. Smith TO, Davies L. Do exercises improve outcome following fixation of ankle fractures: a systematic review. *Int J Ther Rehabil.* 2006;13(6):273–81.
20. Hintermann B. Biomechanics of the unstable ankle joint and clinical implications. *Med Sci Sports Exerc.* 1999;31 Suppl 7:459–69.

---

## Part II

### Clinics, Lesions, and Diseases

Milena M. Ploeger, Christof Burger,  
and Matthias D. Wimmer

## Abstract

Fractures of the lower leg, ankle, hindfoot, midfoot, and forefoot are common injuries. Etiology involves accidents and fall from heights as well as sports related injuries. Clinical symptoms involve pain, swelling, and hematoma formation as well as limping. A precise and sport discipline focused history and clinical examination is essential. X-Rays are obtained most often, depending on the expected injury, ultrasound, CT or MRI scans can provide helpful additional information to facilitate an appropriate classification. For all fractures it is essential to search for tendon or ligament injuries which need to be addressed as well. An overlooked soft tissue injury poses a potentially irreversible threat to recovery, especially in athletes. Anatomical reconstruction of the joint lines as well as soft tissue repair and balancing is the key to success, allowing a fast individually adapted rehabilitation program bringing our patients back to sports. In professional athletes the rehabilitation time frame might vary strongly and the individual rehabilitation program has to be adjusted to fasten the return to the pre-injury level.

## Keywords

Ankle fracture • Calcaneal fracture • Talar fracture • Jones Fracture • Lisfranc • Chopard

## Acute Fractures (Lower Leg, Ankle, Hindfoot, Midfoot, Forefoot)

### Etiology and Pathomechanism

Fractures of the lower leg, ankle or foot are common [1]. The etiology involves traffic accidents and fall from height but sports related trauma can be involved either. The distinct trauma mechanism, eventually video taped in professional athletes, patients' reports, and X-rays studies, can provide important hints to the injury pattern.

M.M. Ploeger, MD • C. Burger, MD • M.D. Wimmer, MD (✉)  
Department of Orthopaedics and Trauma Surgery, University  
Clinics of Bonn, Sigmund-Freud-Str. 25, Bonn 53113, Germany  
e-mail: [milena.ploeger@ukb.uni-bonn.de](mailto:milena.ploeger@ukb.uni-bonn.de);  
[christof.burger@ukb.uni-bonn.de](mailto:christof.burger@ukb.uni-bonn.de);  
[matthias.wimmer@ukb.uni-bonn.de](mailto:matthias.wimmer@ukb.uni-bonn.de)

### Symptoms

Clinical symptoms involve pain, swelling, and hematoma formation. Especially spontaneous partial or non-weight bearing, limping, and disability for competition may be seen.

### History

Especially in athletes, pain and the pain course (sudden appearance or slowly evolving), training amount, injury mechanism, equipment type, and other informations are essential. Especially in potential chronic cases a profound history evaluation cannot be stressed enough.

### Clinical Investigation

It is essential to investigate the entire, unclothed ankle and lower leg as well as the foot for pain caused by pressure or crepitation. It is important to assess soft tissues injuries and

the status of blood perfusion. Potential nerve injuries have to be checked as well as a sign of a compartment syndrome. The knee joint has to be included in the clinical investigation not to miss the proximal fibula. Severely malposition or dislocation of the joint requires immediate reduction and splinting. Even though, the initial clinical examination should be performed carefully until fractures are ruled out by radiographs.

## Imaging

To evaluate the ankle joint, x-rays in two planes (anterior-posterior (mortise-view) and lateral) should be performed. According to the suspected injury pattern, additional images should be added. A dorso-plantar and lateral projection of the foot and the Saltzman view can provide essential information about foot configuration, potential fractures and inframalleolar alignment [2]. The Harris projection (oblique projection) should be added to evaluate the calcaneus more detailed. If a proximal fibular fracture is suspected, x-rays of the knee in two planes should be performed as well.

In elective or chronic cases x-rays of the ankle joint and the foot are performed with weight bearing. A computer tomography (CT) scan can be very helpful improving classification of the distinct fracture pattern and planning of surgery [3].

In adults magnetic resonance imaging (MRI) is usually not indicated to exclude fractures. Nevertheless to diagnose potential injuries of tendons, ligament and cartilage, MRI is often used as a very powerful additional technique, especially in professional or pediatric athletes [4].

Ultrasound can help in an acute setting to diagnose a tendon, ligament or muscle injury fast.

## Lower Leg and Ankle Fractures

Although sports equipment and preventive sports have reduced the amount of foot and ankle sports lesions in the last years, sports with jumpings and landings, rotational actions, uneven ground, etc, like football, soccer, volleyball, basketball, athletics, etc. can be affected by lower leg and ankle fractures.

### Classification

Lower leg and ankle fractures can be simply described as uni-, bi-, or trimalleolar fractures.

The Danis-Weber classification modified by the AO Foundation is a commonly used classification [5]:

**Danis-Weber Type A:** below the syndesmosis

**Danis-Weber Type B:** at the level of the syndesmosis

**Danis-Weber Type C:** above the syndesmosis (Fig. 15.1).

It is necessary to search for additional injuries:

1. A high fibula fracture (Maisonneuve-fracture)
2. A bony avulsion of the posterior tibia (Volkman-fragment)
3. A bony avulsion of the anterior tibia in adolescents (Tillaux-Chaput-fragment) and
4. An avulsion on the ventral syndesmosis on the fibular side (Wagstaffe-fragment) [6, 7].

A more precise classification (also for studies and international comparison) is the current AO classification for ankle fractures: 44-A1-3 (and subtypes), 44-B1-3 (and subtypes), 44-C1-3 (and subtypes).

Another more pathomechanistically classification is the Lauge-Hansen classification [8–10]:

- Pronation – Abduction injury
- Pronation-External (Eversion) Rotation injury (Fig. 15.1)
- Supination – Adduction injury
- Supination – External (Eversion) Rotation injury

## Therapy

### Conservative Treatment

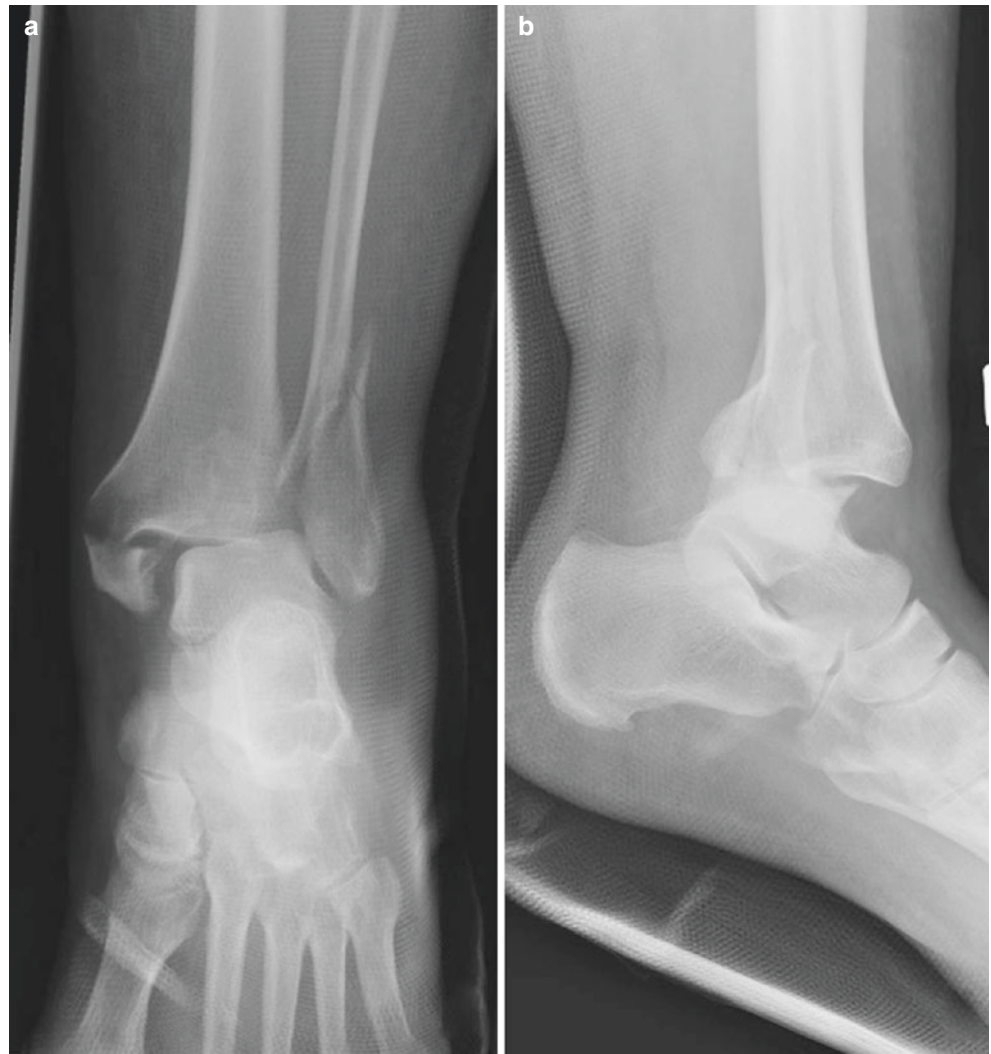
Non-displaced, or stable fractures, which are only minimally displaced, can be treated conservatively. This involves a stabilizing orthosis, walker or cast for Danis-Weber Type-A fractures. Pain adapted weight bearing should be supported by proprioceptive physiotherapy and early functional training. A vacuum walker should be preferred over a cast due to the comfort of the patient [11]. Radiographic controls should be obtained, especially in the first 2 weeks, not to miss a displacement of the fracture.

### Surgery

Anatomical reconstruction of the ankle joint is essential to achieve optimal results and prevention of posttraumatic osteoarthritis. Potentially injured tendons and especially stabilizing ligaments should be reconstructed as well to improve the functional outcome. Therefore surgery is indicated for all unstable and/or severely dislocated fractures (44A1-3 if displaced and unstable, 44B1 fractures is dislocated, and all fractures from 44B1.2 and above (Fig. 15.1). Open reduction and internal fixation (ORIF) should be performed. Time is of the essence and the 4–6 h time slot after the trauma should be used to avoid soft tissue complications and to fasten return to sports.

Surgery of the ankle joint is usually performed via the distal longitudinal lateral approach to the fibula. Additional

**Fig. 15.1** Trimalleolar, luxated ankle fracture Danis-Weber Type C with insufficient initial reduction (**a**: a.p. view, **b**: lateral view)



incisions have to be added as required and demanded by the specific co-injuries. Gentle soft tissue management is essential. Ankle arthroscopy can be very useful to control fracture reduction and to investigate the joint for osteochondral lesions [12].

Type A-fractures can either be stabilized with lag-screws or cannulated screws. Tension-band wiring or plating might be used as well.

Type B and C fractures should be reduced in length and rotation anatomically with reduction forceps. A lag screw can provide interfragmentary compression while a neutralization plate or modern fixed-angle implants should be used to stabilize the reposition. In cases of proximal fibula fractures (Maisonneuve-fractures) an osteosynthesis of the proximal fibula itself is generally not necessary. Nevertheless the interosseus membrane as well as the syndesmosis are often ruptured in these cases and require surgical reconstruction, e.g. by syndesmotic screws or a tight rope system.

### Rehabilitation and Back-to-Sports

Restoring the range of motion is essential for a fast rehabilitation process. Postoperative mobilisation on crutches or walkers should be adapted to the fracture pattern and the treatment concept as described above. In compliance with the surgeon's instructions non weight-bearing or partial weight-bearing should be preferred for 6 weeks. After a follow-up X-ray, weight-bearing can be increased in 10–20 kg per weeks. Especially ligamentous injuries require proprioceptive physiotherapy to avoid the development of chronic ankle instabilities. Depending on the type of injury back to training is possible after 12–16 weeks. Contact sports and competition level should be generally avoided for 6 months after the injury. In professional athletes this time frame might vary strongly and the individual rehabilitation program has to be adjusted to fasten the return to the pre-injury level [13].



## Evidence (Description of Highest Evidence with Mentioning the Level and Grade of Evidence)

The formal level of evidence regarding an optimized treatment algorithm is low by the criteria of evidence based medicine. Nevertheless the results of multiple prospective RCTs are expected to be available soon and will hopefully improve the treatment in the near future.

## Hindfoot Fractures

### Calcaneal Fracture

Calcaneal fractures account for about 2% of all fractures [14] (Fig. 15.2). These injuries often occur after high-energy trauma, for example a fall from height or during a motor vehicle accident. Athletes in disciplines with increased risk of high energetic trauma to the calcaneus, i.e. climbers, ski jumpers, parachuters or acrobats can be affected by this type of injury. Calcaneal fractures are often accompanied by severe soft tissue trauma. Accompanying injuries like fractures of the vertebral column, hip or knee are frequent, thus treatment is challenging.

### Classification

#### Sanders Classification [15]

The Sanders classification is the mostly used classification and is CT-based:

**Type 1:** no displacement regardless of the number of fragments

**Type 2:** two part fractures of the posterior surface

**Type 3:** tree part fractures

**Type 4:** comminuted fractures with significant fragmentation of the posterior articular surf

#### Essex Lopresti Classification

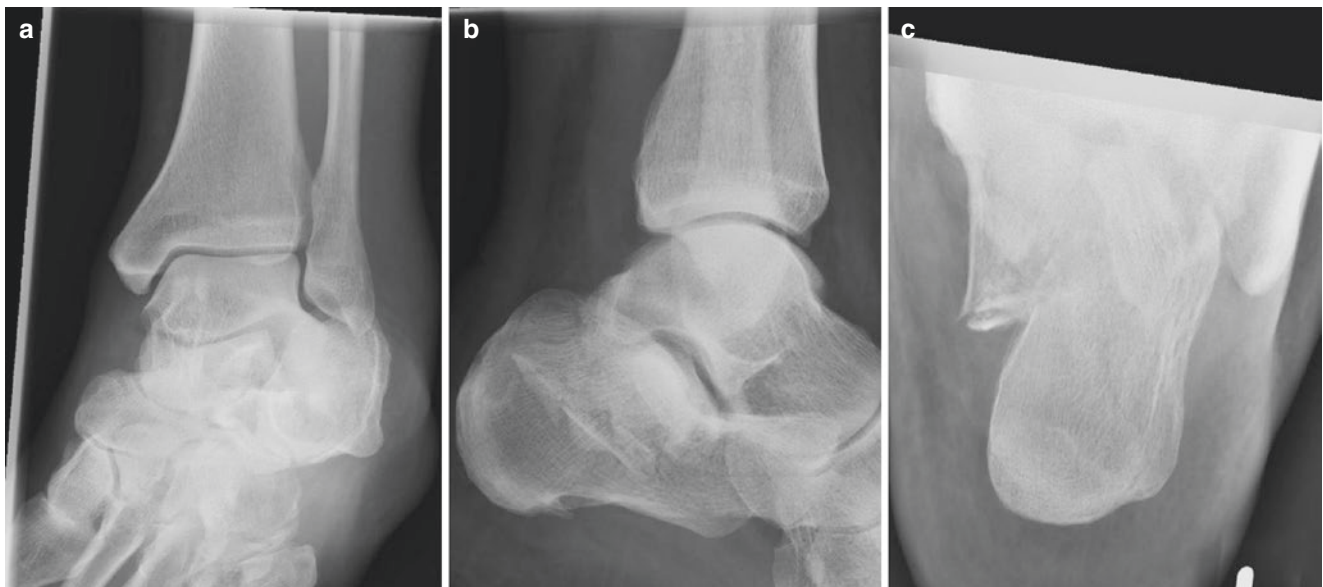
The Essex Lopresti classification is a helpful tool to grade a calcaneal fracture based on X-rays [16]:

- extra-articular
- intra-articular
- tongue fracture
- joint depression fracture

### Therapy: Conservative Treatment and Surgery (Approach, Technique, Risks, Aftertreatment)

#### Conservative Treatment

Conservative treatment is indicated in fractures with a minimal degree of dislocation and lowering of the articular surface (Sanders Type 1) only or in smokers or diabetic patients. The treatment includes cooling and elevation of the limb, as well as immobilization in a removable splint or walker for 6 weeks followed by 6 weeks of partial weight bearing. Control X-rays should be done after a week from injury and at monthly intervals. Conservative treatment might lead to a higher number of patients suffering from osteoarthritis and chronic pain syndromes [17].



**Fig. 15.2** Calcaneal fracture: (Sanders Type II). (a: a.p.view, b: lateral view, c: calcaneal axial view)

### Surgery

ORIF should be performed in Sanders Type 2–4 fractures (Fig. 15.2). The standard extensile lateral hindfoot-approach should be used. Interfragmentary screws as well as angular-stable plates for neutralization and stabilization of the posterior facet, lateral wall and anterior process of the calcaneus can be utilized [18, 19]. Typically bone grafting (autologous, allograft, synthetic fillers) is necessary to fill up the defect within the calcaneus. An alternative approach and treatment technique is the minimal invasive ORIF with an incision over the sinus tarsi and percutaneous incisions, using an angular stable plate on the lateral wall of the posterior facet and cannulated screws [20–22].

The postoperative management includes 6 weeks of non-weight bearing followed by 6 weeks of partial weight bearing adapted to the healing process. Besides general surgery related risks, infections, wound healing complications, osteonecrosis, and posttraumatic osteoarthritis are major risks [19].

### Talar Fractures

Only 3% of all fractures are talar fractures resulting from trauma as described above (see section “**Calcaneal Fracture**”) [23].

Talar fractures are divided in talar head fractures, talar neck fractures, talar process fractures, talar body fracture, and osteochondral lesions. The most common are talar neck fractures (weakest area of the talus), talar process fractures (e.g. lateral talar process fracture: snowboarder’s ankle; see Chap. 68), and osteochondral lesions in the ankle joint (see Chap. 16).

### Talar Neck Fractures

#### Classification

The mostly used classification for talar neck fractures is the Hawkins classification [24]:

**Hawkins I fractures:** nondisplaced vertical fractures of the talus neck

**Hawkins II fractures:** displacement of the neck of the talus and as well as a subluxation of the of the subtalar joint.

**Hawkins III fractures:** displacement of the subtalar and tibiotalar joint, currently with a dislocation of the corpus posteromedial

**Hawkins IV fractures:** Dislocation of the subtalar, tibiotalar and talonavicular joint

#### Therapy: Conservative Treatment and Surgery (Approach, Technique, Risks, Aftertreatment)

##### Conservative Treatment

Nondisplaced fractures (Hawkins Type I) can be treated conservatively in cast or walker immobilization with non-weight bearing for 6 weeks. X-ray controls should be performed.

### Surgery

Since approximately 60% of the talar surface is covered with articular cartilage, any displaced fracture of the neck or body of the talus (Hawkins Type II–IV) requires open reduction and internal fixation [25]. Severe problems of the blood circulation and the constitution of the patient are relative contraindications of a surgical treatment [26]. Severe bone subluxation requires emergent ORIF. Regarding the approaches a combined anteromedial and anterolateral approach or a single dorsal anterior approach might be used [27]. Fixation can be done with cannulated screws anteroposterior or postero-anterior or by angular-stable plates. Avascular necrosis is a major complication since the blood supply of the talus is often compromised, which is seen in X-rays with the so called Hawkins-sign (reduced subchondral talar dome bone density). Further complications are equino-varus-supination malalignment and posttraumatic osteoarthritis.

### Chopart Joint Dislocation Fractures

Nearly a third of injuries involving the Chopart or Lisfranc joints are overlooked or misdiagnosed [28]. They often happen in patients with multiple traumatic injuries, for example after a high energy trauma [29, 30]. Axial load or a forced abduction or adduction can be seen as the typical pathomechanism for Chopart joint dislocation. Uncommon are injuries by axial load of a plantar flexed foot or severe abduction. This leads typically to a lateral luxation of the Lisfranc joints II–V and may occur by falling of a horse with the foot in a stirrup or a fall off the surfboard with the foot still fixed in straps [31].

#### Classification

Zwipps classification disposes fractures of the chopart joint into six different types regarding to their dislocating force [23].

#### Therapy: Conservative Treatment and Surgery (Approach, Technique, Risks, Aftertreatment)

##### Conservative Treatment

Conservative treatment of closed reduction and stabilization in a short leg cast for 6–8 weeks should only be considered for mild subluxations of the Chopart joint or if there are severe contraindications for ORIF only.

##### Surgery

Every luxation of the Chopart joint should be treated surgically.

If closed reduction is not considered to be stable internal fixation by Kirschner wires is used for 6 weeks.

Two approaches, antero-medial and antero-lateral, allow a good visualization for open reduction and internal stabilization by screws or Kirschner wires. ORIF provides a better

outcome concerning the function of the Chopart joint than closed reduction. A primary arthrodesis may be considered in patients with massive irreconstructable bone damage [32].

### Rehabilitation and Back-to-Sports After Hindfoot Fractures

Conservative as well as surgical treatment require 6 weeks of non-weight bearing followed by 6 weeks of partial weight bearing. Supportive is an early gentle mobilization of the subtalar joint and ankle. Sanders Type 1 and 2 fractures have a better outcome compared to Type 3 and 4 fractures [15]. Overall, calcaneal fractures are often a turning point in the career of professional athletes since return to sports is often delayed and it is challenging to reach to pre-injury level.

### Prevention

Preventive training programs and proprioceptive training as well as strength training help to reduce sports injuries [33]. In current studies stretching and proprioceptive training before or after sports does not seem to be effective regarding the prevention of sport related injuries [34].

### Evidence (Description of Highest Evidence with Mentioning the Level and Grade of Evidence)

Current studies show surgical treatment of closed, displaced calcaneal fractures does not improve outcome when compared with non-operative treatment. The outcome of surgical treatment is published to depend mainly on the practice and knowledge of the surgeon [19]

---

## Lisfranc Joint Dislocation

### Classification

Quenu and Küss (1909) classification, modified by Hardcastle (1979) and Myerson (1986) [35, 36]

Type A: total incongruity, either medially or laterally displaced.

Type B: Partial incongruity, either medial (type B1) or lateral (type B2).

Type C: Divergent displacement, either partial (type C1) or total (type C2).

### Therapy: Conservative Treatment and Surgery (Approach, Technique, Risks, Aftertreatment)

#### Conservative Treatment

A closed reduction and cast application should be done in patients with contraindications for surgical treatment only

[37]. A cast is applied for about 6–8 weeks and a strict non-weight-bearing is important.

### Surgery

Surgery is indicated in unstable or dislocated fractures injuries and should be done within the first 24 h after trauma. Closed reduction and percutaneous fixation can be used for isolated simple injuries to the first and second tarsometatarsal joint.

Comminuted fractures or dislocations should be reduced opened and fixed by screws or plates in the 1–3 tarsometatarsal joints and by K-wires in the 4–5 tarsometatarsal joints. The first screw is placed from the medial cuneiform to the base of the second metatarsal, followed by a screw from the base of the first metatarsal to the medial cuneiform and a screw from the third metatarsal oblique to the second cuneiform. The reduction of the fourth and fifth metatarsal is achieved by the reduction of the other metatarsal [37]. Cortical screw fixation in the 1–3 tarsometatarsal joints allows a better and stable anatomic reduction compares to fixations by Kirschner wires [38]. In very comminuted fractures of the 1–3 tarsometatarsal joints a primary arthrodesis might be a treatment alternative.

### Fifth Metatarsal Base Fractures

Fractures of the fifth metatarsal are the most common fractures of metatarsal fractures, frequently seen in sports. Over two third of the sport associated fractures of the fifth metatarsal occur in soccer [35].

### Avulsions Fracture

Sudden inversion of the foot is a typical trauma mechanism of this proximal base fracture of the fifth metatarsal. It accompanies mostly with less dislocation of the fragments [39]. Surgical treatment with screws is necessary for fractures with a displacement >2 mm, involvement of the tarsometatarsal joint >30% or a decelerated healing.

### Jones Fracture

Acute Jones fracture is caused by adduction of the forefoot with a fracture at the proximal metaphyseal-diaphyseal junction extending into the intermetatarsal joint. In contrast to the acute, the chronic Jones fracture is a stress fracture with poor healing potential.

### Classification

#### Dameron-Quill Classification

Dameron-Quill classifications divides fractures of the proximal fifth metatarsal into three anatomic subgroups:

1. Avulsions fractures
2. Fractures at the metaphyseal/diaphyseal junction (true Jones fracture)
3. Proximal diaphyseal stress fracture [40].

### **Torg Classification for Fractures Distal to the Tuberosity**

Type 1: narrow fracture and absence of intramedullary sclerosis

Type 2: widening fracture line and evidence of intramedullary sclerosis

Type 3: non-union and complete obliteration of the medullary canal by sclerotic bone [41].

### **Therapy: Conservative Treatment and Surgery (Approach, Technique, Risks, Aftertreatment)**

#### **Avulsions Fracture**

##### **Conservative Treatment**

All non-displaced or minimal displaced avulsions fractures can be treated conservatively by partial weight bearing or weight bearing if tolerated [42] [43]. Other options are hard sole shoes or a short leg cast, but recent studies showed better results of return to work and sports by using elastig wrapping [44].

##### **Surgery**

Every displacement over 2 mm, comminuted fractures or fractures involving more than 30% of the cubometatarsal joint should be reduced and fixed by small fragment screws [43]. Postoperative treatment includes partial weight bearing and casting for approximately 6 weeks.

#### **Jones Fracture**

##### **Conservative Treatment**

Non-displaced fractures may be treated conservatively by strict non-weight-bearing in a short-leg cast for 6–8 weeks. But because of the poor vascular supply the rate of non-unions and refractures is high [45].

##### **Surgery**

Intramedullary screw fixation combined with use of orthobiologics or bone grafting is the standard for displaced fractures over 2 mm or nonunion of initially conservatively treated fractures.

### **Rehabilitation and Back-to-Sports**

Because of the risk of re-fractures after conservative treatment patients should start weight bearing gradually and pain adapted after 6 weeks. Conservative treatment takes at least twice the time to return back to sports compared to surgical treatment [46].

### **Forefoot Fractures**

Fractures of the forefoot are the most common fractures of the foot with a prevalence of nearly 4% of all fractures in adults. Fractures of the toes mostly cause from stubbing or a crush, fractures of the distal metatarsal bone cause either from direct impact, like vehicle accidents or from indirect force, like repetitive loads which can lead to stress fractures [39, 47]. Most of the toe fractures affect the great toe followed by the fifth toe [47].

Stress fractures mostly occur to athletes of ballet, dancing or running [48, 49], in the second and third metatarsals. Pain increases with weight-bearing activity besides little swelling of the foot. Stress fractures are difficult to see on X-rays until they have started to heal. MRI is more sensitive to detect the fracture. Generally stress fractures are treated conservatively by immobilization or rigid insoles for 5 weeks [50].

### **Classification**

There is no common classification to classify fractures of the forefoot.

### **Therapy: Conservative Treatment and Surgery (Approach, Technique, Risks, Aftertreatment)**

#### **Conservative Treatment**

Most of the phalangeal fractures are non or minimally displaced and are treated conservatively though the joint face is affected. Fractures of the great toe need to be immobilized in a cast for 3 weeks, fractures of the lesser toes are treated with splinting to the adjacent intact toe and wearing a rigid-sole shoe for about 2 weeks [47].

#### **Surgery**

Fractures of the proximal phalanx of the great toe are fixed percutaneous by Kirschner wires or rarely by screws and plates especially by affection of the joint surface [39]. Rehabilitation, back-to-sports depends on the specific type of injury and the individual healing process.

### **Evidence**

Up to date, there is Level I and II Evidence indicating no superiority for surgery in the treatment of lower leg and ankle fractures. Nevertheless current systematic reviews criticize the often poor quality of the studies analyzed. There are several high quality trials which results are expected in the very near future. Still, the treatment strategies presented rep-

resent the clinically proven standard, even though the formal evidence level remains low.

## Summary

- Lower leg and ankle fractures are common.
- A precise and sport discipline focused patient history is essential.
- Fracture patterns as well as tendon and ligament injuries have to be investigated carefully; CT, ultrasound or MRI are powerful tools to facilitate an appropriate classification.
- Anatomical reconstruction of the joint lines as well as soft tissue repair and balancing is the key to success, allowing a fast individually adapted rehabilitation program bringing our patients back to sports and back to the pre-injury level.
- In professional athletes the rehabilitation time frame might vary strongly and the individual rehabilitation program has to be adjusted to fasten the return to the pre-injury level.

## References

1. Shibuya N, Davis ML, Jupiter DC. Epidemiology of foot and ankle fractures in the United States: an analysis of the National Trauma Data Bank (2007 to 2011). *J Foot Ankle Surg.* 2014;53:606–8. doi:10.1053/j.jfas.2014.03.011.
2. Saltzman CL, el-Khoury GY. The hindfoot alignment view. *Foot Ankle Int.* 1995;16:572–6.
3. Black EM, Antoci V, Lee JT, Weaver MJ, Johnson AH, Susarla SM, Kwon JY. Role of preoperative computed tomography scans in operative planning for malleolar ankle fractures. *Foot Ankle Int.* 2013;34:697–704. doi:10.1177/1071100713475355.
4. Stähler A, Szeimies U, Walther M. Radiologische Diagnostik des Fußes. Stuttgart: Thieme; 2012.
5. Weber BG, Colton C. Malleolar fractures. In: *Manual of internal fixation.* Berlin: Springer; 1991. p. 595–612.
6. Park JW, Kim SK, Hong JS, Park JH. Anterior tibiofibular ligament avulsion fracture in weber type B lateral malleolar fracture. *J Trauma.* 2002;52:655–9.
7. Dias LS, Giegerich CR. Fractures of the distal tibial epiphysis in adolescence. *J Bone Joint Surg Am.* 1983;65:438–44.
8. Rodriguez EK, Kwon JY, Herder LM, Appleton PT. Correlation of AO and Lauge-Hansen classification systems for ankle fractures to the mechanism of injury. *Foot Ankle Int.* 2013;34:1516–20. doi:10.1177/1071100713491730.
9. Chen DW, Li B, Yang YF, Yu GR. AO and Lauge-Hansen classification systems for ankle fractures. In: *Foot Ankle Int.* Sage, London. 2013. p. 1750.
10. [www.aofoundation.org](http://www.aofoundation.org). In.
11. Herscovici Jr D, Scaduto JM, Infante A. Conservative treatment of isolated fractures of the medial malleolus. *J Bone Joint Surg Br.* 2007;89:89–93. doi:10.1302/0301-620x.89b1.18349.
12. Bonasia DE, Rossi R, Saltzman CL, Amendola A. The role of arthroscopy in the management of fractures about the ankle. *J Am Acad Orthop Surg.* 2011;19:226–35.
13. Lin CW, Donkers NA, Refshauge KM, Beckenkamp PR, Khera K, Moseley AM. Rehabilitation for ankle fractures in adults. *Cochrane Database Syst Rev.* 2012;11:CD005595. doi:10.1002/14651858.CD005595.pub3.
14. Mitchell MJ, McKinley JC, Robinson CM. The epidemiology of calcaneal fractures. *Foot (Edinb).* 2009;19:197–200. doi:10.1016/j.foot.2009.05.001.
15. Sanders R, Fortin P, DiPasquale T, Walling A. Operative treatment in 120 displaced intra-articular calcaneal fractures. Results using a prognostic computed tomography scan classification. *Clin Orthop Relat Res.* 1993;290:87–95.
16. Lopresti-Essex P. The mechanism, reduction technique, and results in fractures of the Os Calcis.pdf. *Clin Orthop Related Res.* 1993;290:3–16.
17. Bruce J, Sutherland A. Surgical versus conservative interventions for displaced intra-articular calcaneal fractures. *Cochrane Database Syst Rev.* 2013;1:CD008628. doi: 10.1002/14651858.CD008628.pub2.
18. Eastwood DM, Langkamer VG, Atkins RM. Intra-articular fractures of the calcaneum. Part II: open reduction and internal fixation by the extended lateral transcalcaneal approach. *J Bone Joint Surg Br.* 1993;75:189–95.
19. Griffin D, Parsons N, Shaw E, Kulikov Y, Hutchinson C, Thorogood M, Lamb SE, Investigators UHFTUH. Operative versus non-operative treatment for closed, displaced, intra-articular fractures of the calcaneus: randomised controlled trial. *BMJ.* 2014;349:g4483.
20. Zhang T, Su Y, Chen W, Zhang Q, Wu Z, Zhang Y. Displaced intra-articular calcaneal fractures treated in a minimally invasive fashion: longitudinal approach versus sinus tarsi approach. *J Bone Joint Surg Am.* 2014;96:302–9. doi:10.2106/jbjs.1.01215.
21. Kikuchi C, Charlton TP, Thordarson DB. Limited sinus tarsi approach for intra-articular calcaneus fractures. *Foot Ankle Int.* 2013;34:1689–94. doi:10.1177/1071100713510267.
22. Nosewicz T, Knupp M, Barg A, Maas M, Bolliger L, Goslings JC, Hintermann B. Mini-open sinus tarsi approach with percutaneous screw fixation of displaced calcaneal fractures: a prospective computed tomography-based study. *Foot Ankle Int.* 2012;33:925–933. doi:10.3113/fai.2012.0925.
23. Zwipp H. *Chirurgie des Fusses.* 1994.
24. Hawkins LG. Fractures of the neck of the talus. *J Bone Joint Surg Am.* 1970;52:991–1002.
25. Kopp L, Obruba P, Riegl J, Meluzinová P, Edelmann K. Surgical management of talus fractures: mid-term functional and radiographic outcomes. *Acta Chir Orthop Traumatol Cech.* 2013;80:165–70.
26. Rammelt S, Winkler J, Zwipp H. Operative treatment of central talar fractures. *Oper Orthop Traumatol.* 2013;25:525–41. doi:10.1007/s00064-013-0245-4.
27. Boack D-H, Manegold S, Haas NP. Operative Technik bei Talusfrakturen. In: *Der Unfallchirurg.* Springer, Heidelberg; 2004. p. 515–520.
28. Mittlmeier T, Beck M. Injuries of the midfoot. *Chirurg.* 2011;82:169–86. doi:10.1007/s00104-009-1866-x; quiz 187–68.
29. Richter M, Wippermann B, Krettek C, Schratz HE, Hufner T, Therman H. Fractures and fracture dislocations of the midfoot: occurrence, causes and long-term results. *Foot Ankle Int.* 2001;22:392–8.
30. Rammelt S, Grass R, Schikore H, Zwipp H. Injuries of the Chopart joint. *Unfallchirurg.* 2002;105:371–83; quiz 384–75.
31. Mittlmeier T, Beck M. Tarsometatarsal injuries—an often neglected entity. *Ther Umsch.* 2004;61:459–65.
32. Richter M, Therman H, Huefner T, Schmidt U, Goesling T, Krettek C. Chopart joint fracture-dislocation: initial open reduction provides better outcome than closed reduction. *Foot Ankle Int.* 2004;25:340–8.
33. Lauenstein JB, Bertelsen DM, Andersen LB. The effectiveness of exercise interventions to prevent sports injuries: a systematic review and meta-analysis of randomised controlled trials. *Br J Sports Med.* 2014;48:871–7. doi:10.1136/bjsports-2013-092538.
34. Leppanen M, Aaltonen S, Parkkari J, Heinonen A, Kujala UM. Interventions to prevent sports related injuries: a systematic review

- and meta-analysis of randomised controlled trials. *Sports Med.* 2014;44:473–86. doi:[10.1007/s40279-013-0136-8](https://doi.org/10.1007/s40279-013-0136-8).
35. Shuen WM, Boulton C, Batt ME, Moran C. Metatarsal fractures and sports. *Surgeon.* 2009;7:86–8.
  36. Myerson MS, Fisher RT, Burgess AR, Kenzora JE. Fracture dislocations of the tarsometatarsal joints: end results correlated with pathology and treatment. *Foot Ankle.* 1986;6:225–42.
  37. Mulier T. The treatment of Lisfranc injuries: review of current Literature. *Eur J Trauma Emerg Surg.* 2010;36(3):206–16.
  38. Lee CA, Birkedal JP, Dickerson EA, Vieta PA, Webb LX, Teasdall RD. Stabilization of Lisfranc joint injuries: a biomechanical study. *Foot Ankle Int.* 2004;25:365–70.
  39. Richter M, Zech S, Hildebrand F, et al. Fuss Fractures of the forefoot. In: *Fuss und Sprunggelenk.* Springer, Heidelberg. 2007. p. 155–166. doi:[10.1007/s10302-007-0286-9](https://doi.org/10.1007/s10302-007-0286-9).
  40. Dameron TB. Fractures and anatomical variations of the proximal portion of the fifth metatarsal. *J Bone Joint Surg Am.* 1975;57:788–92.
  41. Torg JS, Balduini FC, Zelko RR, Pavlov H, Peff TC, Das M. 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.* 1984;66:209–14.
  42. Zwitser EW, Breederveld RS. Fractures of the fifth metatarsal; diagnosis and treatment. *Injury.* 2010;41:555–62. doi:[10.1016/j.injury.2009.05.035](https://doi.org/10.1016/j.injury.2009.05.035).
  43. Rammelt S, Heineck J, Zwipp H. Metatarsal fractures. *Injury.* 2004;35 Suppl 2:SB77–86. doi:[10.1016/j.injury.2004.07.016](https://doi.org/10.1016/j.injury.2004.07.016).
  44. Zenios M, Kim WY, Sampath J, Muddu BN. Functional treatment of acute metatarsal fractures: a prospective randomised comparison of management in a cast versus elasticated support bandage. *Injury.* 2005;36:832–5. doi:[10.1016/j.injury.2004.12.001](https://doi.org/10.1016/j.injury.2004.12.001).
  45. Nunley JA. Fractures of the base of the fifth metatarsal: the Jones fracture. *Orthop Clin North Am.* 2001;32:171–80.
  46. Mologne TS, Lundeen JM, Clapper MF, O'Brien TJ. Early screw fixation versus casting in the treatment of acute Jones fractures. *Am J Sports Med.* 2005;33:970–5. doi:[10.1177/0363546504272262](https://doi.org/10.1177/0363546504272262).
  47. Van Vliet-Koppert ST, Cakir H, Van Lieshout EM, De Vries MR, Van Der Elst M, Schepers T. Demographics and functional outcome of toe fractures. *J Foot Ankle Surg.* 2011;50:307–10. doi:[10.1053/j.jfas.2011.02.003](https://doi.org/10.1053/j.jfas.2011.02.003).
  48. Gross TS, Bunch RP. A mechanical model of metatarsal stress fracture during distance running. *Am J Sports Med.* 1989; 17:669–74.
  49. Eisele SA, Sammarco GJ. Fatigue fractures of the foot and ankle in the athlete. *J Bone Joint Surg Am.* 1993;75:290–8.
  50. Gehrman RM, Renard RL. Current concepts review: stress fractures of the foot. *Foot Ankle Int.* 2006;27:750–7.

Martin Wiewiorski, Alexej Barg, Markus Wurm,  
and Victor Valderrabano

## Abstract

Acute chondral or osteochondral lesions of the ankle joint are typically co entities of ankle sprains and ankle fractures. The exact incidence is not clear, however clinical and radiological studies suggest that this entity is more common than assumed and probably frequently missed in an acute trauma situation. The initial clinical examination and conventional radiographs are often inconclusive. In a severe ankle injury, CT scans and MRI are useful diagnostic tools to assess chondral or osteochondral injury. If diagnosed, surgical treatment consists of either debridement and bone marrow stimulation, or refixation. Return to sports depends on additional injury to the joint as well as patients and sports specific condition.

## Keywords

Ankle • Acute injury • Chondral lesion • Osteochondral lesion • Cartilage

## Introduction

Acute ankle osteochondral and chondral lesions are common injuries in Foot & Ankle sports orthopaedics. To date there is no clear data on incidence for this special lesions. These lesions are typically co-entities in severe ankle sprains and ankle fractures (see Chaps. 15 and 27). Pain, joint locking or inability to walk are just some of possible manifesting symptoms. Even with accurate and fast clinical examination most of these

lesions are missed at the initial trauma and they may cause as hidden and undiagnosed lesions posttraumatic pain problems, chronic osteochondral lesions, and osteoarthritis. CT-scans and MR-imaging are two potential diagnostic tools however they are seldom used in acute situations. A variety of treating options are known today. Conservative treatment as well as surgical techniques i.e. bone marrow stimulation or fixation of osteochondral flakes can be considered. Only physicians and surgeons with longstanding experience on osteochondral as well as chondral lesions should be in charge of treatment. Time until return to sports should be adapted to potential additional injury as well as patients- and sports specific conditions.

M. Wiewiorski, MD (✉)

Orthopaedic Department, Kantonsspital Winterthur,  
Winterthur, Switzerland  
e-mail: [mwiewiorski@gmail.com](mailto:mwiewiorski@gmail.com)

A. Barg, MD

Department of Orthopaedics, University of Utah, Salt Lake City, USA  
e-mail: [alexej.barg@hsc.utah.edu](mailto:alexej.barg@hsc.utah.edu)

M. Wurm, MD

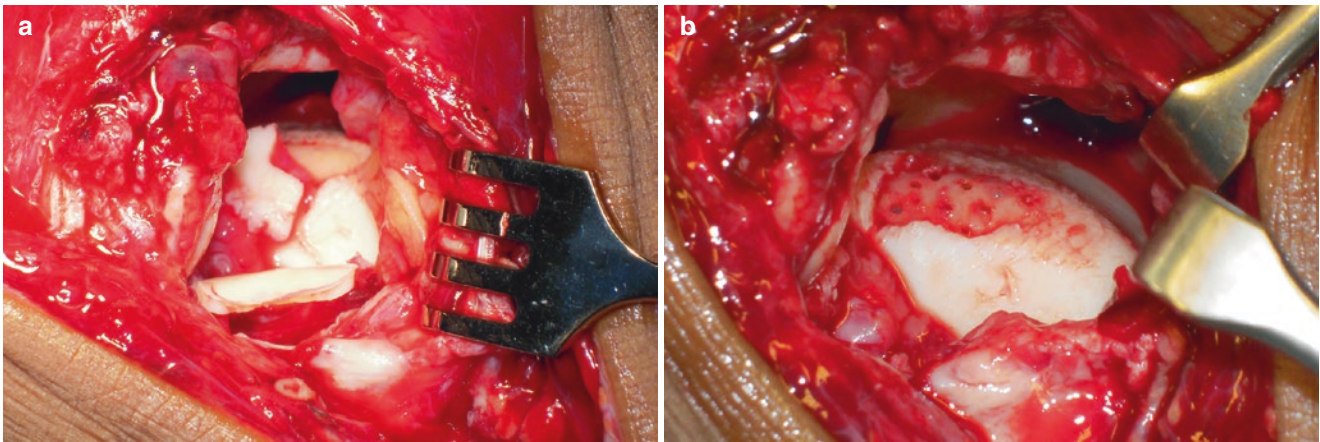
Department of Orthopaedics and Traumatology, University  
Hospital Basel, Basel, Switzerland  
e-mail: [wurmarkus@gmail.com](mailto:wurmarkus@gmail.com)

V. Valderrabano, MD, PhD

Orthopaedic Department, SWISS ORTHO CENTER,  
Schmerzklinik Basel, Swiss Medical Network, Basel, Switzerland  
e-mail: [vvalderrabano@gsmn.ch](mailto:vvalderrabano@gsmn.ch)

## Etiology and Pathomechanism

Talar tilt in the malleolar mortise during ankle sprains and ankle fractures has been shown to induce cartilage damage and osteochondral fractures in ex- as well as in-vivo studies [1, 2]. Laboratory investigations by Bernd and Harty in 1959 on cadaver ankle joints showed trauma to the upper talar surface, when exposed to inversion or eversion forces [3]. Based on those experiments, a radiological classification was developed, which described a chain of consecutive morphological



**Fig. 16.1** Acute chondral lesion. This patient sustained a severe eversion trauma with luxation of the ankle joint during a soccer match resulting in a syndesmotic injury, rupture of the interosseous membrane

and high fibular fracture. The open approach revealed several chondral fragments on the medial talar edge (a). The chondral flakes were removed, and the subchondral bone was microdrilled (b)

changes induced by traumatic forces to the ankle joint. These ranged from compression of subchondral bone to occurrence and displacement of an osteochondral fragment (as depicted in Fig. 16.1a, b). The incidence of acute chondral/osteochondral lesions is not well researched. Most less severe ankle sprains and ankle fractures do not undergo arthroscopy or further diagnostic imaging other than conventional radiography. Arthroscopical studies suggest that the incidence for chondral/osteochondral lesions of the ankle joint (tibial/fibular/talar joint surface) after ankle sprain is 33–89%, and 79.2% after ankle fracture. No study is known to the authors, which would specify the incidence of those lesions for an individual sport activity. Several authors have noted cartilage damage in ankles with acute lateral ligament injuries ranging from an incidence of 25–77% [4–6].

## Symptoms

There are no specific symptoms for acute chondral or osteochondral injuries. General symptoms after ankle trauma vary depending on the severity of the injury, and can include swelling, pain, joint locking, hematoma, and inability of weight bearing.

## Diagnostics, Classification

Clinical examination for chondral/osteochondral lesion in an acute trauma situation is often not conclusive. Symptoms which might be generated by a lesion, would be masked by the general symptoms of a sprain or fracture. Planar radiography is indicated as a primary imaging modality to rule out fractures. However, planar radiographs would not show always the osteochondral lesions or even the chondral

lesions. Larger osteochondral fractures can be occasionally seen. Computertomography (CT) and/or magnetic resonance imaging (MRI) in the acute trauma situation are rarely performed, unless it's a severe complex injury requiring immediate surgical attention. MRI has shown its ability to depict acute osteochondral lesions. Brown et al. evaluated MRI of patients with acute syndesmotic injuries and found osteochondral lesions in 26% of the patients [7]. Lohmann et al. performed MRI in 60 children after acute lateral ligament injury and physeal ankle fractures [8]. Twenty-two (37%) of the 60 patients had bone bruises, of which 83% occurred in patients with ligament disruption and without any fracture on MRI. Of the bone bruises, 11 occurred in the talus, seven in the distal tibia and five in the distal fibula.

Arthroscopy showed to be a reliable method to depict chondral lesions in the acute stage after ankle injury. Taga et al. performed a diagnostic arthroscopy in nine patients with acute lateral ankle instability due to a flexion-inversion ankle sprain (4). They found medial chondral lesions in eight patients (89%) and lateral lesions in three patients (33%). Hintermann et al. assessed 288 patients with acute ankle fractures for arthroscopical findings of chondral lesions (2). Lesions of the cartilage were found in total of 228 ankles (79.2%), more often on the talus (69.4%) than on the distal tibia (45.8%), the fibula (45.1%), or the medial malleolus (41.3%).

## Therapy: Conservative Treatment and Surgery

### General Considerations

Most acute chondral lesions after ankle injury stay undiagnosed and untreated. In such cases, conservative treatment might be carried out because of the apparent injuries to bone



(fracture) or soft tissue (ligaments, tendons), and varies depending on injury pattern and severity.

### Debridement Followed by Bone Marrow Stimulation, Fixation

In cases where acute chondral/osteochondral lesions are detected by advanced imaging techniques (MRI, CT), or ankle arthroscopy, the lesions should be addressed. In cases of purely chondral lesions, where the cartilage is sheared of the subchondral bone, the flakes should be removed, and bone marrow stimulation should be performed (microdrilling/microfracturing). This can be either performed arthroscopically, or in cases of extensive defects through a mini-open procedure. In cases of osteochondral fractures  $\geq 1$  cm in diameter, the flake should be reduced and fixated. This can be done either with a resorbable pin/screw (less stable, no need for removal) [9], or a regular screw (more stable fixation, needs removal) [10].

### Augmented Bone Marrow Stimulation

Most advanced cartilage reconstruction methods (ACI, MACI, osteochondral transplantation) in the acute setting are not available, or not feasible. However, matrix augmentation of the debrided and microdrilled/microfractured area can be easily performed. A synthetic [11] or allograft [12] matrix can be cut to match the defect size and is typically glued onto the defect with fibrin glue. The matrix seals the defect and allows mesenchymal stem cells from the bone marrow to migrate into the matrix and re-differentiate into chondrocytes. Only case reports are available describing this approach for professional athletes [13, 14]. The advantages are a one-step approach, off-the-shelf availability of the matrices, and low costs. The matrix can be inserted either arthroscopically [15], or through a mini-open approach [16].

### Rehabilitation and Back-to-Sports

The rehabilitation protocol depends on the severity of the injury, and the surgical reconstruction technique. Immediate postoperative care consists of immobilization

using an ankle walker boot (e.g. Aircast Walker, DJO Global, Vista, USA) and functional physiotherapy with 15 kg partial weight bearing starting on the second postoperative day for 8 weeks. The range of motion is restricted to 20° with use of a continuous passive motion machine, and lymphatic drainage massage for the first 8 weeks. For complete rehabilitation algorithm please see Chap. 31, Table 16.1.

### Prevention

In the acute injury, no preventive measures can be undertaken.

### Evidence

For literature and evidence see Table 16.1. Several surgical techniques show satisfactory clinical outcome in case series, however, based on level of evidence criteria, there is no evidence of superiority of any surgical technique over debridement and bone marrow stimulation. The overall level of evidence in the existing literature is low. No randomized controlled studies exist, and recommendations can only be made upon level 4 studies or expert consensus.

### Summary

- Acute chondral/osteochondral lesions are frequent, but rarely diagnosed at initial ankle trauma.
- Acute chondral/osteochondral lesions are commonly seen in sports types causing ankle sprains and fractures.
- Preoperative CT or MRI diagnostics or intraoperatively ankle arthroscopy help to visualize the hidden chondral/osteochondral lesions.
- If diagnosed, the treatment consists of debridement of the chondral flakes and bone marrow stimulation, or in cases of osteochondral fractures, debridement or refixation. Bone marrow stimulation can be augmented with a matrix, if available.
- Undiagnosed chondral/osteochondral lesions may cause untreated chronic ankle pain, chronic osteochondral lesions, and osteoarthritis, and therefore endanger the athlete's sports career.

**Table 16.1** Overview of important publications on acute chondral/osteochondral talar lesions

Author	Topic	Level of evidence
Valderrabano et al. (2014) [14]	AMIC technique for treatment of fresh chondral lesions	4
Hintermann et al. (2000) [2]	Arthroscopic findings in acute fractures of the ankle	4
Berndt/Harty (1959) [3]	Injury mechanism of osteochondral fractures of the talus	/
Brown et al. (2004) [7]	Cartilage lesions in MRI in acute ankle trauma	4

## References

1. Alanen V, et al. Incidence and clinical significance of bone bruises after supination injury of the ankle. A double-blind, prospective study. *J Bone Joint Surg Br.* 1998;80(3):513–5.
2. Hintermann B, et al. Arthroscopic findings in acute fractures of the ankle. *J Bone Joint Surg Br.* 2000;82(3):345–51.
3. Berndt AL, Harty M. Transchondral fractures (osteochondritis dissecans) of the talus. *J Bone Joint Surg Am.* 1959;41-A:988–1020.
4. Komenda GA, Ferkel RD. Arthroscopic findings associated with the unstable ankle. *Foot Ankle Int.* 1999;20(11):708–13.
5. Ogilvie-Harris DJ, Gilbert MK, Chorney K. Chronic pain following ankle sprains in athletes: the role of arthroscopic surgery. *Arthroscopy.* 1997;13(5):564–74.
6. Sugimoto K, et al. Chondral injuries of the ankle with recurrent lateral instability: an arthroscopic study. *J Bone Joint Surg Am.* 2009;91(1):99–106.
7. Brown KW, et al. MRI findings associated with distal tibiofibular syndesmosis injury. *AJR Am J Roentgenol.* 2004;182(1):131–6.
8. Lohman M, et al. Acute paediatric ankle trauma: MRI versus plain radiography. *Skeletal Radiol.* 2001;30(9):504–11.
9. Kristensen G, et al. Fracture stage 4 of the lateral talar dome treated arthroscopically using Biofix for fixation. *Arthroscopy.* 1990;6(3):242–4.
10. Dodd A, Simon D, Wilkinson R. Arthroscopically assisted trans-fibular talar dome fixation with a headless screw. *Arthroscopy.* 2009;25(7):806–9.
11. Giannini S, et al. Surgical treatment of osteochondral lesions of the talus in young active patients. *J Bone Joint Surg Am.* 2005;87 Suppl 2:28–41.
12. Wiewiorski M, et al. Autologous matrix-induced chondrogenesis aided reconstruction of a large focal osteochondral lesion of the talus. *Arch Orthop Trauma Surg.* 2011;131(3):293–6.
13. Bark S, Riepenhof H, Gille J. AMIC cartilage repair in a professional soccer player. *Case Rep Orthop.* 2012;2012:364342.
14. Valderrabano V, et al. Osteochondral lesions of the ankle joint in professional soccer players: treatment with autologous matrix-induced chondrogenesis. *Foot Ankle Spec.* 2014;7(6):522–8.
15. Sadlik B, et al. Dry arthroscopy with a retraction system for matrix-aided cartilage repair of osteochondral lesions of the talus. *Foot Ankle Int.* 2014;3(1):e141–4.
16. Valderrabano V, et al. Autologous matrix-induced chondrogenesis-aided repair of osteochondral lesions of the talus. *Techn Foot Ankle Surg.* 2011;10(4):159–62. doi:[10.1097/BTF.0b013e318237c1b0](https://doi.org/10.1097/BTF.0b013e318237c1b0).

William Melton and J. Benjamin Jackson III

**Abstract**

Stress fractures were first described over 250 years ago and are believed to be the result of repetitive, prolonged muscular action on bone that has yet to accommodate or remodel. There is a close association with several well-known groups: military recruits, runners, and ballet dancers. Despite the easily identifiable populations, stress fractures are difficult to diagnose due to late appearance on standard radiograph. Therefore, a thorough history, a detailed physical exam, and a high level of suspicion are required to diagnose in a timely manner. Foot and ankle stress fractures make up a large percentage of the overall incidence of stress fractures with the tibia, fibula, metatarsals (MT), and calcaneus being more common and the navicular and sesamoid bones being less common. Although each bone has a slightly different etiology, presentation, and radiographic tendency, stress fractures of the foot and ankle are generally treated with activity modification and protected weight bearing, a difficult concept for the highly-active patient that usually presents. Prevention of stress fractures focuses on optimizing bone quality, sound training technique, and quality training equipment.

**Keywords**

Foot • Ankle • Stress • Fracture • Military • Ballet • Runner

**Etiology and Pathomechanism**

Breithaupt, a German military surgeon, first described stress fractures in 1855 and demonstrated two common themes in foot and ankle stress fractures: little need for standard radiographs and strong connection to the military [1]. Foot and ankle stress fractures make up a large percentage of the overall incidence of stress fractures with the tibia, fibula, metatarsals (MT), and calcaneus being more common and the navicular and sesamoid bones being less common. Stress

fractures are generally seen in very active individuals, and are classically associated with runners, ballet dancers, orientierers, and military personnel. Each group is predisposed to different types of stress fractures based on the stress the activity applies to the foot and ankle. Stress fractures are believed to be the result of repetitive, prolonged muscular action on bone that has yet to accommodate or remodel. Under normal circumstances, bone remodels based on the stress applied to it in accordance with Wolf's Law. However, muscle accommodates faster than bone creating an unbalanced pull [2]. Risk factors generally fall under 3 categories: changes in anatomy, decreased biomechanical alignment, and training related errors.

Known anatomic risk factors including:

- Pes cavus
- Flatfoot
- Morton's foot (long 2nd metatarsal relative to the 1st)
- Metatarsus adductus

---

W. Melton, MD  
Department of Orthopaedics, Palmetto Health Richland,  
Columbia, SC, USA  
e-mail: [wmelton@gmail.com](mailto:wmelton@gmail.com)

J.B. Jackson III, MD (✉)  
USC Department of Orthopaedics, Two Medical Park, Suite 404,  
Columbia, SC 29204, USA  
e-mail: [jbenjackson@gmail.com](mailto:jbenjackson@gmail.com)

Metabolic factors that can predispose patients to stress fractures include:

- Osteopenia or Osteoporosis
- Hormonal disorders such as menopause and hyperthyroidism
- The female athletic triad: amenorrhea, eating disorders, decreased bone mineral density

Modifiable risk factors of patient habits and training include:

- Tobacco
- Alcohol use
- Nutritional deficiencies
- Use of steroids
- Poor form/technique
- Poor equipment

When these risk factors are combined with increased physiologic stress, a stress fracture can develop. For example, this combination can be seen when a workout is initiated and progressed too quickly, as in running 1 mile a day 1 week then running 10 miles a day the next. However, this scenario is not unique to changes in workout intensity. Using the runner example, changes in shoe or running surface can lead to stress fractures by the same mechanism of changing muscle action too quickly. Multiple cycles of stress loading at fatigue levels, but not at levels high enough to create plastic deformation, create microfractures in the bone that grow as the stress repeats. These microfractures are caused by an abundance of osteoclast activity compared to osteoblast activity when there is insufficient period of rest and recovery [3]. Eventually, the microfractures can grow to cause mechanical failure.

Although each bone has unique stress fracture characteristics, there are two broad categories based on etiology: fatigue and insufficiency. A fatigue stress fracture results when an abnormal force is applied to bone with normal elastic resistance as in a new, strenuous, and repeated activity. An insufficiency fracture results when a normal muscular force is applied to bone with abnormal elastic resistance as in osteoporosis. Although these are logical general categories, they are not absolutes and there will be overlap as in the ballerina who subjects her potentially weak bone (female athlete triad) to abnormal muscular forces [4, 5].

---

## Symptoms

High clinical suspicion and a detailed history are required to diagnose stress fractures. Important points in the history include onset or changes in level or type of activity and pain with activity that is relieved with rest. There may or may not be an inciting event that is obvious to the patient, as not all causative activity is high-impact. Classically, groups like ballerinas, runners, and military recruits are at high risk for stress fracture and should raise clinical suspicion. Also, previously mentioned hormonal imbalances, eating disorders,

and nutritional deficiencies should be noted. This can be especially important in young female athletes involved in high-impact sports focusing on intense fitness-level and low weight such as ballet, gymnastics, and cheerleading.

Stress fractures often initially present as a stress reaction: no radiographic evidence of stress fracture with increased uptake seen on MRI [6]. These patients will present with varying degrees of swelling but usually will exhibit tenderness over the fracture site. The nature of the swelling should be carefully elucidated. Cardiovascular, lymphatic, or other systemic causes of edema can present bilaterally compared to the expected unilateral swelling with stress fractures. Also, a systematic approach to bony palpation is suggested. One possible approach includes a circular pattern starting at the medial 1st toe, moving proximal to the calcaneus, and moving back distally along the lateral aspect to the 5th toe [7]. As with any exam of the foot and ankle, it is recommended that the patient be questioned about their footwear, especially in runners as shoes older than 6 months have been found to be a risk factor for developing stress fractures [8].

They could potentially still be participating in the offending activity, and a differential diagnosis would include tendinitis, periostitis, bursitis, and nerve injury [9]. As the microfractures grow in size and number, the resulting pain and tenderness should correlate. As with other foot and ankle pathology, there is rarely referred pain, and the area of tenderness is most likely to bone involved. However, each bone has unique characteristics that will be addressed in the appropriate section.

---

## Diagnostics, Classification

Radiographic findings were first described by Kirchner in 1905, 50 years after stress fractures were first described highlighting their somewhat limited role. The findings on radiographs (XR) are usually not helpful for a quick diagnosis and may not even be present in 30–70% of cases [10]. Radiographic findings of stress fracture usually indicate a more long standing problem or a more severe stress fracture. Other imaging modalities, such as US, MRI, and CT, may be more accurate in the acute diagnosis. In a study of 320 stress fractures in athletes, Matheson reports an average of 10–21 days from onset of clinical symptoms to bony changes evident on radiographs [5]. Focal linear sclerosis is often evident first and is perpendicular to the trabeculae. Other radiographic findings include periosteal reaction, new bone formation, and cortical thickening [6]. In more severe cases cortical disruption and even complete fractures can also be seen [10]. Technetium-122 bone scans (T-122) are much more sensitive than conventional radiographs, and radio-nucleotide uptake can be evident within 24 h of injury. However, it is not very specific and is not a good modality to monitor fracture healing. In contrast,



**Fig. 17.1** Calcaneus MRI: The black stress fracture line can be appreciated as it is perpendicular to the calcaneal trabeculae

MRI is very sensitive, very specific, and is not associated with radiation exposure. CT imaging is best used for defining the fracture line and to determine a complete vs. an incomplete fracture [11, 12]. Also see stress fracture imaging (Chap. 5, pages 51–52).

Levy described two stress fracture patterns based on location in bone and characteristics seen on imaging [10]. Metatarsals two to five often have diaphyseal fractures with cortical disruption followed by dense callus. The first metatarsal and calcaneus often have fractures in cancellous bone seen as lines perpendicular to the applied stress as seen in Fig. 17.1 [13]. This classification was expanded by Keading in an attempt to find a more reproducible classification system based on grades with high intraserver and interobserver reliability. This study uses 5 grades increasing in severity from stress reaction (Grade I) to non-union (Grade V), and it reports excellent interobserver and intraobserver reliability between 15 clinicians [14].

## Treatment

There is currently no proven benefit for the use of medication in treating stress fractures. Although non-steroidal Anti-Inflammatories (NSAIDs) reduce the associated inflammation, they may impede bone healing and have been shown to do so in animal models [15]. Other modalities with potential theoretical application include bisphosphonates, teriparatide (recombinant human parathyroid hormone analog), calcitonin, bone stimulation, and shock therapy. All of these treatment options require more extensive study to prove their efficacy.

Conservative treatment, including activity modification with protected weight bearing, is the treatment of choice in a majority of stress fractures. The degree of protected weight bearing depends on the evidence of a fracture line. In a stress reaction with no evidence of a fracture line, 6–8 weeks of activity modification with orthotics or shoe modifications is appropriate. When there is evidence of a fracture line, more caution is required with a boot or cast for protected weight bearing with serial exams to rule out non-union or mal-union. In both scenarios, activity can continue but should be changed to less impact, i.e. aquatic sports or biking instead of running. The patient can return to their previous level of activity when they are pain-free and without radiographic evidence of fracture. This time consuming treatment plan can be frustrating for active patients who are often at a highly competitive level of activity. Although the vast majority of stress fractures can be treated conservatively, some require surgical intervention, and they will be addressed as they present for specific bones and fracture patterns.

## Prevention

Conservative care should also focus on prevention of future stress fractures. Prevention of stress fractures can be focused on two of the three previously mentioned main groups of risk factors: bone quality and training education. Improving bone quality begins with nutrition and slowing the progression of osteoporosis. A study by Wentz on dietary habits and training predictors of stress fractures in female athletes showed the most predictive factors of stress fractures to be decreased bone mineral density, current calcium intake, irregular menstrual cycles, number of years running, and history of training on hard surfaces [16].

Calcium intake is associated with both and, along with vitamin D homeostasis, is a well-known necessity for quality bone. Two prospective studies of athletes and calcium intake suggest high calcium intake of 1500–2000 mg a day could be protective against stress fractures and a 2 year prospective study of young female cross-country runners found that a high-dairy and low-fat diet led to a 68% reduction in stress fracture incidence [17–19]. In young females presenting with stress fractures and low BMIs, eating disorder counseling and referral to primary care may be necessary. Smoking cessation is another modifiable risk factor to improve bone mineral density [20–22].

Training education is also important to ensure the patient is using satisfactory equipment and understands pacing the progression of their workouts. Maintaining a base level of fitness can also be protective, especially before initiating a high intensity work-out regimen [8]. The quality of training equipment, especially shoes, should also be stressed. This multi-aspect training education is especially relevant today

with the contemporary high intensity, work-to-failure training fads.

### Calcaneal Stress Fracture

Calcaneal stress fractures are common with incidence of tarsal bone fractures as high as 21%. They were first described in military recruits and are associated with excessive marching [23]. Risk factors include increased force during the gait cycle at heel strike, as seen in marching, running, or jumping.

Patients present with tenderness to palpation at the posterior superior calcaneus and with swelling at the precalcaneal bursa. However, they often present later in the course of the injury when swelling could have decreased. There is also pain with pressure applied to the medial and lateral calcaneal tuberosities. These stress fractures can be misdiagnosed as plantar fasciitis due to similar pain with weight bearing and onset associated with changes in activity. However, imaging can differentiate between the two.

Calcaneal stress fractures have a characteristic appearance best seen on lateral views of standard radiographs: a fracture line at the posterior-superior calcaneus perpendicular to the trabeculae stress line again seen best in Fig. 17.2 [13, 24]. The same general principles of utilizing advanced imaging including MRI, bone scans, and CT also apply to calcaneal stress fractures.

Unlike other foot and ankle stress fractures, the treatment for calcaneal stress fractures is symptomatic. The patient can bear weight in a cast or boot, but running, training, or high-

impact activity should be avoided. Tenderness to palpation and swelling should typically resolve by 2–4 weeks. Unlike fractures of the metatarsals, serial radiographs are not required. Also there are no absolute surgical indications. Fracture displacement and recurrence are not commonly seen. The patient's symptomatic pain with activity dictates the return to activity.

### Metatarsal Stress Fractures

Metatarsal (MT) stress fractures are one of the more common foot and ankle stress fractures accounting for as many as 20% of cases [9]. Of these, the 2nd is the most common (52%) followed by the 3rd (35%), 1st (8%), 4th (5%), and 5th (5%). Keeping with Levy's previously mentioned classification, metatarsals 2–4 are diaphyseal fractures, and the 1st metatarsal typically involves cancellous metaphyseal bone on the medial aspect as seen in Fig. 17.3 [13]. The 5th metatarsal



**Fig. 17.2** Lateral Calcaneus Radiograph: The arrows indicate the stress fracture perpendicular to the calcaneal trabeculae



**Fig. 17.3** 1st Metatarsal Radiograph: The fracture line perpendicular to the 1st metatarsal trabeculae can be appreciated along its medial as well as lateral central third

stress fracture will be discussed separately as it has a unique mechanism when compared to the other four MTs.

Risk factors for developing metatarsal stress fractures are related to biomechanical alignment of the foot, especially the plantar arch. Running and jumping rely on the plantar arch, and peak strains are increased in the MT during these activities. Although the ligamentous anchoring between the heads of the MTs protect against fracture displacement, they also increase plantar-oriented forces during weight bearing [25]. Specifically, the 2nd and 3rd MTs have the highest peak pressure during weight bearing [26]. Barefoot activities (no arch support) and pes planus also impart a higher strain on the MT [27, 28]. The plantar fascia specifically has been shown to play an important role in stress on the MTs based on reports that strain increased 100% with sectioning [28].

Other than the plantar arch, relative lengths of the 1st and 2nd metatarsal have been shown to play a role [29, 30]. Chuckpaiwong et al. hypothesized that the short 1st MT in Morton's foot leads to abnormal overloading of stress along the full length of the 2nd MT. Also, foot muscle fatigue has been shown to increase strain on the 2nd MT leading to a fourfold reduction in the number of cycles needed to cause failure [31]. This is an important point as it highlights the inverse relationship of intensity to cycle load: increased intensity and associated muscle fatigue leads to a decreased number of cycles required to produce a stress fracture.

MT stress fractures present with dorsal swelling and tenderness. This tenderness differs in 1st MT vs. 2–4 MT based on the previously mentioned fracture patterns [32]. These stress fractures often have a very rapid onset and may only need one stressor for the injury to occur. Despite this rapid onset, the average time to presentation has been reported as 2–6 weeks [27]. History is again very important, and these fractures are often seen in groups with heavy stress on the plantar arch: military recruits, ballet dancers, and athletes in running and cutting sports. A differential diagnosis includes metatarsalgia, synovitis, Morton neuroma, and other arthritic conditions.

As previously seen, bone scans and MRIs are useful for acute diagnosis. As in other stress fractures described by Levy, radiographs can detect these fractures in the subacute phase with characteristic findings based on fracture patterns. The 1st MT often shows linear sclerosis perpendicular to the line of stress and periosteal reaction is rare [13]. However, the 2–4th MT often shows periosteal reaction. Specifically, the 2nd and 3rd MTs demonstrate changes along the medial diaphysis and the lateral proximal diaphyseal-metaphyseal junction [33].

In general MT stress fractures, a proximal location is less common than non-proximal and is associated with worse outcomes. Chuckpaiwong et al. noted that these fractures are also associated with chronic symptoms, Achilles contracture, Morton's foot, and low bone mass. They also found a low training volume (<0.5 h a week), and none of the sports-related injuries in their study were in ballet dancers [31].

Therefore, the clinician must always maintain a high level of suspicion and not rely on common history points like high training volume or participation in ballet.

The majority of MT stress fractures are treated by activity restriction with or without immobilization. The patient is able to return to activity when the radiographs show healing and there is no tenderness to palpation on physical examination. Serial radiographs can be utilized to document bony union. Surgical indications include significantly increased dorsiflexion and non-union. Plantar flexion MT osteotomy minimizes the risk of malunion and transfer lesions. A transfer lesion occurs when there is increased strain and risk of stress fracture at the 3rd MT after 2nd MT stress fracture. Since the 2nd and 3rd MTs have the highest peak pressure with weight bearing, the 3rd MT must take a greater share of the load when the 2nd MT is injured [26]. This increases the strain seen by the 3rd MT head potentially predisposing these patients to adjacent fracture of the 3rd MT. Non-unions are treated by take-down of the non-union and rigid internal fixation with possible bone grafting [34].

---

## Ballet

Ballet Dancers are a unique population with a higher risk for MT stress fractures due to the combined effect of unusual muscle usage while en pointe and increased risk for the female athlete triad with subsequent poor bone quality. Unlike other metatarsal stress fractures, 2nd MT stress fractures typically occur at the proximal base.

The en pointe position increases the stress on the plantar arch through extremes of plantar flexion for sustained time intervals. Through the previously mentioned ligamentous attachments, this increased plantar arch stress is transmitted to the 2nd and 3rd MTs. This increased stress, along with the peroneus longus and posterior tibial tendon tendon insertions, focuses increased stress at the 2nd MT base.

These patients have an average presentation delay of 2.5–6.5 weeks, but present like other MT stress fractures with swelling and tenderness to palpation at the fracture site [35]. In addition to the standing AP and lateral radiographs, the diagnosis can be made with the Dancer's View: a PA view with the dorsum of the foot against the cassette. The characteristic finding is an oblique fracture line along the proximal aspect of the 2nd MT. Treatment consists of immobilization and temporary cessation of dancing.

---

## Navicular Stress Fractures

Towne et al. first described navicular stress fractures in humans in 1970 [36]. Although not as common as other tarsal stress fractures, they require a high degree of suspicion

due to potentially poor outcomes related to vascular anatomy. Small branches of the posterior tibial artery and dorsalis pedis enter the navicular at the insertion of posterior tibialis tendon, anastomose, and radiate toward the center of the bone. This distribution leads to a relatively avascular central third of the navicular.

Due to its anatomic location, the navicular undergoes physical compression from the talus proximally and the cuneiforms distally with load bearing in plantar flexion. The increased torque with push-off in sprinting and jumping exaggerates this mechanism and would be expected in sports with excessive cycles of these two activities: hurdles, basketball, football, etc. [37]. Although no statistically significant associations have been identified, anything leading to increased stress across the midfoot could logically be considered a relative risk factor such as an Achilles contracture, pes cavus, pes planus, Morton's foot, bony prominences, and limited dorsiflexion due to osteophytes on the distal tibia or talar neck.

They usually present with vague foot pain, and, as with others, the pain is activity related and progresses the longer the patient performs the activity. Due to the anatomic location of the navicular, the pain can be initially described as "ankle pain," and can obscure the diagnosis. Furthermore, range of motion is usually normal with little to no swelling or ecchymosis. However there are some unique aspects of the physical exam that suggest navicular stress fracture. Khan et al. [38] coined the term "N spot" for the tender area over the dorsal center navicular, and this pain can radiate into the medial arch. The symptoms can be reproduced when hopping on the plantar flexed foot [39].

The diagnosis can be delayed from 4 to 7 months by the vague symptomatic complaints as well as by difficulty visualizing the injury on standard radiograph [38, 40]. A true and clear AP of the navicular is very difficult to obtain which limits radiographs efficacy [41]. MRI is often used as a screen due to its higher specificity, but, if edema is present, CT is warranted to define both the fracture pattern and to differentiate between avascular necrosis and stress fractures.

Treatment depends on the fracture characteristics: displaced vs. non-displaced and complete vs. partial. Khan et al. showed non-weight bearing to be statistically significantly superior to weight bearing, and found no difference between non-weight bearing and surgery [42]. Therefore, associated general surgical complications should be avoided if possible. Non-displaced fractures can be treated conservatively with non-weight bearing activity modification followed by re-evaluation in 6–8 weeks. If pain persists, then a repeat CT should be obtained for further evaluation. If there is no pain, then the patient can begin rehabilitation and physical therapy. However, high-level athletes may not be amenable to the long conservative treatment course that can last 6–8 months.

Surgical indications include displaced fractures, non-displaced complete fractures with sclerotic changes, incom-

plete fractures with evidence of propagation, those that fail conservative treatment, and those that cannot tolerate the duration of conservative treatment [39, 43]. Screws can be placed percutaneously for incomplete or complete non-displaced fractures, but displaced fractures or those needing bone graft require open fixation. For 4–6 weeks post-operatively, displaced fractures or complete fractures with sclerotic changes should have protected weight bearing, and incomplete fractures should have a walker boot and be allowed to bear weight. Orthotic devices can be used when activity resumes particularly those aimed at correcting the patients underlying foot morphology, typically cavo-varus. Complications from surgery include non-union or malunion, failed screw fixation, and AVN. Due to the ramifications of these complications, CT should not be delayed if symptoms persist [44, 45].

---

## Sesamoid Stress Fractures

Despite the multiple variations of sesamoids, this section will focus on the two hallux sesamoids. The lateral of fibular sesamoid is smaller, rounder, and more proximal in relation to the medial or tibial sesamoid. The tibial sesamoid is larger, longer, and more distal. It also experiences more stress during weight-bearing, and therefore is more often injured [25, 46, 47]. These two bones have three functions to assist with push-off power [48, 49]. They dissipate forces within the hallux metatarsalphalangeal (MTP) joint and elevate the metatarsal head to increase the mechanical advantage of the flexor hallucis brevis tendon while simultaneously protecting and maintaining the position of the flexor hallucis longus tendon.

Patients often present with pain localized over the plantar hallux MTP joint that worsens with weight-bearing. The associated pain varies slightly based on the sesamoid. The tibial pain is often plantar medial while the fibular is often directly plantar. Either way, the aforementioned plantar pain is usually present. As with other stress fractures, there is variable presence of edema and erythema.

In addition to standing AP and lateral radiographs, oblique views can be obtained to specifically evaluate the tibial sesamoid. A radio-opaque marker at the site of tenderness can be useful to differentiate the two sesamoids as well as a sesamoid from a flexor tendon. Sesamoid fractures can also be confused with bipartite sesamoids. The fracture will have irregular borders vs. the smooth cortical borders of intact bones as seen in Fig. 17.4. Also, bipartite sesamoids are often bilateral, and a contralateral radiograph could be obtained for comparison [46].

Conservative treatment is usually indicated and can include permissive weight bearing, taping, orthotics, and/or casting. A toe spica addition to a cast can relieve stress





**Fig. 17.4** Sesamoid Radiograph: The black transverse horizontal fracture line can be appreciated in the AP of the 1st MT sesamoid

on the sesamoid with mild plantar-flexion of the hallux. Taping compresses the fracture and limits movement, and it can be considered for mild symptoms or athletes. Chronic symptoms generally require orthoses or footwear modifications, and these can be either over the counter or custom made. In athletes that continue activity, serial radiographs can be useful to screen for a possible complication of diastasis with cock-up deformity of the hallux.

Surgical indications include pain for more than 6 months, functional loss, and failure of non-operative management. Two techniques are commonly employed: sesamoidectomy and bone grafting. Sesamoidectomy, either partial or total, carries the risk of loss of push-off strength, hallux valgus, and bunion deformity. A distal metatarsal osteotomy should be considered if a hallux valgus deformity is present prior to surgery. Anderson and McBryde [25, 47] developed a bone grafting procedure that gives favorable results for tibial sesamoid stress fractures with less than 2 mm diastasis with no gross motion between the two fragments.

### Fibular Stress Fracture

Detelfsen first described fibular stress fractures in 1937. A few years later, in 1940, Burrows described two groups classically seen with this injury: young male athletes and middle

aged females. The middle-aged females are more likely to develop a fracture distally in cancellous bone, most likely related to bone mineral density. However, young male athletes, specifically runners and skaters in the Burrows description, tended to have a more proximal fracture in denser cortical bone. This group had a stronger correlation with increased activity [50].

Despite these clear descriptions, there is no universally accepted theory for etiology. Richmond and Shafar describe eversion of the hindfoot that results in opposition of the pull of the tibiofibular interosseous ligament. This medial pull of the ligament imparts a lateral force on the distal portion of the fibula proximal to the malleolus [51]. Devas described another mechanism of repetitive plantar flexor and ankle and toe flexor contraction during running. With repetitive cycles, this motion at the fibula creates the microfractures associated with stress fractures.

As with other stress fractures, fibular stress fractures are often seen in runners and military recruits [52, 53]. The onset of pain is usually quick, often within 10 days of change in activity. However, the pain is generalized, the tenderness is difficult to pinpoint, and the edema is diffuse. As in other stress fractures around the ankle, these findings obscure the diagnosis. The tenderness, when present, may be on the postero-lateral boarder of the fibula roughly 4–7 cm from the tip of the malleolus. A more characteristic finding is seen in the sub-acute presentation: a palpable prominence from the abundance of callus [50].

This is the one foot and ankle stress fracture that rarely needs bone scan or MRI as a screen. The radiographs will show findings at roughly 3–4 weeks. Typically, there will be the aforementioned abundant callus as well as a transverse fracture line [50]. These stress fractures are generally treated conservatively with rest and restricted physical activity. Devas recommends weight bearing but no resumption of activity until symptom free [52]. The typical time to return to play was 6–8 weeks.

### Evidence

- Level 1:

References 17, 19:

17. J W Nieves, K Melsop, M Curtis, J L Kelsey, L K Bachrach. **Nutritional factors that influence change in bone density and stress fracture risk among young female cross-country runners.** PM & R 2010, Aug; 2 [8]: 740–50.
19. Lappe J, Cullen D, Haynatzki G, Recker R, Ahlf R, Thompson K. **Calcium and vitamin d supplementation decreases incidence of stress fractures in female navy recruits.** J Bone Miner Res 2008, May;23(5):741–9.

- Level 2:

none

## Summary

1. The history is the most vital aspect of diagnosing stress fractures and should focus on high risk groups (ballerinas, military personnel, runners), females, changes in activity, and factors relating to bone mineral density.
2. The physical exam for stress fractures should focus on bony palpation, edema, and pain with weight bearing.
3. Diagnosing stress fractures of the foot and ankle require a high clinical suspicion because radiographs are benign until the subacute phase and MRI is an expensive and unconventional initial screening test.
4. Rehabilitation of stress fractures focuses on conservative treatment including limited weight bearing and activity modification. Surgery is often not indicated, and return to previous activity is generally advised when the patient is symptom free with no radiographic evidence of stress fracture.
5. Prevention of stress fractures should focus on bone mineral density improvement (nutrition, calcium, osteoporosis) and education of training technique, equipment, and regimen.

## References

1. Breithaupt J. Zur pathologie des menschlichen fusses. *Medizin Zeitung*. 1855;24:169–77.
2. Daffner RH, Martinez S, Gehweiler JA, Harrelson JM. Stress fractures of the proximal tibia in runners. *Radiology*. 1982;142(1):63–5.
3. Beck BR. Tibial stress injuries. An aetiological review for the purposes of guiding management. *Sports Med*. 1998;26(4):265–79.
4. Daffner RH, Pavlov H. Stress fractures: current concepts. *AJR Am J Roentgenol*. 1992;159(2):245–52.
5. Matheson GO, Clement DB, McKenzie DC, Taunton JE, Lloyd-Smith DR, MacIntyre JG. Stress fractures in athletes. A study of 320 cases. *Am J Sports Med*. 1987;15(1):46–58.
6. Brockwell J, Yeung Y, Griffith JF. Stress fractures of the foot and ankle. *Sports Med Arthrosc*. 2009;17(3):149–59.
7. Hoppenfeld S, Hutton RJA. *Physical examination of the spine and extremities*. New York: Appleton-Century-Crofts; 1976.
8. Gardner LI, Dziados JE, Jones BH, Brundage JF, Harris JM, Sullivan R, Gill P. Prevention of lower extremity stress fractures: a controlled trial of a shock absorbent insole. *Am J Public Health*. 1988;78(12):1563–7.
9. McBryde AM. Stress fractures in runners. *Clin Sports Med*. 1985;4(4):737–52.
10. Levy JM. Stress fractures of the first metatarsal. *AJR Am J Roentgenol*. 1978;130(4):679–81.
11. Spitz DJ, Newberg AH. Imaging of stress fractures in the athlete. *Radiol Clin North Am*. 2002;40(2):313–31.
12. Lee JK, Yao L. Stress fractures: MR imaging. *Radiology*. 1988;169(1):217–20.
13. LEABHART JW. Stress fractures of the calcaneus. *J Bone Joint Surg Am*. 1959;41-A:1285–90.
14. Kaeding CC, Miller T. The comprehensive description of stress fractures: a new classification system. *J Bone Joint Surg Am*. 2013;95(13):1214–20.
15. Harder AT, An YH. The mechanisms of the inhibitory effects of nonsteroidal anti-inflammatory drugs on bone healing: a concise review. *J Clin Pharmacol*. 2003;43(8):807–15.
16. Wentz L, Liu PY, Ilich JZ, Haymes EM. Dietary and training predictors of stress fractures in female runners. *Int J Sport Nutr Exerc Metab*. 2012;22(5):374–82.
17. Nieves JW, Melsop K, Curtis M, Kelsey JL, Bachrach LK. Nutritional factors that influence change in bone density and stress fracture risk among young female cross-country runners. *PM R*. 2010;2(8):740–50.
18. Tenforde AS, Sayres LC, Sainani KL, Fredericson M. Evaluating the relationship of calcium and vitamin D in the prevention of stress fracture injuries in the young athlete: a review of the literature. *PM R*. 2010;2(10):945–9.
19. Lappe J, Cullen D, Haynatzki G, Recker R, Ahlf R, Thompson K. Calcium and vitamin d supplementation decreases incidence of stress fractures in female navy recruits. *J Bone Miner Res*. 2008;23(5):741–9.
20. Altarac M, Gardner JW, Popovich RM, Potter R, Knapik JJ, Jones BH. Cigarette smoking and exercise-related injuries among young men and women. *Am J Prev Med*. 2000;18(3 Suppl):96–102.
21. Jones BH, Cowan DN, Tomlinson JP, Robinson JR, Polly DW, Frykman PN. Epidemiology of injuries associated with physical training among young men in the army. *Med Sci Sports Exerc*. 1993;25(2):197–203.
22. Reynolds KL, Heckel HA, Witt CE, Martin JW, Pollard JA, Knapik JJ, Jones BH. Cigarette smoking, physical fitness, and injuries in infantry soldiers. *Am J Prev Med*. 1993;10(3):145–50.
23. Greaney RB, Gerber FH, Laughlin RL, Kmet JP, Metz CD, Kilcheski TS, et al. Distribution and natural history of stress fractures in U.S. Marine recruits. *Radiology*. 1983;146(2):339–46.
24. D'Ambrosia RD, Drez D. *Prevention and treatment of running injuries*. Thorofare: C.B. Slack; 1982.
25. Anderson RB, McBryde AM. Autogenous bone grafting of hallux sesamoid nonunions. *Foot Ankle Int*. 1997;18(5):293–6.
26. Griffin NL, Richmond BG. Cross-sectional geometry of the human forefoot. *Bone*. 2005;37(2):253–60.
27. Milgrom C, Finestone A, Sharkey N, Hamel A, Mandes V, Burr D, et al. Metatarsal strains are sufficient to cause fatigue fracture during cyclic overloading. *Foot Ankle Int*. 2002;23(3):230–5.
28. Donahue SW, Sharkey NA. Strains in the metatarsals during the stance phase of gait: implications for stress fractures. *J Bone Joint Surg Am*. 1999;81(9):1236–44.
29. Drez D, Young JC, Johnston RD, Parker WD. Metatarsal stress fractures. *Am J Sports Med*. 1980;8(2):123–5.
30. HARRIS RI, BEATH T. The short first metatarsal; its incidence and clinical significance. *J Bone Joint Surg Am*. 1949;31A(3):553–65.
31. Chuckpaiwong B, Cook C, Pietrobon R, Nunley JA. Second metatarsal stress fracture in sport: comparative risk factors between proximal and non-proximal locations. *Br J Sports Med*. 2007;41(8):510–4.
32. Lucas MJ, Baxter DE. Stress fracture of the first metatarsal. *Foot Ankle Int*. 1997;18(6):373–4.
33. Meurman KO. Less common stress fractures in the foot. *Br J Radiol*. 1981;54(637):1–7.
34. Porter DA, Foulk DM, Rund AM. Intramedullary screw fixation for chronic proximal fourth metatarsal stress fractures: a new technique for the fourth metatarsal jones. *Tech Foot Ankle Surg*. 2010;9(3):147–53.
35. O'Malley MJ, Hamilton WG, Muniyakk J, DeFranco MJ. Stress fractures at the base of the second metatarsal in ballet dancers. *Foot Ankle Int*. 1996;17(2):89–94.
36. Towne LC, Blazina ME, Cozen LN. Fatigue fracture of the tarsal navicular. *J Bone Joint Surg Am*. 1970;52(2):376–8.
37. Bennell KL, Malcolm SA, Thomas SA, Wark JD, Brukner PD. The incidence and distribution of stress fractures in competitive track and field athletes. A twelve-month prospective study. *Am J Sports Med*. 1996;24(2):211–7.

38. Khan KM, Fuller PJ, Brukner PD, Kearney C, Burry HC. Outcome of conservative and surgical management of navicular stress fracture in athletes. Eighty-six cases proven with computerized tomography. *Am J Sports Med.* 1992;20(6):657–66.
39. Fitch KD, Blackwell JB, Gilmour WN. Operation for non-union of stress fracture of the tarsal navicular. *J Bone Joint Surg Br.* 1989;71(1):105–10.
40. Torg JS, Pavlov H, Cooley LH, Bryant MH, Arnoczky SP, Bergfeld J, Hunter LY. Stress fractures of the tarsal navicular. A retrospective review of twenty-one cases. *J Bone Joint Surg Am.* 1982;64(5):700–12.
41. Pavlov H, Torg JS, Freiburger RH. Tarsal navicular stress fractures: radiographic evaluation. *Radiology.* 1983;148(3):641–5.
42. Khan KM, Brukner PD, Kearney C, Fuller PJ, Bradshaw CJ, Kiss ZS. Tarsal navicular stress fracture in athletes. *Sports Med.* 1994;17(1):65–76.
43. Saxena A, Fullem B. Comment on torg et al, “management of tarsal navicular stress fractures: conservative versus surgical treatment”. *Am J Sports Med.* 2010;38(10):NP3.
44. McCormick F, Nwachukwu BU, Provencher MT. Stress fractures in runners. *Clin Sports Med.* 2012;31(2):291–306.
45. Kiss ZS, Khan KM, Fuller PJ. Stress fractures of the tarsal navicular bone: CT findings in 55 cases. *AJR Am J Roentgenol.* 1993;160(1):111–5.
46. Gould JS. *Operative foot surgery.* Philadelphia: W.B. Saunders Co; 1994.
47. McBryde AM, Anderson RB. Sesamoid foot problems in the athlete. *Clin Sports Med.* 1988;7(1):51–60.
48. Aper RL, Saltzman CL, Brown TD. The effect of hallux sesamoid resection on the effective moment of the flexor hallucis brevis. *Foot Ankle Int.* 1994;15(9):462–70.
49. Aper RL, Saltzman CL, Brown TD. The effect of hallux sesamoid excision on the flexor hallucis longus moment arm. *Clin Orthop Relat Res.* 1996;325:209–17.
50. BURROWS HJ. Fatigue fractures of the fibula. *J Bone Joint Surg Br.* 1948;30B(2):266–79.
51. Richmond DA, Shafar J. A case of bilateral fatigue fracture of the fibula. *Br Med J.* 1955;1(4908):264.
52. DEVAS MB. Stress fractures of the tibia in athletes or shin soreness. *J Bone Joint Surg Br.* 1958;40-B(2):227–39.
53. Miller MD, Marks PH, Fu FH. Bilateral stress fractures of the distal fibula in a 35-year-old woman. *Foot Ankle Int.* 1994;15(8):450–3.

Jonathan A. Godin, Travis J. Dekker, and C.T. Moorman III

### Abstract

Compartment syndrome of the lower leg or foot is usually seen in the setting of high-energy trauma. However, chronic exertional compartment syndrome (CECS) is most frequently seen in athletes. The diagnosis is largely based on clinical examination, and the most sensitive clinical symptom of compartment syndrome is severe pain. Intracompartmental pressure measurements can be obtained to confirm diagnosis. Once the diagnosis is made, immediate fasciotomy of all involved compartments is required. Fasciotomy of the lower leg can be performed by one lateral incision or by medial and lateral incisions. Endoscopic fasciotomy is also described in cases of CECS. Foot compartment syndrome requires thorough examination of all compartments with special focus on the calcaneal compartment. Depending on the injury, clinical examination, and compartment pressure, fasciotomy is recommended via a dorsal and/or medial plantar approach. Surgical intervention does not eliminate the risk of developing nerve and muscle dysfunction. If left untreated, poor outcomes characterized by sensory deficits, weakness, paralysis, contractures, and toe deformities can be expected.

### Keywords

Compartment • Syndrome • Acute • Chronic • Exertional • Leg • Foot

## Introduction

Compartment syndrome can present in either an acute or a chronic timeframe. Acute compartment syndrome is usually due to trauma to, or reperfusion of, the extremity. Chronic exertional compartment syndrome (CECS) is often associated with the repetitive loading or microtrauma of endurance activities. Both acute and chronic compartment syndromes are due to increased interstitial pressure within a compartment, resulting in decreased perfusion and ischemia of soft tissues. In contrast to the reversible nature of CECS, acute compartment syndromes progress rapidly and

require urgent fasciotomy to avoid irreversible soft tissue necrosis in the affected compartment. Clinical manifestations of compartment syndrome include pain, pallor, poikilothermia, pulselessness, swelling, numbness, and weakness of the extremity [1, 2]. The reported incidence ranges between 14 and 33 % among individuals with lower leg pain [3–5].

While a number of case reports have been published highlighting acute compartment syndrome of the leg and foot, the majority of sports-related compartment syndrome cases are due to CECS [6–13]. Wilson first described the concept of CECS in 1912, but Mavor was the first to successfully treat a patient with anterior compartment syndrome of the leg using a fasciotomy [14]. CECS is often bilateral and is equally prevalent among males and females. Moreover, diabetic patients may be at increased risk of developing CECS [15]. Case reports of CECS of the forearm, thigh, and gluteal regions exist, but are rare [16–18]. The leg is the most

J.A. Godin, MD, MBA • T.J. Dekker, MD  
• C.T. Moorman III, MD (✉)  
Duke University Medical Center, Department of Orthopaedic  
Surgery, Duke Sports Science Institute, Durham, NC 27710, USA  
e-mail: [jonathan.godin@duke.edu](mailto:jonathan.godin@duke.edu); [travis.dekker@dm.duke.edu](mailto:travis.dekker@dm.duke.edu);  
[t.moorman@dm.duke.edu](mailto:t.moorman@dm.duke.edu)

common site, with the anterior and lateral compartments most frequently affected.

Foot compartment syndrome (FCS) is most often associated with trauma of the foot. Typical injuries that make a patient more susceptible to FCS include calcaneus fractures, Lisfranc injuries, and multiple metatarsal neck or shaft fractures [19]. CECS of the foot is most often specific to the medial compartment of the foot [20–25]. Foot trauma leads to severe edema in relatively small tissue compartments that can incite FCS. However, CECS of the foot has been observed in patients exercising on their feet for long periods of time (i.e. long distance runners/triathletes, infantrymen, dancers) [21, 24, 25]. In addition, acute exertional compartment syndromes of the foot have been described in basketball players, football players and marathon runners [26].

## Etiology and Pathomechanism

### Anatomy

The leg contains four compartments: anterior, lateral, superficial posterior, and deep posterior. The anterior compartment contains the anterior tibial artery, the deep peroneal nerve, and four muscles (tibialis anterior, extensor digitorum longus, extensor hallucis longus, and peroneus tertius). Its borders are the tibia, fibula, interosseus membrane, anterior intermuscular septum, and deep fascia of the leg. The lateral compartment contains the superficial peroneal nerve and two muscles (peroneus longus and peroneus brevis). Its borders are the anterior intermuscular septum, the fibula, the posterior intermuscular septum, and the deep fascia. The common peroneal nerve branches into the superficial and deep peroneal nerves within the substance of the peroneus longus after passing along the neck of the fibula. The superficial peroneal nerve continues within the lateral compartment, while the deep peroneal nerve wraps around the fibula deep to the extensor digitorum longus until reaching the anterior surface of the interosseus membrane. The lateral compartment does not contain a large artery; the peroneal muscles receive their blood supply via several branches of the peroneal artery.

The superficial posterior compartment contains the sural nerve and three muscles (gastrocnemius, soleus, and plantaris) and is surrounded by the deep fascia of the leg. The deep posterior compartment contains the posterior tibial and peroneal arteries, tibial nerve, and four muscles (flexor digitorum longus, flexor hallucis longus, popliteus, and tibialis posterior). It is bordered anteriorly by the tibia, fibula, and interosseus membrane, and posteriorly by the deep transverse fascia. A fifth compartment that encloses the tibialis posterior muscle has been described, but its existence is controversial [27]. It has been suggested that the presence of an

extensive fibular origin of the flexor digitorum longus muscle may create a subcompartment within the deep posterior compartment that may develop elevated pressures [28].

The identification of clinically significant compartments of the foot has changed over time. Initially, four fascial compartments were identified: medial, lateral, central and interosseous [29]. Manoli and Weber identified nine compartments of the foot, dividing the previous central compartment into a superficial and deep compartment and also increasing the interosseous compartment from one to four functional compartments [19]. The deep compartment of the foot encompasses the plantar surface of the calcaneus. The deep/calcaneal compartment contains the quadratus plantae and posterior tibial neurovascular bundle to include the lateral plantar nerve often associated with the sequelae of FCS. The deep compartment of the foot communicates with the deep posterior compartment of the leg.

Due to their communication, authors have documented FCS with no history of foot trauma [30]. More recently, clinically important foot compartments were identified based on FCS sequelae. Ling and Kumar concluded that three vertical fascial septae extended from the hindfoot to the midfoot on the plantar surface of the foot creating a medial, intermediate, and lateral compartment. The medial compartment contains the abductor hallucis and is bound by skin medially and the medial septum laterally. The intermediate compartment contains the flexor digitorum brevis and quadratus plantae. Lastly, the lateral compartment contains the abductor digiti minimi. The intermediate and lateral compartments are rigidly bound by fascial layers and require decompression to prevent FCS sequelae [31]. Compartment syndrome of the deep calcaneal compartment has commonly been associated with 10% of calcaneus fractures, half of which develop sequelae of foot compartment syndrome: claw toes, neurovascular dysfunction and stiffness [32, 33].

### Pathomechanism

The pathophysiologic mechanism that causes compartment syndromes is increased tissue pressure and the resulting development of ischemia, which leads to irreversible muscle damage. Cellular anoxia is the final common pathway of all of the varieties of compartment syndrome. However, the interaction between increased compartment pressure, blood pressure, and blood flow are incompletely understood. Muscle ischemia can lead to the release of myoglobin from damaged muscle cells. During reperfusion, myoglobin is released into the circulation with other inflammatory and toxic metabolites. Myoglobinuria, metabolic acidosis, and hyperkalemia can lead to renal failure, shock, hypothermia, and cardiac arrhythmias and/or failure [34]. The development and extent of these systemic effects depends on the

severity and duration of compromised tissue perfusion and the size and number of muscle compartments involved.

Similarly, CECS is thought to be due to an abnormal increase in intramuscular pressure during exercise resulting in impaired local perfusion, tissue ischemia, and pain. One study demonstrated a 20% increase in muscle volume during exercise [35]. Elevated lactate levels and water content have been documented in muscle biopsies from compartments with elevated pressures following exercise [4]. Muscle hypertrophy and increased perfusion volume with exertion do not explain the elevated resting pressure seen in patients with CECS, however. The mechanical damage theory hypothesizes that heavy exertion results in myofibril damage, release of protein-bound ions, increased osmotic pressure in the interstitial space, and, therefore, decreased arteriolar flow in the compartment.

Additionally, in some cases, focal fascial defects may be a contributing factor. Anterolateral fascial hernias are present in 39–46% of patients with CECS, as compared to less than 5% of asymptomatic individuals [36, 37]. These defects typically are located near the anterior intermuscular septum between the anterior and lateral compartments, and they can entrap the superficial peroneal nerve exiting the junction of the middle and distal thirds of the leg. Evidence suggests that patients with CECS have a lower capillary density in relation to muscle fiber size compared to controls. This decreased capillarity leads to decreased structural capacity for muscle blood flow [38]. None of the existing theories explain all of the available data on the etiology of CECS. Most likely, the pathogenesis of the elevated intracompartmental pressures seen in CECS is multifactorial.

## Symptoms

The classic clinical diagnosis encompasses the six Ps: pain, pressure, pulselessness, paralysis, paresthesias, and pallor [34]. Pain out of proportion to the injury, aggravated by passive stretching of muscle groups in the corresponding compartment, is one of the earliest and most sensitive clinical signs of compartment syndrome. However, pain may be an unreliable indicator and may be absent in an established compartment syndrome [34]. Pressure or firmness in the compartment, a direct manifestation of increased intracompartmental pressure, is the earliest and may be the only objective finding of early compartment syndrome. Peripheral pulses are palpable and, unless a major arterial injury is present, capillary refill is routinely present [34]. Patients may develop numbness and weakness over time. Pallor is uncommon and is associated with the rare instance where arterial inflow is occluded.

With CECS, these symptoms develop at a predictable interval after initiation of a repetitive, endurance-type activ-

ity and resolve with rest. The following symptoms may be present upon exertion and resolve with rest: cramping, burning, aching, or tightness in the region of the affected compartment(s); numbness, paresthesias or weakness in the extremity; a transient foot drop may develop if the deep peroneal nerve is affected; a temporary loss of eversion strength may occur if the superficial peroneal nerve is affected. Physical examination of the resting lower extremity often is unremarkable. Examination following exercise may reveal: tightness or tenderness to palpation of the involved compartments. If a fascial defect is present, a focal area of tenderness and swelling may develop as the underlying muscle bulges through the defect. A positive Tinel's sign may be present over the defect if the superficial peroneal nerve is compressed. In addition, numbness and/or motor weakness may be present in the superficial peroneal, deep peroneal, or tibial nerve distributions. The vibration test consists of placing a vibrating tuning fork over bone at the area of suspected stress; an elicitation of pain is consistent with a stress fracture. Pain when performing resisted ankle dorsiflexion and inversion is consistent with tibialis posterior tendinitis or posteromedial periostitis.

In the setting of trauma, increased awareness of FCS is necessary in order to adequately evaluate the patient as many symptoms from the inciting incident typically overlap with FCS symptoms. The pain seen in FCS is described as a relentless burning pain of the entire foot [19]. CECS symptoms of the foot include tightness developing on the medial side of the foot with burning and throbbing pain in the setting of exercise [25]. Furthermore, the patient often complains of a cramp-like sensation along the medial longitudinal arch or hindfoot. Pain with passive dorsiflexion of the toes has been found to be the most reliable physical exam finding when attempting to diagnose foot compartment syndrome [39]. This maneuver decreases the overall volume of the intrinsic muscles of the foot and passively places them on stretch. Myerson's series in 1991 demonstrated the following: 12 of 14 ft had pain with passive dorsiflexion, 7 of 11 demonstrated decreased loss of two-point discrimination and 7 of 13 ft showed loss of light touch sensation [29]. Paresthesias, numbness, change in color/pallor or swelling over the abductor hallucis are all further symptoms/signs often seen in the setting of chronic exertional compartment syndrome of the foot. The pain is relieved after a certain amount of time and activity cessation [21].

---

## Diagnostics, Classification

### Imaging and Other Diagnostic Studies

When the history and physical examination findings are consistent with compartment syndrome, the diagnosis should be confirmed with compartment pressure measurements. In the

setting of CECS, pre- and post-exercise compartment pressure measurements should be assessed. For acute compartment syndrome, a perfusion pressure (the difference between diastolic blood pressure and compartment pressure) of 20 mmHg has been shown to be an absolute indicator for fasciotomy [34]. For CECS, most clinicians follow the diagnostic criteria of Pedowitz et al, in which a resting pressure greater than or equal to 15 mmHg or a 1-min post-exercise measurement greater than or equal to 30 mmHg or a 5-min post-exercise measurement greater than or equal to 20 mmHg is considered abnormal and diagnostic of CECS [40]. The exercise performed at the time of testing must be intense enough to reproduce the patient's symptoms; otherwise, the post-exercise pressure measurements may result in a false negative result.

Several methods for measuring compartment pressures have been described in the literature. These include the slit catheter, wick catheter, needle manometry, digital pressure monitor, microcapillary infusion, and solid-state transducer intracompartmental catheter methods [17, 37, 41–44]. The Stryker intracompartmental pressure monitor (Kalamazoo, MI) is a handheld digital monitor that can be used to check multiple compartments. It can be used either with a side port needle or an indwelling slit catheter to obtain serial measurements in a single compartment. A handheld digital device recently developed by Synthes (Paoli, PA) also allows placement of indwelling catheters and may be useful for obtaining serial measurements. Near-infrared spectroscopy has been used to determine tissue oxygen saturation [45]. This may be a noninvasive, painless alternative to intracompartmental pressures in the diagnosis of CECS, but is not currently standardized or readily available.

When pressure measurements are not consistent with CECS, further diagnostic studies may be necessary to explore the differential diagnosis. Plain radiographs may demonstrate a periosteal reaction in patients with tibial stress fractures or posteromedial tibial periostitis. A bone scan will show increased uptake, and MRI may show edema or a black line, at the site of a stress fracture. Ultrasound may play a role in diagnosis by identifying anterior compartment thickness [46]. Tingling, numbness, or a positive Tinel's sign at a specific location may warrant an electromyogram (EMG) and nerve conduction velocity (NCV) studies to evaluate for peripheral nerve entrapment. Pain and coolness with paradoxical claudication may warrant an angiogram to evaluate for popliteal artery entrapment.

In the setting of trauma, there is no consensus on methods of measuring the various compartments of the foot except one: the deep calcaneal compartment. This compartment has been documented as consistently having higher baseline pressures than the others and must be monitored serially. As many of the anatomical landmarks are obscured with severe swelling in the setting of foot trauma, the calcaneal compartment can be measured by inserting the needle 5 cm distal and

2 cm inferior to the medial malleolus and directing the needle towards the posterior tuberosity of the calcaneus [39]. CECS of the foot is most commonly associated with the medial compartment of the foot and is consistently described in all foot compartment anatomic configurations [21–24]. Mollica et al. demonstrated that post-exercise pressures of the foot could be correlated to the diagnostic criteria described by Pedowitz et al for CECS of the leg. Furthermore, the study showed that resting pressures were not associated with increased likelihood of development of foot CECS [21]. The medial foot compartment can be reliably measured using a Stryker intracompartmental pressure monitor (Kalamazoo, MI) and inserting an 18-gauge needle just inferior to the medial cuneiform [21].

## Differential Diagnosis

- Tibial stress fractures
- Posteromedial tibial periostitis
- Tenosynovitis of posterior tibialis or ankle dorsiflexors
- Peripheral nerve entrapment
- Radiculopathy secondary to lumbar pathology
- Complex regional pain syndrome
- Peripheral vascular disease
- Popliteal artery entrapment syndrome
- Deep venous thrombosis
- Calcaneus Fracture
- Lisfranc Injury
- Metatarsal fractures/dislocations
- Crush injury of the foot
- Peripheral vascular disease
- Neuropraxia

---

## Therapy: Conservative Treatment and Surgery (Approach, Technique, Risks, After Treatment)

### Non-operative Management

In the acute setting, any splints or occlusive dressings should be removed. The extremity should be elevated, albeit not above the level of the patient's heart to both maximize perfusion and minimize swelling. When non-operative treatment measures have failed to ameliorate symptoms and the clinical diagnosis of compartment syndrome remains clear, emergent and complete fasciotomy of all compartments with elevated pressures is necessary to reliably normalize compartment pressures and restore perfusion to the affected tissues.

The non-operative management of CECS usually requires activity limitation or cessation. Adjuncts to activity modifi-

cation include anti-inflammatory medications, stretching and foot orthotics. Symptoms usually return with resumption of prior activity level. Surgery, therefore, is indicated in patients who cannot tolerate activity restriction.

## Surgical Management

Surgical treatment involves fasciotomy of the affected compartments, sometimes with partial fasciectomy.

### Preoperative Planning

It is critical to identify which compartments are affected. All symptomatic compartments should be addressed at the time of surgery. It is common for a failed index procedure to be due to a failure to release all affected compartments. The appropriate approach should be selected based on the compartments that need to be released.

### Positioning

The patient is placed in the supine position for each technique.

### Approach

A single- or dual-incision technique can be used to release the lateral and anterior compartments. The perifibular approach can be used to access all four compartments. A posteromedial approach offers easier access to the superficial and deep posterior compartments. Endoscopically-assisted fasciotomies allow access to the entire length of the compartment, allow visualization of fascial hernias, and may minimize surgical complications such as postsurgical fibrosis and injury to the superficial peroneal nerve. The safety and effectiveness of endoscopically-assisted compartment release have been demonstrated in multiple studies [47]. A technique using balloon dissectors and carbon dioxide insufflation is described in the technique section below [47, 48].

## Techniques

### Single-Incision Lateral Approach for Anterior and Lateral Compartment Fasciotomy

The patient is placed in the supine position on the operating table. A 5-cm vertical incision is made halfway between the fibular shaft and the tibial crest at the mid-portion of the leg. The incision should lie over the anterolateral intermuscular septum. If a focal fascial defect is present, the incision should be adjusted so that the defect can be incorporated. A small transverse incision is made through the fascia, and the septum and superficial peroneal nerve, which lie near the septum in the lateral compartment and exit the fascia near the distal aspect of the incision, are identified. Longitudinal

releases of the anterior and lateral compartments are performed using long Metzenbaum scissors in a proximal and distal direction from the transverse incision in the fascia that crosses over the anterolateral intermuscular septum. A partial fasciectomy may be performed, particularly in cases of recurrence following a prior fasciotomy. Thereafter, the fascia is left open. The subcutaneous tissue is approximated using 2-0 absorbable suture materials. The skin is closed with a running subcuticular 4-0 non-absorbable suture material and Steri-strips.

### Dual-Incision Lateral Approach for Anterior and Lateral Compartment Fasciotomy

The patient is placed in a supine position. The leg is divided into thirds, and two 3-cm incisions are placed at the junction of the thirds over the anterolateral intermuscular septum. The superficial peroneal nerve is identified as it exits the fascia near the distal incision. Fasciotomies of the anterior and lateral compartments are performed on each side of the intermuscular septum. The incisions in the fascia are connected using Metzenbaum scissors to divide the fascia from the proximal incision toward the knee, then from the proximal incision toward the distal incision, and finally from the distal incision toward the ankle. Distally, the fasciotomy should extend to 4–6 cm proximal to the ankle. At the distal aspect of the anterior compartment, the release should be directed toward the midline to minimize risk of injuring cutaneous sensory nerves in the lateral aspect of the compartment. The distal aspect of the lateral compartment fasciotomy should be directed laterally. The subcutaneous tissue is closed with 2-0 absorbable suture materials. The skin is closed with running subcuticular 4-0 sutures and Steri-strips.

### Perifibular Approach for Four-Compartment Fasciotomy

The patient is placed in the supine position. A 10-cm incision is made directly over the mid-portion of the fibula. The skin is retracted anteriorly and the fascia of the anterior and lateral compartments is released longitudinally in a proximal and distal direction. The skin is retracted posteriorly, and the fascia overlying the lateral head of the gastrocnemius is released. The fascia over the superficial posterior compartment is incised for a distance of about 15 cm. Next, the anterior and lateral compartments are retracted anteriorly and the superficial posterior compartment is retracted posteriorly. The soleal bridge must be released from the fibula. The fascia over the flexor hallucis longus is then identified and incised. The gastrocnemius is retracted posteriorly and the flexor hallucis longus laterally to expose the posterior tibial artery, tibial nerve, and peroneal artery overlying the tibialis posterior. The fascia is incised around the tibialis posterior and the interval between the muscle and the origins of the flexor hallucis longus is widened if constrictive. The subcu-



taneous tissue is approximated with 2-0 absorbable sutures, and the skin is closed with running subcuticular non-absorbable 4-0 suture.

### **Posteromedial Incision for Fasciotomy of the Posterior Compartments**

A vertical incision 8–10 cm in length is made over the mid-portion of the leg approximately 1 cm posterior to the posteromedial edge of the tibia. The saphenous vein and nerve are identified in the subcutaneous tissue and retracted anteriorly. The fascia over the superficial posterior compartment is incised for a distance of about 15 cm. To fully access the deep posterior compartment, the origin of the soleus from the proximal tibia and fibula must be detached. The deep fascia can then be sharply divided with Metzenbaum scissors. The fasciotomy should extend distally to 8–10 cm above the ankle. The opening between the origins of the flexor hallucis longus and the tibialis posterior is enlarged if constrictive. The subcutaneous tissue is closed with 2-0 absorbable sutures. The skin is closed with running subcuticular non-absorbable 4-0 suture.

### **Endoscopically-Assisted Compartment Release**

The patient is placed in a supine position. Balloon dissectors can be used to create an optical cavity at the fascial cleft, which is the potential space between the superficial fascia (the deepest layer of the skin and subcutaneous tissue) and the deep fascia (the fascia overlying a muscle compartment). To insert the balloon dissector, a 2-cm transverse incision is made either at the anterolateral aspect of the knee between the fibular head and Gerdy's tubercle or at the posteromedial aspect of the knee at the level of the tibial crest. Dissection is carried down through the subcutaneous fat and superficial fascia until the deep fascia overlying the muscle is visualized. The balloon dissector with a sheath around it is inserted between the superficial and deep fascial layers under direct observation and manual palpation to the level of the ankle. The sheath is removed and the balloon is inflated to create a cavity within the fascial cleft. The balloon is then deflated and removed. The optical cavity is maintained using towel clips. Alternatively, the optical cavity between the superficial and deep fascial layers can be maintained with 15 mmHg of carbon dioxide insufflation to allow adequate visualization of the fascia to be released and to allow adequate space to perform soft tissue dissection with the endoscopic equipment. A one-way cone-shaped cannula is inserted in the skin at the site of balloon insertion.

Next, the fascia overlying the anterior compartment is released with endoscopic scissors down to the level of the ankle under direct vision. The intermuscular septum between the anterior and lateral compartments, as well as the superficial peroneal nerve, can be visualized. If a lateral compartment release is indicated, perform a second fasciotomy

posterior to the intermuscular septum. If posterior compartment releases are indicated, make a 2 cm transverse incision proximally along the medial aspect of the leg just posterior to the edge of the tibia. The balloon dissector and sheath are inserted into the fascial cleft overlying the superficial and deep posterior compartments to the level of the ankle. As described above, the balloon is inflated, deflated, and removed. Towel clips are used to maintain the cavity. The fascia of the deep posterior compartment is released directly off the posterior medial border of the tibia, anterior to the intermuscular septum, from proximal to distal under direct visualization with endoscopic scissors. The fascia of the superficial posterior compartment posterior to the intermuscular septum is released in the same fashion. If necessary, a distal instrument portal with a pneumatic lock can be placed, but the fasciotomies usually are carried out proximal to distal through the initial portal. After the release, the cannula is removed and the cavity is deflated. The wound is closed in a two-layer fashion with 2-0 Vicryl for the deep layer and a running subcuticular stitch for the skin over a medium Hemovac drain.

### **Three Incision Approach of the Foot**

This technique is based on the nine-compartment model of the foot and is best utilized in the setting of trauma/fractures [30]. The medial incision allows release of the medial, superficial and deep central, and lateral compartments. The medial incision landmarks are as follows: incision starts 4 cm anterior to the posterior aspect of the heel and 3 cm superior to the plantar surface and is extended distally approximately 6 cm. The incision is carried down to the fascia of the medial compartment. Elevating the subcutaneous tissues both superiorly and inferiorly then exposes the plantar aponeurosis. A 1 cm longitudinal incision is made in line with the aponeurosis 1 cm from the inferior border allowing for decompression of the medial compartment. The abductor hallucis is lifted superiorly and released from its attachment to the lateral fascia of the medial compartment.

The same 1 cm strip created above is followed laterally for the release of the deeper compartments. A small nick is made in the fascia just superior to the fascial strip. Care must be taken as the lateral plantar nerve and artery lie just deep to this fascia. Extending this incision distally allows for decompression of the calcaneal compartment. To release the superficial deep compartment, a similar nick is made in the fascia inferior to the strip – extending the fascial incision the length of the skin fully decompresses this compartment. At the most proximal portion of the incision, access to the lateral compartment occurs through the origin of the abductor digiti quinti (ADQ). Release of the inferomedial overlying fascia of the ADQ extending laterally allows for completion of the release of the lateral compartment.

The interosseous and adductor compartments are released through dual dorsal incisions. The first incision is made just

medial to the second metatarsal and the other just lateral to the fourth metatarsal. Subcutaneous tissues are undermined medially and laterally to allow for access to each respective interosseous compartment. The thin fascia is incised longitudinally allowing complete release. Spacing these incisions in this manner allows for adequate skin bridge between the two to minimize risk of skin necrosis. All wounds are initially left open wrapped in loose sterile dressings. Dorsal incisions typically require split thickness skin grafts for final coverage 5–7 days after fasciotomy, while the medial incision can be closed in a delayed fashion or primarily [29, 49, 50].

### The Single Incision Medial Approach

Bonutti and Bell describe a single incision extensile approach to release all of the compartments of the foot. In the absence of fracture, a medial incision approach allows for a potentially faster return to normal compartment pressure levels [51]. Selective decompression of specific compartments, specifically the medial compartment in CECS of the foot, is reported in multiple case reports with good result [20–22, 24, 52, 53].

A curvilinear incision is made from the first metatarsophalangeal joint distally through the navicular tuberosity proximally to the calcaneus (~8 cm in length). The deep fascia is incised allowing for complete decompression of the abductor hallucis. The decompression can be extended laterally by incising the medial extension of the plantar fascia and the intermuscular septum laterally; this allows for decompression of the central compartments [24]. Sterile dressings are placed over the incision. After swelling subsides, soft tissues are evaluated, freshened and then re-approximated for primary closure [53].

### Risks

- Superficial peroneal nerve injury – Identify the nerve as it exits the fascia at the junction of the distal and middle thirds of the leg; direct the anterior fasciotomy medially and the lateral fasciotomy posteriorly at the distal extent.
- Saphenous vein and nerve injury – Identify the structures in the subcutaneous tissue at the medial aspect of the leg. Avoid excessive traction on the saphenous nerve, which results in traction paresthesias.
- Incomplete fascial release – Muscle herniates at the bottom of the “V” of the fasciotomy, resulting in pain. Extend lateral and anterior fasciotomies to 4–6 cm above the ankle and posterior fasciotomies to 8–10 cm above the ankle.
- Medial calcaneal branch of the posterior tibial nerve injury- susceptible to injury at the proximal aspect of the medial fasciotomy incision
- Lateral plantar nerve branch injury- the nerve travels just distal to the calcaneus medial tuberosity coursing between

the flexor digitorum brevis and quadratus plantae (the superficial and deep central compartments respectively).

### Post-operative Care

Active range of motion at the ankle and knee should begin immediately. Crutches can be used as needed in the initial postoperative period, but patients are encouraged to bear weight as tolerated and perform light activities. Elevation of the legs while at rest may help to decrease pain and swelling. Full activity usually can be resumed 6–8 weeks after surgery.

### Complications

Recurrence rates of 3–17% have been reported after fasciotomy [54–57]. Recurrence may be due to a number of factors, including inadequate fascial releases, failure to decompress a compartment that was believed to be asymptomatic, nerve compression by an unrecognized fascial hernia, and the development of prolific scar tissue [58]. Other reported complications of fasciotomies with some degree of subcutaneous or blind dissection include arterial injury, hematoma or seroma formation, superficial wound infections, peripheral cutaneous nerve injuries, and deep venous thromboses [36, 47, 54, 59]. The superficial peroneal nerve is particularly vulnerable as it exits the fascia over the lateral aspect of the leg at the junction of the middle and distal thirds.

Reports of surgical sequelae following foot fasciotomies are limited in comparison to fasciotomy of the lower leg. Furthermore, there is even less data in regards to surgical complications associated with chronic exertional compartment syndrome of the foot. Studies associated with FCS state that complications exist, such as decreased range of motion of the foot and toes, pain and numbness associated with surgical scars, and the need for free flaps for soft tissue coverage [60]. Furthermore, known surgical complications such as damage to the medial plantar nerve lying in the calcaneal compartment or superficial compartment exist but have not been quantified [61].

### Rehabilitation and Back-to-Sports

Athletes can participate in sports as pain and disability allow. The following post-operative rehabilitation protocol may be used:

- Range of motion exercises of the knee and ankle in the immediate post-operative period
- Three to five days of protected weight bearing, followed by progression to weight bearing as tolerated

- Physical therapy for strengthening
- Gradual return to jogging between 4 and 6 weeks after surgery
- Full return to sports participation is anticipated at 8 weeks following surgery if one compartment is released. Return to sport is anticipated at approximately 12 weeks if multiple compartments are treated operatively.

A recent study of a military population showed that 78 % of patients remained in the military and 41 % returned to full military duty following elective fasciotomy for CECS [62]. Another study in an active military population reported symptom recurrence in 44.7 % of the patients, and 27.7 % were unable to return to full activity [63]. Surgical complications were documented for 15.7 % of the patients, 5.9 % underwent surgical revision, and 17.3 % were referred for medical discharge because of chronic exertional compartment syndrome [63]. Moreover, a cohort study of 73 athletes with CECS compared outcomes for patients who underwent isolated anterior compartment fasciotomy versus combined anterior and lateral compartment fasciotomies [64]. Sixty-nine percent of patients in the anterior compartment fasciotomy group returned to sports within 3 months, and all patients in this group eventually returned to sport [64]. Conversely, only 33 % of patients in the anterior and lateral compartment fasciotomies group returned to sports within 3 months, and 27 % of patients in this group did not return to sport [64].

In the series of various case reports with chronic exertional compartment syndrome of the foot in athletes, most patients recovering from FCS treated by fasciotomy are able to walk by post-operative day 3 with gradual and full return to activity typically occurs by 8 weeks [24]. Extrapolating data from acute foot compartment syndrome due to trauma would point towards better results with earlier recognition and definitive treatment by fasciotomy. In a prospective study by Han et al., patients followed at a mean of 2 years demonstrated that earlier fasciotomy for definitive treatment leads to better American Orthopaedic Foot and Ankle Society (AOFAS) scores, visual analog scale (VAS) pain scores, and a greater likelihood of being able to wear shoes comfortably [65].

---

## Prevention

The best prevention of sports-related compartment syndromes is the education of athletes on the signs and symptoms of acute and chronic compartments syndromes of the leg and foot.

---

## Evidence

Various techniques of compartment release have reports of success rates ranging from 78 to 100 % [5, 36, 37, 40, 47, 54–57]. These techniques include open fasciotomies, one- or

two-incision minimally invasive subcutaneous fasciotomies, and fasciotomies with partial fasciectomy. Adequate long-term follow-up is lacking in the literature.

Slimmon et al reported on long-term follow-up of patients treated with fasciotomy with partial fasciectomy and noted a good or excellent outcome in 60 % at a mean follow-up of 51 months. Thirteen of 62 had reduced activity levels due to recurrence of symptoms or development of a different lower extremity compartment syndrome [59]. Fasciotomy appears to be less effective in alleviating pain in the deep posterior compartment than in other compartments. Some authors have postulated that failure of the fasciotomy may be due to an incomplete fasciotomy or not identifying and releasing the fascia around the tibialis posterior [27, 55, 56].

The literature on compartment release for CECS of the foot is quite limited. Case reports state good relief of symptoms with fasciotomy [21, 22, 24, 25, 53]. Jowett et al demonstrated 6 of 7 ft had complete resolution of symptoms at approximately 21 months from surgery [25]. Long-term consequences of failure to treat CECS of the foot have not been clearly defined and likely result in persistence of symptoms alone.

---

## Summary

- A predictable history of exertional leg pain followed by gradual dissipation of the pain with rest, especially in a runner, should raise a clinician's suspicion for CECS.
- Intracompartmental pressure testing at rest and after symptomatic, sport-specific exercise is still considered the gold standard for diagnosis.
- For those athletes with persistent symptoms and the desire to continue with their sport, fasciotomy of the documented involved compartments of the lower leg is the intervention of choice.
- Dynamic compartment pressure monitoring with exercise is the gold standard for diagnosis for exertional compartment syndrome of the foot
- Despite limited data, fasciotomy for exertional compartment of the syndromes of the foot demonstrate reliable relief of symptoms.

---

## References

1. French EB, Price WH. Anterior tibial pain. *BMJ*. 1962;2:1290–6.
2. Reneman RS. The anterior and the lateral compartment syndrome of the leg due to intensive use of muscles. *Clin Orthop Rel Res*. 1975;113:69–80.
3. Clanton TO, Solcher BW. Chronic leg pain in the athlete. *Clin Sports Med*. 1994;4:743–59.
4. Qvarfordt P, Christenson JT, Eklof B, et al. Intramuscular pressure, muscle blood flow, and skeletal muscle metabolism in chronic anterior tibial compartment syndrome. *Clin Orthop Relat Res*. 1983;179:284–90.

5. Styf JR, Korner LM. Chronic exertional compartment syndrome of the leg: results of treatment by fasciotomy. *J Bone Joint Surg Am*. 1986;68A:1338-47.
6. Arciero RA, Shishido NS, Parr TJ. Acute anterolateral compartment syndrome secondary to rupture of the peroneus longus muscle. *Am J Sports Med*. 1984;12:366-7.
7. Jarolem KL, Wolinsky PR, Savenor A, et al. Tennis leg leading to acute compartment syndrome. *Orthopedics*. 1994;17:721-3.
8. McHale KM, Prahinski JR. Acute exertional compartment syndrome occurring after performance of the army physical fitness test. *Orthop Rev*. 1994;23:749-53.
9. McKee MD, Jupiter JB. Acute exercise-induced bilateral anterolateral leg compartment syndrome in a healthy young man. *Am J Orthop*. 1995;24:862-4.
10. Nicholson P, Devitt A, Stevens M, et al. Acute exertional peroneal compartmental syndrome following prolonged horse riding. *Injury*. 1998;29:643-4.
11. Johnson J, Becker J. Bilateral acute compartment syndrome in a football player: a case report. *Curr Sports Med Rep*. 2012;6:287-9.
12. Assenmacher JA, Hunter RE. Acute nonexertional lateral compartment syndrome from snowboarding. A case report. *Am J Sports Med*. 2002;5:754-6.
13. Stollsteimer GT, Shelton WR. Acute atraumatic compartment syndrome in an athlete: a case report. *J Athl Train*. 1997;3:248-50.
14. Mavor GE. The anterior tibial syndrome. *J Bone Joint Surg Br*. 1956;38B:513-7.
15. Edmundsson D, Toolanen G. Chronic exertional compartment syndrome in diabetes mellitus. *Diabet Med*. 2011;28:81-5.
16. Hallock GG. An endoscopic technique for decompressive fasciotomy. *Ann Plast Surg*. 1999;43:668-70.
17. Rorabeck CH, Castle GS, Hardie R, et al. Compartment pressure measurements: an experimental investigation using the slit catheter. *J Trauma*. 1981;21:446-9.
18. Kutz JE, Singer R, Lindsay M. Chronic exertional compartment syndrome of the forearm: a case report. *J Hand Surg Am*. 1985;10:302-4.
19. Manoli 2nd A, Weber TG. Fasciotomy of the foot: an anatomical study with special reference to release of the calcaneal compartment. *Foot Ankle*. 1990;10:267-75.
20. Cortina J, Amat C, Selga J, Corona PS. Isolated medial foot compartment syndrome after ankle sprain. *Foot Ankle Surg*. 2014;20:e1-2. doi:10.1016/j.fas.2013.08.006.
21. Mollica MB, Duyshart SC. Analysis of pre- and postexercise compartment pressures in the medial compartment of the foot. *Am J Sports Med*. 2002;30:268-71.
22. Muller GP, Masquelet AC. Chronic compartment syndrome of the foot. A case report]. *Rev Chir Orthop Reparatrice Appar Mot*. 1995;81:549-52.
23. Seiler R, Guziec G. Chronic compartment syndrome of the feet. A case report. *J Am Podiatr Med Assoc*. 1994;84:91-4.
24. Lokiec F, Siev-Ner I, Pritsch M. Chronic compartment syndrome of both feet. *J Bone Joint Surg Br*. 1991;73:178-9.
25. Jowett A, Birks C, Blackney M. Chronic exertional compartment syndrome in the medial compartment of the foot. *Foot Ankle Int*. 2008;29:838-41.
26. Chambers L, Hame SL, Levine B. Acute exertional medial compartment syndrome of the foot after playing basketball. *Skeletal Radiol*. 2011;40:931-5. doi:10.1007/s00256-011-1157-8.
27. Davey JR, Rorabeck CH, Fowler PJ. The tibialis posterior muscle compartment. An unrecognized cause of exertional compartment syndrome. *Am J Sports Med*. 1984;12:391-7.
28. Hislop M, Tierney P, Murray P, et al. Chronic exertional compartment syndrome: the controversial "fifth" compartment of the leg. *Am J Sports Med*. 2003;31:770-6.
29. Myerson MS. Management of compartment syndromes of the foot. *Clin Orthop Relat Res*. 1991;271:239-48.
30. Manoli 2nd A, Fakhouri AJ, Weber TG. Concurrent compartment syndromes of the foot and leg. *Foot Ankle*. 1993;14:339.
31. Ling ZX, Kumar VP. The myofascial compartments of the foot: a cadaver study. *J Bone Joint Surg Br*. 2008;90:1114-8. doi:10.1302/0301-620X.90B8.20836.
32. Myerson M, Manoli A. Compartment syndromes of the foot after calcaneal fractures. *Clin Orthop Relat Res*. 1993;290:142-50.
33. Kalsi R, Dempsey A, Bunney EB. Compartment syndrome of the foot after calcaneal fracture. *J Emerg Med*. 2012;43:e101-6. doi:10.1016/j.jemermed.2009.08.059.
34. Olson SA, Glasgow RR. Acute compartment syndrome in lower extremity musculoskeletal trauma. *J Am Acad Orthop Surg*. 2005;13:436-44.
35. Lundvall J, Mellander S, Westling H, et al. Fluid transfer between blood and tissues during exercise. *Acta Physiol Scand*. 1972;2:258-69.
36. Fronek J, Mubarak SJ, Hargens AR, et al. Management of chronic exertional compartment syndrome of the lower extremity. *Clin Orthop Relat Res*. 1987;220:217-27.
37. Styf JR, Korner LM. Microcapillary infusion technique for measurement of intramuscular pressure during exercise. *Clin Orthop Rel Res*. 1986;207:253-62.
38. Edmundsson D, Toolanen G, Thornell L, et al. Evidence for low muscle capillary supply as a pathogenic factor in chronic compartment syndrome. *Scand J Med Sci Sports*. 2010;6:805-13.
39. Haddad S L, Managing risk: compartment syndromes of the foot. American Academy of Orthopaedics Surgeons Now. 2007. <http://www.aaos.org/news/bulletin/janfeb07/clinical1.asp>.
40. Pedowitz RA, Hargens AR, Mubarak SJ, et al. Modified criteria for the objective diagnosis of chronic compartment syndrome of the leg. *Am J Sports Med*. 1990;18:35-40.
41. Awbrey BJ, Sienkiewicz PS, Mankin HJ. Chronic exercise-induced compartment pressure elevation measured with a miniaturized fluid-pressure monitor. A laboratory and clinical study. *Am J Sports Med*. 1988;16:610-5.
42. Brace RA, Guyton AC, Taylor AE. Reevaluation of the needle method for measuring interstitial fluid pressure. *Am J Physiol*. 1976;229:603-7.
43. McDermott AG, Marble AE, Yabsley RH, Phillips MB. Monitoring dynamic anterior compartment pressures during exercise: a new technique using the STIC catheter. *Am J Sports Med*. 1982;10:83-9.
44. Murabak SJ, Hargens AR, Owen CA, et al. The wick catheter technique for measurement of intramuscular pressure: a new research and clinical tool. *J Bone Joint Surg Am*. 1976;58A:1016-20.
45. Van den Brand JGH, Verleisdonk EJMM, van der Werken C. Near infrared spectroscopy in the diagnosis of chronic exertional compartment syndrome. *Am J Sports Med*. 2004;32:452-6.
46. Rajasekaran S, Beavis C, Aly AR, Leswick D. The utility of ultrasound in detecting anterior compartment thickness changes in chronic exertional compartment syndrome: a pilot study. *Clin J Sports Med*. 2013;4:305-11.
47. Wittstein J, Moorman CT, Levin LS. Endoscopic compartment release for chronic exertional compartment syndrome. *Am J Sports Med*. 2010;8:1661-6.
48. Zobrist R, Aponte R, Levin LS. Endoscopic access to the extremities: the principle of fascial clefts. *J Orthop Trauma*. 2002;16:264-71.
49. Fulkerson E, Razi A, Tejwani N. Review: acute compartment syndrome of the foot. *Foot Ankle Int*. 2003;24:180-7.
50. Fakhouri AJ, Manoli 2nd A. Acute foot compartment syndromes. *J Orthop Trauma*. 1992;6:223-8.
51. Myerson MS. Experimental decompression of the fascial compartments of the foot--the basis for fasciotomy in acute compartment syndromes. *Foot Ankle*. 1988;8:308-14.
52. Izadi FE, Richie Jr DH. Exertional compartment syndrome of the medial foot compartment--diagnosis and treatment: a case report. *J Am Podiatr Med Assoc*. 2014;4:417-21. doi:10.7547/0003-0538-104.4.417.

53. Miozzari HH, Gerard R, Stern R, Toman J, Assal M. Acute, exertional medial compartment syndrome of the foot in a high-level athlete: a case report. *Am J Sports Med.* 2008;36:983–6.
54. Detmer DE, Sharpe K, Sufit RL, et al. Chronic compartment syndrome: diagnosis, management, and outcomes. *Am J Sports Med.* 1985;13:162–70.
55. Rorabeck CH, Bourne RB, Fowler PJ. The surgical treatment of exertional compartment syndrome in athletes. *J Bone Joint Surg Am.* 1983;65A:1245–51.
56. Rorabeck CH, Fowler PJ, Nott L. The results of fasciotomy in the management of chronic exertional compartment syndrome. *Am J Sports Med.* 1988;16:224–7.
57. Schepesis AA, Martini D, Corbett M. Surgical management of exertional compartment syndrome of the lower leg: long term follow up. *Am J Sports Med.* 1993;21:811–7.
58. Schepesis AA, Fitzgerald M, Nicoletta R. Revision surgery for exertional compartment syndrome of the lower leg. *Am J Sports Med.* 2005;33:1040–7.
59. Slimmon D, Bennell K, Bruncker P, et al. Long-term outcome of fasciotomy with partial fasciectomy for chronic exertional compartment syndrome of the lower leg. *Am J Sports Med.* 2002;30:581–8.
60. Frink M, Hildebrand F, Krettek C, Brand J, Hankemeier S. Compartment syndrome of the lower leg and foot. *Clin Orthop Relat Res.* 2010;468:940–50. doi:10.1007/s11999-009-0891-x.
61. Ojike NI, Roberts CS, Giannoudis PV. Foot compartment syndrome: a systematic review of the literature. *Acta Orthop Belg.* 2009;75:573–80.
62. McCallum JR, Cook JB, Hines AC, Shaha JS, Jex JW, Orchowski JR. Return to duty after elective fasciotomy for chronic exertional compartment syndrome. *Foot Ankle Int.* 2014;9:871–5.
63. Waterman B, Laughlin M, Kilcoyne K, Cameron K, Owens B. Surgical treatment of chronic exertional compartment syndrome of the leg: Failure rates and postoperative disability in an active patient population. *J Bone Joint Surg Am.* 2013;95:592–6.
64. Packer JD, Day MS, Nguyen JT, Hobart SJ, Hannafin JA, Metzl JD. Functional outcomes and patient satisfaction after fasciotomy for chronic exertional compartment syndrome. *Am J Sports Med.* 2013;2:430–6.
65. Han F, Daruwalla ZJ, Shen L, Kumar VP. A prospective study of surgical outcomes and quality of life in severe foot trauma and associated compartment syndrome after fasciotomy. *J Foot Ankle Surg.* 2014;14:S1067–2516. doi:10.1053/j.jfas.2014.09.015.

Sampat S. Dumbre Patil

**Abstract**

Common overuse injuries in athletes are -shin splints, stress fractures, compartment syndrome, nerve entrapment, etc. The term 'shin splints' is used broadly to describe many conditions causing exercise induced pain in sporting personnel. True shin splints is also labeled as Medial Tibial Stress Syndrome (MTSS), describing a syndrome in which leg pain and discomfort in distal posteromedial aspect of leg is caused by repetitive activity and it excludes causes of pain due to stress fractures or due to ischemia.

Inadequate warm-up, sudden increase in training mileage and hyperpronation of foot are some of the predisposing factors. Diagnosis is mainly clinical and is supported by investigations like MRI and Bone scan. Conservative treatment in the form of Rest, Physiotherapy and Orthotics is usually successful and these measures are also helpful in Prevention of MTSS.

**Keywords**

Shin splints • Medial Tibial Stress Syndrome • Chronic overuse injuries leg

**Introduction**

Chronic overuse injuries may prove distressing to the elite athletes as well as weekend warriors. Tibial stress fracture, chronic compartment syndrome and medial tibial stress syndrome (MTSS) are few common causes of exercise induced leg pain. MTSS is the commonest of all and is referred to as true shin splints.

The American Medical association in 1966 has defined shin splints syndrome as 'pain and discomfort in the leg from repetitive activity on hard surfaces or due to forcible, excessive use of foot flexors.' The diagnosis excludes stress fractures or ischemic disorders and is limited only to the musculotendinous inflammations [1].

It has been described as "Spike Soreness" in runners in 1913 [2]. It is a very common injury experienced by runners

and military personnel accounting for 13.2–17.3% [3, 4] of all running injuries and up to 22% of injuries seen in aerobic dancers [5]. Players participating in tennis, volley-ball, basketball and long jumping may also suffer from MTSS.

**Etiology and Pathomechanism**

The exact etiology of shin splints syndrome is not known. The causes are broadly divided in acute and chronic conditions. The acute conditions include bone stress reaction, periostitis, fibrositis, bone strains, tenosynovitis, tendonitis of tibialis anterior, tibialis posterior, soleus and flexor hallucis longus muscles. Detmer [6] proposed periostalgia as the likely cause of shin splints whereas Johnell et al. [7] studied 37 limbs of MTSS and found osseous metabolic changes and no inflammatory reaction in the biopsies. He proposed bone stress reaction as the underlying cause of MTSS which is also supported by recent studies. The chronic conditions include periosteal reaction, traction periostalgia, chronic tendonitis, fatigue tear of bridging collagen fibers between

S.S.D. Patil, MBBS, D, DNB, MNAMS  
Consultant Orthopedic Surgeon, Noble Hospital, 153, Magarpatta  
City Road, Pune, Maharashtra 411013, India  
e-mail: [sampatdumbre@gmail.com](mailto:sampatdumbre@gmail.com)

muscle and bone and chronic compartment syndrome [6, 8–10].

Certain metabolic changes are observed in the tibia when a person begins exercise. These are in the form of initial bone porosity due to increased osteoclastic activity on the compressed concave posteromedial border. This is followed by increased osteoblastic activity and deposition of new bone. Tibia becomes stronger on the posteromedial border than the pre exercise state. However, in long standing MTSS, tibia becomes 15% more porous than in control group and 23% less than athletic control group [11].

Various studies have been published to highlight the underlying risk factors for shin splints. Some of the proposed intrinsic risk factors are lack of running experience, inadequate warm-up, incomplete stretching, increased running frequency, excessive weekly running distances, sudden increase in training mileage and lack of flexibility training. Other intrinsic factors which may contribute are navicular drop, hyperpronation of foot, increased femoral neck anteversion, greater plantarflexion ROM, increased hip external rotation ROM and imbalance between quadriceps and hamstring muscles.

Few extrinsic or environmental risk factors for shin splints are type of sport, time of day, always running on the same side of the road, hard running surface, uneven terrain, shoes, in-shoe orthoses, climate, weather conditions etc.

---

## Symptoms

There is recurrent dull ache to intense pain associated with repetitive strenuous activity lasting for several hours or days. The pain is usually on the middle one third or lower one third of the tibial crest. Pain increases on running and decreases with rest. Discomfort may persist during normal activities of daily living particularly in severe cases.

---

## Diagnostics, Classification

Diagnosis of shin tibia is particularly based on thorough clinical history and physical examination. On clinical examination, usually pain increases on passive dorsiflexion of foot with great toe hyperextension and also on extreme passive plantar flexion. Range of motion of the ankle may be decreased, limited to 20° dorsiflexion and 40–50° plantar flexion [12]. There are no sensory, motor or muscular abnormalities.

Diffuse area of tenderness can be elicited on palpation of the posteromedial border of the tibia particularly along the middle or distal third of its length. The bone surface may feel uneven.

Investigations like Magnetic Resonance Imaging (MRI), bone scintigraphy (bone scan) or plain radiographs may be useful in nonspecific cases as well as to rule out other forms of exercise induced leg pain. If one is sure of the diagnosis on clinical examination, these investigations are not required [13].

Plain radiographs are usually normal initially but may show localized cortical thickening and scalloping of the posteromedial surface of the tibia if the symptoms persist for more than 3–4 weeks [9].

Bone scintigraphy demonstrates a linear vertical uptake involving more than one third of the posterior tibial cortex in the third phase [14] (Fig. 19.1a, b). Angiograms and blood pool images are normal in shin splints, differentiating it from the stress fracture [15]. Magnetic Resonance Imaging (MRI) demonstrates periosteal or marrow edema and helps in diagnosing stress injury before the development of a stress fracture [16–18] (Fig. 19.2a–c). MRI is recommended over bone scan as it is more accurate in correlating the degree of bone involvement with clinical symptoms, lack of exposure to ionizing radiation and significantly less imaging time compared to three phase bone scintigraphy [19]. MRI is also useful in monitoring the follow up and allows more accurate recommendation for rehabilitation [19] (Fig. 19.3a, b).

Ultrasonography and Computed Tomography (CT scan) are of limited value in diagnosing shin splints but help to rule out stress fracture and more research has to be done on their sensitivity and specificity.

---

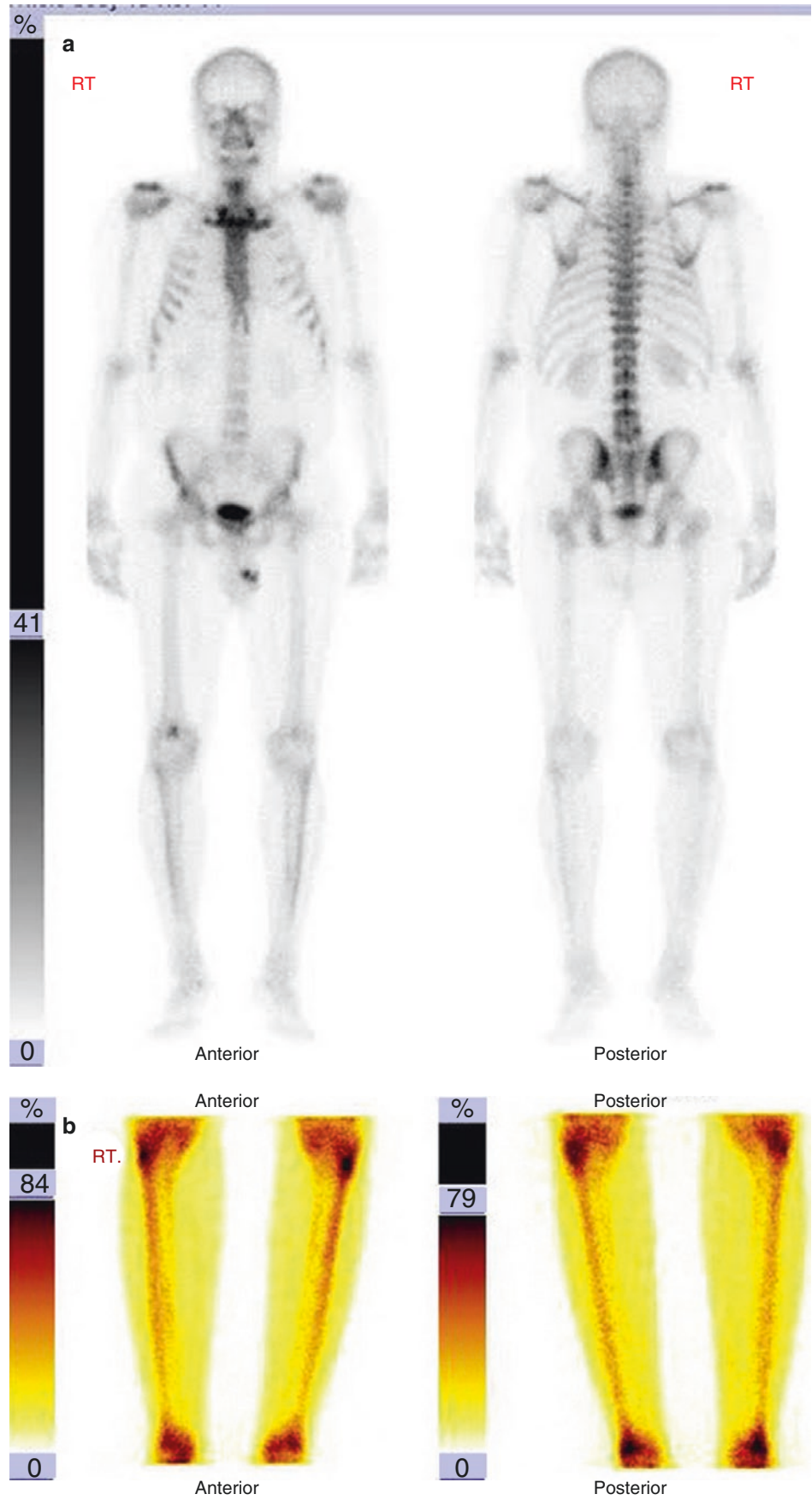
## Classification

Tibial stress injuries are graded on bone scan and MRI. Zwaset et al. [20] proposed four stage grading system based on bone scan findings as-

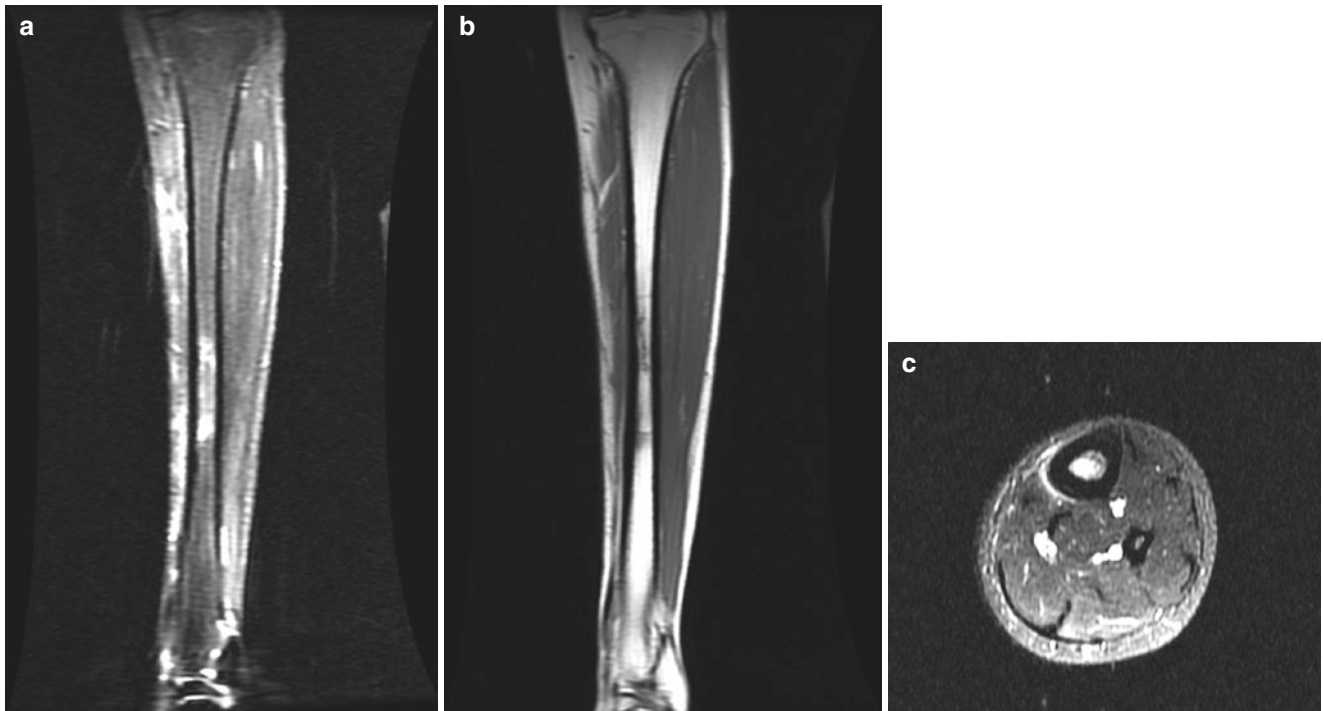
- Grade 1 – Small, ill-defined cortical area of mildly increased activity
- Grade 2 – Better defined cortical area of moderately increased activity
- Grade 3 – Wide to fusiform, cortical-medullary area of highly increased activity
- Grade 4 – Transcortical area of intensely increased activity

Michael et al. [19] proposed a grading system based on MRI findings and compared it with the bone scan grading scale by Zwas et al. Their grading system showed that periostitis or shin splints correspond to Grade 1 stress injury. Grade 2 and 3 injuries were more severe injuries manifested by bone marrow edema. Fracture line is clearly visible in grade 4 tibial stress injuries. In all the grades from 1 to 4, they constantly observed periosteal edema at the attachments of tibialis posterior, flexor digitorum longus and soleus muscles [19].

**Fig. 19.1** (a, b) Intravenous injection of  $^{99m}\text{Tc}$ -MDP and static imaging 3 h later reveals, linear increased tracer uptake along the tibial shins (without any focal or fusiform uptake) which is characteristic of “Shin Splints” (a, b courtesy of Dr. Sameer Sonar, Fellow, Harvard Medical School, USA)

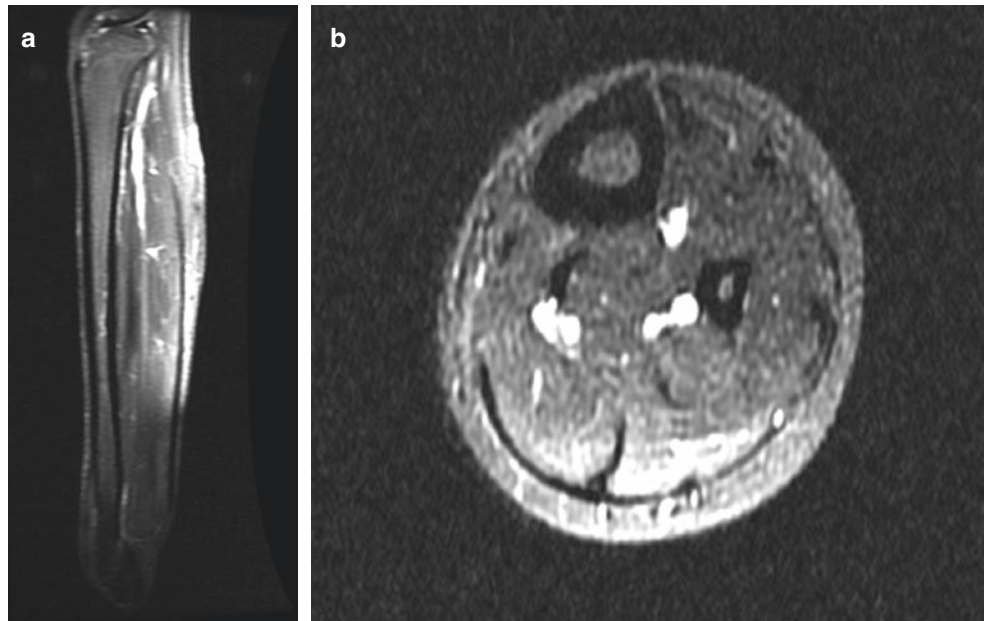






**Fig. 19.2** (a–c) MRI Scan of a young female athlete showing bone marrow edema appreciated as hypointense signal on T1 weighted and hyperintense signal on T2 weighted images. Note the periosteal edema on STIR axial images

**Fig. 19.3** (a, b) MRI scan of the same patient at 3 months follow up demonstrating resolution of previously seen marrow edema on STIR images



## Therapy

Various treatment modalities have been recommended by different authors. Most of the patients are relieved with the conservative mode of treatment and surgical intervention is rarely needed [13, 21–25].

## Conservative Treatment

### Rest

Rest is essential in the acute phase followed by gradual return to the sports activity. MRI grading system may be referred to decide the duration of rest which is generally 2–3

weeks in Grade 1, 4–6 weeks in grade 2 and 6–9 weeks in grade 3 tibial stress injuries. Individuals with Grade 4 injuries are treated in a cast [19].

When the acute phase subsides, training program is modified by reducing the weekly running distance, intensity and frequency by half [26–29]. It is advised to avoid running on hills or uneven surfaces [30].

### Ice Fomentation

Application of ice directly on the affected part immediately after exercise for 15–20 min provides immediate local anesthesia and reduces the spasticity of muscle tissue [12, 31].

### Physiotherapy

#### Exercises

The principles of exercise program are to stretch the plantar flexors and strengthen the dorsiflexors of ankle [12]. Strengthening core hip muscles is also encouraged to develop core stability and running mechanics [31].

Cross training like running in the pool or use of floating device play an important role in maintaining the aerobic conditioning. These activities can be supplemented using stationary bicycle, cross-country skiing machine, stair climber etc. [19].

Extracorporeal shock wave therapy (ESWT), local ultrasound has been used as supportive conservative measures but their efficacy is not proved due to limited research.

#### Orthotics

Placement of orthotic support inside the shoes have been recommended by Jukka et al. [32]. This method limits the excessive movement of the subtalar joint and it has been successfully tried in many athletes in outpatient sports clinic of Turku.

#### Fascial Distortion

Christoph et al. has described treatment of MTSS according to the Fascial Distortion Model [33]. In this method, he uses strong local pressure by fingertips on painful areas to reduce pain.

#### Others

Some of the conservative modalities like taping of longitudinal arch of the foot, cast immobilization in severe cases, acupuncture [34], use of nonsteroidal anti-inflammatory medication or steroid injections are used in practice but with limited evidence. Newer methods such as dry-needling, autologous blood injection, platelet-rich plasma, and prolotherapy [31] have been described in literature.

## Surgical Treatment

Ben et al. performed surgical intervention in 78 patients of which 46 patients kept the follow up [35]. The surgery was performed only after conservative treatment had failed for a minimum of 12 months. On mean postoperative follow up of 30 months, he observed significant reduction in pain associated with MTSS. According to the technique described [35], a longitudinal linear incision is made along the middle and distal thirds of the inner tibial border. A deep posterior compartment fasciotomy is performed by dividing the fascia at the fascia-bone interface. The most important step is the release of the soleus bridge which is the part of distal fascia proximal to flexor retinaculum. A two cm wide strip of periosteum is then removed from along the inner tibial border. Patient is advised to walk with crutches for 2 weeks after which normal walking is allowed but no sports activity for 6 weeks [35]. The study concluded that surgery significantly reduced pain levels ( $p < 0.001$ ) by an average of 72% as indicated on the visual analog pain scale [35].

## Rehabilitation and Back-to-Sports

Specific rehabilitation program should be planned for individual sports person, taking into consideration various factors like grade of injury, its duration, kind of sports activity etc. Proprioceptive exercises are vital and strengthening exercises are started according to the pain tolerance.

Proper training plan is implemented which slowly increases in duration, frequency and intensity. Sports specific activities, impact activities like jumping, hill running are started in graded manner [26].

## Prevention

Many measures have been recommended by various authors to prevent occurrence of shin splints. However, limited study has been performed to prove effectiveness of any of these measures. A systematic review of four randomized controlled trials has been published by Stephen et al. [36]. Based on this review, he recommends use of shock absorbent orthoses inserts in young male athletes as a preventive measure. The orthosis stabilizes the subtalar joint and decreases pronation.

Other preventive measures include warm-up exercises, good running techniques, minimization of running on hills and hard surfaces, the time of exercise and the use of running shoes.

A good exercise program is suggested to strengthen and stretch the appropriate muscle group. This includes Achilles

tendon stretching, dorsiflexor muscle strengthening with weights on toes and isometric exercises for developing arch strength [12].

## Evidence

The highest level of evidence supporting the explanation in this article is Level-1, i.e. Systematic review of Level-1 randomized controlled trials.

## Summary

- True Shin Splints (Medial Tibial Stress Syndrome) is a symptom complex characterized by pain and discomfort in the distal posteromedial aspect of leg after repetitive overuse in walking and running.
- Diagnosis of shin splints is particularly based on thorough clinical history and physical examination.
- Investigations like plain radiograph, bone scintigraphy and MRI are recommended in nonspecific cases and to rule out other forms of exercise induced leg pain.
- Conservative treatment is effective method of management while rarely surgical intervention is needed.
- Many preventive modalities have been suggested by various authors but limited study has been performed to prove their effectiveness.

**Acknowledgements** I want to acknowledge contribution of my wife, Dr. Mrs Vaishali Sampat Dumbre Patil, who is a consultant radiologist, Pune, India, for her assistance in radiological investigations in shin splints.

## References

1. American Medical Association, Committee on the medical aspects of sports, subcommittee on classification of sports injuries. Standard nomenclature of athletic injuries. Chicago: AMA; 1966. p. 126.
2. Hutchins CP. Explanation of spike soreness in runners. *Am Phys Ed Rev.* 1913;18:31–5.
3. Clement D, Taunton J, Smart G. A survey of overuse running injuries. *Phys Sports Med.* 1981;9:47–58.
4. Epperly T, Fields K. Epidemiology of running injuries. In: O'Connor F, Wilder R, editors. *Textbook of running medicine.* New York: McGraw-Hill; 2001. p. 1–11.
5. Taunton JE, McKenzie DC, Clement DB. The role of biomechanics in the epidemiology of injuries. *Sports Med.* 1988;6:107–120.
6. Detmer D. Chronic shin splints: classification and management of medial tibial stress syndrome. *Sports Med.* 1986;3:436–46.
7. Johnell O, Wendeberg M, Westlin N. Morphological bone changes in shin splints. *Clin Orthop.* 1982;167:180–4.
8. Beck BR, Osternig LR. Medial tibial stress syndrome. *J Bone Joint Surg.* 1994;76:1057–61.
9. Mubarak SJ, Gould RN, Lee YF, Schmidt DA, Hargens AR. The medial tibial stress syndrome: a cause of shin splints. *Am J Sports Med.* 1982;10:201–5.
10. Paul WD, Soderberg GL. The shin splint confusion. In: *Proceedings of the 8th National Conference on Aspects of Sports,* Chicago, Am Med Assoc. 1967;19–24.
11. Magnusson H, Westlin N, Nyqvist F, et al. Abnormally decreased regional bone density in athletes with medial tibial stress syndrome. *Am J Sports Med.* 2001;29:712–5.
12. Wayne Rasmussen, Rpt. Shin Splints: definition and treatment. *J Sports Med.* 1974;2(2):111–7.
13. Ugalde V, Batt M, Chir MBB. Shin splints: current theories and treatment. *Crit Rev Phys Rehab Med.* 2001;13:217–53.
14. Holder LE, Michael RH. The specific scintigraphic pattern of “shin splints in the lower leg”: concise communication. *J Nucl Med.* 1984;25:865–9.
15. Love C, Din AS, Maria B, Tomas MB, et al. Radionuclide bone imaging: an illustrative review. *Radiographics.* 2003;23(2):341–58.
16. Lee JK, Yao L. Stress fractures MR imaging. *Radiology.* 1988;169:217–20.
17. Martin SD, Healey JH, Horowitz S. Stress fracture MRI. *Orthopedics.* 1993;16:75–8.
18. Stafford SA, Rosenthal DI, Gebhardt MC, et al. MRI in stress fracture. *AJR.* 1986;147:553–6.
19. Michael Fredericson A, Bergman AG, Hoffman KL, et al. Tibial stress reaction in runners correlation of clinical symptoms and scintigraphy with a new magnetic resonance imaging grading system. *Am J Sports Med.* 1995;23(4):472–81.
20. Zwas ST, Elkanovitch R, Frank G. Interpretation and classification of bone scintigraphic findings in stress fractures. *J Nucl Med.* 1987;28:452–7.
21. Abramowitz AJ, Schepsis A, McArthur C. The medial tibial syndrome. The role of surgery. *Orthop Rev.* 1994;23:875–81.
22. Clanton TO, Solcher BW. Chronic leg pain in the athlete. *Clin Sports Med.* 1994;13:743–59.
23. Moore MP. Shin splints. Diagnosis, management, prevention. *Postgrad Med.* 1988;83:199–210.
24. Clement DB. Tibial stress syndrome in athletes. *J Sports Med.* 1974;2:81–5.
25. Rzonca EC, Baylis WJ. Common sports injuries to the foot and leg. *Clin Podiatr Med Surg.* 1988;5:591–612.
26. Beck B. Tibial stress injuries: an aetiological review for the purposes of guiding management. *Sports Med.* 1998;26(4):265–79.
27. Kortebein P, Kaufman K, Basford J, Stuart M. Medial tibial stress syndrome. *Med Sci Sports Exerc.* 2000;32(3 suppl):S27–33.
28. Couture C, Karlson K. Tibial stress injuries: decisive diagnosis and treatment of ‘shin splints’. *Phys Sportsmed.* 2002;30(6):29–36.
29. DeLee J, Drez D, Miller M. DeLee and Drez’s orthopaedic sports medicine principles and practice. Philadelphia: Saunders; 2003. p. 2155–9.
30. Wilder R, Seth S. Overuse injuries: tendinopathies, stress fractures, compartment syndrome, and shin splints. *Clin Sports Med.* 2004;23:55–81.
31. Michael Galbraith R, Lavalley ME. Medial tibial stress syndrome: conservative treatment options. *Curr Rev Musculoskelet Med.* 2009;2:127–33.
32. Jukka T, Viitasalo JT, Kvist M. Some biomechanical aspects of the foot and ankle in athletes with and without shin splints. *Am J Sports Med.* 1983;11(3):125–30.
33. Schulze C, Finze S, Bader R, et al. Treatment of medial tibial stress syndrome according to the fascial distortion model: a prospective case control study. *Sci World J.* 2014; Article ID 790626:1–6.
34. Callison M. Acupuncture and tibial stress syndrome (shin splints). *J Chinese Med.* 2002;70:24–7.
35. Yates B, Allen MJ, Barnes MR. Outcome of surgical treatment of medial tibial stress syndrome. *J Bone Joint Surg.* 2003;85-A(10):1974–80.
36. Thacker SB, Gilchrist J, Stroup DF, et al. The prevention of shin splints in sports: a systematic review of literature. *Med Sci Sports Exer.* 2001;32–41.

Matthijs Jacxsens, Lukas Weisskopf, Victor Valderrabano,  
and Claudio Rosso

#### Abstract

Achilles tendon disorders are most common in individuals who participate in endurance sports that involve repetitive loading of the foot. The increasing incidence is correlated with an expanding participation of the population in recreational and competitive sports and is therefore one of the more common disorders seen in sports medicine. In the following chapter both acute Achilles tendon disorders such as the partial and full Achilles tendon ruptures as well as more chronic disorders such as Achilles tendinopathy is thoroughly discussed with special attention for elite sportsmen.

#### Keywords

Achilles tendon • Achilles tendinopathy • Achilles tendinitis • Mid-portion tendinopathy • Achilles insertional tendinopathy • Paratenonitis • Achilles tendon rupture • Achilles tendon tear • Achilles tendon repair

Achilles tendon injuries are among the more common disorders seen in sports medicine. As a result of an expanding participation of the population in recreational and competitive sports, an increasing incidence is noticed during the last decades [1]. Achilles disorders are most common in individuals who participate in endurance sports that involve repetitive, eccentric impact loading and jumping. Therefore, the highest prevalence of Achilles tendon injuries is seen in

track and field athletes [2–6]. Other athletes that suffer of this pathology mostly participate in racquet sports, dancing or ball games such as football, soccer, volleyball, basketball, and rugby [7–14].

The Achilles tendon is the largest and strongest tendon of the body and is formed by the confluence of fibers derived of the gastrocnemius and soleus muscles [15, 16]. In 93 % of the population, these muscles are accompanied medially by the plantaris tendon [17]. Towards its insertion on the posterior surface of the calcaneus distal to the posterior-superior calcaneal tuberosity, the fibers twist 90° to help tendon elongation, elastic recoil and release of stored energy during movement so the tendon can act as a spring [18]. The retrocalcaneal bursa is located deep and proximal to this insertion [16]. The Achilles tendon is surrounded by a paratenon. This richly vascularized tissue is responsible for the main blood supply to the tendon [19]. The sensory innervations of the tendon is derived from the attaching muscles and cutaneous nerves, particularly the sural nerve [20]. The musculotendinous unit (MTU) of the gastrocnemius and soleus (the conjunction of tendon, their aponeurosis and muscles) function as the main plantar flexor of the ankle joint. It provides the primary propulsive force for movement during walking, running

---

M. Jacxsens, MD (✉)  
Department of Orthopaedic Surgery, University Hospital Basel,  
Spitalstrasse 21, Basel CH-4031, Switzerland  
e-mail: [matthijs.jacxsens@usb.ch](mailto:matthijs.jacxsens@usb.ch)

L. Weisskopf, MD  
Altius Swiss Sportmed Center, Habich Dietschy-Strasse 5A,  
Rheinfelden 4310, Switzerland  
e-mail: [lukas.weisskopf@altius.ag](mailto:lukas.weisskopf@altius.ag)

V. Valderrabano, MD, PhD  
Orthopaedic Department, SWISS ORTHO CENTER,  
Schmerzklinik Basel, Swiss Medical Network, Basel, Switzerland  
e-mail: [vvalderrabano@gsmn.ch](mailto:vvalderrabano@gsmn.ch)

C. Rosso, MD, MSc, PD Dr. med.  
Shoulder and Elbow Center, Arthro Medics, Basel, Switzerland  
e-mail: [c.rosso@arthro.ch](mailto:c.rosso@arthro.ch)

and jumping. Furthermore, the Achilles tendon provides a supination of the subtalar joint as the Achilles tendon inserts medially of the axis of the hindfoot [14]. Despite the frequency of Achilles tendon disorders, there is still discussion about the terminology. The terminology applied in this chapter is based on anatomic location, symptoms, clinical findings and histopathology as described by van Dijk et al. [21].

## Etiology and Pathomechanism

### Achilles Tendinopathy

Achilles tendinopathy is a typical chronic overuse tendon disease as a result of repeated loading on the tendon. This overuse disorder is most commonly seen in elite endurance runners, with a 1-year prevalence of 11% and a lifetime cumulative incidence of up to 50% [5, 6, 22]. However, 30% of the sedentary population suffers of the same disorder. This condition has been correlated with seronegative arthropathies [23] and is most likely to occur in middle-aged people (30–55 years old). Achilles tendinopathy can be categorized according to the anatomical location as a mid-portion (non-insertional) and an insertional tendinopathy.

The mid-portion tendinopathy is characterized by a combination of pain and swelling located 2–6 cm proximal to the tendon insertion, correlating to the histological pattern of “tendinosis”, which indicates an inadequate healing process with a disorganized collagen structure and possible degenerative non-inflammatory signs. Occasionally, signs of necrosis or calcification can be found within the tendon. Three stages are proposed to classify the pathology: initially a reactive tendinopathy can be seen, followed by a tendon disrepair, finally a degenerative tendon is described [24]. However, it should be mentioned that up to 34% of asymptomatic tendons show the same histopathological changes [25, 26]. These morphological changes lead to stiffness of the tendon, predisposing the elderly population secondary to overuse microtraumata.

The etiology of the disease is unclear but is seen as multifactorial. Several theories have been proposed, such as the neurogenic, angiogenic, impingement and “iceberg” hypotheses [27–31]. The latter describes the hypothesis that the disease consists out of different thresholds with the symptomatic patient as the end stage. The cause of pain has been correlated with neovascularization and nerve ingrowth, which could trigger neurogenic-mediated inflammation pain [26]. Besides these local tissue changes, peripheral and central nociceptive mechanisms might be involved in the pain mechanism [32]. The role of inflammation in tendinopathy in general has been controversial, recently infiltration of inflammatory cells has been found in the early phase of tendinopathy. However, no histological evidence of inflamma-

tion has been found in the advanced phase of the disease where the patient is symptomatic [31, 33]. Individual predisposing risk factors, both intrinsic as well as extrinsic, may influence the onset of the pathology. The main intrinsic predisposing risk factors are genetic predisposition, age, gender, endocrine/metabolic diseases, lower extremity malalignment with hyperpronation of the foot as the most important contributing one, leg length discrepancy, chronic ankle instability, and muscular dysbalance and insufficiency. The main extrinsic factors contributing to Achilles tendon tendinopathy are medication (steroids, fluoroquinolone antibiotics), environmental factors such as hard training surface and most of all excessive loading because of intensive training, rapid progressive training, improper footwear, fatigue and inadequate warm-up or stretching [34–37].

Enthesiopathies such as the Achilles insertional tendinopathy (AIT) take place on the junction between the tendon and the bone is located at the insertion of the tendon onto the calcaneus. The pathomechanism is similarly as seen in jumper’s knee, tennis or golfer’s elbow [38]. Because of the typical twist of 90° at the insertion of the Achilles tendon, the junction is exposed to shear strains, as the tendon fibers do not enter the bone perpendicular [39]. The described factors leading to AIT are muscle fatigue by altering muscle-tendon stiffness [40], training on hard surfaces so that shock waves are absorbed by the junction [41], contracture, dysbalance and/or insufficiency of the gastrocnemius, soleus and tibialis anterior muscles and stress-shielding, where the posterior portion is exposed to significantly more strain as the anterior portion which corresponds with the pathological area in AIT [42].

AIT is highly associated with retrocalcaneal bursitis. Up to 60% of patients also have Haglund’s deformity, which leads to mechanical bony impingement causing an exacerbating of the pathology.

Bone spurs and calcifications in the tendon at the insertion site can be found often. Histopathologically, there is ossification of enthesial fibrocartilage, sometimes accompanied with small tendon tears occurring at the tendon-bone junction.

Paratendinitis/Paratenonitis is a more acute tendon disorder occurring in most of all long-distance runners and is defined by an inflammation of the paratenon around the Achilles tendon. This inflammatory condition of the paratenon can be associated with rheumatoid arthritis, seronegative arthropathies, infection, and most of all with overuse injuries of the Achilles tendon [43]. In acute paratenonitis, edema and hyperaemia of the paratenon are typically seen. Infiltration of inflammatory cells is described and may possibly lead to the production of a fibrinous exudate located between the tendon sheath and the tendon itself, causing palpable crepitations on physical examination. In chronic Achilles paratenonitis, the paratenon becomes more thickened as a result of persistent fibrinous

exudate. The proliferation of (myo-) fibroblasts leads to the formation of new connective tissue and adhesions between tendon, paratenon and the crural fascia. Therefore, the crepitations and swelling are less pronounced but pain at the mid-portion of the tendon induced by physical activity is more prominent [44].

## Achilles Tendon Rupture

Although the Achilles tendon is the strongest tendon in the human body, it is also the most frequently ruptured one with increasing incidence [23, 45–47]. In contrast to other tendon ruptures, the Achilles tendon rupture is highly correlated with physical activity, with more than 75% of the Achilles tendon ruptures occurring during sports activity. In the past decades, an increasing total number of Achilles tendon ruptures has been noticed. This increase has been attributed to an increase in sports participation of the middle aged sedentary population, mostly in the third or fourth decade of life [14, 36, 48]. However, elite athletes have an elevated risk with rupture occurring earlier in life because of the high physical demands put on the Achilles tendon [6].

Although the pathomechanism of Achilles tendon ruptures is not clear, a consensus about a multifactorial process exists. A combination of degenerative changes and mechanical stress are described as causal processes. The most common site for acute tears is the area about 2–6 cm proximal to the insertion, correlating with a hypovascularized mucoid degenerative zone of the tendon. Failure of tendon repair because of repetitive loading and disuse could lead to microtrauma of the tendon, even in healthy tendons without signs of degeneration [49]. An ultimate stress then leads to failure, resulting in a partial or full-thickness tear. An acute tear is mostly related to a sudden forced plantar flexion during weight bearing with a fully extended knee. Therefore, athletes who participate in sports requiring explosive acceleration, sudden changes in direction, jumping and sprinting are at higher risk. Also a violent dorsiflexion in a plantar flexed foot which occurs when falling has been described as a mechanism of injury [6, 50].

The same triggering intrinsic and extrinsic risk factors as those for mid-portion tendinopathy, are described, with overtraining, hyperpronation, gastrocnemius/soleus contracture/insufficiency and excessive eccentric loading as main attributing factors [51]. However, only 5% of the patients with an Achilles tendon rupture had a diagnosis of Achilles tendinopathy before the tendon failure. It seems that most of the ruptures occurs in subjects who were asymptomatic before the tear, although in most Achilles tendon rupture signs of degeneration and tendinopathy can be found [52].

Several drugs such as anabolic steroids and fluoroquinolones are related to spontaneous ruptures as they can cause

dysplasia of collagen fibrils and increase the risk of tendon rupture [53, 54].

## Symptoms

### Achilles Tendinopathy and Paratenonitis

Mid-portion Achilles tendinopathy is a clinical syndrome characterized by pain and a diffuse or localized swelling of the tendon located at 2–6 cm from the insertion onto the calcaneus leading to an impaired performance [21]. Mid-portion Achilles tendinopathy can be initially asymptomatic with a typically gradual progress of the symptoms.

In AIT, the patient will describe a combination of pain, morning ankle stiffness and sometimes swelling that worsens with activity, localized at the insertion of the tendon. An athlete may describe these symptoms getting worse after exercise but becoming eventually constant. The symptoms can be aggravated by uphill running and interval training.

The most important symptoms in paratendinitis/paratenonitis are exercise-induced pain and edema of the tendon leading to a swelling. However, athletes will often complain of stiffness and pain at the beginning of their sports activity. This discomfort then may resolve after a period of running.

### Partial and Complete Achilles Tendon Rupture

Patients with an Achilles tendon rupture usually describe a history of acute intense, mostly sports-related or pain in the mid-portion of the Achilles tendon comparable to the feeling of being shot or kicked above the heel. A popping sound is often described by the athlete. Also physical dysfunction, weakness or stiffness of the affected ankle is commonly described. Patients with a partial tear do not often complain of severe pain. Above all, an increased swelling or dysfunction of the ankle during weight bearing may be presented.

## Diagnostics

First, a thorough history and physical examination is mandatory. Further evaluation can be obtained by imaging. Ultrasonography (US) and Magnetic Resonance Imaging (MRI) are used to confirm a clinical suspicion of an Achilles tendon disorder. US is more cost-effective and has the advantage of being a dynamic examination, however it is reader-dependent so that an experienced user is favorable [55]. On the other hand, MRI leads to identification of the various stages of the pathology and can identify concomitant pathology. MRI can be preferred for preoperative planning in chronic tendon disorders as it allows for multiplanar imaging

[56]. Radiographs can be useful for demonstrating additional pathology and malalignment of the foot and ankle.

### Achilles Tendinopathy and Paratenonitis

In mid-portion Achilles tendinopathy, the pain may be exacerbated by loaded dorsiflexion of the foot. In the clinical examination, the dorsiflexion of the ankle joint is often impaired and the intratendinous swelling moves typically with the Achilles tendon. In some cases, ossifications can be palpated in the tendon. The imaging findings correlate with the different stages of the pathology. Degeneration with a correlating tendon thickening can be visualized by both MRI as well as US. In a degenerated tendon, US shows a hypoechoic tendon. Progression of the pathology with collagen fiber separation and disruption can be visualized as intrasubstance tears or clefts [43]. An additional Doppler scanning can determine the neovascularisation of the tendon. Calcifications and bony spurs can hinder US evaluation. Plain radiographs are then indicated for further assessment of the tendon. On MRI, the following findings are associated with the disease: tendon thickening, edema of Kager's fat pad, peritendinous edema and tendon disruption. During progression of the disease, an increasing high signal is seen in the T2 weighted images [57]. All these features are found in asymptomatic and symptomatic patients, so that both clinical presentations and imaging findings have to be seen as a whole picture. It should be noticed that an abnormal tendon signal without tendon thickening should be analyzed with care as the magic angle phenomenon can lead to false-positive high signals of the tendon [58].

In case of paratenonitis (also called paratendinitis), a diffuse swelling can be palpated in combination with typical crepitations along the tendon. Also tender nodules can be noticed in some cases. These findings are less pronounced in case of chronic paratenonitis because new connective tissue and adhesions are formed between tendon, paratenon, and crural fascia. In the acute phase, US may reveal fluid between the tendon and the paratenon most likely at the posterior side of the tendon, leading to a hypoechoic signal combined with a thickened paratenon. This fluid leads to a hyperintense signal on T2 weighted MR images [57]. Doppler imaging is helpful in order to detect increased vascularity [43].

In AIT, examination reveals tenderness at the insertion of the tendon, with swelling and possible calcifications or a bony spur within the tendon. The pain can be worse by passive dorsiflexion and a limited dorsiflexion can be found secondary to this pain. All these findings may be combined with the palpation of a prominence of the posterosuperior lateral aspect of the calcaneus, also known as a Haglund's deformity. Other possible diagnosis of poste-



**Fig. 20.1** The morphology of posterior aspect of the calcaneus can be visualized by use of plain radiographs. Also the morphology of the Achilles tendon insertion can be determined, a bony spur can be possibly visualized in case of AIT

rior heel pain, such as os trigonum or posterior impingement should be ruled out. Imaging is not necessary for the diagnosis but plain radiographs can be helpful to determine the morphology of the posterior aspect of the calcaneus and the Achilles tendon insertion as the previously described deformities can be visualized (Fig. 20.1). US and/or MRI may be used to determine the degree of degenerative changes of the tendon at its insertion and to visualize concomitant pathologies such as retrocalcaneal bursitis or posterior impingement.

### Achilles Tendon Rupture

The diagnosis of an acute Achilles tendon rupture is first of all based on clinical findings [56]. Nevertheless, the diagnosis may be overlooked at the first presentation at the physician in up to 23 % of the cases [59]. In most of the patients, ankle dorsiflexion is impaired, although some patients are still able to plantarflex the ankle by use of the remaining intact tendons of the foot.

Inspection and palpation may show edema, bruising and a gap along the course of the tendon. Ankle plantarflexion is typically decreased so that a heel rise becomes impossible. To confirm the diagnosis, many physical examination maneuvers are described. The most sensitive and specific test is the (Simmonds-) Thompson test, hereby the patient is lying in the supine position and the knee is in 90° of flexion. Squeezing the calf leads to plantar flexion of the foot if the Achilles tendon is intact. Diminished or absent plantar flexion is suggestive for a rupture [60, 61]. Other helpful tests are the Matles (Fig. 20.2), Copeland and O'Brien tests [62–64].



**Fig. 20.2** During the Matles test, the patient lies in a prone position, active or passively flexing the knee to 90° with both feet and ankles in a neutral position. When an absence of plantar flexion is observed, the test proves positive. The rupture will tend the foot more into dorsal flexion as seen on the left foot of this patient



**Fig. 20.3** T2 weighted MRI of an acute rupture showing MRI tendon discontinuity with possible degenerative changes of the underlying tendon

Alterations in the clinical examination findings can be found because of an intact part of the tendon in partial tears or interposed scar tissue in chronic tears. Imaging evaluation is not necessary to make a diagnosis but is helpful in therapeutic decision making. US is useful in the acute setting as it can

asses a dynamic evaluation of the tear. A hematoma in the interposing gap can be well visualized but may lead to a difficult visualization of the tendon ends [65]. A disadvantage of US is that the differentiation between partial tears and degeneration can be difficult, so that in this case further assessment by use of a MRI is indicated [66, 67]. MRI of an acute rupture demonstrates tendon discontinuity with possible degenerative changes of the underlying tendon and edema or hematoma between the retracted tendon parts (Fig. 20.3). In chronic full-thickness tears, the tendon gap may consist out of scar tissue. Partial tears can be visualized as incomplete disruption of tendon fibers and often extend to the tendon surface [43].

## Therapy

### Mid-portion Achilles Tendinopathy

Mid-portion Achilles tendinopathy is characterized by disrepair of the tendon leading to degenerative changes, including disorganized collagen fibers and neovascularity. Treatment therefore should focus on reversing the disrepair of the tendon.

The mid-portion Achilles tendinopathy usually responds well to non-operative treatment measures with a success rate of up to 75 % [68]. In athletes, an initial activity modification is generally accepted as primary treatment measure. The role of non-steroidal anti-inflammatory drugs (NSAID) is controversial as inflammation is only seen in the initial phase of the disease, moreover this inflammation could be necessary for tendon healing. Therefore, NSAID could have a negative effect on tendon healing so that the role of these drugs seems to be obsolete [69, 70]. Analgesia by use of paracetamol and ice application should be encouraged. Besides treating the intrinsic and extrinsic causing factors by modifying or correcting malalignments, muscular dysbalance, footwear, inadequate stretching and training errors, eccentric exercises are the gold standard therapy. The most used training protocol is based on an eccentric heel drop on the affected leg, the non-affected leg is then used to return to the start position. For a period of 12 weeks, three sets of 15 repetitions should be done twice daily (3 × 15 with the knee in extension and 3 × 15 with the knee in flexion) [68]. These exercises have shown to be superior to a wait-and-see treatment or concentric exercise [71]. Only low-energy shock wave therapy (SWT) can be a potential alternative for such an exercise program. Nevertheless, the combination of both eccentric exercises and low-energy SWT seems to be the most effective [71–76]. Additional therapy measures such as night splints, low-level laser therapy and injection of low-dose heparin, glycosaminoglycans, aprotinin, topical glyceryl trinitrate and sclerosing agents are described and may induce pain relief, however



there is no definitive evidence whether these may lead to tendon healing [77–81]. Also corticosteroid injections have lack of clinical evidence and have the risk to induce Achilles tendon ruptures, so that the use of corticosteroid injection are generally not recommended in the treatment of mid-portion tendinopathy [82].

Platelet-rich plasma (PRP)-infiltrations are gaining popularity and mild to moderate clinic benefits are described. However, similar outcomes were noted in placebo and control groups, so there is no clear evidence that PRP-infiltrations have superior clinical outcomes [83, 84].

Failure of conservative treatment is correlated with the degree of tendon degeneration, patient age, and duration of symptoms. Generally, after a period of 3–6 months of unsuccessful conservative management, an operative treatment is indicated [68].

Operative management consists out of a longitudinal incision of the tendon in order to debride the diseased portion along with adjacent involved fibrous tissue and bony spurs. The longitudinal repair is performed by use of tubularization. In severe stages of degeneration or irreparable tendons, augmentation repair with the plantaris longus tendon or a tendon transfer can be a solution [1].

### Insertional Achilles Tendinopathy

Similarly as the other overuse Achilles tendon disorders, AIT should be initially treated by a non-operative treatment, although the success rate is less than in mid-portion tendinopathy. The treatment consists of the same measures as previously described for mid-portion tendinopathy. A relative rest, ice applications, stretching and modifications of training errors, activity and footwear, including heel lifts or open-back shoes to avoid pressure on the heel, are part of the treatment [85]. Eccentric training of the tendon is favored and forms the basis of the treatment, however patient satisfaction is worse than in mid-portion Achilles tendinopathy [86–88]. It has been thought that full range of motion exercises lead to mechanical impingement of the tendon, with persisting pathology and lower patient satisfaction as a consequence. Therefore, floor level exercises are described with promising results [89]. Concomitant low-energy SWT can optimize the clinical outcomes [88]. Also in the treatment of AIT, injection of several substances such as hyperosmolar dextrose or polidocanol may lead to pain relief, however, there is a clear lack of level I studies to confirm the clinical evidence [89].

Non-operative measures should be exhausted before heading to surgery. The surgical goals consist of removal of degenerative portions of the tendon and associated calcifications or bony spurs. Secondly, the retrocalcaneal bursa and a prominent posterior calcaneal prominence can be resected.



**Fig. 20.4** Intraoperative fluoroscopy of a reattached Achilles tendon by use of two anchors after Haglund's deformity (pump bump) resection and intratendinous debridement with partial detachment of the Achilles tendon

Several approaches are described: a medial incision, lateral incision, a combination of both and a posterior longitudinal midline incision [90–94]. A medial incision with a longitudinal midline tendon incision is the preferred approach by the authors. After skin incision and a thorough inspection, the tendon is tenotomised longitudinally so that scar mucoid tissue and bony parts can be excised. If necessary, the tendon is partially or fully detached in order to resect a prominent posterior prominence. Tendon reattachment is done by use of bone anchors (Fig. 20.4). If a large portion of the tendon is resected, an augmentation or tendon transfer can be done.

Surgical treatment has shown to have relative good outcomes with up to 80% patient satisfaction. A complication rate of 23% with 3% major complications after operative treatment is reported [89].

### Paratenonitis

Most of the patients can be treated successfully with conservative measures. These include relative rest and immobilization by means of a sports activity limitation and a night splint. As infiltration of inflammatory cells is described in this Achilles tendon disorder, non-steroidal anti-inflammatory medications probably will not only lead to pain relief but may be influence the disease to a healing process. Also ice applications can lead to more pain relief in the acute phase [44].

If these conservative measures don't help, a technique called brisement can be performed. Local anesthetics is hereby infiltrated in the space between the paratenon and the Achilles tendon to release the scar tissue. These infiltrations probably has to be repeated for two or three times and can be beneficial in earlier stages of the problem up to 50 percent of the time. We don't recommend to add cortisone as it is associated with an increased risk of Achilles tendon rupture. Physiotherapy combining stretching with strengthening exercises, is usually beneficial.

Surgical therapy consists of debridement of the surrounding thickened and scarred sheath. The tendon itself is also explored and any associated disorder is treated. To prevent any recurrent scarring of the tendon to the paratenon, mobilization is started almost immediately postoperatively. Full weight-bearing is allowed as soon as pain and swelling is reduced, usually within the first 2 weeks after surgery [14].

## Achilles Tendon Rupture

The treatment of choice, conservative versus operative, in acute Achilles tendon ruptures is controversial and the subject of many debates. The treatment should not only restore tendon length and tension but also strength of the MTU resulting in good clinical function [95, 96]. In competitive athletes, younger patients as well as active elderly, a consensus exists for the operative because of a lower described rerupture and tendon lengthening risk in comparison to a non-operative treatment. On the other hand, surgery is associated with an increased risk of perioperative complication [97, 98]. Because of these perioperative complications, conservative treatment is preferred in patients with multiple comorbidities [99, 100].

### Surgical Treatment

In the classic open approach, a medial incision is preferred to reduce the risk of sural nerve lesions. A meticulous prepara-

tion with respect for the surrounding soft-tissue, in particularly the preservation of the paratenon, is necessary to prevent healing complications. After a longitudinal incision of the paratenon, the Achilles tendon can be exposed. After gentle debridement of the tendon ends, the Achilles tendon is repaired by open end-to-end non-absorbable sutures (Fig. 20.5). Multiple suture techniques can be used such as the Bunnell, Kessler, Krackow, giftbox and triple bundle technique, in order to reduce the tendon gap [95]. These may vary from 2-, 4-, or 6-strand repairs. One of the great advantages of the classic open approach is the re-attachment of the Soleus tendon, which is very important for plantar flexion force and is often missed in percutaneous surgical techniques. The contralateral ankle can be used to compare and ensure proper length and tension. Additional augmentation with the plantaris tendon or a gastrocnemius turndown flap can be performed and are helpful in complex or chronic ruptures [101–103].

The percutaneous repair introduced by Ma and Griffith, forms an alternative for the open repair technique [104]. By use of small stab incisions under local anesthesia infiltration at the medial and lateral side of the tendon, sutures are passed through the tendon first proximally of the rupture, afterwards the same is repeated distally. Several sutures are passed through the tendon, next the proximal and distal sutures are tied with the ankle in maximal plantar flexion. Several variations on this principle using different kind of devices are described going from a technique with fewer incisions to a mini open technique [105, 106].

The role of PRP application combined with surgical repair is controversial, however, recently a meta-analysis provides evidence for platelet concentrates in the treatment of Achilles tendon ruptures in vivo, so that PRP may be a useful additional therapy for the management of Achilles tendon ruptures [84].

Postoperative treatments vary from complete immobilization with casting to immediate weight-bearing. It has been shown and recently confirmed that functional rehabilitation



**Fig. 20.5** (a) The Achilles tendon rupture is exposed after a medial incision. Proximally and distally of the rupture, degenerative tissue can be visualized and is gently debrided. (b) The Achilles tendon is repaired

by multiple open end-to-end non-absorbable sutures. (c) In order to restore tendon length and tension, Achilles tendon repair is done in plantarflexion

is advantageous as compared to immobilization [100]. In athletes, we prefer a walker or shoe and partial weight bearing because early mobilization and functional bracing has shown to have better patient satisfaction, earlier return to work and pre-injury activities [107]. In the first 2 weeks, partial weight-bearing with a walker in 30° of plantar flexion is allowed. In weeks 3 and 4, patients are allowed to progress to full weight-bearing with 20° of plantar flexion. In the following 2 weeks, the heel pad is reduced to 10° of plantar flexion. After that, plantigrade weightbearing in a walker is allowed for another 2 weeks, resulting in functional bracing of 8 weeks in total. During these 8 weeks, the patient is asked to wear a night splint in 30° of plantar flexion, as lengthening of the tendon has to be prevented.

The most common complications of surgical repair include infection and wound healing problems. Other described complications are rerupture, lengthening of the Achilles tendon, sural nerve lesions, deep venous thrombosis (DVT), pulmonary embolus and scar formation [51]. The outcomes of both surgical repair techniques have shown to have similar rerupture rates [108, 109]. The short and midterm results of recent clinical reports could show a beneficial trend toward faster return to work and better torque per muscle volume in case of an open surgery technique [96, 110]. However, open repair is correlated with higher deep-infection rates and wound healing problems [100, 108]. On the other hand, percutaneous repair is correlated with a possible iatrogenic sural nerve lesions leading to anesthesia of the lateral aspect of the foot or a painful neuroma [111].

Despite the higher risk of infections and wound healing problems, the authors prefer an open technique using a small incision to reconstruct the whole Achilles tendon (Gastrocnemii and Soleus). This technique has multiple advantages such as the possibility to correct asymmetrical ruptures, correct alignment of the torn tendon, low rerupture rates, superior strength and early active mobilization.

With any of the above-described approaches, the principles of stable fixation, proper tendon length, rigorous soft tissue handling, and protection of nervous structures must be kept in mind.

### Conservative Treatment

Conservative treatment is a viable alternative for surgical treatment in the elderly with multiple comorbidities. In the active person, surgical repair is preferred because of the early active mobilization and less rerupture rates. However, clinical results and muscle atrophy are comparable to surgical treatment [112]. Conservative treatment classically consists of cast immobilization in equines position for 4 weeks, followed by a more neutral position for the following 4 week

with typically 6 weeks of non-weight-bearing [113, 114]. More recent protocols include functional bracing, early weight-bearing and mobilization. These protocols allow partial or full weight-bearing in a functional brace or stability boot, initially in an equines position, followed by a gradually decreasing plantar flexion to neutral position over 6–12 weeks. Often active plantarflexion is allowed with restricted dorsiflexion [115, 116]. Patients are then progressed to more aggressive strengthening protocols.

---

## Rehabilitation and Back-to-Sports

### Achilles Tendonitopathy

Return to full sports in athletes dealing with Achilles tendonopathy is variable and can take as long as 6–12 months. Initially, the athlete can maintain his fitness by pain-free, non weight-bearing activities and may benefit from aquatics-based exercises, swimming or cycling. Once the athlete is pain-free, cross-training and exercises with a gradual increase in weight-bearing can be started. Once the patient can handle these basic exercises pain-free, a graded return to running can be initiated. Athletes should take rest days on a regular basis in order to give the tendon time to adapt to the increased stress. Furthermore, an athlete should listen to his body and take symptoms seriously to prevent a recurrence of the overuse tendon injury. The results of conservative rehabilitation are good with up to 84 % of the patients returning to the normal level of physical activity.

In case of a surgical tendon debridement, early weight-bearing in a cam boot and ankle motion exercises are initiated after the postoperative swelling has diminished. In the 3th week postoperative, if the wound is healed, the athletes can start with gentle isometric and concentric exercises. Also low resistance exercises on a stationary bike and aquatics-based exercises are allowed. A progress to eccentric calf muscle can be started at 5 weeks postoperative. At 7 weeks postoperative, the cam boot can be changed for normal footwear. The stability boot is worn for a total of 12 weeks. Continue calf strengthening exercises are performed in order to progress to gentle running exercises at 12 weeks postoperative. At 3 months postoperative, unrestricted sports activities are allowed if the patient is pain-free.

In case of tendon reconstruction by use of anchors, the postoperative rehabilitation program as described for Achilles tendon ruptures is initiated. The return to sport at the same level has been described to be 75 % in long-distance runners with an average period of recovery in performance to 9.3 months [117].

## Achilles Tendon Ruptures

Physiotherapy plays an important role in recovery of patients after Achilles tendon surgery. Early weight-bearing and early ankle motion exercises lead to better and more rapid functional recovery compared to immobilization after Achilles tendon repair. Moreover, this rehabilitation regimen leads to less postoperative complications [100, 118, 119]. Athletes and ambitious patients are encouraged to mobilize the operated ankle early on a regular basis. After the postoperative swelling has diminished, athletes are allowed to train on a stationary bicycle, only by exerting hind-foot pressure to move the pedals with a low resistance. Also proprioception exercises in the boot are allowed. At 4 weeks postoperative, athletes are encouraged to perform non weight-bearing active dorsiflexion exercises with a flexed knee in order to stimulate collagen healing [120]. Active plantarflexion is not allowed yet. Recoil plantarflexion brings the foot back in a neutral position. After 8 weeks postoperative, the cam boot is changed for a normal shoe and the patient can convert to full weight-bearing. Barefoot proprioception, coordination, stretching and isometric exercises are allowed. Strengthening exercises by means of double heel rises can be started. Further exercises with a cross trainer, aquatics and a stationary bike with forefoot pedaling are introduced in the rehabilitation regime. Jogging and sport specific basic exercises are allowed at 3 months postoperative. At 6 months postoperative, the athlete can pick up his normal sports activities. Regaining normal maximal performance capacities can take up to 1 year. A return to sports rate between 67% and 96% has been described [107, 109]. However, in athletes doing sports with a high physical demand of the Achilles tendon, a tendon rupture can be a career ending injury [8, 13].

Also in non-surgical therapy, early functional rehabilitation have shown to be superior than cast immobilization or non-weight-bearing protocols [121]. We prefer an initial partial weight-bearing for 2 weeks in a stability boot in 30° of plantarflexion. After 2 weeks, patients are progressed to full weight-bearing. Every second week the heel lift is reduced with 10°, leading to plantigrad weight-bearing in a stability boot after 6 weeks postoperative for a total of 12 weeks. The first 4 weeks range of motion is limited and only plantarflexion, eversion and inversion of the ankle joint is allowed. After 2 weeks, exercises on a stationary bike with boot at low resistance are allowed. After 6 weeks, stretching exercises, isometric strengthening exercises and proprioceptive training in the boot can be initiated. After 12 weeks, the stability boot treatment is completed, during this time passive dorsiflexion is not allowed as it can lead to Achilles tendon lengthening. At this point, concentric strengthening of the Achilles tendon is started. Next, double heel raises will be recommended

with finally single heel raises. A crosstrainer or stationary bike provides additional exercise at home. Low intensity jogging is allowed after 3 months. Return to pre-injury sports activities may be possible after 6 months in ambitious patients with a high motivation.

---

## Prevention

Prevention measures should focus on the intrinsic and extrinsic contributing risk factors attributing to Achilles tendon injuries. When doing sports, a gradual warming-up followed by stretching of the Achilles tendon and hamstring is recommended. Limited passive dorsiflexion of the ankle has been correlated with Achilles tendon disorders so that stretching may be more important as some people think as it restores tendon length and assists in the restoration of biomechanical properties of the tendon. Stretching is therefore recommended as a preventive measure [122]. In a biomechanical point of view, malalignments, especially hyperpronation, and a leg length discrepancy of 5 mm is correlated with Achilles tendon disorders and should therefore be correct with orthotic insoles [35, 37]. The most logical prevention measure in an overuse injury of the Achilles tendon is addressing and correcting training errors: excessive long distance runs, frequent high intensive runs, running at increasing intensity or distance too early, frequent uphill or downhill running or training on uneven or hard surfaces. In track and field athletes, only a change in direction of running on the track can already initiate symptom relief as it leads to a less monotonous, asymmetric loading of the calf muscles and preventing muscle dysbalance [36]. Medication such as corticosteroids and fluoroquinolones should be taken with care and only if the medical indication is clear and evidence based.

---

## Evidence

### Level I

de Jonge, S., et al., *One-year follow-up of a randomised controlled trial on added splinting to eccentric exercises in chronic midportion Achilles tendinopathy*. Br J Sports Med, 2010. **44**(9): p. 673–7.

de Vos, R.J., et al., *The additional value of a night splint to eccentric exercises in chronic midportion Achilles tendinopathy: a randomised controlled trial*. Br J Sports Med, 2007. **41**(7): p. e5

Stergioulas, A., et al., *Effects of low-level laser therapy and eccentric exercises in the treatment of recreational athletes with chronic achilles tendinopathy*. Am J Sports Med, 2008. **36**(5): p. 881–7.

- de Vos, R.J., et al., *Platelet-rich plasma injection for chronic Achilles tendinopathy: a randomized controlled trial*. JAMA, 2010. **303**(2): p. 144–9.
- Willberg, L., et al., *Sclerosing injections to treat midportion Achilles tendinosis: a randomised controlled study evaluating two different concentrations of Polidocanol*. Knee Surg Sports Traumatol Arthrosc, 2008. **16**(9): p. 859–64.
- Sadoghi, P., et al., The role of platelets in the treatment of Achilles tendon injuries. J Orthop Res, 2013. **31**(1): p. 111–8.
- Rompe, J.D., J. Furia, and N. Maffulli, Eccentric loading compared with shock wave treatment for chronic insertional achilles tendinopathy. A randomized, controlled trial. J Bone Joint Surg Am, 2008. **90**(1): p. 52–61.
- Khan, R.J., et al., Treatment of acute achilles tendon ruptures. A meta-analysis of randomized, controlled trials. J Bone Joint Surg Am, 2005. **87**(10): p. 2202–10.
- Lim, J., R. Dalal, and M. Waseem, Percutaneous vs. open repair of the ruptured Achilles tendon--a prospective randomized controlled study. Foot Ankle Int, 2001. **22**(7): p. 559–68.
- Olsson, N., et al., Stable surgical repair with accelerated rehabilitation versus nonsurgical treatment for acute Achilles tendon ruptures: a randomized controlled study. Am J Sports Med, 2013. **41**(12): p. 2867–76.
- Huang, J., et al., Rehabilitation Regimen After Surgical Treatment of Acute Achilles Tendon Ruptures: A Systematic Review With Meta-analysis. Am J Sports Med, 2014.
- Young, S.W., et al., Weight-Bearing in the Nonoperative Treatment of Acute Achilles Tendon Ruptures: A Randomized Controlled Trial. J Bone Joint Surg Am, 2014. **96**(13): p. 1073–1079.

## Summary

- Achilles tendon disorders are among the most common injuries in athletes with an increasing incidence. These disorders are typically overuse injuries and can be categorized in acute ruptures or more chronic tendinopathies.
- These disorders are clinical diagnoses based on a thorough history and physical examination. Further evaluation can be obtained by imaging and may be helpful in determining additional pathologies or in preoperative planning.
- In Achilles tendinopathy, the corner stone of the treatment is based on conservative measures such as eccentric exercises, low-energy shock wave therapy and pain relieving

measures with additional corrections of the predisposing extrinsic and intrinsic risk factors.

- The best treatment for Achilles tendon ruptures remains controversial. A consensus exists in athletes and active patients toward surgical treatment. Non-operative, open repair and percutaneous repair techniques have good clinical results, with each type of treatment correlating with specific complication risks. The clinical evidence for early weight-bearing in both non-surgical as well as surgical treatment is high and should be implemented in the rehabilitation program.
- Prevention measures should focus on the intrinsic and extrinsic contributing risk factors attributing to Achilles tendon injuries, with correcting training errors and hyperpronation of the foot as the most important factors.

## References

1. Krahe MA, Berlet GC. Achilles tendon ruptures, re rupture with revision surgery, tendinosis, and insertional disease. Foot Ankle Clin. 2009;**14**(2):247–75.
2. Longo UG, Ronga M, Maffulli N. Acute ruptures of the achilles tendon. Sports Med Arthrosc. 2009;**17**(2):127–38.
3. Lysholm J, Wiklander J. Injuries in runners. Am J Sports Med. 1987;**15**(2):168–71.
4. Ganse B, et al. Impact of age, performance and athletic event on injury rates in master athletics - first results from an ongoing prospective study. J Musculoskelet Neuronal Interact. 2014;**14**(2):148–54.
5. Jacobsson J, et al. Prevalence of musculoskeletal injuries in Swedish elite track and field athletes. Am J Sports Med. 2012;**40**(1):163–9.
6. Kujala UM, Sarna S, Kaprio J. Cumulative incidence of achilles tendon rupture and tendinopathy in male former elite athletes. Clin J Sport Med. 2005;**15**(3):133–5.
7. Maffulli N, Sharma P, Luscombe KL. Achilles tendinopathy: aetiology and management. J R Soc Med. 2004;**97**(10):472–6.
8. Amin NH, et al. Performance outcomes after repair of complete achilles tendon ruptures in national basketball association players. Am J Sports Med. 2013;**41**(8):1864–8.
9. Parekh SG, et al. Epidemiology and outcomes of Achilles tendon ruptures in the National Football League. Foot Ankle Spec. 2009;**2**(6):283–6.
10. Fahlstrom M, Lorentzon R, Alfredson H. Painful conditions in the Achilles tendon region in elite badminton players. Am J Sports Med. 2002;**30**(1):51–4.
11. Fahlstrom M, Lorentzon R, Alfredson H. Painful conditions in the Achilles tendon region: a common problem in middle-aged competitive badminton players. Knee Surg Sports Traumatol Arthrosc. 2002;**10**(1):57–60.
12. Fernandez-Palazzi F, Rivas S, Mujica P. Achilles tendinitis in ballet dancers. Clin Orthop Relat Res. 1990;**257**:257–61.
13. Sankey RA, et al. The epidemiology of ankle injuries in professional rugby union players. Am J Sports Med. 2008;**36**(12):2415–24.
14. Schepsis AA, Jones H, Haas AL. Achilles tendon disorders in athletes. Am J Sports Med. 2002;**30**(2):287–305.
15. Maffulli N, et al. Chronic rupture of tendo Achillis. Foot Ankle Clin. 2007;**12**(4):583–96, vi.

16. O'Brien M. The anatomy of the Achilles tendon. *Foot Ankle Clin.* 2005;10(2):225–38.
17. Cummins EJ, Anson BJ, et al. The structure of the calcaneal tendon (of Achilles) in relation to orthopedic surgery, with additional observations on the plantaris muscle. *Surg Gynecol Obstet.* 1946;83:107–16.
18. Arndt A, et al. Asymmetrical loading of the human triceps surae: I. Mediolateral force differences in the Achilles tendon. *Foot Ankle Int.* 1999;20(7):444–9.
19. Carr AJ, Norris SH. The blood supply of the calcaneal tendon. *J Bone Joint Surg Br.* 1989;71(1):100–1.
20. Stilwell Jr DL. The innervation of tendons and aponeuroses. *Am J Anat.* 1957;100(3):289–317.
21. van Dijk CN, et al. Terminology for Achilles tendon related disorders. *Knee Surg Sports Traumatol Arthrosc.* 2011;19(5):835–41.
22. Longo UG, et al. No influence of age, gender, weight, height, and impact profile in achilles tendinopathy in masters track and field athletes. *Am J Sports Med.* 2009;37(7):1400–5.
23. Ames PR, et al. Achilles tendon problems: not just an orthopaedic issue. *Disabil Rehabil.* 2008;30(20–22):1646–50.
24. Cook JL, Purdam CR. Is tendon pathology a continuum? A pathology model to explain the clinical presentation of load-induced tendinopathy. *Br J Sports Med.* 2009;43(6):409–16.
25. Maffulli N, Khan KM, Puddu G. Overuse tendon conditions: time to change a confusing terminology. *Arthroscopy.* 1998;14(8):840–3.
26. van Sterkenburg MN, van Dijk CN. Mid-portion Achilles tendinopathy: why painful? An evidence-based philosophy. *Knee Surg Sports Traumatol Arthrosc.* 2011;19(8):1367–75.
27. Riley G. The pathogenesis of tendinopathy. A molecular perspective. *Rheumatology (Oxford).* 2004;43(2):131–42.
28. Andersson G, et al. Presence of substance P and the neurokinin-1 receptor in tenocytes of the human Achilles tendon. *Regul Pept.* 2008;150(1–3):81–7.
29. Andersson G, et al. Tenocyte hypercellularity and vascular proliferation in a rabbit model of tendinopathy: contralateral effects suggest the involvement of central neuronal mechanisms. *Br J Sports Med.* 2011;45(5):399–406.
30. Pufe T, et al. The role of vasculature and angiogenesis for the pathogenesis of degenerative tendons disease. *Scand J Med Sci Sports.* 2005;15(4):211–22.
31. Abate M, et al. Pathogenesis of tendinopathies: inflammation or degeneration? *Arthritis Res Ther.* 2009;11(3):235.
32. Rio E, et al. The pain of tendinopathy: physiological or pathophysiological? *Sports Med.* 2014;44(1):9–23.
33. Millar NL, et al. Inflammation is present in early human tendinopathy. *Am J Sports Med.* 2010;38(10):2085–91.
34. Mahieu NN, et al. Intrinsic risk factors for the development of achilles tendon overuse injury: a prospective study. *Am J Sports Med.* 2006;34(2):226–35.
35. Kannus P. Etiology and pathophysiology of chronic tendon disorders in sports. *Scand J Med Sci Sports.* 1997;7(2):78–85.
36. Jarvinen TA, et al. Achilles tendon disorders: etiology and epidemiology. *Foot Ankle Clin.* 2005;10(2):255–66.
37. Kvist M. Achilles tendon injuries in athletes. *Sports Med.* 1994;18(3):173–201.
38. Rufai A, Ralphs JR, Benjamin M. Structure and histopathology of the insertional region of the human Achilles tendon. *J Orthop Res.* 1995;13(4):585–93.
39. Maganaris CN, et al. Biomechanics and pathophysiology of overuse tendon injuries: ideas on insertional tendinopathy. *Sports Med.* 2004;34(14):1005–17.
40. Horita T, et al. Exhausting stretch-shortening cycle (SSC) exercise causes greater impairment in SSC performance than in pure concentric performance. *Eur J Appl Physiol.* 2003;88(6):527–34.
41. Nichols AW. Achilles tendinitis in running athletes. *J Am Board Fam Pract.* 1989;2(3):196–203.
42. Lyman J, Weinhold PS, Almekinders LC. Strain behavior of the distal achilles tendon: implications for insertional achilles tendinopathy. *Am J Sports Med.* 2004;32(2):457–61.
43. Harris CA, Peduto AJ. Achilles tendon imaging. *Australas Radiol.* 2006;50(6):513–25.
44. Paavola M, Jarvinen TA. Paratendinopathy. *Foot Ankle Clin.* 2005;10(2):279–92.
45. Maffulli N, et al. Changing incidence of Achilles tendon rupture in Scotland: a 15-year study. *Clin J Sport Med.* 1999;9(3):157–60.
46. Lantto I, et al. Epidemiology of Achilles tendon ruptures: increasing incidence over a 33-year period. *Scand J Med Sci Sports.* 2015;25(1):e133–8.
47. Huttunen TT, et al. Acute achilles tendon ruptures: incidence of injury and surgery in sweden between 2001 and 2012. *Am J Sports Med.* 2014;42(10):2419–23.
48. Moller A, Astron M, Westlin N. Increasing incidence of Achilles tendon rupture. *Acta Orthop Scand.* 1996;67(5):479–81.
49. Saltzman CL, Tearse DS. Achilles tendon injuries. *J Am Acad Orthop Surg.* 1998;6(5):316–25.
50. Arner O, Lindholm A. Subcutaneous rupture of the Achilles tendon; a study of 92 cases. *Acta Chir Scand Suppl.* 1959;116(Supp 239):1–51.
51. Longo UG, et al. Acute achilles tendon rupture in athletes. *Foot Ankle Clin.* 2013;18(2):319–38.
52. Longo UG, et al. Tendon augmentation grafts: a systematic review. *Br Med Bull.* 2010;94:165–88.
53. Bernard-Beaubois K, et al. In vitro study of cytotoxicity of quinolones on rabbit tenocytes. *Cell Biol Toxicol.* 1998;14(4):283–92.
54. Fredberg U, et al. Ultrasonography as a tool for diagnosis, guidance of local steroid injection, and together with pressure algometry, monitoring of the treatment of athletes with chronic jumper's knee and Achilles tendinitis: a randomized, double-blind, placebo-controlled study. *Scand J Rheumatol.* 2004;33(2):94–101.
55. Bleakney RR, et al. Long-term ultrasonographic features of the Achilles tendon after rupture. *Clin J Sport Med.* 2002;12(5):273–8.
56. Garras DN, et al. MRI is unnecessary for diagnosing acute Achilles tendon ruptures: clinical diagnostic criteria. *Clin Orthop Relat Res.* 2012;470(8):2268–73.
57. Schweitzer ME, Karasick D. MR imaging of disorders of the Achilles tendon. *AJR Am J Roentgenol.* 2000;175(3):613–25.
58. Kader D, et al. Achilles tendinopathy: some aspects of basic science and clinical management. *Br J Sports Med.* 2002;36(4):239–49.
59. Maffulli N. Rupture of the Achilles tendon. *J Bone Joint Surg Am.* 1999;81(7):1019–36.
60. Kou J. AAOS Clinical Practice Guideline: acute Achilles tendon rupture. *J Am Acad Orthop Surg.* 2010;18(8):511–3.
61. Scott BW, al Chalabi A. How the Simmonds-Thompson test works. *J Bone Joint Surg Br.* 1992;74(2):314–5.
62. O'Brien T. The needle test for complete rupture of the Achilles tendon. *J Bone Joint Surg Am.* 1984;66(7):1099–101.
63. Matles AL. Rupture of the tendo achilles: another diagnostic sign. *Bull Hosp Joint Dis.* 1975;36(1):48–51.
64. Copeland SA. Rupture of the Achilles tendon: a new clinical test. *Ann R Coll Surg Engl.* 1990;72(4):270–1.
65. Fessell DP, et al. US of the ankle: technique, anatomy, and diagnosis of pathologic conditions. *Radiographics.* 1998;18(2):325–40.
66. Paavola M, et al. Ultrasonography in the differential diagnosis of Achilles tendon injuries and related disorders. A comparison between pre-operative ultrasonography and surgical findings. *Acta Radiol.* 1998;39(6):612–9.

67. Kayser R, Mahlfeld K, Heyde CE. Partial rupture of the proximal Achilles tendon: a differential diagnostic problem in ultrasound imaging. *Br J Sports Med.* 2005;39(11):838–42; discussion 838–42.
68. Alfredson H, Cook J. A treatment algorithm for managing Achilles tendinopathy: new treatment options. *Br J Sports Med.* 2007;41(4):211–6.
69. Dimmen S, et al. Negative effects of parecoxib and indomethacin on tendon healing: an experimental study in rats. *Knee Surg Sports Traumatol Arthrosc.* 2009;17(7):835–9.
70. Li Z, et al. Inflammatory response of human tendon fibroblasts to cyclic mechanical stretching. *Am J Sports Med.* 2004;32(2):435–40.
71. Rompe JD, Furia J, Maffulli N. Eccentric loading versus eccentric loading plus shock-wave treatment for midportion achilles tendinopathy: a randomized controlled trial. *Am J Sports Med.* 2009;37(3):463–70.
72. Rompe JD, et al. Eccentric loading, shock-wave treatment, or a wait-and-see policy for tendinopathy of the main body of tendo Achillis: a randomized controlled trial. *Am J Sports Med.* 2007;35(3):374–83.
73. Mafi N, Lorentzen R, Alfredson H. Superior short-term results with eccentric calf muscle training compared to concentric training in a randomized prospective multicenter study on patients with chronic Achilles tendinosis. *Knee Surg Sports Traumatol Arthrosc.* 2001;9(1):42–7.
74. Silbernagel KG, et al. Eccentric overload training for patients with chronic Achilles tendon pain—a randomised controlled study with reliability testing of the evaluation methods. *Scand J Med Sci Sports.* 2001;11(4):197–206.
75. Rasmussen S, et al. Shockwave therapy for chronic Achilles tendinopathy: a double-blind, randomized clinical trial of efficacy. *Acta Orthop.* 2008;79(2):249–56.
76. Alfredson H, et al. Heavy-load eccentric calf muscle training for the treatment of chronic Achilles tendinosis. *Am J Sports Med.* 1998;26(3):360–6.
77. de Jonge S, et al. One-year follow-up of a randomised controlled trial on added splinting to eccentric exercises in chronic midportion Achilles tendinopathy. *Br J Sports Med.* 2010;44(9):673–7.
78. de Vos RJ, et al. The additional value of a night splint to eccentric exercises in chronic midportion Achilles tendinopathy: a randomised controlled trial. *Br J Sports Med.* 2007;41(7):e5.
79. Roos EM, et al. Clinical improvement after 6 weeks of eccentric exercise in patients with mid-portion Achilles tendinopathy – a randomized trial with 1-year follow-up. *Scand J Med Sci Sports.* 2004;14(5):286–95.
80. Stergioulas A, et al. Effects of low-level laser therapy and eccentric exercises in the treatment of recreational athletes with chronic achilles tendinopathy. *Am J Sports Med.* 2008;36(5):881–7.
81. Willberg L, et al. Sclerosing injections to treat midportion Achilles tendinosis: a randomised controlled study evaluating two different concentrations of Polidocanol. *Knee Surg Sports Traumatol Arthrosc.* 2008;16(9):859–64.
82. Metcalfe D, Achten J, Costa ML. Glucocorticoid injections in lesions of the achilles tendon. *Foot Ankle Int.* 2009;30(7):661–5.
83. de Vos RJ, et al. Platelet-rich plasma injection for chronic Achilles tendinopathy: a randomized controlled trial. *JAMA.* 2010;303(2):144–9.
84. Sadoghi P, et al. The role of platelets in the treatment of Achilles tendon injuries. *J Orthop Res.* 2013;31(1):111–8.
85. Heckman DS, Gluck GS, Parekh SG. Tendon disorders of the foot and ankle, part 2: achilles tendon disorders. *Am J Sports Med.* 2009;37(6):1223–34.
86. Fahlstrom M, et al. Chronic Achilles tendon pain treated with eccentric calf-muscle training. *Knee Surg Sports Traumatol Arthrosc.* 2003;11(5):327–33.
87. Jonsson P, et al. New regimen for eccentric calf-muscle training in patients with chronic insertional Achilles tendinopathy: results of a pilot study. *Br J Sports Med.* 2008;42(9):746–9.
88. Rompe JD, Furia J, Maffulli N. Eccentric loading compared with shock wave treatment for chronic insertional achilles tendinopathy. A randomized, controlled trial. *J Bone Joint Surg Am.* 2008;90(1):52–61.
89. Wiegerinck JJ, et al. Treatment for insertional Achilles tendinopathy: a systematic review. *Knee Surg Sports Traumatol Arthrosc.* 2013;21(6):1345–55.
90. Elias I, et al. Outcomes of chronic insertional Achilles tendinosis using FHL autograft through single incision. *Foot Ankle Int.* 2009;30(3):197–204.
91. McGarvey WC, et al. Insertional Achilles tendinosis: surgical treatment through a central tendon splitting approach. *Foot Ankle Int.* 2002;23(1):19–25.
92. Johnson KW, Zalavras C, Thordarson DB. Surgical management of insertional calcific achilles tendinosis with a central tendon splitting approach. *Foot Ankle Int.* 2006;27(4):245–50.
93. Maffulli N, et al. Calcific insertional Achilles tendinopathy: reattachment with bone anchors. *Am J Sports Med.* 2004;32(1):174–82.
94. Yodlowski ML, Scheller Jr AD, Minos L. Surgical treatment of Achilles tendinitis by decompression of the retrocalcaneal bursa and the superior calcaneal tuberosity. *Am J Sports Med.* 2002;30(3):318–21.
95. Sadoghi P, et al. Initial Achilles tendon repair strength—synthesized biomechanical data from 196 cadaver repairs. *Int Orthop.* 2012;36(9):1947–51.
96. Rosso C, et al. Long-term biomechanical outcomes after Achilles tendon ruptures. *Knee Surg Sports Traumatol Arthrosc.* 2015;23(3):890–8.
97. Bhandari M, et al. Treatment of acute Achilles tendon ruptures: a systematic overview and metaanalysis. *Clin Orthop Relat Res.* 2002;400:190–200.
98. Holm C, Kjaer M, Eliasson P. Achilles tendon rupture - treatment and complications: a systematic review. *Scand J Med Sci Sports.* 2015;25(1):e1–10.
99. Khan RJ, Carey Smith RL. Surgical interventions for treating acute Achilles tendon ruptures. *Cochrane Database Syst Rev.* 2010;9:CD003674.
100. Khan RJ, et al. Treatment of acute achilles tendon ruptures. A meta-analysis of randomized, controlled trials. *J Bone Joint Surg Am.* 2005;87(10):2202–10.
101. Maffulli N, et al. Peroneus brevis tendon transfer for reconstruction of chronic tears of the Achilles tendon: a long-term follow-up study. *J Bone Joint Surg Am.* 2012;94(10):901–5.
102. Maffulli N, et al. Free gracilis tendon graft for reconstruction of chronic tears of the Achilles tendon. *J Bone Joint Surg Am.* 2012;94(10):906–10.
103. Ponnappula P, Aaranson RR. Reconstruction of achilles tendon rupture with combined V-Y plasty and gastrocnemius-soleus fascia turndown graft. *J Foot Ankle Surg.* 2010;49(3):310–5.
104. Ma GW, Griffith TG. Percutaneous repair of acute closed ruptured achilles tendon: a new technique. *Clin Orthop Relat Res.* 1977;128:247–55.
105. Carmont MR, Maffulli N. Modified percutaneous repair of ruptured Achilles tendon. *Knee Surg Sports Traumatol Arthrosc.* 2008;16(2):199–203.
106. Webb JM, Bannister GC. Percutaneous repair of the ruptured tendo Achillis. *J Bone Joint Surg Br.* 1999;81(5):877–80.
107. Brumann M, et al. Accelerated rehabilitation following Achilles tendon repair after acute rupture - Development of an evidence-based treatment protocol. *Injury.* 2014;45(11):1782–90.

108. Lim J, Dalal R, Waseem M. Percutaneous vs. open repair of the ruptured Achilles tendon—a prospective randomized controlled study. *Foot Ankle Int.* 2001;22(7):559–68.
109. Cretnik A, Kosanovic M, Smrkolj V. Percutaneous versus open repair of the ruptured Achilles tendon: a comparative study. *Am J Sports Med.* 2005;33(9):1369–79.
110. Olsson N, et al. Stable surgical repair with accelerated rehabilitation versus nonsurgical treatment for acute Achilles tendon ruptures: a randomized controlled study. *Am J Sports Med.* 2013;41(12):2867–76.
111. Majewski M, et al. Avoiding sural nerve injuries during percutaneous Achilles tendon repair. *Am J Sports Med.* 2006;34(5):793–8.
112. Rosso C, et al. Long-term outcomes of muscle volume and Achilles tendon length after Achilles tendon ruptures. *Knee Surg Sports Traumatol Arthrosc.* 2013;21(6):1369–77.
113. Jacobs D, et al. Comparison of conservative and operative treatment of Achilles tendon rupture. *Am J Sports Med.* 1978;6(3):107–11.
114. Persson A, Wredmark T. The treatment of total ruptures of the Achilles tendon by plaster immobilisation. *Int Orthop.* 1979;3(2):149–52.
115. Hufner TM, et al. Long-term results after functional nonoperative treatment of achilles tendon rupture. *Foot Ankle Int.* 2006;27(3):167–71.
116. McComis GP, Nawoczenski DA, DeHaven KE. Functional bracing for rupture of the Achilles tendon. Clinical results and analysis of ground-reaction forces and temporal data. *J Bone Joint Surg Am.* 1997;79(12):1799–808.
117. Rousseau R, et al. Results of surgical treatment of calcaneus insertional tendinopathy in middle- and long-distance runners. *Knee Surg Sports Traumatol Arthrosc.* 2015;23(9):2494–501.
118. Huang J, et al. Rehabilitation regimen after surgical treatment of acute Achilles tendon ruptures: a systematic review with meta-analysis. *Am J Sports Med.* 2015;43(4):1008–16.
119. Majewski M, et al. Postoperative rehabilitation after percutaneous Achilles tendon repair: early functional therapy versus cast immobilization. *Disabil Rehabil.* 2008;30(20–22):1726–32.
120. Speck M, Klaue K. Early full weightbearing and functional treatment after surgical repair of acute achilles tendon rupture. *Am J Sports Med.* 1998;26(6):789–93.
121. Young SW, et al. Weight-bearing in the nonoperative treatment of acute Achilles tendon ruptures: a randomized controlled trial. *J Bone Joint Surg Am.* 2014;96(13):1073–9.
122. Kvist M, Jarvinen M. Clinical, histochemical and biomechanical features in repair of muscle and tendon injuries. *Int J Sports Med.* 1982;3 Suppl 1:12–4.



Yousef Alrashidi, Alexej Barg, Manuel Kampmann,  
and Victor Valderrabano

## Abstract

Plantar fasciitis is a common musculoskeletal condition among sport-active people. Careful history taking and appropriate clinical examination is essential to exclude other causes of plantar heel pain. Following a step-wise approach is recommended in plantar fasciitis treatment, as it is usually self-limiting condition. Inadequate response to non-operative treatment and persistence of symptoms more than 6 months may warrant surgical intervention. Provision of health instructions to athletes would help in prevention of plantar fasciitis. Athletes should watch for their training mistakes and should maintain good muscular strength and balance.

## Keywords

Plantar fasciitis • Plantar fasciopathy • Plantar fasciosis • Plantar fasciitis in athletes • Overuse foot injuries • Foot injuries • Heel pain in athletes

## Introduction

Plantar fasciitis (PF) is a common musculoskeletal condition among athletic and non-athletic people. It is characterized by the presence of morning plantar heel pain that is located at

Y. Alrashidi, MBBS, SB-Orth (✉)  
Orthopedic Surgery Department, Taibah University,  
College of Medicine, Almadinah Almunawrah, Saudi Arabia

Orthopaedic Department, SWISS ORTHO CENTER,  
Schmerzlinik Basel, Swiss Medical Network, Basel, Switzerland  
e-mail: [yalrashidi@gmail.com](mailto:yalrashidi@gmail.com)

A. Barg, MD  
Department of Orthopaedics, University of Utah,  
590 Wakara Way, Salt Lake City, UT 84108, USA  
e-mail: [alexejbarg@mail.ru](mailto:alexejbarg@mail.ru)

M. Kampmann  
Department of Musculoskeletal Radiology, Basel University  
Hospital, Petersgraben 4 CH-4031, Basel, Switzerland  
e-mail: [manuel.kampmann@usb.ch](mailto:manuel.kampmann@usb.ch)

V. Valderrabano, MD, PhD  
Orthopaedic Department, SWISS ORTHO CENTER,  
Schmerzlinik Basel, Swiss Medical Network, Basel, Switzerland  
e-mail: [vvalderrabano@gsmn.ch](mailto:vvalderrabano@gsmn.ch)

the inferomedial calcaneus, increases with walking initiation, disappears shortly after few steps, and goes back at the end of ambulation activity and at rest [1–3]. The onset of symptoms is usually preceded by activity change, shoe modification, weight change, or simple pedal trauma. Moreover, gastrocnemius-soleus contracture, tibio-talar joint stiffness and calcaneal spurs may be found in common with chronic cases that may lead to physical and financial negative consequences. Numbness, nocturnal pain, and swelling are occasionally present. [4]

## Incidence

It has been reported that one-tenth of people may suffer from symptoms of plantar fasciitis during their lifetime [5]. Bilateral PF may present in less than one-third of the cases [4]. Moreover, such a condition is one of the common chronic pedal overuse injuries especially in sports that involve repetitive trauma and complex motions [6, 7]. Up to authors' knowledge, there is not clear reported figures showing the incidence rate among non-elite and elite athletes [6].

## Etiology and Pathomechanism

It is generally agreed that PF is a result of chronic process of frequent micro-tearing and inflammation due to tensile overload at the longitudinal plantar fascia, but recently it is more accepted to be a result of multiple factors [4, 8]. Histologically, the inflammatory cells are not seen in advanced cases. Failed attempts of tissue healing would result in disorganised vascularity along with areas of both regeneration and degradation. That also would result in additive damage at the cellular level [4]. The limited length of such a fascia and the tendency to plantar flex the ankle joint during dorsiflexion of the first toe could increase the loading forces on the plantar fascial insertion. Consequently, forefoot loading forces and time would increase. As a result, micro-injury may persist and the process becomes chronic [9].

According to a level II prognostic study, it has been observed that PF is found in common with ankle dorsiflexion limitation as in gastrocnemius-soleus shortening, high body mass index, and jobs necessitating prolonged weight bearing (Table 21.1). Riddle et al described the association between PF with dorsiflexion limitation and body mass index as “dose dependent” [5]. Cavus deformity of foot may predispose to PF owing to the low flexibility of medial arch [10]. Moreover, longitudinal plantar arch morphology and hindfoot malalignment, which were believed to be important factors in development of PF, have been studied but conclusions were not decisive [2]. Some studies suggest that leg length inequality may predispose to development of compensatory motions that might increase the risk of pedal injuries in runners [11]. Other studies found that competitive athletes are more liable to PF due to the use of spike sport-shoes and not due to grade of activity [10]. Overuse appears to be the likely factor in development of PF in sporty people [12].

When we look at PF as an overuse sport injury, there is no scientific agreement on how much sport exposure time that is considered as a risk factor for the development of such a condition. In other words, several previous reports represented exposure time units as years or seasons that do not necessarily indicate the actual time [6]. In fact, such types of studies may be liable to bias and inaccurate measurements [7].

**Table 21.1** Risk factors of plantar fasciitis

1. Ankle dorsiflexion limitation (e.g. gastrocnemius-soleus shortening)
2. High body mass index
3. Jobs necessitating prolonged weight bearing
4. Competitive sports using spike sport-shoes
5. Overuse
6. Cavus foot (not fully proved)
7. Flatfoot (not fully proved)
8. Limb length discrepancy in athletes

## Symptoms

Patients usually report plantar-medial heel pain, typically after a long weight-bearing phase. PF shows also a starting pain after resting or in the first steps in the morning, which alleviates after warming-up and, however, gets worse after significant stress load.

## Classification

In literature, up to authors' knowledge, there is not yet a clear useful classification for PF. However, it is generally agreed to label a patient with persistent symptoms for more than 6 months in spite of conservative therapy as “recalcitrant”. Moreover, a recent study proposed to classify PF as “insertional” or “non-insertional” depending upon the ultrasonic picture. Such a classification may help revisit the current therapeutic guidelines [13].

## Diagnostics

PF is an important differential diagnosis for plantar heel pain. So, careful history taking, appropriate physical examination and investigations should aim to rule out other causes such as stress fracture, compression neuropathies (e.g. Baxter's nerve entrapment), infection of calcaneus, calcaneal bursitis, systemic arthritis syndromes, localised osteoarthritis [14]. The classical tender point of PF is plantar-medial, distally of the plantar medial calcaneal tuberosity. Compared to it, Baxter's nerve entrapment (first branch of the lateral plantar nerve) has the typical tender point more proximally and medially [15]. In junior athletes, PF could be mistaken for apophysitis of calcaneus where the tender point is clear on the calcaneus [16]. One of the useful tests is the toes dorsiflexion test, which cause a stretch pain on the plantar fascia. Further examination shall include assessment of a possible heel cord contracture (with knee extension and heel varisation), hindfoot alignment and foot arch evaluation.

Plain radiographs are less likely to help in reaching the diagnosis. However, calcaneal spur may be found radiologically at the medial calcaneal tuberosity, anatomically corresponding to the origin area of the flexor digitorum brevis muscle and not plantar fascia (Fig. 21.1). But its presence may not indicate the origin of patient's complaint [16]. However, presence of calcaneal spur may reflect chronicity of the problem and longer time needed for recovery [17].

Magnetic resonance imaging (MRI) can confirm PF diagnosis and rule other causes of plantar heel pain because of its significantly high sensitivity and specificity. Thickening,



**Fig. 21.1** Conventional X-ray. Lateral view in a 51-year-old female which shows a calcaneal spur

partial tears and edema of the fascia are characteristic MRI changes in PF (Fig. 21.2) [4].

Bone scan (Technetium-99) or single photon emission computerised tomography (SPECT-CT) may help to differentiate PF from calcaneal stress fracture (Fig. 21.3) [4, 18]. It may show localised high uptake over the medial calcaneal surface in case of PF but non-localized with higher intensity in calcaneal stress fracture. Bone scan is less specific than MRI [4].

Ultrasound is a very useful and promising modality of imaging. It is a fast tool to confirm the diagnosis and not painful. It can be used for follow-up and monitoring the improvement after initiation of therapy. However, it is operator-dependent. The presence of hypoechoic foci and discontinuity of fascial bundles are characteristic ultrasonic changes for PF [4].

Laboratory workup should be considered in bilateral cases, in case of failed non-operative therapy and if a systemic disease is suspected. Moreover, advanced neurological assessment has to be kept in mind in the presence of significant neurological findings [4, 17].



**Fig. 21.2** MRI diagnostics. Sagittal PD fs image in a 52-year-old female that demonstrates calcaneal spur, ill-defined calcaneal bone marrow edema, diffuse soft-tissue edema superficial to origin of plantar fascia, and hyperintense signal within plantar fascia close to its origin

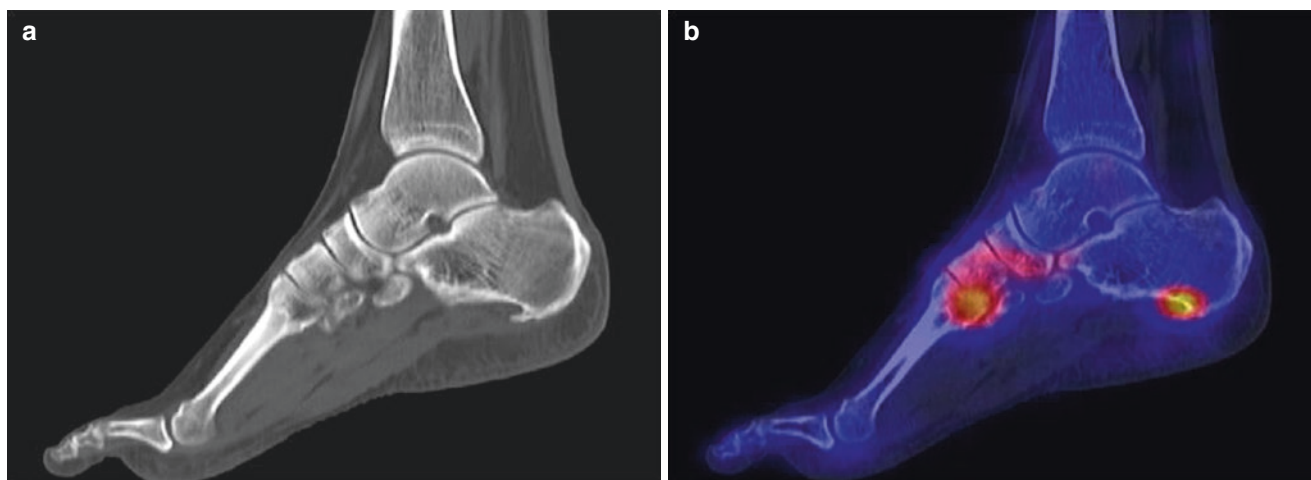
## Therapy

PF is well known to be usually a self-limited condition that may resolve within 12 months. So, efforts should be directed towards a simple, a safe and low-cost way of treatment [8]. In spite of use of different combination of treatments, about one tenth of cases may fail to respond to conservative treatment [4]. In fact, there is not enough studies to support superiority of one modality of treatment over others (Table 21.2).

For plantar heel pain management, the first level of treatment would include the use of non-steroidal anti-inflammatory drugs (NSAIDs), specific physical exercises, foot insoles, night splints, and patient's instructions to lose weight, restrict some activities, and not to use flat shoes or walk barefoot [17].

According to a randomized clinical study, it has been suggested that the use of NSAIDs in the non-operative protocol might help decrease suffering of patients in terms of good pain relief and better function [19].

Because of the negative impact of heel cord tightness on the plantar fascia through windlass phenomenon and through increasing forefoot loading, heel cord-specific stretching exercises have been recommended [9]. However, those exercises are not unique for recreating windlass phenomenon and are usually done after the initiation of walking. In other



**Fig. 21.3** SPECT-CT diagnostics. (a) Sagittal reconstruction of CT scan and (b) fused colored SPECT-CT in a 53-year-old female. Note the calcaneal spur. The scan also demonstrates increased bone turnover at the origin of the plantar fascia related to a plantar fasciitis. Also, there is

an incidental finding of degenerative changes of TMT joint. *Abbreviations:* MRI magnetic resonance imaging, SPECT-CT single photon emission computerised tomography, CT computerized tomography, PDfs proton density weighted fat saturated, TMT tarso-meta-tarsal

**Table 21.2** Treatment recommendation

Step-wise recommended approach for plantar fasciitis treatment		Evidence: grade of recommendation [14]
First level	1. NSAIDs	B
	2. Physiotherapy (Achilles and plantar fascia specific exercises)	A
	3. Insoles to deload the calcaneal plantarprint and correct medical arch deformities	B
Second level	1. ESWT	A
Third level	2. PRP injection	C
Fourth level	3. Corticosteroid injection (only once)	B
Fifth level	4. Temporary immobilization (cast/walker) with PWB	C [17]
Sixth level	5. Surgery	C

NSAIDs non-steroidal anti-inflammatory drugs, ESWT extracorporeal shock wave therapy, PRP Platelet Rich Plasma, PWB partial weight bearing

words, according to a level I randomised controlled trial, it is recommended to do plantar fascia-specific stretching maneuvers before initiation of walking in the morning in order to help inhibit the process of inflammation and micro tearing [20]. Moreover, those specific maneuvers are so advantageous in terms of improving the compliance of patients because those maneuvers can be done at any time before initiation of walking and they are not weight bearing [20].

The goal of prescribing foot insoles in PF is to lower stress loading on the longitudinal plantar fascia through hindfoot elevation and supporting the medial arch. In addition, they help in distributing and absorbing weight of the body over the plantar fascia attachments [8]. The aim of night splints is to keep the Achilles tendon and longitudinal fascial length in an extended position during night [17]; so, that would give a chance for tissue healing at a good length and appropriate tension [12].

There is not enough high level studies to support significantly the use of acupuncture, ice therapy, heat therapy, or magnetic insoles in PF. [8].

The extracorporeal shock wave therapy (ESWT) is considered after exhausting all of the previous conservative modalities over more than a half a year [17]. ESWT is considered to be very advantageous and a good alternative to the surgical options. According to recent high-level studies, ESWT is a safe and effective way of treatment in chronic PF. In addition, it has minor complications and short time to recovery as it is a non-invasive modality [21–23]. Moreover, it can be used as an outpatient treatment [22]. Consequently, it can help in reducing health care expenses and jeopardy of work-time [23]. It was proposed that patients during or after ESWT treatment are not in need to stop their daily life works or even their athletic activities [22, 23]. The effective and safe therapeutic protocol of radial ESWT has been found to be three consecutive doses ( $3 \times 2000$  impulses,  $0.16 \text{ mJ/mm}^2$ ) focused on the area of maximum tenderness without the need for any anaesthesia [22]. In addition, according to level I studies, the utilization of focused ESWT has been found to be effective in PF treatment especially if given in high doses [21].

Platelet Rich Plasma (PRP) injection seems to be a safer modality of injection than corticosteroids. It is suggested that PRP effect addresses the collagen catabolism and disturbed vascularity in chronic PF [4]. According to a recent randomised blinded study, it is recommended to continue on specific stretching exercises after PRP injection in order to get the best outcome. A recent systematic review suggested that PRP injection may be of a superiority over a pure non-operative therapy in the course of treatment PF [24]. However, further high-level studies are needed to estimate the appropriate candidate, timing, injection dose, local efficacy and systematic impacts on the body [4, 24].

Corticosteroid injection is suggested that it modulates the ultrasonic picture of the disease by decreasing the thickness of plantar fascia and formation of hypoechoic foci. Corticosteroids can be administered in the form of a single dose injected from medial to avoid fat pad atrophy. [14]. However, potentially serious complications have been documented like plantar fascia rupture [4]. Other possible complications include fat pad atrophy post-procedure pain and calcifications of the plantar longitudinal fascia [14].

If minimal or no positive response to previous modalities, limb immobilization for 4–6 weeks can be considered by using a splint or a walker [14].

---

## Surgical Treatment

The necessity of surgical treatment in PF is rare (around in 5 % of the cases). Inadequate response to non-operative treatment and persistence of symptoms more than half a year may warrant surgical intervention [15, 17, 25]. Presence of medical comorbidities, infection, inadequate limb vascularity, short or deficient conservative course of treatment, non-compliance of patient to non-operative treatment, or unclear patient's expectations are relative contraindications for surgery. However, the need for nerve decompression should be considered in the presence of neurologic manifestations [15]. Plantar fascia release can be done by an open or minimally invasive techniques [25].

The open technique may involve debridement, extensive or partial release of the plantar fascia [25]. Such a technique offers generous view of the anatomic structures [26]. Under general or local anaesthesia, a transverse 3 cm skin incision is made over the medial side of the hindfoot. Then, careful dissection is carried out until the medial part of the plantar fascia is reached. Once the plantar fascia is identified, a transverse incision of about one third of the medial part is made using a blade or Mayo scissors. Then, irrigation and closure in layer is done. Such a procedure may also involve excision of calcaneal spurs in the area of the flexor digitorum brevis origin [27]. Possible complications are not limited to nerve injury, pain syndromes, infection (superficial or deep), wound dehiscence, stress fracture (calcaneal or metatarsal) or iatrogenic

flatfoot [25]. It has been suggested to limit the fascial release up to 40% to minimize the risk of medial arch instability, lateral column pain, and consequently gait changes [25]. However, complete release of plantar fascia with proximal and distal release of tarsal tunnel is recommended in patients with history of partial release surgery or if plantar fascia is attenuated. This way may address the underlying pathological aetiology [15].

Minimally invasive techniques are advantageous over open technique in terms of simplicity, cost-effectiveness, faster recovery, fewer complications and have a good to excellent outcome [25]. Intra-operatively, percutaneous release of medial part of the plantar fascia has to be done while toes are in a dorsiflexed position and confirmed by feeling the fall of medial band tension [27]. Moreover, endoscopic release is a promising alternative to open procedures in PF and seems to be a safe modality [25]. Postoperative complications of endoscopic techniques may include persistent hindfoot pain, stress fractures, skin problems, entrapment of nerves and infection [28]. Moreover, many of relevant studies have some limitations; so, there is a need for further high-quality studies to ascertain the best procedure candidates, best portal system and timing of intervention [28].

Outcome studies of plantar fasciitis release in athletes are limited [29]. In a study by Leach et al, fourteen out fifteen athletes were able return to full sport activity within 9 weeks after they underwent open plantar fascia release followed by 2 weeks of limb immobilisation postoperatively [30]. It is still believed that limb immobilization after surgery may play a major role in decreasing the after-surgery consequences [29]. In a comparative study by Zimmerman et al, it has been found that patients who started immediate postoperative weight bearing in a cast for 2 weeks after endoscopic release had a shorter time to return to sports and favourable patient's satisfaction [31].

## Rehabilitation and Back-to-Sports

After surgery, weight-bearing is allowed as tolerated according to individual patient's pain. Slight careful physiotherapy exercises are important to maintain the fascia release. After 2 weeks of sports break, low-impact sports are permitted, individually increasing to force and endurance training and high-impact sports.

## Prevention

As any overuse injury, PF prevention in sport active people is recommended. Athletes should watch for training mistakes and not use extremes of load. Good muscular strengthening of lower limbs may help prevent muscular imbalance that

could be a factor in overuse injuries. Furthermore, full recovery from any injury is advised before engaging in any competitive sport [32]. An appropriate clinical assessment of sport-active people and providing health instructions would help to prevent plantar fasciitis especially in athletes who have a history of such a condition [33].

## Evidence

Table 21.2 shows grades of evidence in Plantar Fasciitis different modalities of treatment.

*Literature source for evidence:*

- Martinelli N, Bonifacini C, Romeo G. *Current therapeutic approaches for plantar fasciitis. Orthop Res Rev. 2014;6:33–40.*
- Thomas JL, Christensen JC, Kravitz SR, Mendicino RW, Schuberth JM, Vanore JV, et al. *The diagnosis and treatment of heel pain: a clinical practice guideline-revision 2010. J Foot Ankle Surg: Off Publ Am Coll Foot Ankle Surg. 2010;49 (3 Suppl):S1–19.*

## Summary

1. Plantar fasciitis is a common musculoskeletal condition among sport-active people.
2. Careful history taking and appropriate clinical examination is essential to exclude other causes of plantar heel pain.
3. Following a step-wise approach is recommended in plantar fasciitis treatment, as it is usually self-limiting condition.
4. Inadequate response to non-operative treatment and persistence of symptoms more than 6 months may warrant surgical intervention
5. Provision of health instructions to athletes would help in prevention of plantar fasciitis. Athletes should watch for their training mistakes and should maintain good muscular strength and balance.

## References

1. Greve JM, Grecco MV, Santos-Silva PR. Comparison of radial shockwaves and conventional physiotherapy for treating plantar fasciitis. *Clinics. 2009;64(2):97–103.*
2. Ribeiro AP, Trombini-Souza F, Tessutti V, Rodrigues Lima F, Sacco Ide C, Joao SM. Rearfoot alignment and medial longitudinal arch configurations of runners with symptoms and histories of plantar fasciitis. *Clinics. 2011;66(6):1027–33.*
3. Kindred J, Trubey C, Simons SM. Foot injuries in runners. *Curr Sports Med Rep. 2011;10(5):249–54.*
4. Monto RR. Platelet-rich plasma and plantar fasciitis. *Sports Med Arthrosc Rev. 2013;21(4):220–4.*
5. Riddle DL, Pulisic M, Pidcoe P, Johnson RE. Risk factors for Plantar fasciitis: a matched case-control study. *J Bone Joint Surg Am. 2003;85-A(5):872–7.*
6. Sobhani S, Dekker R, Postema K, Dijkstra PU. Epidemiology of ankle and foot overuse injuries in sports: a systematic review. *Scand J Med Sci Sports. 2013;23(6):669–86.*
7. Knobloch K, Yoon U, Vogt PM. Acute and overuse injuries correlated to hours of training in master running athletes. *Foot Ankle Int. 2008;29(7):671–6.*
8. Stuber K, Kristmanson K. Conservative therapy for plantar fasciitis: a narrative review of randomized controlled trials. *J Can Chiropr Assoc. 2006;50(2):118–33.*
9. Harty J, Soffe K, O’Toole G, Stephens MM. The role of hamstring tightness in plantar fasciitis. *Foot Ankle Int. 2005;26(12):1089–92.*
10. Francesco D, Caprio RB, Massimiliano M, Antonino C, Sandro G. Foot and lower limb diseases in runners: assessment of risk factors. *J Sports Sci Med. 2010;9:10.*
11. Messier SP, Pittala KA. Etiologic factors associated with selected running injuries. *Med Sci Sports Exerc. 1988;20(5):501–5.*
12. Karagounis P, Tsironi M, Prionas G, Tsiganos G, Baltopoulos P. Treatment of plantar fasciitis in recreational athletes: two different therapeutic protocols. *Foot Ankle Spec. 2011;4(4):226–34.*
13. Jeong E, Afolayan J, Carne A, Solan M. Ultrasound scanning for recalcitrant plantar fasciopathy. Basis of a new classification. *Skeletal Radiol. 2013;42(3):393–8.*
14. Martinelli N, Bonifacini C, Romeo G. Current therapeutic approaches for plantar fasciitis. *Orthop Res Rev. 2014;6:33–40.*
15. DiGiovanni BF, Dawson LK, Baumhauer JF. Plantar Heel Pain. In: Coughlin MJ, Saltzman CL, Anderson RB, editors. *Mann’s Surgery of the Foot and Ankle. 1. 9 ed. Philadelphia: Elsevier Saunders; 2014. p. 688.*
16. Omev ML, Micheli LJ. Foot and ankle problems in the young athlete. *Med Sci Sports Exerc. 1999;31(7 Suppl):S470–86.*
17. Thomas JL, Christensen JC, Kravitz SR, Mendicino RW, Schuberth JM, Vanore JV, et al. The diagnosis and treatment of heel pain: a clinical practice guideline-revision 2010. *J Foot Ankle Surg Off Publ Am Coll Foot Ankle Surg. 2010;49(3 Suppl):S1–19.*
18. Nathan M, Mohan H, Vijayanathan S, Fogelman I, Gnanasegaran G. The role of 99mTc-diphosphonate bone SPECT/CT in the ankle and foot. *Nucl Med Commun. 2012;33(8):799–807.*
19. Donley BG, Moore T, Sferra J, Gozdanovic J, Smith R. The efficacy of oral nonsteroidal anti-inflammatory medication (NSAID) in the treatment of plantar fasciitis: a randomized, prospective, placebo-controlled study. *Foot Ankle Int. 2007;28(1):20–3.*
20. DiGiovanni BF, Nawoczinski DA, Lintal ME, Moore EA, Murray JC, Wilding GE, et al. Tissue-specific plantar fascia-stretching exercise enhances outcomes in patients with chronic heel pain. A prospective, randomized study. *J Bone Joint Surg Am. 2003;85-A(7):1270–7.*
21. Speed C. A systematic review of shockwave therapies in soft tissue conditions: focusing on the evidence. *Br J Sports Med. 2014;48(21):1538–42.*
22. Gerdesmeyer L, Frey C, Vester J, Maier M, Weil Jr L, Weil Sr L, et al. Radial extracorporeal shock wave therapy is safe and effective in the treatment of chronic recalcitrant plantar fasciitis: results of a confirmatory randomized placebo-controlled multicenter study. *Am J Sports Med. 2008;36(11):2100–9.*
23. Kudo P, Dainty K, Clarfield M, Coughlin L, Lavoie P, Lebrun C. Randomized, placebo-controlled, double-blind clinical trial evaluating the treatment of plantar fasciitis with an extracorporeal shockwave therapy (ESWT) device: a North American confirmatory study. *J Orthop Res Off Publ Orthop Res Soc. 2006;24(2):115–23.*
24. Franceschi F, Papalia R, Franceschetti E, Paciotti M, Maffulli N, Denaro V. Platelet-rich plasma injections for chronic plantar fasciopathy: a systematic review. *Br Med Bull. 2014;112(1):83–95.*

25. Nery C, Raduan F, Mansur N, Baunfeld D, Del Buono A, Maffulli N. Endoscopic approach for plantar fasciopathy: a long-term retrospective study. *Int Orthop*. 2013;37(6):1151–6.
26. Mook WR, Gay T, Parekh SG. Extensile decompression of the proximal and distal tarsal tunnel combined with partial plantar fascia release in the treatment of chronic plantar heel pain. *Foot Ankle Spec*. 2013;6(1):27–35.
27. Fallat LM, Cox JT, Chahal R, Morrison P, Kish J. A retrospective comparison of percutaneous plantar fasciotomy and open plantar fasciotomy with heel spur resection. *J Foot Ankle Surg Off Publ Am Coll Foot Ankle Surg*. 2013;52(3):288–90.
28. Hogan KA, Webb D, Shereff M. Endoscopic plantar fascia release. *Foot Ankle Int*. 2004;25(12):875–81.
29. Saxena A. Uniportal endoscopic plantar fasciotomy: a prospective study on athletic patients. *Foot Ankle Int*. 2004;25(12):882–9.
30. Leach RE, Seavey MS, Salter DK. Results of surgery in athletes with plantar fasciitis. *Foot Ankle*. 1986;7(3):156–61.
31. Zimmerman BJ, Cardinal MD, Cragel MD, Goel AR, Lane JW, Schramm KA. Comparison of three types of postoperative management for endoscopic plantar fasciotomy. A retrospective study. *J Am Podiatr Med Assoc*. 2000;90(5):247–51.
32. Fredericson M, Misra AK. Epidemiology and aetiology of marathon running injuries. *Sports Med*. 2007;37(4–5):437–9.
33. Rome K, Howe T, Haslock I. Risk factors associated with the development of plantar heel pain in athletes. *The Foot*. 2001;11:119–25.

Abdulaziz Almaawi, Andrzej Marcin Boszczyk,  
and Timothy R. Daniels

---

## Abstract

Osteochondroses are characterized by disorderliness of endochondral ossification in the growing skeleton affecting various joint developments throughout the body. This process, which starts in adolescence, can cause permanent bony and/or articular alterations that require management in the adult phase of life. Examples in the foot and ankle include Freiberg's infraction, Köhler disease, sesamoid osteochondrosis, Sever's disease and Iselin's disease. Etiology, symptoms, diagnostics, non-operative management and surgical options are discussed. Haglund's deformity of the posterior calcaneus is a predisposing factor for painful retrocalcaneal bursitis. Diagnosis relies on weight-bearing lateral radiographs of the foot; numerous radiological parameters have been developed. Operative options include calcaneoplasty (open or arthroscopic) and calcaneal osteotomy. Most of these conditions can be treated non-operatively. Symptom control, immobilization, activity modification and appropriate rehabilitation can all play a role in a safe and timely return to sport.

---

## Keywords

Osteochondrosis • Freiberg's • Köhler • Sesamoid • Sever's • Iselin's • Hugland

The term exostosis encompasses any benign formation of new bone or cartilaginous tissue on the surface of a bone, usually due to the formation of excess calcium. An exostosis can cause chronic pain, from mild to severely debilitating, depending on its shape, size, and location. This chapter will discuss three types of exostoses that commonly affect the foot: (A) osteochondroses, (B) apophysites, and (C) Haglund's deformity.

---

A. Almaawi, MD, FRCSC • T.R. Daniels, MD, FRCSC (✉)  
St. Michael's Hospital, 800-55 Queen Street East,  
Toronto, ON M5C 1R6, Canada  
e-mail: [abdulaziz.almaawi@mail.mcgill.ca](mailto:abdulaziz.almaawi@mail.mcgill.ca); [danielst@smh.ca](mailto:danielst@smh.ca)

A.M. Boszczyk, MD, PhD  
Adam Gruca Autonomous Public Clinical Hospital,  
Konarskiego Str. 13, Otwock PL05-400, Poland  
e-mail: [ortopeda@boszczyk.pl](mailto:ortopeda@boszczyk.pl)

---

## Osteochondroses

Osteochondrosis is a group of conditions characterized by disorderliness of endochondral ossification, including both osteogenesis and chondrogenesis, in the growing skeleton [1]. The clinical presentation of osteochondrosis can vary, depending on the anatomic location and whether it occurs in the epiphysis or apophysis. Significant morbidity can result from involvement of weight-bearing areas and joints in the foot.

Overuse injuries occur in the growing skeleton, mainly during growth spurts or with intense athletic participation. Among athletic injuries, the foot is the third most common cause of time lost in sport, with the ankle and knee being more common [2].

Osteochondroses of the foot include a wide range of pathologies affecting cartilage growth in joints, physeal lines, and tendon and ligament attachments. Specific examples include Freiberg's infraction (epiphyseal osteonecrosis



of the second metatarsal), Köhler disease (osteonecrosis of the tarsal navicular bone), sesamoid osteochondrosis, Sever's disease (apophysitis of calcaneus), and Iselin's disease (apophysitis of the tuberosity of the fifth metatarsal bone).

### **Etiology and Pathomechanism**

The exact etiology of osteochondroses is unknown. Repetitive trauma, vascular abnormalities, mechanical factors, and hormonal imbalances may all play a role. Acute or chronic repetitive trauma is the most commonly accepted theory [3]. Impairment of the blood flow in an area of skeletal growth is thought to also be a contributing factor. These disorders result from abnormal growth, injury, or overuse of the developing growth plate and surrounding ossification centres [4].

Osteochondrosis initially starts with necrosis of bone and cartilage, followed by revascularization, reorganization with granulation tissue formation and invasion, osteoclast reabsorption of necrotic segments, and finally osteoid replacement and formation of mature lamellar bone. During growth, there are rapid changes in the musculoskeletal system, and weight-bearing areas in the foot can be affected greatly by acute and repetitive stress injuries. Additionally, abnormalities in the bone and cartilage that may remain asymptomatic and undiagnosed in inactive children can become symptomatic with activities and sports as increased stress is placed on the bone or joint [2].

### **Classification**

A classification system for osteochondroses described by R. S. Siffert is intended to focus on anatomic and clinical parameters [1]. The classification of the osteochondroses presented includes three fundamental areas of disorderly endochondral ossification: the articular epiphysis, the nonarticular epiphysis, and the physal epiphysis. Articular osteochondroses are either primary, involving articular and epiphyseal cartilage and adjacent endochondral ossifications (e.g., humeral condylosis, Freiberg's disease, also known as Freiberg's infraction), or secondary as a consequence of avascular necrosis of subchondral bone (e.g., Kohler's disease, osteochondritis dissecans). Nonarticular osteochondroses occur at tendon attachments (Iselin's disease), ligament attachments (e.g., vertebral ring, epicondyles), or impact sites (e.g., Sever's Disease). The third type includes physal osteochondroses in long bones (e.g., tibia vara), and vertebrae (Scheuermann's Disease).

### **Freiberg's Infraction**

Osteochondrosis of the lesser metatarsal heads most commonly involves the second (68%) or third (27%) and fourth

(5%) metatarsal heads [5]. It is uncommon in the fifth metatarsal head. This condition was first described by Freiberg in 1914 in patients who presented with pain over the second metatarsal during activity.

### **Etiology and Pathomechanism**

There is debate in the literature as to the true etiology of the disease. Suggested etiologies include osteonecrosis of the metatarsal head, acute or repetitive trauma, stress overloading, and avascular necrosis following acute trauma [2, 6].

Freiberg's infraction is most prevalent in athletic adolescents, with predominance in females at a ratio of 5:1. The symptoms are often in the forefoot and centered at the joint involved. The pain is exacerbated by activity and weight-bearing and is described as metatarsalgia. There is often swelling around the involved joint and tenderness, which is exacerbated by range of motion of the joint, at the toe and surrounding tissue.

### **Symptoms**

The initial presentation is generally temporary and self-limiting, but can often return due to exacerbation by trauma, change in shoe wear or increased activity. Plain radiographs in early stages are normal, but MRI will demonstrate hypo-intensity of the second metatarsal head. As soon as 7 months later, radiographs will reveal progression with a flattened, condensed, fragmented metatarsal head [7, 8].

### **Diagnostics, Classification**

Smillie has classified Freiberg's infraction into five stages depending on metatarsal head degeneration [9]. In stage 1, there is a fissure in the epiphysis, with sclerosis between cancellous surfaces. In stage 2, there is absorption of cancellous bone on the proximal side, with sinking of the articular cartilage dorsally. Further absorption and sinking of the articular surface with bony projections medially and laterally represent stage 3, with exostoses on the dorsal proximal metatarsal head. In stage 4, the articular surface is sunken so far that restoration of normal anatomy is not possible, and there are associated fractures of the medial and lateral projections. Stage 5 includes arthrosis with flattening and deformity of the metatarsal head with intact plantar cartilage.

### **Therapy**

Non-operative management is rooted in decreasing foot pressure and unloading the affected metatarsal. Smillie reports that spontaneous healing may occur at any stage,

and restoration of a normal metatarsal head may be possible in stages 1–3. A short period of immobilization for 6–12 weeks is often indicated for symptom and pain control. Metatarsal padding and stiff-soled shoes can help decrease the stress on the affected metatarsal head. After symptomatic relief, patients can gradually return to weight-bearing activities. In general, reossification and signs of radiographic healing take between 2 and 3 years [2].

Surgical procedures available to treat Freiberg's disease can be divided into two categories. The first comprises procedures to correct abnormal biomechanics that can worsen the clinical course, such as corrective metatarsal osteotomy and core decompression. The second category comprises procedures that restore articular congruity in the later stages of the disease, including debridement, osteotomy, grafting and older procedures such as metatarsal head excision and arthroplasty that have fallen out of favor due to complications. The ideal method to treat late stage Freiberg's disease remains an unanswered question for surgeons. Dorsal wedge osteotomy is the most common surgical option for late stage disease. It involves excising the area of damaged articular cartilage by performing a closing wedge osteotomy while rotating the lower aspect of the metatarsal head dorsally. This allows the intact plantar cartilage to articulate with the proximal phalanx [6, 10–12].

### Rehabilitation and Back-to-Sports

Rehabilitation following surgical management of Freiberg's infraction involves a period of weight-bearing in a protective shoe or boot. If an osteotomy has been performed and stabilized by a percutaneous pin, this is removed at approximately the fourth postoperative week. If internal fixation has been used, the patient is protected from forced dorsiflexion at the metatarsophalangeal joint for approximately 4 weeks. Then the patient gradually begins to wear a supportive shoe and begins gentle passive/active dorsi- and plantarflexion movements. Activities such as running or jumping are restricted for 12–14 weeks.

### Prevention

No evidence-based preventative measures have been identified.

### Evidence

All available evidence on Freiberg's infraction is level III to level V. No information on the natural history of the disease exists, and clinical series reporting on the surgical outcomes are short in follow-up.

### Summary

1. The etiology of Freiberg's infraction is unknown.
2. The natural history of Freiberg's infraction has not been determined.
3. Surgical management is based on the Smillie's radiographic classification.
4. Surgical procedures that salvage the metatarsal head are preferred.

### Kohler's Disease

Kohler's disease is an osteochondrosis of the navicular. It most commonly affects children between the ages of 4 and 9 years, with a higher prevalence in boys. It presents as mid-foot pain with an antalgic gait. Symptoms increase with weight-bearing and activity, and the child often favors walking on the lateral aspect of the affected foot. Differential diagnoses include os navicularis (accessory navicular) and stress fracture of the navicular [13].

Plain radiographs can reveal sclerosis and flattening of the navicular. Radiographs can help to differentiate Kohler's disease from acute or stress navicular fractures. Although Kohler's can rarely present bilaterally, contralateral radiographs are useful for comparison.

Treatment is always conservative and includes rest, ice, and analgesics with immobilization in a short leg-walking cast for 4–6 weeks [2]. Duration of symptoms is shorter in patients treated in a cast compared to those treated with an arch support.

The majority of cases resolve without long-term sequelae. Subsequent radiographs reveal reorganization of the tarsal navicular, with a small percentage of cases resulting in chronic deformation of the bone after healing, which is most often asymptomatic.

### Osteochondrosis of the Sesamoids

Hallucal sesamoids are intratendinous ossicles that serve as insertion sites for the two muscle bellies of the flexor hallucis brevis plantar to the first metatarsal joint. The intersesamoid ligament and the plantar plate link the sesamoids together. The sesamoids' function is to dissipate weight-bearing pressure and act as a fulcrum to increase the mechanical force of the flexor hallucis brevis tendon. The tibial sesamoid is bipartite in 10% of the population, whereas the fibular sesamoid is rarely bipartite [14].

Although trauma is probably the most frequent cause, osteonecrosis with subsequent regeneration and excessive calcification may be present. Osteochondrosis of the medial sesamoid is more common and more frequent in women, especially between the ages of 18–25 years. Symptoms

consist of persistent localized pain and tenderness under the involved sesamoid [15, 16].

Axial radiograph or CT scan will show an enlarged or deformed sesamoid with irregular areas of increased bone density and fragmentation. Osteochondroses are usually unresponsive to conservative treatment, but all treatment modalities should be exhausted before a surgical option is considered.

Surgery includes total sesamoidectomy. Alternatively, two-thirds of either sesamoid can be removed without disturbing the ligamentous attachments; this may relieve pain while avoiding total sesamoidectomy. The surgical approach depends on which sesamoid is to be resected. For tibial sesamoidectomy, plantar medial incision can be used. The fibular sesamoid can be approached through a dorsal or plantar incision. The dorsal approach is technically demanding because of the depth of the sesamoids; however, with the plantar approach, the proximity of the neurovascular bundle to the first web space and the presence of the flexor hallucis longus tendon between the sesamoids make excision difficult [14].

---

## Apophysites

Apophysites are a subset of osteochondroses occurring at the bony attachment sites of musculotendinous units. An apophysis develops as an accessory ossification center and is evident on plain radiographs. Irritation at this attachment site and protuberance are representative of apophysitis.

## Sever's Disease

Osteochondrosis of the posterior calcaneal apophysis is an injury commonly found in adolescent athletes. Incidence rates vary from 2 to 16%. Symptoms present at the age of 11–15 years in boys and 8–13 years in girls [17, 18].

Apophysites are associated with increased tension or shortness of the Achilles tendon in the rapidly growing adolescent. This soft tissue change may have the potential to place an interim strain or traction on the apophysis at its insertion [19].

Diagnosis is by history and clinical examination. The physical activity reported to produce the highest levels of pain is frequent running and jumping [18]. Radiographs are not diagnostic, but can be used to rule out other causes of heel pain in children such as a calcaneal bone cyst, osteoid osteoma or tarsal coalition [20].

Conservative treatment is the only option, including ice, restriction of sports, stretching and eccentric strengthening exercises [21]. The available evidence indicates orthotics with a brim (heel cup) and medial arch support were more effective in reducing pain in sporting activities compared to heel raises or no treatment (Level 2 evidence). There was also support for heel raises reducing pain in sporting activities compared to no

treatment (Level 2 evidence). Taping also appeared to have some immediate pain relief benefit (Level 3 evidence) [19].

## Iselin's Disease

Iselin's disease refers to traction apophysitis of the tuberosity of the fifth metatarsal bone. The differential diagnosis includes avulsion fracture of the fifth metatarsal base, Jones fracture, metatarsal stress fracture, and os vesalianum pedis. The etiology is proposed to be an overuse injury caused by repetitive microtrauma and traction of tendons inserted to the base of the fifth metatarsal, namely peroneus brevis and peroneus tertius tendons. It occurs during periods of rapid growth and is more common in children involved in sports. The timing of ossification varies, with the mean appearance between the ages of 12 and 13 years, but it can be seen as early as 10 years in boys and 8 years in girls. Fusion is usually complete by 17–18 years [22].

Iselin's disease can present with lateral foot pain with weight-bearing and swelling at the base of the fifth metatarsal. On physical exam, tenderness is noted at the enlarged fifth metatarsal base, which can rub on shoe wear. Discomfort is reproduced with stressed plantar flexion and inversion. Plain radiographs show an enlarged apophysis compared with the opposite side, with irregular ossification and slight separation of the chondro-osseous junction [23].

Treatment includes immobilization in a short-leg walking cast or boot for 2 weeks. Repeat examination and x-rays follow this brief period of immobilization. If there are no radiographic signs of acute fracture (avulsion) and the patient is symptom free, immobilization can be discontinued; however, most patients require an average of 4 weeks of immobilization for acute symptom control, followed by progressive mobilization. A gradual progression of activity should be guided by the lack of pain and limping. An exercise program for acute ankle sprains can be used with emphasis on strengthening, stretching and proprioception. Iselin's disease may last several months to years, but it always resolves when ossification is complete [2].

---

## Haglund's Deformity

Haglund's deformity can be defined as a painful conflict between the back of the shoe and the posterosuperior margin of the calcaneus. The terminology related to this entity is, however, highly inconsistent. According to van Dijk, the term Haglund syndrome was coined by Pavlov in 1982 [24]. Other names followed: Haglund's syndrome, achillobursitis, achillobursitis, achillobursitis, retrocalcaneal bursitis and Haglund's disease, the last name being the most confusing as it has been already attributed to osteochondrosis of the accessory navicular bone

[24]. Popular names for this condition include: pump bump (from recurrent friction encountered from women's dress shoes), winter heel, knobby heel, calcaneal altus, highbrow heels and cucumber heel [25].

According to the terminology proposed by van Dijk et al., all previously mentioned definitions fall into the category of retrocalcaneal bursitis. The authors proposed abandoning previously used names [24]. In this chapter, however, we will use the term Haglund's deformity, as it emphasizes calcaneal abnormality as a focal point of the problem.

### Etiology and Pathomechanism

The pain in Haglund's deformity is generated by inflammation of the bursa separating the tendon and the posterior calcaneal wall – retrocalcaneal bursitis—and accompanying Achilles tendon lesion or tendinopathy. This is brought about by mechanical conflict between the Achilles tendon and the posterosuperior heel. Patrick Haglund correlated painful heel deformity with wearing constricting shoes, and this etiology certainly remains relevant. Often the conflict is aggravated by sports requiring repeated bouts of ankle dorsiflexion, for example running uphill [26]. The pathology of Haglund's deformity is therefore clearly different from other conditions of the Achilles tendon. The distinction, however, between Haglund's deformity and insertional tendinitis is not very clear, as the two often coexist.

Bony predisposition to Haglund's deformity can be viewed as a variation in calcaneal shape (i.e., enlargement of the posterosuperior corner of the calcaneus) or calcaneal position (i.e., high calcaneal pitch). It appears that the normally shaped calcaneus can be symptomatic when it is positioned vertically, and the abnormally shaped calcaneus can remain asymptomatic by virtue of a horizontal position [27, 28]. Consequently, it has been difficult to develop universal radiological parameters for Haglund's deformity. A great proportion of patients with pain consistent with Haglund's deformity do not meet the radiological criteria for this condition.

### Symptoms

Haglund's deformity is characterized by a posterior heel pain aggravated by walking and sports. Women are more frequently affected, and the condition is aggravated by shoe wear. Typical symptoms include skin irritation, local erythema and swelling. Pain is located on the superior eminence of the calcaneus, lateral to the Achilles tendon [29]. Patients with painful calcaneal eminence are younger (in the range of 15–30 years of age) than patients with insertional tendinitis.

### Diagnostics, Classification

The cornerstone of diagnosis remains weight-bearing lateral radiographs of the foot. Several radiographic parameters are

available to quantify Haglund's deformity. The two parameters with the highest sensitivity are parallel pitch lines (63% sensitivity) (Fig. 22.1A) and Chaveaux-Liet angle (73%) (Fig. 22.1B) [27]. Calcaneal pitch angle (Fig. 22.1C) is important for the identification of patients potentially requiring osteotomy. Other parameters, less useful for clinical decision making, include Philip and Fowler angle (Fig. 22.1D), total angle of Ruch (Fig. 22.1E), Steffensen and Evensen angle (Fig. 22.1F), Dijan angle (Fig. G), and Denis and Huber-Levernieux test (Fig. 22.1H) [25, 27, 30]. In many clinical situations, however, the patient is symptomatic, but the radiographic parameters are within normal ranges [25, 27].

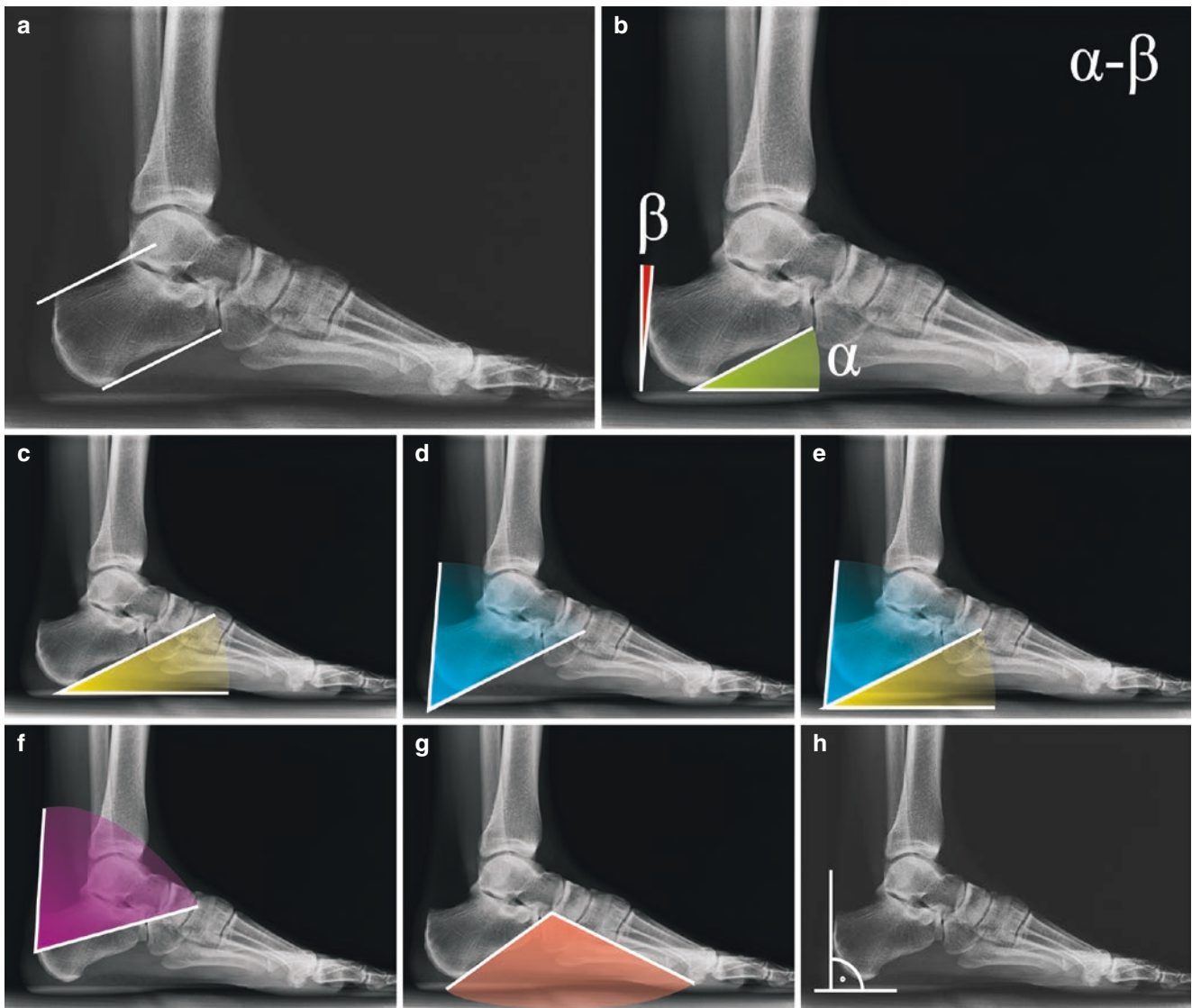
The parallel pitch lines of Henegan and Pavlov consist of two lines (Fig. 22.1A). The first line is defined by the inferior margin of the calcaneocuboid joint and plantar part of the calcaneal tuberosity. The second line, parallel to the first, is constructed through the posterior lip of the talar articular surface [27]. If the posterosuperior edge of the calcaneus remains below the second line, then the parallel pitch line is negative. If it protrudes above this line, then the parallel pitch line is positive, and this parameter reflects the height of the calcaneal prominence.

The Chaveaux-Liet angle is calculated as the difference between two angles,  $\alpha$  and  $\beta$ . The  $\alpha$  angle is consistent with the calcaneal pitch angle (described below). The  $\beta$  angle is formed by a vertical line and the line defined by the most posterior part of the calcaneal tuberosity and the posterosuperior corner of the calcaneus. The normal value of the the Chaveaux-Liet angle is less than  $12^\circ$ . A Chaveaux-Liet angle greater than  $18^\circ$  is considered a strong indication for operative treatment. The strength of this measurement is that it takes into account the position of the calcaneus and the shape of the posterior wall, thus reflecting both abnormal posterior morphology and abnormal inclination of the calcaneus [27, 29]. The Chauveaux-Liet angle is also significantly elevated in patients with insertional tendinitis, which may reflect a partial concurrence of these pathologies [29].

The calcaneal pitch angle (Fig. 22.1C) is the angle between the line defined by the inferior margin of the calcaneocuboid joint and the plantar part of the calcaneal tuberosity and a horizontal line. Normal values range from ten to  $30^\circ$ , with a more narrowly defined norm of  $15\text{--}17^\circ$  [27]. This angle takes into account the position of the calcaneus.

Retrocalcaneal bursitis can be detected with a simple parameter developed by van Sterkenburg et al. [31]. They observed that obliteration of the retrocalcaneal recess in lateral weight-bearing radiographs represented retrocalcaneal bursitis with 80% sensitivity and 98–100% specificity.

Other modalities, while not necessary for the diagnosis of Haglund deformity, can be used to confirm the diagnosis and rule out coexisting insertional Achilles tendinopathy



**Fig. 22.1** Radiographic parameters for assessing Haglund's deformity: (a) Henegan and Pavlov parallel pitch lines, (b) Chaveaux-Liet angle, (c) calcaneal pitch angle, (d) Philip and Fowler angle, (e) total angle of

Ruch, (f) Steffensen and Evensen angle, (g) Dijan angle, (h) Denis and Huber-Levernieux test

(Fig. 22.2). Ultrasound can detect retrocalcaneal bursitis [32] and reveal deformity of the bony shape [24]. Marrow edema in the posterior superior calcaneal eminence, evident by magnetic resonance imaging, is seen almost exclusively in symptomatic patients [29, 33].

### Therapy

Conservative treatment commences with activity and shoe wear modification (i.e., backless shoes) [34, 35]. The latter is especially important given the shoe-related nature of the condition. In runners, reduction in mileage and avoiding workouts on hills can be helpful [26]. The use of heel lifts and topical and/or oral anti-inflammatory agents is advised. The use of corticosteroid injection is controversial and brings

about the risk of tendon rupture. Physiotherapy, particularly with stretching and eccentric-loading exercises, is recommended [36–39].

Operative treatment is indicated in patients who do not respond to 3–6 months of conservative therapy [34]. First-line operative treatment consists of posterior bump removal (calcaneoplasty) with bursectomy. If indicated, Achilles tendon debridement and reconstruction, as well as repair of a longitudinal tear or re-insertion to the calcaneus, should be performed. As a second line of treatment, calcaneal osteotomy (vertical or closing wedge) may be considered. The optimal indication for calcaneoplasty is pure Haglund's deformity without insertional tendinopathy and with normal calcaneal pitch angle. The calcaneoplasty can be performed as open surgery or arthroscopically.

The available evidence for calcaneoplasty is mainly level IV studies, with one level III study [40] supporting the open intervention and one level II study [41] supporting the arthroscopic intervention.

Open calcaneoplasty is the standard surgery for symptomatic Haglund exostosis. The open surgery can be performed with good results using a medial [26], lateral [42], or central tendon-splitting approach [40, 43, 44]. The key to successful calcaneoplasty is removal of sufficient bone to prevent tendon impingement [42, 45, 46]. The drawback of open calcaneoplasty is the potential for skin breakdown and is thus contraindicated in patients with a compromised soft tissue envelope. With either a lateral or medial approach, there is a risk of under-resection on the opposite side of the calcaneus. Therefore a medial skin incision with a central

tendon-split approach provides the best overview and allows debridement of the Achilles tendon mucoid degeneration and solid reconstruction of the tendon by suturing or reinsertion to the calcaneus with anchors. Surgery is performed prone, and skin incisions are performed directly down to the paratenon without dissecting the skin. Once the central split of the tendon is complete, the posterior part of the calcaneus is removed. All degenerated tendon must be removed. If more than 50% of the insertion is compromised, re-insertion with anchors is necessary (Fig. 22.3).

The postoperative protocol for when only calcaneoplasty is performed, or with suturing of longitudinal Achilles tendon tears, begins with full weight-bearing in an Aircast walker with 30° plantar flexion for 2 weeks, then 20° plantar flexion for 2 weeks. Physiotherapy is initiated with lymphatic drain massage, then slight range of motion exercises are introduced, with strengthening after boot removal, i.e. at week seven. Following calcaneoplasty combined with Achilles tendon reattachment (anchors), the postoperative protocol begins with full weight-bearing in an Aircast walker with 30° plantar flexion for 2 weeks, then 20° plantar flexion for 2 weeks, and then 10° plantar flexion 2 weeks. Physiotherapy follows the same protocol: initiated with lymphatic drainage, slight range of motion exercises, and strengthening after boot removal.

In 2001, van Dijk reported on a group of 21 patients with Haglund's deformity treated by arthroscopic calcaneoplasty [46]. All patients met the radiographic criteria for Philip-Fowler angle and parallel pitch lines for Haglund's deformity. No complications were observed, and 19 patients had good and excellent results. The authors underscored the necessity of removing sufficient bone from the posterior calcaneus to achieve favorable results [46]. Their results were confirmed in 33 heels by Leitze et al. [41]. In 2007, Ortmann et al. reported on a prospective study of 30 heels that underwent three-portal arthroscopic calcaneoplasty [35]. They observed 29 good and excellent results, with one major complication, an Achilles tendon rupture that was primarily repaired with a



**Fig. 22.2** A 47 year-old patient, 10 months after arthroscopic calcaneoplasty. No improvement was observed and magnetic resonance imaging shows Achilles tendinopathy as a source of refractory symptoms



**Fig. 22.3** Open calcaneoplasty. A 49-year-old female with symptomatic Haglund exostosis and Achilles tendinopathy with tendon-split lesion. Conservative treatment was not successful. (a). Magnetic reso-

nance image, (b). intraoperative photograph, (c). intraoperative radiograph after exostosis removal (Courtesy of V. Valderrabano)

good result. Return to weight-bearing was at a mean of 4 weeks, and return to sports was on average at 12 weeks.

Arthroscopic calcaneoplasties have shown good results. However, they are more technically demanding, and the good results reported by experienced arthroscopists may not be easily transferable to casual arthroscopists. In a cadaver study comparing open and arthroscopic resection, the remaining rim of bone was found to be smaller after open resection [45].

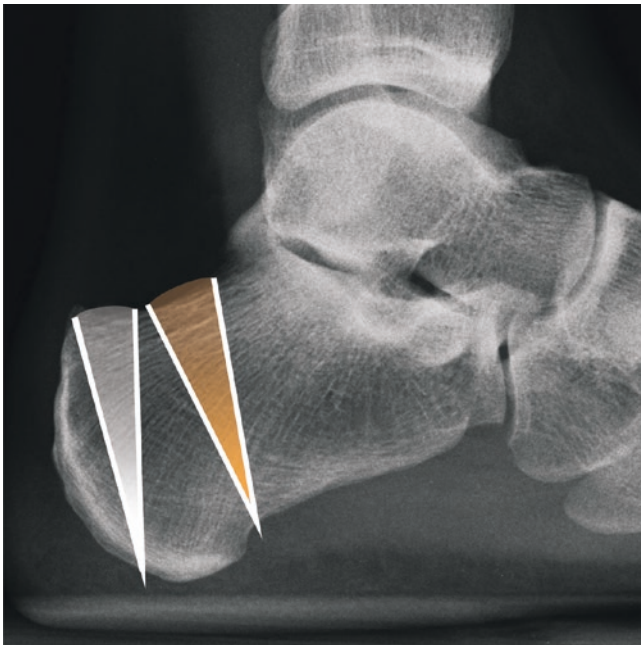
Arthroscopic calcaneoplasty is indicated in patients with pain related to retrocalcaneal bursitis. Pathology of the Achilles tendon itself is a relative contraindication for arthroscopic treatment, as it needs to be properly addressed for a favourable outcome. Two portals, located medial and lateral to the Achilles tendon, are used, and the bursa, synovitis and posterior part of the calcaneus are removed [47]. As the tendon is not affected, the postoperative regimen allows weight-bearing, shoe choice and sports resumption as tolerated.

Calcaneal osteotomy for Haglund's deformity has several theoretical advantages over calcaneoplasty. The Achilles insertion is spared from scarring, and the healing potential of the bone is greater than at the tendon insertion. It is also possible to globally reshape the posterior part of the calcaneus [48]. In North America, the Keck and Kelly osteotomy is more popular, whereas in Europe the osteotomy designed by Zadek is generally used. Apart from being more invasive than calcaneoplasty, the osteotomy techniques share the disadvantage of changing the biomechanics of Achilles pull. The lowered lever arm may lead to increased work of the Achilles and increased symptoms postoperatively. There is

scarce literature regarding calcaneal osteotomy, and all available evidence is Level IV.

Keck and Kelly proposed a closing wedge vertical calcaneal osteotomy in 1965 [49]. The best indication for this procedure is posterosuperior calcaneal prominence combined with elevated pitch angle in cases with and without insertional tendinopathy. After meticulous templating, surgery is performed with a lateral approach. A proximally based wedge with the plantar apex anterior to the Achilles insertion is removed (Fig. 22.4). The osteotomy is then closed and stabilized. The osteotomy exits the calcaneal tubercle dorsally to the weight-bearing part of the calcaneal tubercle and plantar aponeurosis insertion, such that no plantar structures are affected. The osteotomy tilts the posterior prominence of the heel anteriorly and slightly elevates the insertion of the Achilles tendon [48]. This addresses both the posterior prominence and the gastrocnemius tightness that is often observed in these patients. Postoperatively, 6 weeks of a non-weight-bearing regimen with removable cast is instituted. The original series reported by Keck and Kelly consisted of five heels in four patients. They observed one good, two fair and one poor result [49]. Miller and Vogel reported good results using the same technique in 18 heels [50].

The calcaneal osteotomy developed by Zadek in 1934 is a closing wedge osteotomy located anteriorly to the Achilles tendon insertion and anteriorly to the insertion of the plantar fascia (Fig. 22.4). It offers the advantage of being less technically demanding than the Keck and Kelly closing wedge osteotomy, at the cost of affecting plantar structures. Indications include insertional tendinosis, Haglund's deformity and retrocalcaneal bursitis. Using a lateral or medial approach, the proximally based wedge is removed, and the osteotomy is stabilised with staples. Postoperative treatment requires casting and a non-weight-bearing regimen. Good results have been reported with the Zadek osteotomy by Kelsall et al. in 23 cases [51], Taylor in 8 cases [52], and Maynou et al. in patients after failed calcaneoplasty [53]. Disadvantages of this procedure are the prolonged recovery time compared to simple calcaneoplasty and its influence on the Achilles tendon lever arm.



**Fig. 22.4** The difference between Keck and Kelly (gray) and Zadek (orange) osteotomy

### Rehabilitation and Back-to-Sports

Fast recovery following calcaneoplasty with sport resumption after an average of 12 weeks has been reported [35]. However, patients should be informed of possible lengthy recovery times. In slowly progressing cases, especially those with coexisting insertional tendinitis, recovery can take between 6 months and 2 years [54].

### Prevention

As the major causative factor of Haglund's deformity is constricting shoes, a suitable choice of shoe wear can be

viewed as a preventive measure. Stretching of the gastrocnemius-soleus complex is crucial to avoid Achilles contracture and excessive pressure on the calcaneal process [38, 39].

### Summary

1. Haglund's deformity of the posterior calcaneus is a predisposing factor for painful retrocalcaneal bursitis.
2. Numerous radiological parameters have been developed. Paralell pitch lines, Chaveaux-Liet angle and obliteration of retrocalcaneal space are most useful.
3. Operative options include calcaneoplasty (open or arthroscopic) and calcaneal osteotomy. Simple exostosis is well adressed with calcaneoplasty, while a vertically aligned calcaneus requires osteotomy.
4. In calcaneoplasty (open or arthroscopic), a key to success is sufficient removal of bony impingement. For successful calcaneal osteotomy, patient selection is critical.
5. For a good operative outcome, it is crucial to recognize and address coexisting insertional Achilles tendinitis.

### Conclusions

Clinicians should be familiar with conditions that affect the developing skeleton. These conditions, while frequently asymptomatic, can be exacerbated by increased physical activity. Diagnosis is usually made by a clinical picture of inflammation and radiographic evidence of fragmentation, osteonecrosis and joint collapse. Most of these conditions can be treated non-operatively. Symptom control, immobilization, activity modification, and appropriate rehabilitation can all play a role in a safe and timely return to sport.

### References

1. Siffert RS. Classification of the osteochondroses. *Clin Orthop Relat Res.* 1981;158:10–8.
2. Gillespie H. Osteochondroses and apophyseal injuries of the foot in the young athlete. *Curr Sports Med Rep.* 2010;9(5):265–8.
3. Douglas G, Rang M. The role of trauma in the pathogenesis of the osteochondroses. *Clin Orthop Relat Res.* 1981;158:28–32.
4. Duthie RB, Houghton GR. Constitutional aspects of the osteochondroses. *Clin Orthop Relat Res.* 1981;158:19–27.
5. Binek R, Levinsohn EM, Bersani F, Rubenstein H. Freiberg disease complicating unrelated trauma. *Orthopedics.* 1988;11(5):753–7.
6. Talusan PG, Diaz-Collado PJ, Reach Jr JS. Freiberg's infraction: diagnosis and treatment. *Foot Ankle Spec.* 2014;7(1):52–6.
7. Helal B, Gibb P. Freiberg's disease: a suggested pattern of management. *Foot Ankle.* 1987;8(2):94–102.
8. Katcherian DA. Treatment of Freiberg's disease. *Orthop Clin North Am.* 1994;25(1):69–81.
9. Smillie IS. Treatment of Freiberg's infraction. *Proc R Soc Med.* 1967;60(1):29–31.
10. Lee HJ, Kim JW, Min WK. Operative treatment of Freiberg disease using extra-articular dorsal closing-wedge osteotomy: technical tip and clinical outcomes in 13 patients. *Foot Ankle Int.* 2013;34(1):111–6.
11. Kim J, Choi WJ, Park YJ, Lee JW. Modified Weil osteotomy for the treatment of Freiberg's disease. *Clin Orthop Surg.* 2012;4(4):300–6.
12. Kilic A, Cepni KS, Aybar A, Polat H, May C, Parmaksizoglu AS. A comparative study between two different surgical techniques in the treatment of late-stage Freiberg's disease. *Foot Ankle Surg.* 2013;19(4):234–8.
13. Waugh W. The ossification and vascularisation of the tarsal navicular and their relation to Kohler's disease. *J Bone Joint Surg Br.* 1958;40-B(4):765–77.
14. Richardson EG. Hallucal sesamoid pain: causes and surgical treatment. *J Am Acad Orthop Surg.* 1999;7(4):270–8.
15. Jahss MH. Spontaneous hallux varus: relation to poliomyelitis and congenital absence of the fibular sesamoid. *Foot Ankle.* 1983;3(4):224–6.
16. Leventen EO. Sesamoid disorders and treatment. An update. *Clin Orthop Relat Res.* 1991;269:236–40.
17. Wiegerinck JI, Yntema C, Brouwer HJ, Struijs PA. Incidence of calcaneal apophysitis in the general population. *Eur J Pediatr.* 2014;173(5):677–9.
18. Suzue N, Matsuura T, Iwame T, Hamada D, Goto T, Takata Y, Iwase T, Sairyō K. Prevalence of childhood and adolescent soccer-related overuse injuries. *J Med Investig JMI.* 2014;61(3–4):369–73.
19. James AM, Williams CM, Haines TP. Effectiveness of interventions in reducing pain and maintaining physical activity in children and adolescents with calcaneal apophysitis (Sever's disease): a systematic review. *J Foot Ankle Res.* 2013;6(1):16.
20. Kose O, Celiktas M, Yigit S, Kisin B. Can we make a diagnosis with radiographic examination alone in calcaneal apophysitis (Sever's disease)? *J Pediatr Orthop B.* 2010;19(5):396–8.
21. Elengard T, Karlsson J, Silbernagel KG. Aspects of treatment for posterior heel pain in young athletes. Open access *J Sports Med.* 2010;1:223–32.
22. Deniz G, Kose O, Guneri B, Duygun F. Traction apophysitis of the fifth metatarsal base in a child: Iselin's disease. *BMJ Case Rep.* 2014;2014.
23. Lehman RC, Gregg JR, Torg E. Iselin's disease. *Am J Sports Med.* 1986;14(6):494–6.
24. van Dijk CN, van Sterkenburg MN, Wiegerinck JI, Karlsson J, Maffulli N. Terminology for Achilles tendon related disorders. *Knee Surg Sports Traumatol Arthrosc.* 2011;19(5):835–41.
25. Lu CC, Cheng YM, Fu YC, Tien YC, Chen SK, Huang PJ. Angle analysis of Haglund syndrome and its relationship with osseous variations and Achilles tendon calcification. *Foot Ankle Int.* 2007;28(2):181–5.
26. Sammarco GJ, Taylor AL. Operative management of Haglund's deformity in the nonathlete: a retrospective study. *Foot Ankle Int.* 1998;19(11):724–9.
27. Singh R, Rohilla R, Siwach RC, Magu NK, Sangwan SS, Sharma A. Diagnostic significance of radiologic measurements in posterior heel pain. *Foot (Edinb).* 2008;18(2):91–8.
28. McCabe LR. Understanding the pathology and mechanisms of type I diabetic bone loss. *J Cell Biochem.* 2007;102(6):1343–57.
29. Sundararajan PP, Wilde TS. Radiographic, clinical, and magnetic resonance imaging analysis of insertional Achilles tendinopathy. *J Foot Ankle Surg.* 2014;53(2):147–51.
30. Kang S, Thordarson DB, Charlton TP. Insertional Achilles tendinitis and Haglund's deformity. *Foot Ankle Int.* 2012;33(6):487–91.
31. van Sterkenburg MN, Muller B, Maas M, Sierevelt IN, van Dijk CN. Appearance of the weight-bearing lateral radiograph in retrocalcaneal bursitis. *Acta Orthop.* 2010;81(3):387–90.
32. Lienau J, Schell H, Duda GN, Seebeck P, Muchow S, Bail HJ. Initial vascularization and tissue differentiation are influenced by fixation stability. *J Orthop Res.* 2005;23(3):639–45.



33. Haims AH, Schweitzer ME, Patel RS, Hecht P, Wapner KL. MR imaging of the Achilles tendon: overlap of findings in symptomatic and asymptomatic individuals. *Skeletal Radiol*. 2000;29(11):640–5.
34. Irwin TA. Current concepts review: insertional achilles tendinopathy. *Foot Ankle Int*. 2010;31(10):933–9.
35. Ortmann FW, McBryde AM. Endoscopic bony and soft-tissue decompression of the retrocalcaneal space for the treatment of Haglund deformity and retrocalcaneal bursitis. *Foot Ankle Int*. 2007;28(2):149–53.
36. Giannoudis PV, Einhorn TA, Marsh D. Fracture healing: a harmony of optimal biology and optimal fixation? *Injury*. 2007;38 Suppl 4:S1–2.
37. Laporte S, Mismetti P. Epidemiology of thrombotic risk factors: the difficulty in using clinical trials to develop a risk assessment model. *Crit Care Med*. 2010;38(2 Suppl):S10–7.
38. Calori GM, Phillips M, Jeetle S, Tagliabue L, Giannoudis PV. Classification of non-union: need for a new scoring system? *Injury*. 2008;39 Suppl 2:S59–63.
39. Caprini JA, Arcelus JI, Reyna JJ. Effective risk stratification of surgical and nonsurgical patients for venous thromboembolic disease. *Semin Hematol*. 2001;38(2 Suppl 5):12–9.
40. Anderson JA, Suero E, O'Loughlin PF, Kennedy JG. Surgery for retrocalcaneal bursitis: a tendon-splitting versus a lateral approach. *Clin Orthop Relat Res*. 2008;466(7):1678–82.
41. Leitze Z, Sella EJ, Aversa JM. Endoscopic decompression of the retrocalcaneal space. *J Bone Joint Surg Am*. 2003;85–A(8):1488–96.
42. Huber HM. Prominence of the calcaneus: late results of bone resection. *J Bone Joint Surg Br*. 1992;74(2):315–6.
43. Fridrich F. Tendon-splitting approach for the surgical treatment of Haglund's deformity and associated condition. Evaluation and results. *Acta Chir Orthop Traumatol Cech*. 2009;76(3):212–7.
44. Johnson KW, Zalavras C, Thordarson DB. Surgical management of insertional calcific achilles tendinosis with a central tendon splitting approach. *Foot Ankle Int*. 2006;27(4):245–50.
45. Lohrer H, Nauck T, Dorn NV, Konerding MA. Comparison of endoscopic and open resection for Haglund tuberosity in a cadaver study. *Foot Ankle Int*. 2006;27(6):445–50.
46. van Dijk CN, van Dyk GE, Scholten PE, Kort NP. Endoscopic calcaneoplasty. *Am J Sports Med*. 2001;29(2):185–9.
47. Russell MW, Taylor DC, Cummins G, Huse DM. Use of managed care claims data in the risk assessment of venous thromboembolism in outpatients. *Am J Manag Care*. 2002;8(1 Suppl):S3–9.
48. Boffeli TJ, Peterson MC. The Keck and Kelly wedge calcaneal osteotomy for Haglund's deformity: a technique for reproducible results. *J Foot Ankle Surg*. 2012;51(3):398–401.
49. Keck SW, Kelly PJ. Bursitis of the posterior part of the heel; evaluation of surgical treatment of eighteen patients. *J Bone Joint Surg Am*. 1965;47:267–73.
50. Miller AE, Vogel TA. Haglund's deformity and the Keck and Kelly osteotomy: a retrospective analysis. *J Foot Surg*. 1989;28(1):23–9.
51. Kelsall KN, Chapman WA, Sangar A, Farrar MJ, Taylor HP. Zadek's calcaneal osteotomy for insertional achilles pathology. *Bone Joint J Orthop Proc Suppl*. 2014;296–B(Suppl 2):19.
52. Taylor GJ. Prominence of the calcaneus: is operation justified? *J Bone Joint Surg Br*. 1986;68(3):467–70.
53. Maynou C, Mestdagh H, Dubois HH, Petroff E, Elise S. Is calcaneal osteotomy justified in Haglund's disease? *Rev Chir Orthop Reparatrice Appar Mot*. 1998;84(8):734–8.
54. Brunner J, Anderson J, O'Malley M, Bohne W, Deland J, Kennedy J. Physician and patient based outcomes following surgical resection of Haglund's deformity. *Acta Orthop Belg*. 2005;71(6):718–23.

Yousef Alrashidi, Hasan N. Alsayed, Hamza M. Alrabai, and Victor Valderrabano

## Abstract

The rate of occurrence of Posterior Tibial Tendon (PTT) injuries and insufficiency in sports is not yet clear based on the available literature. However, literature review may point to a link between some sports with specific lesions such as PTT dislocation, PTT tear, and PTT Insufficiency (PTTI) stage I or II. High index of suspicion is needed to diagnose and treat those lesions to prevent their progression to advanced stages.

## Keywords

Calcaneal osteotomy • Flatfoot surgery • Flatfoot in sport • Posterior tibial tendon insufficiency • Posterior tibial tendon dislocation • Posterior tibial tendon injury • PTT • Pes planovalgus • Pes planus • Unhealed ankle sprain

## Acute PTT Injuries

### Introduction

Posterior Tibial Tendon (PTT) is a vital structure that highly contributes to the dynamic and passive stability of medial aspect of the ankle joint and hindfoot [1]. PTT muscle originates from the lower part of the deep posterior crural compartment. Just 6 cm above the medial malleolus, PTT is

covered by a synovial sheath for 7–9 cm which continues distally towards its navicular insertion [1]. Acute injury to PTT could result from direct or indirect trauma during athletic activities. Such a trauma may lead to partial or complete rupture of the tendon. PTT could dislocate and also may become incarcerated in the ankle or distal tibiofibular space preventing successful closed reduction of associated bony injury [2, 3].

Y. Alrashidi, MBBS, SB-Orth (✉)

Orthopedic Surgery Department, Taibah University,  
College of Medicine, Almadinah Almunawwrah, Saudi Arabia

Orthopaedic Department, SWISS ORTHO CENTER,  
Schmerzklinik Basel, Swiss Medical Network, Basel, Switzerland  
e-mail: [yalrashidi@gmail.com](mailto:yalrashidi@gmail.com)

H.N. Alsayed, MBBS, SB-Orth  
Department of Orthopaedics, University of Dammam,  
Dammam, Saudi Arabia  
e-mail: [hsayed@uod.edu.sa](mailto:hsayed@uod.edu.sa)

H.M. Alrabai, MBBS, SB-Orth  
College of Medicine, King Saud University, Riyadh, Saudi Arabia  
e-mail: [hamzarabai@gmail.com](mailto:hamzarabai@gmail.com)

V. Valderrabano, MD, PhD  
Orthopaedic Department, SWISS ORTHO CENTER,  
Schmerzklinik Basel, Swiss Medical Network, Basel, Switzerland  
e-mail: [vvalderrabano@gsmn.ch](mailto:vvalderrabano@gsmn.ch)

### Incidence

No clear consensus found in literature about incidence rate of acute PTT injuries in population [1]. It has been reported that 58.5% of initial PTT dislocations were triggered by an athletic activity [4]. PTT dislocations were reported in common with some sport activities (Table 23.1) [1, 5–9]. Deland and Hamilton reported five cases with partial PTT tendon tears in dancers [6].

### Aetiology and Pathomechanism

Lacerations of PTT may follow some sport activities such as ice skating (with high-speed), which may be associated with

**Table 23.1** List of sports which may be associated with risk of PTT dislocation

Motorcycling [7]
Dancing [6]
Tae-Kwon-Do [9]
Running [4]
Rock-climbing [8]
Snowboarding [5]

traumatic lesions of adjacent bony or soft tissue structures [1]. Acute ruptures may accompany ankle fractures resulting from pronation-external rotation (PER) pedal injury, and to a less extent from direct ankle trauma [10, 11]. Spontaneous ruptures may occur in elderly; obese patients; in patients with co-morbid disorders such as diabetes mellitus (DM), hypertension, or rheumatoid arthritis; or in patients on corticosteroid therapy [11]. However, they also may occur in athletes after a corticosteroid injection, typically happening later on with an eversion trauma.

Acute PTT dislocation is believed to be, commonly, a result of excessive dorsiflexion or plantar flexion of the tibiotalar joint in an inverted foot with simultaneous PTT contraction [12]. Depending on several case series, factors that predispose to dislocation include flexor retinaculum incompetency due to tear, avulsion, laxity, or the elevation over a “retinacular-periosteal sleeve”. Other factors include shallowness of the retro-malleolar groove, hypoplasia of the malleolar sulcus, or as a consequence of repetitive corticosteroid injection, tarsal tunnel surgical decompression, or old medial malleolar injury [5, 13, 14].

In cases of ankle joint fracture dislocation, PTT might dislocate and get entrapped into the joint or in the distal tibiofibular syndesmotoc space preventing anatomical closed reduction of such a bony injury [2, 3, 15].

Moreover, we think, PTT injury may follow surgical treatment (iatrogenic) of a medial malleolar fracture where PTT may get entrapped into the fracture site, penetrated by misplaced screw, or damaged by an errant retractor placement.

## Symptoms

Acute closed PTT injuries tend to be missed due to failure to examine patients adequately in the presence of painful and swollen ankle [5, 11]. Adding to the rarity of those injuries, they may be misdiagnosed as medial ankle sprain [5]. In a systematic review of PTT dislocations, Lohrer and Nauck reported that about 53 % of cases were overlooked at the initial hospital’s visit [4]. Attention to the description of injury events may give a clue for predicted soft tissue injuries including PTT. So, diagnosis of such an injury is believed to require high index of suspicion.

Patient with PTT injury may present with a retro-malleolar swelling, variable pain and difficulty in bearing weight. Clinically, the picture may mimic medial ankle sprain, Sudeck’s sympathetic syndrome, or tibial nerve neuralgia [12, 16].

## Diagnostics

Any patient with sport-related injury should undergo a primary systematic assessment depending upon the mechanism of injury and presence of other body system involvement.

Suspicion of PTT injury should be raised in patients with significant closed ankle injury associated with medial symptoms or cases of “non-healed medial ligamentous sprain”, which did not respond to conservative treatment. In particular, PTT dislocation patient may report a feeling of a ‘pop’ at the time of injury, “recurrent snapping” and/or feeling of a cord-like structure over the medial malleolus [4]. Careful local examination, including neurovascular structures, should be carried out. Further diagnostic workup, as by MRI, may be warranted in equivocal cases.

PTT injury should be differentiated from flexor hallucis longus (FHL) tenosynovitis in sporty people especially dancers [6].

Marcus and Pfister suggested that the presence of the following clinical findings may raise the suspicion of PTT rupture: Eversion ankle injury; diffuse medial ankle pain and/swelling; flexible, asymmetric flatfoot and forefoot pronation; and loss of PTT function on clinical examination (more specific) [17].

Adequate plain X-rays are essential to rule out associated bony injuries. However, plain radiographs are not specific for acute tibialis posterior injury. According to Lohrer and Nauck, medial malleolar chip fracture may be seen in only 14.7 % of PTT dislocations [4].

MRI imaging is known to be highly sensitive and specific in detecting PTT injuries. Ultrasound imaging is useful in evaluation of the integrity and relationship of PTT to other structures in the retromalleolar groove, but excessive subcutaneous fat and post-traumatic swelling may obscure the view. Furthermore, ultrasound is an operator dependent modality [18, 19].

## Therapy

After administration of appropriate antibiotic and tetanus prophylaxis, significant open injuries of the ankle joint should undergo surgical exploration in the operating room. Repair of tendon is essential to prevent later development of tibialis posterior dysfunction, and it is important to check the integrity of flexor sheath as it plays an important role in keeping the tendon in normal track and position.

In literature overview, the trend to treat PTT dislocations or entrapment non-operatively is not sufficiently supported. However, Deland and Hamilton reported successful conservative treatment of partial PTT tear in a dancer with cavus foot by immobilisation and physiotherapy protocol and on the other hand, another three cases which did not respond to conservative treatment and subsequently required surgical intervention [6].

Up to our knowledge, there is no scientific evidence supporting surgical correction of hindfoot malalignment at the same time of repair or reconstruction of acute PTT ruptures or tears. Surgical treatment of PTT dislocations has to address flexor retinaculum and retromalleolar groove abnormalities. Such a treatment may consist of direct repair or reefing of the retinaculum. Augmentation is possible with surrounding soft tissues (e.g. deltoid ligament and periosseum), and/or retromalleolar groove deepening [4].

## Chronic PTT Insufficiency

### Introduction

PTT dysfunction or acquired flatfoot is a very common progressive PTT lesion that may give rise to several sequelae such as medial chronic ankle instability, Spring ligament injury, plantar fasciitis, subfibular lateral bony impingement, syndesmotic insufficiency, subtalar osteoarthritis, lateral valgus ankle osteoarthritis, Lisfranc joint osteoarthritis, and Achilles tendon tendinopathy.

### Incidence

PTTI is common among athletes. The authors believe that most of PTTI cases in sport are of stage I Johnson & Strom (tenosynovitis). According to cross-sectional studies on special Olympic athletes, it has been found that 38.1–38.5 % of subjects had flatfoot [20].

### Aetiology and Pathomechanism

It seems that PTT dysfunction is a multifactorial disorder and could be linked to patient's specific aetiology [21]. Several theories have been suggested to elaborate PTT dysfunction pathogenesis such as hypo-vascular, vascular, inflammatory, structural, hormonal, and traumatic [10, 22–25].

Stage I PTTI (tenosynovitis) in sport patients are mainly related to athletic activities without having an appropriate protection and suitable shoes, which may lead to irritation of the tendon. For instance, it has been found that it may occur

in ballet dancers due to overuse of PTT (sickling in a valgus position) [26].

Frey et al. suggested the presence of a relative hypovascular PTT segment that makes the tendon more liable to degeneration. They found such a segment is situated dorsal and distal to the medial malleolar level; and anatomically, it is more subject to chronic irritation and micro-injury from any medial malleolar bony prominence [25, 27]. According to Peterson et al. this area is confined to PTT retromalleolar part that is rich with fibrocartilagenous tissue [23].

Inflammatory process in the form of seronegative tenosynovitis is thought to be more common in young adults, which may predispose to tenosynovitis and development of pes planovalgus deformity [28].

Bridgeman et al. examined a possible link between estrogen hormone receptors and development of PTT insufficiency in a limited number of subjects, and the study did not show any significant association. So, there is a need for larger cohort studies to prove or disprove this association [29].

In an epidemiological analytical study of 67 adults with chronic PTT rupture, Holmes and Mann found that 52 % of subjects had positive history of diabetes mellitus (DM), high blood pressure, or high body mass index (BMI); also, they found a significant correlation between PTT rupture and both high blood pressure and high body mass index [24].

Development of pes planus may be a stem from a missed PTT disruption in association with ankle fractures (especially pronation-external rotation) or eversion ankle sprain [10, 17].

Degenerative changes observed in PTT dysfunction cases might be a result of change of collagen distribution and composition leading to a reduction in the mechanical resistance of tendon tissue and consequently affecting its compliance [17, 22].

Pisani suggested that the overall pathological image of degenerative flatfoot is related to “degenerative glenopathy of coxa pedis (talo-calcaneo-navicular joint)”. Further, presence of accessory navicular bone between PTT and glenoid of coxa pedis may explain the evolution of microtraumatic lesions on glenoid cartilage. So, excision of accessory navicular as soon as possible may help prevent development of such lesions [30, 31].

In our opinion, the presence of concomitant **spring ligament** (plantar calcaneonavicular ligament) lesion should be kept in mind in all patients undergoing any kind of flatfoot reconstruction as missing treatment of such a lesion may cause failure of surgical treatment. Owing to the fact that spring ligament is an important contributor to the stability of the medial longitudinal arch, Spring ligament affection may happen throughout PTT disease course. [32]. Spring ligament lesions can be brought about by PTT pathology and vice versa. In other words, Pisani suggested that the presence of fibrocartilagenous tissue in parts of glenoid of “coxa pedis

(talo-calcaneo-navicular joint)“ with a “limited repairing capacity”, which makes glenoid more liable to frequent microtrauma; may explain primary degeneration of spring ligament and later PTT affection [30, 31, 33]. In recent scientific literature in English, several pes planovalgus cases with spring ligament lesion and intact PTT were reported. So, early diagnosis of isolated spring complex lesion as the main reason of the deformity may help avoid unnecessary surgical interventions [34, 35].

## Symptoms

Patients with PTT dysfunction may present with pain and/or swelling that is localised over medial ankle joint. Symptoms may interfere with physical activities such as sport, and may make patients change into low impact activities [36].

In advanced stages of the disease, patients may also suffer from lateral ankle pain that may be attributed to development of subfibular impingement or sinus tarsi impingement. Nevertheless, the actual aetiology of this pain is not yet fully proven [37]. Pathobiomechanically, the authors believe that valgus hindfoot cause valgisation of the talus leading to medial talar impingement against medial malleolus. Consequently, that cause frequent injury to medial aspect articular cartilage of talus giving rise to early ankle degeneration and/or medial talar osteochondral lesions development. Feeling of giving way or instability at the ankle joint may follow elongation of the deltoid ligament or syndesmotism insufficiency, both which may result from hindfoot valgus malalignment. In addition, longstanding abduction of the foot and frequent unbalanced load on subluxed Lisfranc joint leads to degeneration, which manifests later with pain at the midfoot especially at the second and third metatarsal bases.

## Classification

Several classifications have been proposed over the last few decades to help understand different bony and soft tissue changes in PTT dysfunction and guide selection of the best treatment options [36, 38–41]. The most commonly used clinical classification of PTT dysfunction was first described by Johnson and Strom in 1989 as three stages. Several years later, a fourth stage was added by Myerson, which involves tibio-talar articulation pathology. The four stages are illustrated in Table 23.2 [36, 38].

Another detailed classification was proposed by Bluman et al. that is believed to be very helpful to surgeons to clinically and radiologically stratify PTT pathology and plan appropriate treatment (Table 23.3) [39]. A classification system (RAM: rearfoot, ankle, and Midfoot) was suggested by

Raikin et al. in 2012, which links pathological changes related to PTT dysfunction in the hindfoot, ankle joint and midfoot [41].

## Diagnostics

Cozen has rightly said that “ unfamiliarity with the syndrome can be embarrassing to the physician and painful for the patient” when he reported his PTT dysfunction case series in 1965. **PTTI diagnosis is based mainly on history taking and clinical examination.** After reviewing patient’s history, the clinician has to be aware of importance of clinical examination in picking up early stages of PTT dysfunction to prevent its progression and choose the appropriate treatment modality adapted to the stage of the disease. Different clinical presentations and radiological signs correlated to disease stages are illustrated in Tables 23.2 and 23.3. The foot should be inspected for any forefoot, midfoot, and/or hindfoot deformities. “ Too many toes” sign represents disease stage II and higher, and indicates significant forefoot abduction. Inability or difficulty to perform **single heel raise test** is an indicator of weakness of PTT. PTT normally varisates and locks the hindfoot, allowing for pull of calcaneus by the Achilles tendon [36]. Hintermann and Gächter described and validated a first metatarsal rise test that was found to be positive in all PTT dysfunction patients involved in their study. This test can be performed in standing or sitting position. The examiner rotates passively the leg and notice the first metatarsal head relationship with the ground. The test is considered positive if metatarsal head rises off the ground [42].

Adequate weight bearing radiographs of foot and ankle have to be obtained to assess the stage of the disease. On the antero-posterior (AP) view of foot, talo-navicular joint alignment and coverage angle can be assessed. On the lateral view, lateral talo-metatarsal angle and medial arch height can be appreciated. Hindfoot alignment view/Saltzman view is used to assess hindfoot alignment [43]. Ankle radiographs (AP, lateral, and mortise views) are used to rule out arthritic changes and any associated deformity. Harris calcaneal view may be needed to detect subtalar coalition (i.e. as an important differential diagnosis). According to Malicky et al., the presence of cystic lesions in distal fibula or sinus tarsi area on plain radiographs may suggest coexistent impingement [37].

CT is very helpful in assessing the bony anatomical changes, while MRI is highly sensitive and specific in the diagnosis and evaluation of PTTI soft tissue changes. Yao et al. found that MRI is highly specific (up to 100%) but with moderate sensitivity (54–77%) in picking up tears of superomedial component of spring ligament. Adding to that, MRI was not reliable in detecting plantar component tears due to

**Table 23.2** Classification of posterior tibial tendon insufficiency (PTTI) according to Johnson and Strom, and Myerson

	Johnson-Strom	Johnson-Strom	Johnson-Strom	Myerson
PTTI Grade	Grade 1	Grade 2	Grade 3	Grade 4
Posterior Tibial Tendon	Tenosynovitis or degeneration, or both	Elongation, degeneration, partial rupture	Elongation, degeneration, rupture	Elongation, degeneration, rupture
Deformity: Pes planovalgus abductus and supinatus	Absent	Flexible, correctable Planovalgus	Fixed, uncorrectable Planovalgus	Fixed, uncorrectable Planovalgus
Pain	Medial	Medial, lateral, or both	Medial, lateral, or both	Medial, lateral, or both
Too-many-toes sign	Negative	Positive	Positive	Positive
1st Met. rise sign	Negative	Positive	Positive	Positive
Ankle Deformity	None	None	None	Yes

its orientation complexity [44, 45]. Toye et al. reported 79% sensitivity of MRI in detecting surgically visible spring tears as full thickness discontinuity [46]. A prevalence of 81.8–92% of spring ligament injury were reported in association with chronic PTT lesions [47, 48].

The ultrasonic picture of stage I PTT dysfunction was found to be the presence of hypoechoic enlargement of tendon sheath (minimum of 5.9 mm) and hypoechoic tendon distension [49]. In a prospective blinded cadaveric study, Girling et al. found the sensitivity of dynamic ultrasound and MRI imaging in detecting PTT longitudinal tears were 69% and 73%, respectively; and specificity of 81% and 69%, respectively. However, this study could be biased by the presence of previous PTT degeneration in old age specimens, and surgically-induced tears may not reflect radiological signs in living people such as oedema and bleeding [50].

## Therapy

All patients with PTTI should undergo adequate conservative treatment for at least 3–6 months before considering surgical options.

In recent literature, there were a number of studies that investigated effectiveness of non-operative measures on early stages of the disease such as orthotics, bracing, and physiotherapy; but with variable methodology [51–55].

Immobilisation using a splint, stabilizing shoe, or walker must be considered in stage I PTTI to prevent progression of the disease. The ideal position of immobilisation is a slight inversion and plantar-flexion for 4–6 weeks [56]. Non-steroidal anti-inflammatory drugs (NSAIDs) have been found to be effective as analgesic in acute disease but not proven yet to interfere with the inflammatory phases of tendinopathy [57]. Injection of corticosteroids may be effective as anti-inflammatory agent but carries a high risk of tendon rupture. In case of no remarkable response to conservative treatment, surgical intervention in the form of tenosynovectomy may be warranted.

The aim of bracing in early stages of PTTI appears to be maintaining the correction of the deformity and subsequently diverting the high loads off the diseased PTT during ambulation [51].

It has been observed that all long muscle groups of foot and ankle have a significant weakness in stages I & II PTTI. So, a structured physiotherapy program, in addition to the use of appropriate varisating insoles or braces, is preferable in those stages to achieve satisfactory outcome and avoid possible surgical intervention complications [54].

Alvarez et al. described a structured PTT rehabilitation program for PTTI stages I & II, and reported a clinical improvement in 89% of subjects. Such a program involves patient education and assessment and orthotic prescription. The physiotherapy protocol for PTT rehabilitation consisted of three phases that can be performed under supervision over 6 days and phase II is repeated if discharge criteria have not been met. The protocol involved gradual participation in focused exercises such as heel rises, sole-to-sole, and active range of motion exercises [54].

In a randomised controlled study, it has been found that a combination of orthotics, stretching exercises, and progressive PTT resistive exercises is very effective in improving function and pain relief in patients with disease stages I and II [58]. In a case series study, a physiotherapy structured program for 12 weeks was found to be effective in combination with an orthotic support in a cohort of 10 subjects with stage I flatfoot. In the latter study, stretching exercises of gastrocnemius and soleus muscles were started 2 weeks earlier than beginning of formal PTT eccentric exercises, and then continue in parallel with PTT exercises for 10 weeks to help decrease the load on PTT and provide more flexibility to gastroc-soleus complex throughout the whole program [59]. However, there is a need for more high-level trials to prove effectiveness and determine the best physiotherapy protocol.

O' Connor et al. in a retrospective comparative investigation, suggested that successful non-operative treatment can be predicted in non-obese patients without prior corticosteroid injection or orthotics use, and short duration before

**Table 23.3** Classification of PTTI according to Bluman et al. [39]

Stage	Substage	Most characteristic clinical findings	Most characteristic radiographic findings	Treatment
I	A	Normal anatomy	Normal	Immobilization, NSAIDs, Cryotherapy, Orthoses, and/or Tenosynovectomy
		Tenderness along PTT		± Systemic disease-specific pharmacotherapy
	B	Normal anatomy	Normal	Immobilization, NSAIDs, Cryotherapy, Orthoses, and/or Tenosynovectomy
		Tenderness along PTT		
	C	Slight HF valgus	Slight HF valgus	Immobilization, NSAIDs, Cryotherapy, Orthoses, and/or Tenosynovectomy
		Tenderness along PTT		
II	A1	Supple HF valgus	HF valgus	Orthoses
		Flexible forefoot varus	Meary's line disruption	MDCO
		Possible pain along PTT	Loss of calcaneal pitch	TAL or Strayer and FDL transf. if deformity corrects only with ankle plantarflexion
	A2	Supple HF valgus	HF valgus	Orthoses
		Fixed forefoot varus	Meary's line disruption	MDCO and FDL transf.
		Possible pain along PTT	Loss of calcaneal pitch	Cotton osteotomy
	B	Supple HF valgus	HF valgus	Orthoses
		Forefoot abduction	Talonavicular uncovering	MDCO and FDL transf.
			Forefoot abduction	Lateral column lengthening
	C	Supple HF valgus	HF valgus	MDCO and FDL transf.
		Fixed forefoot varus	First TMT plantar gapping	Cotton osteotomy or medial column fusion
		Medial column instability		
		First ray dorsiflexion with HF correction		
		Sinus tarsi pain		
	III	A	Rigid HF valgus	Subtalar joint space loss
Pain in sinus tarsi			HF valgus	Triple arthrodesis
			Angle of Gissane sclerosis	
B		Rigid HF valgus	Subtalar joint space loss	Custom bracing if not surgical candidate
		Forefoot abduction	HF valgus	Triple arthrodesis ± lateral column lengthening
		Pain in sinus tarsi	Angle of Gissane sclerosis	
		Forefoot abduction		
IV	A	Supple tibiotalar valgus	Tibiotalar valgus	Surgery for HF valgus and associated deformity
			HF valgus	Deltoid reconstruction
	B	Rigid tibiotalar valgus	Tibiotalar valgus	TTC fusion or pantalar fusion
			HF valgus	

HF hind foot, FDL transf. flexor digitorum longus transfer, MDCO medial displacing calcaneal osteotomy, NSAID nonsteroidal anti-inflammatory drugs, PTT posterior tibial tendon, TAL tendo Achilles lengthening, TMT tarsometatarsal joint, TTC tibiotalar canal

consulting an orthopaedic surgeon. Nevertheless, lacking one or more of the previous criteria cannot be assumed as predictors for the need for surgical management at initial patient's visit [60].

## Surgical Treatment

Unsuccessful conservative treatment for a period of 3–6 months may necessitate surgical management. The main aim of surgery is to maintain stable, painless and plantigrade foot. See Table 23.4 for an overview of aims of some surgical tactics in flatfoot.

Synovectomy is indicated in cases of failed conservative treatment in stage I PTTI. During the procedure, the tendon should be inspected for any evidence of degeneration that may necessitate debridement, shortening, repair, or reconstruction.

In a significant flatfoot deformity (PTTI II–IV), understanding the patho-mechanics of the three dimensional deformity is the key of successful surgical planning. In such cases, reconstruction of the soft tissue alone may carry a high tendency to failure. So, the necessity of bony deformity correction together with soft tissue reconstruction should be addressed properly (Fig. 23.1). A summary of current treatment options for PTTI stages II–IV is shown in Table 23.5.

Spring ligament integrity should be checked intraoperatively. It may be found attenuated rather than disruption, and repair or reconstruction is essential as it is a major pathologic contributor to pes planovalgus deformity.

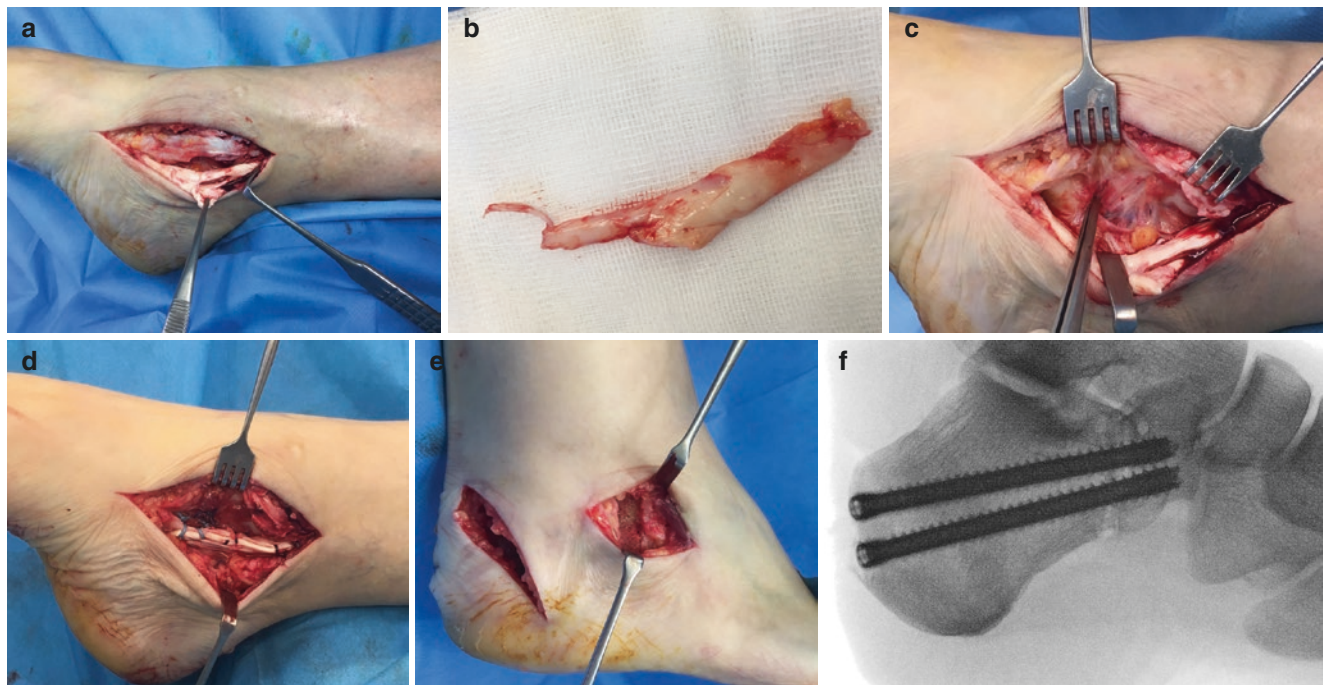
Selection of the appropriate type/types of osteotomy is dictated by the amount of correction needed according to the three dimensional image of deformity (hindfoot valgus, forefoot abduction, and medial arch flattening). Combination of more than one type of osteotomy may be required (see Table 23.6). **Medial Calcaneal Displacing Osteotomy**

**Table 23.4** Goals of surgical treatment of PTTI

Surgical strategy	Aim
Soft tissue reconstruction	Pain ↓, PTT-Function ↑ Medial Stability ↑
Bone reconstruction	Medial Soft tissue stress ↓ Correction of: Planus Abductus Supinatus Peritalar Luxation (TN) Long term Stability & Alignment

An arrow pointing up: increase; An arrow pointing down: decrease; *PTT* posterior tibial tendon, *TN* talonavicular joint

(**MDCO**) can be used in both flexible and rigid hindfeet and it aims at a significant correction of hindfoot valgus and redirection of the line of Achilles tendon pull; so, it may help lessen the impact of Achilles action as a deforming force for valgus hindfoot [61]. According to a biomechanical study, MDCO with 10 mm translation was not significant to affect the Achilles tendon length [62]. However, another study suggested that MDCO could help decrease the amount of forces exerted on first metatarsal and talonavicular articulation moment whereas it may increase forces exerted on the fifth metatarsal and calcaneo-cuboid articulation [63]. **Lateral Calcaneal Lengthening Osteotomy (LCLO)** requires a flexible deformity (i.e. some subtalar motion preservation) as a prerequisite. As the surgical approach for this osteotomy involves an exposure of sinus tarsi, it is advised to preserve fat pad at that area because it is thought to contain proprioceptive neural tissue (“**cerebellum pedis**”) [64]. Moreover, care should be taken not to sacrifice interosseous ligament in sinus tarsi as it may destabilise the subtalar joint. The value of such an osteotomy is in generous correction of forefoot abduction and moderate effect on correcting hindfoot valgus and medial arch of the foot (Fig. 23.2) [61]. **Cotton (medial cuneiform open dorsal wedge) osteotomy** is useful for substantial restoration of the medial foot arch and minimal



**Fig. 23.1** Intraoperative illustration of surgical reconstruction of a PTTI stage IIA in a young adult. (a) A medial incision was used to approach the PTT tendon, which starts from the medial malleolus and extends distally to the navicular bone. Note hypertrophy and degeneration of PTT tendon with complex tear. (b) The excised degenerative part of PTT tendon is shown. (c) Spring ligament was found to be affected. A softening and a gap were noted and indicates tear. (d) Spring ligament was repaired. A tenodesis of Flexor digitorum longus (FDL)

to PTT was done. (e) A lateral incision is used to approach lateral calcaneal lengthening osteotomy (LCLO) site just below the sinus tarsi. Note the inserted allograft at the osteotomy site. A second incision was used to perform the additional medial displacing calcaneal osteotomy (MDCO) as more correction of hindfoot valgus was needed. (f) The fixation of both osteotomies was performed with two cannulated CCS 7.0 screws (Medartis, Switzerland). Note the configuration of screws in the lateral x-ray



**Table 23.5** Overview of surgical options in treatment of grade II-IV PTTI

	Medial soft tissue reconstruction	Osteotomies hindfoot/midfoot	Arthrodeses hindfoot/midfoot	Supramalleolar OT, total ankle arthroplasty
Grade II	+ (PTT, Spring, Deltoid)	+		
Grade III	+ (Deltoid)		+	
Grade IV	+ (Deltoid)		+	+

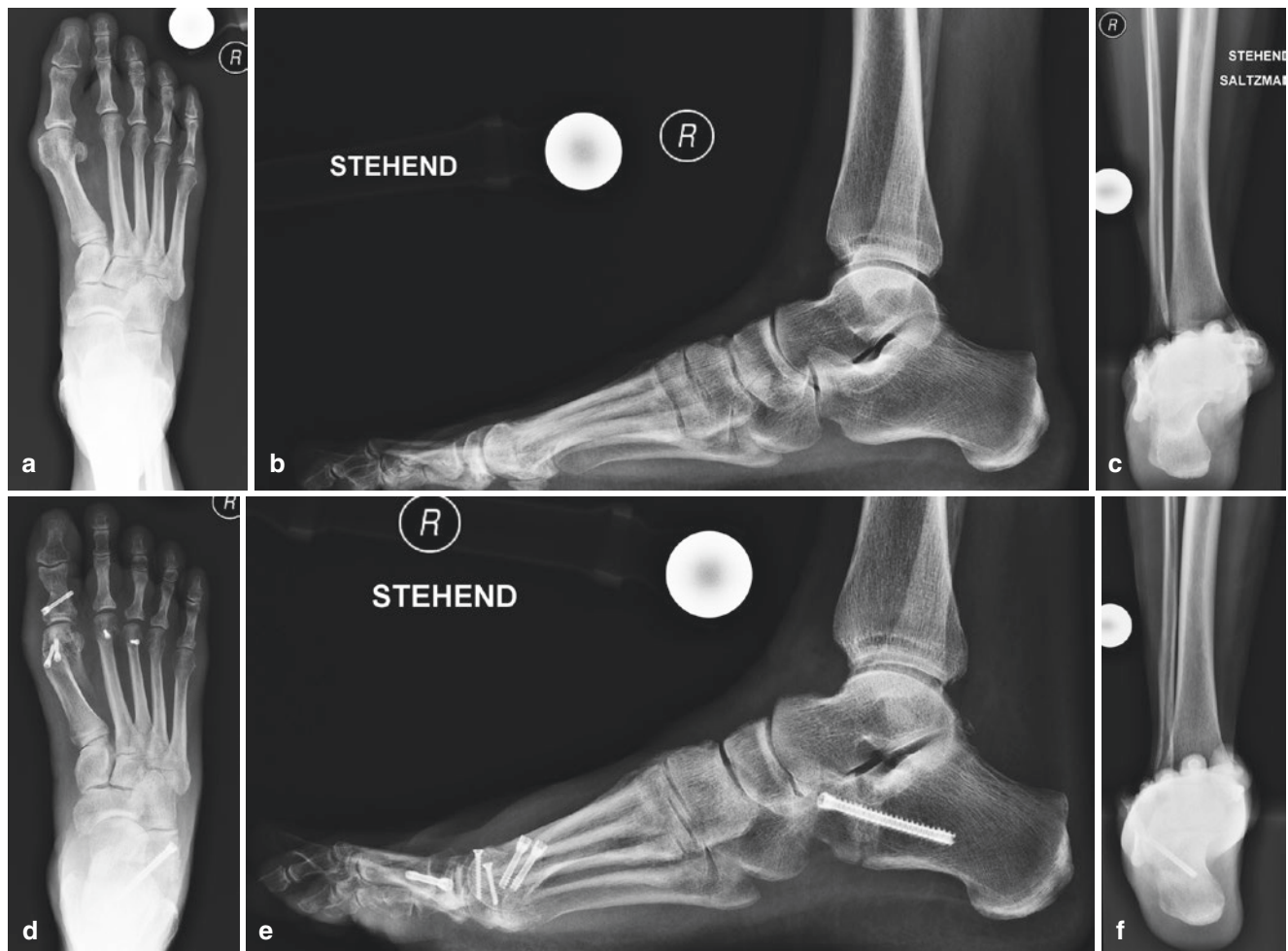
OT osteotomy, PTT posterior tibial tendon

**Table 23.6** Amount of expected flatfoot deformity correction in three different osteotomies

	MDCO	LCLO	Cotton
Hindfoot varisation	+++	++	-
Forefoot adduction	+	+++	+
Medial arch restoration	+	++	+++

Adapted from Valderrabano et al. [61]

LCLO lateral calcaneal lengthening osteotomy, MDCO medial displacing calcaneal osteotomy



**Fig. 23.2** Radiographic illustration of an adult patient who underwent a lateral calcaneal lengthening osteotomy (LCLO) for correction of PTTI stage IIA: (a) A preoperative anteroposterior (AP) view of foot. Note the talonavicular subluxation. Also, hallux valgus is seen which appears to be a result of PTTI deformity. (b) A preoperative lateral view of the foot which shows abnormal talo-first metatarsal angle. (c) A preoperative Saltzman view of foot which shows a significant hindfoot valgus. (d) A postoperative anteroposterior (AP) view of foot, which

shows a good correction of talonavicular subluxation and coverage. Forefoot osteotomies are noted aimed at correction of hallux valgus and transfer metatarsalgia (Modified Chevron's osteotomy, Akin's osteotomy, and Weil osteotomy for 2nd and 3rd metatarsals). (e) A postoperative lateral view of foot, which shows a normalisation of the talo-first metatarsal angle. (f) A postoperative Saltzman view of foot, which shows a significant correction of the hindfoot alignment

forefoot adduction correction [61]. **The authors think that performing osteotomies in elite athletes is better to be avoided as they might be a possible end of their career.**

In stage IV PTTI, where tibiotalar joint and/or deltoid ligament are involved, has to be treated as a valgus malaligned ankle osteoarthritis (OA). **Joint preserving surgery, by supramalleolar medial closing wedge osteotomy**, may be appropriate if a significant part of the articular cartilage is still intact prior to considering total ankle arthroplasty or fusion of ankle joint (e.g. ankle arthrodesis, pantalar arthrodesis, or tibio-talo-calcaneal arthrodesis).

In advanced PTTI stages, deltoid ligament may become attenuated and requires reconstruction. Adding to that, long-standing valgus malalignment may result in overload of distal tibiofibular joint and lead to **syndesmotic insufficiency**. Such an insufficiency may need to be considered to be treated during operative treatment. Moreover, seldom, Tibialis anterior rupture may also occur in longstanding PTTI cases.

In cases where medial soft tissue procedure (e.g. FDL transfer and spring ligament reconstruction) together with lateral calcaneal osteotomy is indicated, our senior author (V.V.) recommends to begin the surgical preparation for medial soft tissue reconstruction followed by calcaneal osteotomy and lastly finalizing medial soft tissue procedures. This strategy may help in avoiding redundancy of medial

soft tissue. Gastrocnemius-Soleus release or Achilles tendon lengthening may be indicated in case of limited ankle dorsiflexion (contracture) and in severe deformities.

In addition to bone reconstruction, the authors recommend to do a PTT to FDL transfer in stages III and IV to aid in dynamic correction and recovery of PTT due to the fact that recovery of degenerated PTT is still possible, even in late repair cases [65].

Possible complications of surgical intervention may include prolonged foot edema, recurrence of deformity, overcorrection, undercorrection, fixation failure, non-union of osteotomy, and non-union of arthrodesis.

## Rehabilitation and Back-to-Sports

In acute and chronic PTT lesions, we recommend immobilization using a pneumatic walker. 15 kg-weight bearing on crutches is permitted for the first 6 weeks. Then, gradual progression to full weight bearing for the following 6 weeks. Structured physical therapy program is instituted from the first postoperative day until at least 3–6 months to achieve good muscular strength and proprioceptive control. Low-impact sports are allowed after 3 months and gradual progression for more high-intensity sports.

**Table 23.7** Illustration of selected best evidence available on therapy of posterior tibial tendon lesions and insufficiency

Authors	Journal, year	Study topic	Number of patients	Evidence level
Deland and Hamilton	<i>Clin Sports Med</i> , 2008	Posterior tibial tendon tears in dancers	4	IV
Lohrer and Nauck	<i>Br J Sports Med</i> , 2010	Posterior tibial tendon dislocation: a systematic review of the literature and presentation of a case	59	IV
Holmes and Mann	<i>Foot Ankle</i> , 1992	Possible epidemiological factors associated with rupture of the posterior tibial tendon	67	
Krause et al	<i>Foot Ankle Int</i> , 2008	Shell brace for stage II posterior tibial tendon insufficiency	18	II
Kulig et al	<i>Phys ther</i> , 2009	Nonsurgical management of posterior tibial tendon dysfunction with orthoses and resistive exercise: a randomized controlled trial	40	I
Alvarez et al	<i>Foot Ankle Int</i> , 2006	Stage I and II Posterior Tibial Tendon Dysfunction Treated by a Structured Nonoperative Management Protocol: An Orthosis and Exercise Program	47	III
O' Connor et al	<i>Foot Ankle Int</i> , 2006	Patient factors in the selection of operative versus nonoperative treatment for posterior tibial tendon dysfunction	166	III
Marks et al	<i>Gait Posture</i> , 2009	Surgical reconstruction of posterior tibial tendon dysfunction: Prospective comparison of flexor digitorum longus substitution combined with lateral column lengthening or medial displacement calcaneal osteotomy	20	II
Dolan et al	<i>Foot Ankle Int</i> , 2007	Randomized prospective study comparing tri-cortical iliac crest autograft to allograft in the lateral column lengthening component for operative correction of adult acquired flatfoot deformity	33	I

Long rehabilitation period may be expected in cases involving corrective osteotomies or joint fusion due to the time taken by the nervous system to be adapted to the new hindfoot alignment [66].

## Prevention

Injuries of PTT can be prevented by the use of safe sport equipments and incorporation of training of the Posterior Tibial muscle in the regular sports training. In athletes with PTTI, it is recommended to do Posterior Tibial muscle exercises to varisate the hindfoot, use of antipronation sports shoes, and insoles with medial arch support.

## Evidence

Level of the best available evidence on PTT lesions and insufficiency are shown in Table 23.7.

## Summary

1. Although there is not clear estimate of its incidence, acute posterior tibial tendon injury commonly follow sport.
2. Acute PTT injury should be considered in the differential diagnosis of “unhealed medial ankle sprain” especially when PTT function testing is questionable.
3. Posterior tibial tendon deficiency is common among athletes, mainly as a stage I Johnson and Strom. Treatment of choice is physiotherapy with Posterior Tibial muscle exercises to varisate the hindfoot, use of antipronation sports shoes, and insoles with medial arch support
4. The presence of concomitant spring ligament lesion should be kept in mind in all patients undergoing flatfoot reconstruction as missing treatment of such a lesion may cause failure of surgical treatment.
5. Selection of the appropriate type/types of osteotomy is dictated by the amount of correction needed according to the three dimensional image of deformity (hindfoot valgus, forefoot abduction, and medial arch flattening).
6. Long rehabilitation period may be expected in cases involving corrective osteotomies or joint fusion due to the time taken by the nervous system to be adapted to the new hindfoot alignment.

## References

1. Ribbans WJ, Garde A. Tibialis posterior tendon and deltoid and spring ligament injuries in the elite athlete. *Foot Ankle Clin.* 2013;18(2):255–91.
2. Trividi M, Brown E, Lese A, Katz LD. Lateral dislocation and incarceration of the posterior tibial tendon through the distal tibio-fibular syndesmosis. *Skeletal Radiol.* 2014;43(8):1175–8.
3. Lacasse JS, Laflamme M, Penner MJ. Irreducible fracture-dislocation of the ankle associated with interposition of the tibialis posterior tendon in the syndesmosis: a case report. *J Foot Ankle Surg.* 2015;54(5):962–6.
4. Lohrer H, Nauck T. Posterior tibial tendon dislocation: a systematic review of the literature and presentation of a case. *Br J Sports Med.* 2010;44(6):398–406.
5. Gambardella GV, Donegan R, Caminear DS. Isolated dislocation of the posterior tibial tendon in an amateur snowboarder: a case report. *J Foot Ankle Surg Off Publ Am College Foot Ankle Surg.* 2014;53(2):203–7.
6. Deland JT, Hamilton WG. Posterior tibial tendon tears in dancers. *Clin Sports Med.* 2008;27(2):289–94.
7. Nava BE. Traumatic dislocation of the tibialis posterior tendon at the ankle. Report of a case. *J Bone Joint Surg Br.* 1968;50(1):150–1.
8. Loncarich DP, Clapper M. Dislocation of posterior tibial tendon. *Foot Ankle Int.* 1998;19(12):821–4.
9. Olive Vilas R, Redon Montojo N, Pino Sorroche S. Traumatic dislocation of tibialis posterior tendon: a case report in a Tae-Kwon-Do athlete. *Clin J Sport Med Off J Can Acad Sport Med.* 2009;19(1):68–9.
10. Schaffer JJ, Lock TR, Saliccioli GG. Posterior tibial tendon rupture in pronation-external rotation ankle fractures. *J Trauma.* 1987;27(7):795–6.
11. Formica M, Santolini F, Alessio-Mazzola M, Repetto I, Andretta A, Stella M. Closed medial malleolar multifragment fracture with a posterior tibialis tendon rupture: a case report and review of the literature. *J Foot Ankle Surg Off Publ Am College Foot Ankle Surg.* 2015.
12. Goucher NR, Coughlin MJ, Kristensen RM. Dislocation of the posterior tibial tendon: a literature review and presentation of Two cases. *Iowa Orthop J.* 2006;26:122–6.
13. Ouzounian TJ, Myerson MS. Dislocation of the posterior tibial tendon. *Foot Ankle.* 1992;13(4):215–9.
14. Langan P, Weiss CA. Subluxation of the tibialis posterior, a complication of tarsal tunnel decompression: a case report. *Clin Orthop Relat Res.* 1980;146:226–7.
15. Hunter AM, Bowlin C. Posterior tibial tendon entrapment within an intact ankle mortise: a case report. *J Foot Ankle Surg.* 2015;54(1):116–9.
16. Coughlin MJ, Schon LC. Disorders of tendons. In: Coughlin MJ, Saltzman CL, Anderson RB, editors. *Mann’s surgery of the foot and ankle.* 1. 9th ed. Philadelphia: Elsevier; 2014. p. 1275.
17. Marcus RE, Pfister ME. The enigmatic diagnosis of posterior tibialis tendon rupture. *Iowa Orthop J.* 1993;13:171–7.
18. Mansour R, Jibri Z, Kamath S, Mukherjee K, Ostlere S. Persistent ankle pain following a sprain: a review of imaging. *Emerg Radiol.* 2011;18(3):211–25.
19. Linklater JM, Read JW, Hayter CL. Imaging of the foot and ankle. In: Coughlin MJ, Saltzman CL, Anderson RB, editors. *Mann’s surgery of the foot and ankle.* 1. 9th ed. Philadelphia: Elsevier; 2014. p. 69–76.
20. Jenkins DW, Cooper K, Heigh EG. Prevalence of podiatric conditions seen in Special Olympics athletes: a comparison of USA data to an international population. *Foot (Edinb).* 2015;25(1):5–11.
21. Haddad SL, Deland JT. Pes planus. In: Coughlin MJ, Saltzman CL, Anderson RB, editors. *Mann’s surgery of the foot and ankle.* 1. 9th ed. Philadelphia: Elsevier; 2014. p. 1299.
22. Goncalves-Neto J, Witzel SS, Teodoro WR, Carvalho-Junior AE, Fernandes TD, Yoshinari HH. Changes in collagen matrix composition in human posterior tibial tendon dysfunction. *Joint Bone Spine Revue du rhumatisme.* 2002;69(2):189–94.

23. Petersen W, Hohmann G, Stein V, Tillmann B. The blood supply of the posterior tibial tendon. *J Bone Joint Surg.* 2002;84(1):141–4.
24. Holmes Jr GB, Mann RA. Possible epidemiological factors associated with rupture of the posterior tibial tendon. *Foot Ankle.* 1992;13(2):70–9.
25. Frey C, Shereff M, Greenidge N. Vascularity of the posterior tibial tendon. *J Bone Joint Surg Am.* 1990;72(6):884–8.
26. Buchhorn T, Sabeti-Aschraf M, Dlaska CE, Wenzel F, Graf A, Ziai P. Combined medial and lateral anatomic ligament reconstruction for chronic rotational instability of the ankle. *Foot Ankle Int.* 2011;32(12):1122–6.
27. Cozen L. Posterior tibial tenosynovitis secondary to foot strain. *Clin Orthop Relat Res.* 1965;42:101–2.
28. Myerson M, Solomon G, Shereff M. Posterior tibial tendon dysfunction: its association with seronegative inflammatory disease. *Foot Ankle.* 1989;9(5):219–25.
29. Bridgeman JT, Zhang Y, Donahue H, Wade AM, Juliano PJ. Estrogen receptor expression in posterior tibial tendon dysfunction: a pilot study. *Foot Ankle Int.* 2010;31(12):1081–4.
30. Pisani G. About the pathogenesis of the so-called adult acquired pes planus. *Foot Ankle Surg Off J Eur Soc Foot Ankle Surg.* 2010;16(1):1–2.
31. Pisani G. The coxa pedis. *Foot Ankle Surg.* 1994;1(2–3):67–74.
32. Huang CK, Kitaoka HB, An KN, Chao EY. Biomechanical evaluation of longitudinal arch stability. *Foot Ankle.* 1993;14(6):353–7.
33. Pisani G. Peritalar destabilisation syndrome (adult flatfoot with degenerative glenopathy). *Foot Ankle Surgery Off J Eur Soc Foot Ankle Surg.* 2010;16(4):183–8.
34. Orr JD, Nunley 2nd JA. Isolated spring ligament failure as a cause of adult-acquired flatfoot deformity. *Foot Ankle Int.* 2013;34(6):818–23.
35. Tryfonidis M, Jackson W, Mansour R, Cooke PH, Teh J, Ostlere S, et al. Acquired adult flat foot due to isolated plantar calcaneonavicular (spring) ligament insufficiency with a normal tibialis posterior tendon. *Foot Ankle Surg Off J Eur Soc Foot Ankle Surg.* 2008;14(2):89–95.
36. Johnson KA, Strom DE. Tibialis posterior tendon dysfunction. *Clin Orthop Relat Res.* 1989;239:196–206.
37. Malicky ES, Crary JL, Houghton MJ, Agel J, Hansen ST, Jr., Sangeorzan BJ. Talocalcaneal and subfibular impingement in symptomatic flatfoot in adults. *J Bone Joint Surg Am.* 2002;84-a(11):2005–9.
38. Myerson MS. Adult acquired flatfoot deformity: treatment of dysfunction of the posterior tibial tendon. *Instr Course Lect.* 1997;46:393–405.
39. Bluman EM, Title CI, Myerson MS. Posterior tibial tendon rupture: a refined classification system. *Foot Ankle Clin.* 2007;12(2):233–49, v.
40. Bluman EM, Myerson MS. Stage IV posterior tibial tendon rupture. *Foot Ankle Clin.* 2007;12(2):341–62, viii.
41. Raikin SM, Winters BS, Daniel JN. The RAM classification: a novel, systematic approach to the adult-acquired flatfoot. *Foot Ankle Clin.* 2012;17(2):169–81.
42. Hintermann B, Gachter A. The first metatarsal rise sign: a simple, sensitive sign of tibialis posterior tendon dysfunction. *Foot Ankle Int.* 1996;17(4):236–41.
43. Saltzman CL, el-Khoury GY. The hindfoot alignment view. *Foot Ankle Int.* 1995;16(9):572–6.
44. Williams G, Widnall J, Evans P, Platt S. MRI features most often associated with surgically proven tears of the spring ligament complex. *Skeletal Radiol.* 2013;42(7):969–73.
45. Yao L, Gentili A, Cracchiolo A. MR imaging findings in spring ligament insufficiency. *Skeletal Radiol.* 1999;28(5):245–50.
46. Toye LR, Helms CA, Hoffman BD, Easley M, Nunley JA. MRI of spring ligament tears. *AJR Am J Roentgenol.* 2005;184(5):1475–80.
47. Gazdag AR, Cracchiolo 3rd A. Rupture of the posterior tibial tendon. Evaluation of injury of the spring ligament and clinical assessment of tendon transfer and ligament repair. *J Bone Joint Surg Am.* 1997;79(5):675–81.
48. Balen PF, Helms CA. Association of posterior tibial tendon injury with spring ligament injury, sinus tarsi abnormality, and plantar fasciitis on MR imaging. *AJR Am J Roentgenol.* 2001;176(5):1137–43.
49. Chen YJ, Liang SC. Diagnostic efficacy of ultrasonography in stage I posterior tibial tendon dysfunction: sonographic-surgical correlation. *J Ultrasound Med Off J Am Institute Ultrasound Med.* 1997;16(6):417–23.
50. Gerling MC, Pfirrmann CW, Farooki S, Kim C, Boyd GJ, Aronoff MD, et al. Posterior tibialis tendon tears: comparison of the diagnostic efficacy of magnetic resonance imaging and ultrasonography for the detection of surgically created longitudinal tears in cadavers. *Invest Radiol.* 2003;38(1):51–6.
51. Krause F, Bosshard A, Lehmann O, Weber M. Shell brace for stage II posterior tibial tendon insufficiency. *Foot Ankle Int.* 2008;29(11):1095–100.
52. Chao W, Wapner KL, Lee TH, Adams J, Hecht PJ. Nonoperative management of posterior tibial tendon dysfunction. *Foot Ankle Int.* 1996;17(12):736–41.
53. Lin JL, Balbas J, Richardson EG. Results of non-surgical treatment of stage II posterior tibial tendon dysfunction: a 7- to 10-year followup. *Foot Ankle Int.* 2008;29(8):781–6.
54. Alvarez RG, Marini A, Schmitt C, Saltzman CL. Stage I and II posterior tibial tendon dysfunction treated by a structured nonoperative management protocol: an orthosis and exercise program. *Foot Ankle Int.* 2006;27(1):2–8.
55. Augustin JF, Lin SS, Berberian WS, Johnson JE. Nonoperative treatment of adult acquired flat foot with the Arizona brace. *Foot Ankle Clin.* 2003;8(3):491–502.
56. Jones DC. Tendon disorders of the foot and ankle. *J Am Acad Orthop Surg.* 1993;1(2):87–94.
57. Rees JD, Wilson AM, Wolman RL. Current concepts in the management of tendon disorders. *Rheumatology (Oxford).* 2006;45(5):508–21.
58. Kulig K, Reischl SF, Pomrantz AB, Burnfield JM, Mais-Requejo S, Thordarson DB, et al. Nonsurgical management of posterior tibial tendon dysfunction with orthoses and resistive exercise: a randomized controlled trial. *Phys Ther.* 2009;89(1):26–37.
59. Kulig K, Lederhaus ES, Reischl S, Arya S, Bashford G. Effect of eccentric exercise program for early tibialis posterior tendinopathy. *Foot Ankle Int.* 2009;30(9):877–85.
60. O'Connor K, Baumhauer J, Houck JR. Patient factors in the selection of operative versus nonoperative treatment for posterior tibial tendon dysfunction. *Foot Ankle Int.* 2010;31(3):197–202.
61. Valderrabano V, Paul J, Monika H, Pagenstert GI, Henninger HB, Barg A. Joint-preserving surgery of valgus ankle osteoarthritis. *Foot Ankle Clin.* 2013;18(3):481–502.
62. Hadfield MH, Snyder JW, Liacouras PC, Owen JR, Wayne JS, Adelaar RS. Effects of medializing calcaneal osteotomy on Achilles tendon lengthening and plantar foot pressures. *Foot Ankle Int.* 2003;24(7):523–9.
63. Arangio GA, Chopra V, Voloshin A, Salathe EP. A biomechanical analysis of the effect of lateral column lengthening calcaneal osteotomy on the flat foot. *Clin Biomech.* 2007;22(4):472–7.
64. Leumann A, Frigg A, Valderrabano V. Ligamentare Instabilität am oberen Sprunggelenk. In: Valderrabano V, Engelhardt M, H Küster H, editors. *Fuß & Sprunggelenk und Sport.* Köln: Deutscher Ärzte-Verlag; 2009. p. 158.
65. Valderrabano V, Hintermann B, Wischer T, Fuhr P, Dick W. Recovery of the posterior tibial muscle after late reconstruction following tendon rupture. *Foot Ankle Int.* 2004;25(2):85–95.
66. Wiewiorski M, Valderrabano V. Painful flatfoot deformity. *Acta Chir Orthop Traumatol Cech.* 2011;78(1):20–6.

Mario Herrera-Perez and Anna Oller-Boix

**Abstract**

Peroneal tendon pathology is an undervalued source of lateral foot and ankle pain in athletes and can be difficult to distinguish from other lateral ankle injuries. There are three broad categories of peroneal tendon pathology: peroneal tendinitis/tendinopathy, peroneal tendon tears, and peroneal subluxation/dislocation. An understanding of the anatomy and biomechanics of the peroneal tendons is essential for diagnosis and proper treatment of its pathology. Space-occupying conditions such as low-lying muscle belly of the peroneus brevis or peroneus quartus, ankle instability, hindfoot varus, or pes cavovarus must be ruled out in all the cases. Proper conservative treatment is effective in athletes with tendinopathy. On the other hand, gross symptomatic ruptures or subluxation/dislocations should be surgically treated in order to return to their professional activity level.

**Keywords**

Peroneal tendons • Peroneal tendinopathy • Peroneal tendon tears • Peroneal subluxation

**Anatomy and Functional Aspects**

The peroneus longus (PL) and peroneus brevis (PB) originate from the lateral compartment of the leg. From proximal to distal, the tendons run around the posterolateral aspect of the ankle in a common synovial sheath surrounded by a fibro-osseous tunnel. This sheath splits at the distal ankle at the level of the peroneal tubercle [1, 2]. The PB is tendinous, about 2–4 cm proximal to the tip of the fibula, lying anteriorly and medially to the PL at the level of the lateral malleolus, while also inserting into the tuberosity of the fifth metatarsal. The PB is innervated by the superficial peroneal nerve (S1), acting as a primary evolver of the foot. The PL courses medially and posteriorly in the sulcus (longus takes

the long way round), passing between the long plantar ligament and the cuboid groove, aiming for its insertion into the plantar base of the first metatarsal and the lateral portion of the medial cuneiform. The tendon crosses the sole obliquely to insert into the base of the first metatarsal and adjoining part of the medial cuneiform [3]. Innervated by the superficial peroneal nerve (S1), the PL acts as a primary plantar flexion of the foot and first metatarsal, as well as actively everting the foot. The PL is the single most important factor in maintaining the integrity of the lateral longitudinal arch of the foot [3]. The *os peroneum* is a sesamoid located at the lateral aspect of the calcaneo-cuboid joint within the PL tendon. It is present in 3–6% of normal feet. The *retroperoneal sulcus* is formed laterally and posteriorly by the superior peroneal retinaculum (SPR), anteriorly by the concave shaped posterior aspect of the fibula and medially by the posterior inferior tibiofibular, the posterior talofibular and the calcaneofibular ligaments. The *superior peroneal retinaculum (SPR)* originates from the periostium of the posterolateral rim of the fibula and inserts into the lateral calcaneus (peroneal tubercle). Medially it blends

M. Herrera-Perez (✉) • A. Oller-Boix  
Department of Orthopaedic Surgery and Traumatology,  
University Hospital of Canary Islands,  
Ctra. Ofra, s/n, 38320 San Cristóbal de La Laguna,  
Santa Cruz de Tenerife, Canary Islands, Spain  
e-mail: [herrera42@gmail.com](mailto:herrera42@gmail.com); [annaoller@gmail.com](mailto:annaoller@gmail.com)

with the posterolateral ankle ligaments next to their attachments to the calcaneus. Inferiorly, it blends with the inferior peroneal retinaculum. The SPR is the primary restraint of the peroneal tendons within the retromalleolar sulcus. When not bearing weight, the peroneal muscles act as plantar flexors of the ankle, but their main function is hindfoot eversion. In addition, the muscles contract involuntarily in passive dorsal extension; during this movement the tendons act as dynamic ankle stabilizers.

### Peroneal Tendinitis and Tendinopathy

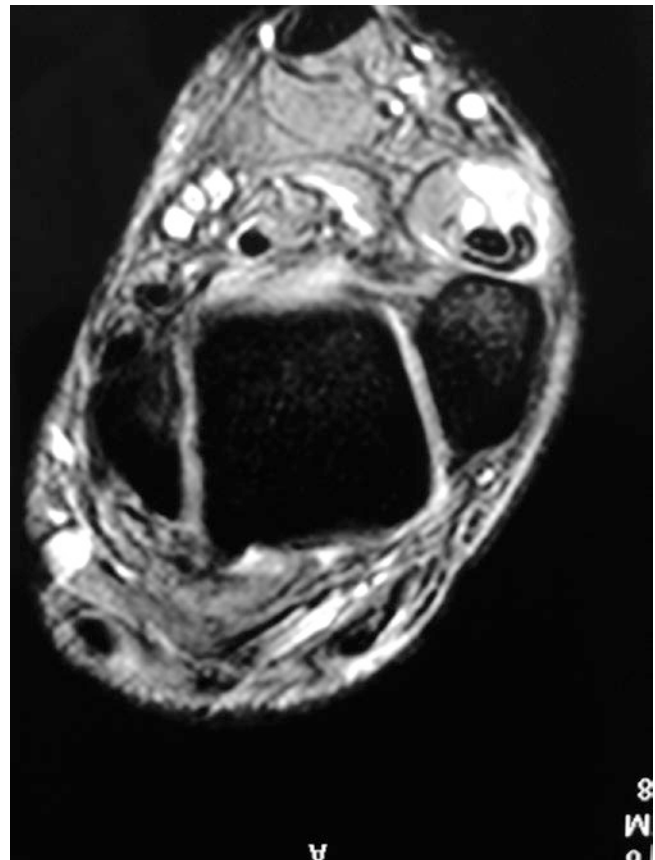
Peroneal tendinitis and tenosynovitis involve inflammation of the tendon or tendon sheath, respectively, affecting both the peroneus longus (PL) and the peroneus brevis (PB) tendons [2]. Overuse is a common cause of lateral ankle pain and should be considered, especially if the athlete has not suffered ankle sprains and instability has been ruled out [4, 5].

### Etiology and Pathomechanism

**Extrinsic factors:** This issue often occurs after the initiation of new, prolonged or repetitive activity, or even after a period of relative inactivity or extended rest [6, 7]. For the athlete, changes in footwear, training regimen or training surface also may contribute to the problem [2]. **Intrinsic factors:** Chronic lateral or rotational ankle instability, hindfoot varus alignment, pes cavo varus, muscle dysbalances, peroneal muscle weakness and acute ankle inversion injuries are also associated [7]. These conditions become symptomatic in runners, ballet dancers, and patients with chronic lateral ankle instability [1, 4]. Other causes include severe ankle sprains, fractures of the ankle or calcaneus, and peroneal tubercle hypertrophy [4, 5]. In areas of abrupt change in tendon direction, as there is in the retroperoneal sulcus, the peroneal trochlea and the lateral edge of the cuboid bone, the tendons flatten and thicken out [6]. At the margin of the cuboid bone the PL forms a sesamoid bone (os peroneum), present in only a quarter of individuals, to withstand the shearing forces of the tendon. These narrow fibro-osseous tunnels bear the risk of stenosing tenosynovitis especially at fulcrum sites. Only a small thickening of ligaments or tendon will start a vicious circle of compression and reactive tendon edema and synovial fluid production. Biomechanically, it is believed to be due to excessive pronation and eversion of the foot. However, congenital prominence of the peroneal trochlea, a descending peroneus brevis belly at the sulcus, additional PB muscle tendons and a peroneus quartus are reasons for development of tenosynovitis or a predisposition for development after minor trauma [6].

**Symptoms and diagnosis** Diagnosis is made clinically. Examination should assess forefoot and hindfoot alignment as well as ankle stability. Physical examination in an acute phase (less than 6 weeks of evolution) reveals swelling and warmth along the tendon sheath behind the lateral malleolus and pain with resisted eversion. If the affected tendon is the PL, the patient presents with pain along the lateral calcaneal wall extending to the cuboid (plantar lateral pain). A pseudo-tumor due to tendon degeneration may be present in chronic cases (more than 6 weeks of evolution).

Radiographs can be helpful to rule out other possible diagnoses and to identify an os peroneum (up to 3–6% of the population) [8]. Peroneal tubercle hypertrophy or spurring of the retromalleolar groove is best identified with a Harris heel radiograph [7]. A hindfoot alignment radiograph (Saltzman view) is helpful to identify a cavovarus foot position. Magnetic resonance image (MRI) is the imaging of choice to confirm this pathology in order to reveal an increase of fluid within the tendon sheath in acute cases (Fig. 24.1) or even intratendinous abnormalities such as partial tears or thickening in chronic cases [2]. Ultrasound study in expert hands should not be dismissed.



**Fig. 24.1** Increased fluid within the tendon sheath in an acute peroneal tendinopathy

**Painful os peroneum syndrome (POPS)** is a spectrum of posttraumatic conditions of the peroneal tendons [9], and includes at least one of the following:

1. Acute fracture of the os peroneum or diastasis of a multipartite os peroneum.
2. Chronic fracture of the os peroneum associated with stenosing tenosynovitis of the peroneus longus.
3. Partial or complete rupture of the peroneus longus tendon near the os peroneum.
4. Entrapment of the peroneus longus tendon and the os peroneum by a hypertrophied peroneal tubercle [10].

---

## Therapy of the Tendinopathy

*Conservative* A conservative approach is the first-line treatment and consists of activity modification, NSAIDs, rest, ice, orthotics/insoles or shoe modification and physical therapy. If symptoms persist, a short period of immobilization in a below knee plaster cast or CAM walker boot (10 days) is prescribed [2]. After symptoms solve, the patient begins a progressive rehabilitation programme of stretching and strengthening of the peroneal musculature. Endurance exercises are added as required for athletic conditioning.

*Platelet-rich therapies (PRT)* Overall, and for the individual clinical conditions, there is currently insufficient evidence to support the use of platelet-rich therapies for treating musculoskeletal soft tissue injuries, including peroneal tendon disorders. Researchers contemplating RCTs should consider the coverage of currently ongoing trials when assessing the need for future RCTs on specific conditions. Moreover, there is need for standardisation of PRP preparation methods [11].

*Surgery* If conservative treatment fails after 3–6 months, surgery is indicated: tenosynovectomy provides a satisfactory outcome [2, 7].

- *Open procedure:* Through a standard retromalleolar posterolateral approach, after protecting the sural nerve, the peroneal tendon sheath is incised. Stenotic areas of the sheath may be excised; degenerated areas of the tendon or partial tears are resected or repaired (with a tubularization technique). Prominent peroneal trochleas are remodelled, while synovial tissue and abnormal muscles such as the peroneus quartus or a descending muscle belly are excised. Rarely additional tendons of the PB are excised as well. The tendon sheath is left unrepaired to prevent a stenosis phenomenon. The SPR, if incised, must be repaired to prevent subluxation. In cases of chronic lateral or rotational ankle instability an approach over the fibula allows the peroneal tendon surgery as well as also the lateral ligament reconstruction.

- *Tendoscopy.* Van Dijk and Kort [12] have described tendoscopy for peroneal tendinopathy. They described the benefit of this treatment for stenosing tenosynovitis, or posttraumatic adhesions, in terms of postoperative follow-up. We find this technique very useful when treating peroneal tendinosis, stenosing synovitis or a low-lying muscle belly. However, some authors have found this technique difficult to use alone regarding suturing the tendons without making further incisions [13].

---

## Treatment of Painful Os Peroneum Syndrome (POPS)

The conservative treatment is the same.

- *Surgery.*
  - *Open procedure:* The PL sheath is identified and incised. The tendon is followed distally to the point where the tendon curves plantar to the cuboid. The os peroneum is identified and carefully shelled out of the peroneus longus tendon. If the tendon is intact, it can be reinforced with sutures. If more than 50% of the tendon is involved in the tear, tenodesis of the peroneus longus to the peroneus brevis can be performed. The proximal segment of the peroneus longus is tenodesed to the peroneus brevis [7].

*Postop, rehab and back to sports* 2 weeks non-weight bearing, followed by a short leg weight bearing cast or boot. Range of motion and strengthening activities are started 2–4 weeks after surgery. Strengthening activities are initiated 6–8 weeks after surgery. Athletic activities are only started once rigorous physical therapy is completed and when strength returns to 90% of the unaffected side [2].

*Evidence* Unfortunately, the treatment recommendations for peroneal tendinopathy are based primarily on case series and expert opinion [7]. More recently, Cychoz et al. [14] published the results of a comprehensive review of the literature, focusing on the use of foot and ankle tendoscopy. Although the current literature suggests that tendoscopy is a safe and effective procedure, this study shows that there is weak evidence (grade Cf) to support the use of tendoscopy on the peroneal tendons, so scientific articles with higher levels of evidence are needed.

---

## Peroneal Tendon Tears

They are frequently overlooked because of the vague nature of pain along the posterolateral ankle region.

**Classifications** Redfern and Myerson developed a treatment algorithm for a tear of both fibular tendons [15]. The tear was classified as Type I in a case where both tendons are repairable, Type II when only one tendon is repairable, Type IIIa is a candidate for tendon transfer is a candidate for tendon transfer with no proximal muscle excursion, and Type IIIb in a candidate for an allograft reconstruction in tears with proximal peroneal muscle excursion. Krause and Brodsky [16] proposed a classification system to guide surgical decision-making in patients with fibular tendon tears. This system is based on the transverse (cross-sectional) area of viable tendon that remains after debridement of the damaged portion of the tendon. This presumes that the retained portion of the tendon has no longitudinal tears. Grade I lesions are less than 50% of the cross-sectional area and tendon repair is recommended. Grade II lesions are more than 50% of the cross-sectional area and tenodesis is recommended.

**Etiology and pathomechanism** Acute ankle inversion injuries produce acute ruptures, as a result of sports injury or trauma [4, 5] but, more frequently, we face with chronic ruptures that can occur with many conditions such as lateral ankle instability, peroneal tendon subluxation, cavovarus foot position, and predisposing anatomic variations that lead to stenosis within the retromalleolar groove: convex or flat fibular groove, low-lying or anomalous muscle belly, SPR incompetence, posterior lateral fibular spurring and a high-arch foot type.

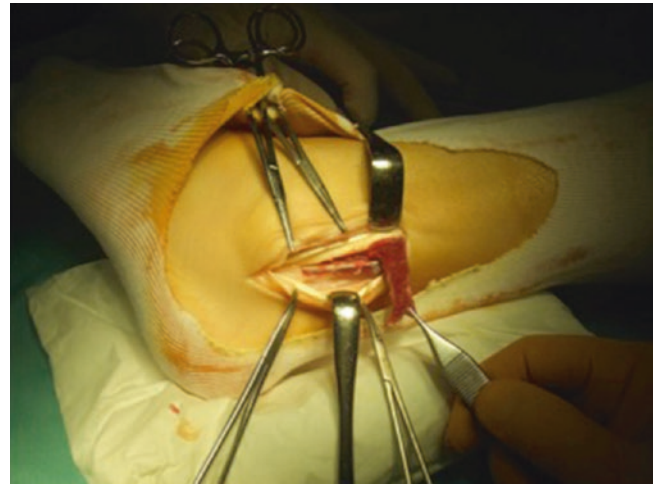
Magnetic resonance imaging (MRI) studies have also showed that anatomic factors, both osseous and soft tissue, contribute to peroneal tendon injury [6–9].

### Peroneus Brevis Tears

**Incidence** Peroneus brevis tears are common [6]. Cadaver studies indicate that incidences range from 11 to 37%. The incidence of clinically significant tears, however, remains unknown. Such injuries are difficult to diagnose with the time from initial injury to diagnosis frequently greater than 12 months.

**Diagnosis** Lateral ankle pain is the most common presenting symptom. Other complaints include ankle instability and lateral ankle swelling. Upon examination, tenderness and swelling over the tendon sheath are consistent findings, and peroneal muscle strength is often decreased [1]. PB tears can be evaluated with the *peroneal tunnel compression test*: pain while applying manual pressure along the peroneal tendon sheath in the retromalleolar groove with the knee flexed 90° and the foot plantar flexed [7]. A history of ankle sprain followed by chronic lateral ankle instability and posterolateral ankle pain is not unusual.

PB tears are usually found within the fibular groove, indicating that they are likely due to mechanical trauma in this region [1]. Clinical and anatomical studies indicate that tears



**Fig. 24.2** Peroneus quartus

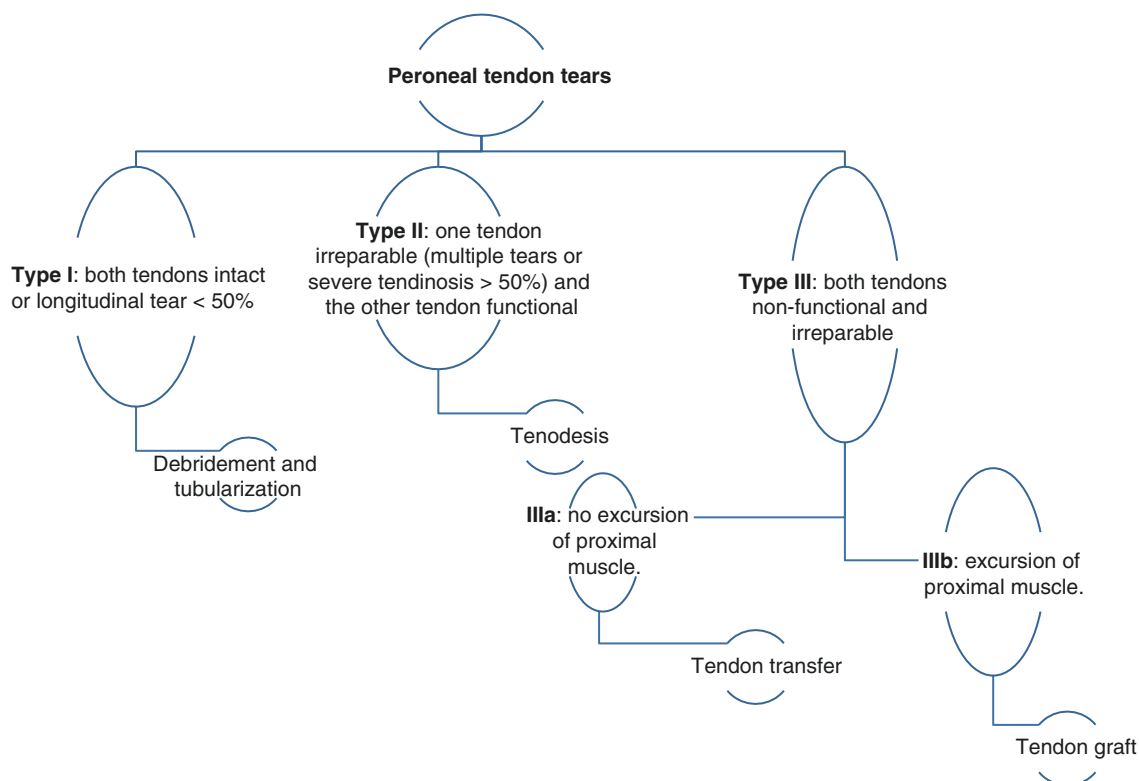
are produced by a combination of factors including compression of the brevis tendon against the posterolateral aspect of the fibula by the longus tendon and intratendinous shear stresses, which allow longitudinal tears in the tendon to develop as it passes beneath the tip of the lateral malleolus. This latter theory is supported by the fact that most tears are found on the undersurface of the tendon.

Laxity of the SPR has also been suggested as a contributing factor. Although subluxation of the tendon over the lateral fibular ridge has also been suggested as a factor, the incidence of this is unknown. A hypertrophied peroneus brevis muscle seen in athletes, as well as an anomalous peroneus quartus muscle may additionally predispose the tendon to tear. In one study, 18% of cadaver specimens with a peroneus quartus (Fig. 24.2) also had a brevis tear present [8, 16–19]. It has been hypothesized that the anomalous muscle and tendon force the brevis tendon laterally against the fibular ridge, which may result in a tear. Even without a tendon tear, an anomalous peroneal muscle may be the cause of chronic ankle pain and affect athletic performance [9]. If an anomalous muscle is suspected, MRI may confirm the diagnosis.

### Peroneus Longus Tears

They are not as common as PB tears and can occur independently or associated with one [6]. Two mechanisms for the development of a PL tear have been suggested: The first being degeneration as a result of high intratendinous shear stress, such as the cuboid tunnel, at the os peroneum, at the peroneal tubercle, or at the tip of the lateral malleolus. The other mechanism relies on evidence that proximal PL tears can be generated in cadavers using a mechanism similar to that which causes an inversion ankle sprain (acute ruptures) [6], in fact, any condition that leads to overuse of the PL tendon may produce a chronic injury





**Fig. 24.3** Peroneal tears treatment algorithm (Original Redfern and Myerson modified)

(such as weakness or absence of the supporting lateral ankle ligament structures). Fragmentation of the os peroneum through direct trauma or as an avulsion fracture is another injury mechanism.

**Symptoms** Clinical examination shows tenderness and occasional swelling along the PL tendon, usually in the cuboid groove or on the plantar aspect of the foot. When an os peroneum is present, tenderness is localized at or just proximal to the bone. With complete rupture of the tendon distal to or through the os peroneum, the bony fragment is pulled proximally with the proximal tendon stump.

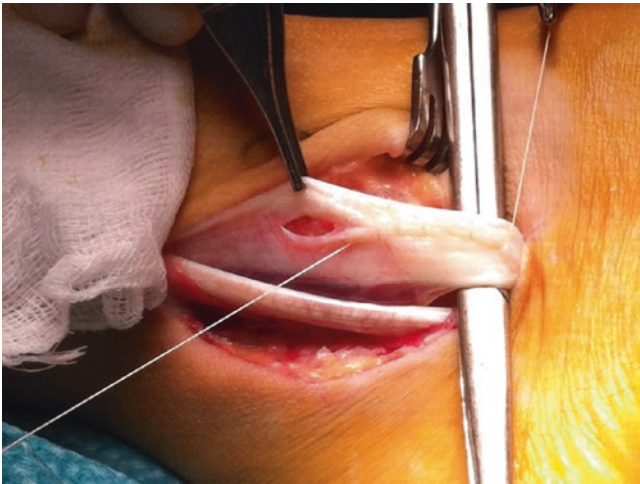
**Diagnosis** Loss or limitation of plantar flexion of the first ray and/or a painful resisted plantar flexion of the first ray and eversion of the foot may indicate a peroneus longus tear [6].

PL tears can be associated with a fracture through the os peroneum, as they are in patients with POPS (Painful Os Peroneum Syndrome) [20].

Radiographs of the foot may demonstrate proximal migration or fracture of the os peroneum, which correlates with the rupture of the PL tendon. In addition, fractures of the base of the fifth metatarsal can indicate avulsion of the PB tendon [1]. The anatomy and tears of peroneal tendons are easily visualized with an MRI. However, ultrasonography, if conducted by experienced radiologists, may be capable of detecting subtler tendon tears [1].

## Therapy

- **Conservative.** Includes NSAIDs, physical therapy, activity modification, and immobilization in a short leg walking cast or a walking boot. However, symptoms frequently persist despite non-operative management, especially in the setting of chronic ankle laxity, peroneal tendon subluxation, hindfoot varus deformity or in high-demand patients, such as athletes [1]. Failure rate of this treatment may be as high as 83%.
- **Surgical**
  - **Open procedure:** we use a modification of the algorithm developed by Redfern and Myerson based of intraoperative findings (Fig. 24.3) [15]. If both tendons are grossly intact (type I) or there's a longitudinal tear in less than 50% of the tendon, we perform a debridement and tubularization of the tendon (Fig. 24.4). If one tendon is irreparable (the tendon has multiple longitudinal tears and significant tendinosis involving more than 50% of the tendon) and the other tendon is functional (type II), a tenodesis between the two peroneal tendons is performed proximal and distal to the tear, carefully considering the location of the anastomosis in order to avoid a new stenosis. If both tendons are non-functional (type III), a reconstruction is indicated, using a tendon graft (semitendinosus) or a tendon transfer (usually FDL to peroneus brevis).



**Fig. 24.4** Debridement and tubularization in a Type I PB tear

– *Tendoscopy*. Technically demanding procedure that requires extensive experience in arthroscopic management of small joints [21]. Some authors have published their own experience in peroneal tenodesis using tendoscopy to identify the tears, while avoiding larger incisions, with excellent results [21, 22].

*Postop, rehab. and back to sports* Postop includes early range of motion and 3 weeks nonweight bearing,

Many studies have suggested that most patients can return to the same level of activity they had before the injury, but in the study by Steel and DeOrio [23], only 46% could return to sports. The patient's gender, body mass index, length of time between onset of symptoms or identifiable injury and operative treatment, and the use of absorbable or nonabsorbable sutures in the tendon did not affect the ability to return to sports. Older age and corrective calcaneal osteotomy had a negative effect on a patient's ability to return to sports [23].

*Evidence* To date all studies outlining the management of peroneal tendon tears are either retrospective reviews (Level IV evidence) or case reviews (Level V evidence). Therefore there's insufficient evidence to recommend for or against any specific treatment (Grade I) [1].

## Subluxation/Dislocations

Results from disruption of the SPR and usually involves avulsion of the retinaculum from the lateral malleolus. Most of these injuries occur in young, active patients.

## Etiology and Pathomechanism

Inversion injury to the dorsiflexed ankle with concomitant forceful reflexive contraction of the PL and PB tendons. Subluxation of the peroneal tendons leads to longitudinal tears over time, which usually involves PB at the fibular groove. There are a number of associated conditions to this pathology such as tears of the peroneus brevis and or longus (longitudinal tears are more common than transverse), inadequate groove for the peroneals in the posterolateral fibula (flat or convex groove), low-lying PB muscle belly, peroneus quartus, hindfoot varus and lateral ankle instability.

## Symptoms

Clicking, popping and feelings of instability or pain on the lateral aspect of the ankle, especially in dorsiflexion movement or exercises.

*Diagnosis* Patients report they felt a pop with a dorsiflexion ankle injury. Upon examination, we find a swelling posterior to the lateral malleolus and tenderness over the tendons. Sometimes we can see a "Pseudotumor" over the peroneal tendons. With active dorsiflexion and eversion manoeuvre the patient can voluntarily subluxate the tendons and a popping sound can be felt.

## Provocation Tests

- *Dorsiflexion standing test*: Subluxation or dislocation of the peroneal tendon through the dorsiflexion of the ankle joint while the patient is standing on both legs and flexing slowly the knees down.
- *Apprehension test*. Forceful eversion and dorsiflexion of the ankle combined with palpation of the tendon may precipitate tendon subluxation during examination.
- *Compression test*. Pain with passive dorsiflexion and eversion of the ankle.

## Imaging

*Radiographs*. Best recognized on an internal rotation AP view of the ankle, a radiograph might show a cortical avulsion off the distal tip of the lateral malleolus (rim fracture).

Saltzman hindfoot view to evaluate for hindfoot varus.

*MRI*: Best evaluated with axial views of a slightly flexed ankle. Can demonstrate anatomic anomalies leading a pathology: peroneus quartus muscle, low-lying peroneus brevis muscle belly, shape of the fibular groove, status of the SPR and tears in the peroneal tendons.

## Classification

Acute dislocation occurs by sudden forced dorsiflexion with concomitant eccentric contraction of the peroneal muscles.

Chronic peroneal tendon dislocation is often associated with recurrent ankle sprains, which lead to incompetency of the SPR, and subsequent tendon subluxation. Eckert and Davies classified SPR pathology in chronic dislocations into three types [1]:

Grade I: SPR is partially elevated off of the fibula allowing subluxation of both tendons.

Grade II: fibrocartilaginous ridge elevated from the fibula with the SPR.

Grade III: cortical fragment avulsed with the SPR.

Grade IV: The SPR is torn from the calcaneus, not the fibula.

More recently, Raikin et al. [24] described a new subgroup of peroneal tendon instability that they called intra-sheath subluxation; these patients described tenderness of the peroneal tendons behind the fibula without a clinically reproducible dislocation, and a palpable and painful clicking during active maximum eversion and dorsiflexion of the foot and ankle. There is no injury of the SPR, and this clinical entity includes a flat or convex peroneal groove, a low-lying PB muscle or the presence of a peroneus quartus. They also describe two subtypes: Type A: with intact tendons and Type B: with a longitudinal split of the PB tendon.

## Therapy

### • *Conservative.*

Short leg cast immobilization or walker with the foot in neutral to slight inversion for 6 weeks is indicated in acute cases: may allow the SPR to adhere to the posterolateral aspect of the fibula.

Indications: all acute grade I and possibly grade III injuries in nonprofessional athletes. Technique: tendons must be reduced at the time of casting.

Outcomes: success rates are only marginally better than 50% [2].

- *Surgery.* Surgical treatment is required to correct chronically subluxating or dislocated peroneal tendons or in acute injuries of professional athletes. If dislocation is diagnosed early, acute repair of the SPR may be beneficial, however, operative intervention often occurs later. Prior to any reconstructive surgery, hindfoot varus deformity or lateral ankle instability must be addressed also.

### • *Open procedures:*

- **Acute repair of SPR and deepening of the fibular groove:**

#### *Indications:*

Acute tendon dislocation in athletes who desire a quick return to a sport or active lifestyle.

Presence of a longitudinal tear.

- **Surgical options for chronic dislocation can be grouped into five categories (in recommended order):**

1. SPR retinaculum repair. Is the most anatomic procedure to perform but sometimes is impossible.
2. Direct and indirect groove-deepening procedures.
3. Bone block procedures.
4. Tendon rerouting.
5. Soft-tissue transfer. Soft-tissue transfers have been described in the literature to reinforce the SPR: Achilles tendon, PB, plantaris.

#### *Indications:*

Chronic/recurrent dislocation.

#### *Technique:*

Less able to reconstruct SPR so treatment focuses on other aspects of peroneal stability.

Typically involves groove-deepening in addition to soft tissue transfer or bone block techniques (osteotomies to further contain the tendons within the sulcus).

- *Tendoscopy.* Vega et al. [25] described the use of tendoscopy using the portals described by Van Dijk and Kort [12], in a series of six cases of intrasheath subluxation they reported excellent functional results with a deepening of the groove in all the patients without space-occupying conditions (peroneus quartus, low-lying muscle belly or both) and just removing these conditions when they do exist, without repairing the SPR. Although most of the published studies report satisfactory results with open surgery procedure, we find the use of tendoscopy particularly helpful in this subgroup because morbidity and pain are lower and patient recovery is faster.

## Postop, Rehab. and Back to Sports

After surgery, the ankle is maintained for 4 weeks in a non-weight bearing cast, followed by 2 weeks in a weight-bearing cast or walker. During immobilization, cardiovascular conditioning is performed along with proximal muscle strengthening. The first few physical therapy treatments are designed to help control pain and swelling from the surgery. Ice and electrical stimulation treatments may be used. The therapist may also use massage and other hands-on treatments to ease

muscle spasm and pain. Soft tissue mobilization around the scar site can be employed to increase the soft tissue mobility [9]. Treatments are also used to help improve ankle range of motion by progressive resistance and ROM exercises without putting too much strain on the area. Active and resisted dorsiflexion and eversion are prevented during the early rehabilitation phase to reduce stress on the SPR (approximately 6–8 weeks) [9]. Talar mobilization exercises and active dorsiflexion and eversion begin when the patient can bear weight without pain [9]. The progression of resisted strengthening, proprioception and agility exercises is initiated when the patient can bear weight without pain and without brace. As strength and proprioception improve, the patient can progress through plyometric and functional activities that lead to a return to competition [9, 26].

## Evidence

Most of the literature regarding peroneal subluxation/dislocation consists of retrospective case series (Level IV evidence), case reports (Level V evidence), and cadaver studies [1].

## Summary

1. Peroneal tendon pathology is an undervalued source of lateral foot and ankle pain in athletes and can be difficult to distinguish from other lateral ankle injuries. There are three broad categories of peroneal tendon pathology: peroneal tendinitis/tendinopathy, peroneal tendon tears, and peroneal subluxation/dislocation.
2. Space-occupying conditions such as low-lying muscle belly of the peroneus brevis or peroneus quartus, ankle instability, hindfoot varus, or pes cavovarus must be ruled out in all the cases. Proper conservative treatment is effective in athletes with tendinopathy.
3. Gross symptomatic ruptures or subluxation/dislocations should be surgically treated in order to return to their professional activity level.
4. Peroneal tendons reconstructions might be accompanied by other surgeries: ankle ligament reconstruction, calcaneal osteotomy (lateral sliding/Dwyer), etc.

## References

1. Selmani E, Gjata C, Gjika E. Current concepts review: peroneal tendon disorders. *Foot Ankle Int.* 2006;27:221–8.
2. Sammarco GJ. Peroneal tendon injuries. *Orthop Clin North Am.* 1994;25:135–45.
3. Jar S, Warner J, Meadows TH. Spontaneous rupture of the peroneus longus tendon with an associated os peroneum fracture: a case report and review of literature. *Foot Ankle Surg.* 1997;2:205–8.
4. Kindred J, Trubey C, Simons S. Foot injuries in runners. *Curr Sports Med Rep.* 2011;10(5):249–54.
5. Barr KP, Harrast MA. Evidence-based treatment of foot and ankle injuries in runners. *Phys Med Rehabil Clin N Am.* 2005;16:779–99.
6. Pagenstert GI, Valderrabano V, Hintermann B. Tendon injuries of the foot and ankle in athletes. *Shweizerische Zeitschrift für "Sportmedizin un Sporttaumatologie".* 2004;52(1):11–21.
7. Heckman DS, Gluck GS, Parekh SG. Tendon disorders of the foot and ankle part 1: peroneal tendon disorders. *Am J Sports Med.* 2009;37:614–25.
8. Wang XT, Rosenberg Z, Mechlin MB, Schweitzer ME. Normal variants and diseases of the peroneal tendons and superior peroneal retinaculum: MR imaging features. *Radiographics.* 2005;25:587–602.
9. Sobel M, Geppert MJ, Warren RF. Chronic ankle instability as a cause of peroneal tendon injury. *Clin Orthop Relat Res.* 1993;296:187–91.
10. Hyer CF, Dawson JM, Philbin TM, Berlet GC, Lee TH. The peroneal tubercle: description, classification, and relevance to peroneus longus tendon pathology. *Foot Ankle Int.* 2005;26:947–50.
11. Moraes VY, Lenza M, Tamaoki MJ, Faloppa F, Belloti JC. Platelet-rich therapies for musculoskeletal soft tissue injuries. *Cochrane Database Syst Rev.* 2013;12:CD010071. doi: [10.1002/14651858.CD010071.pub2](https://doi.org/10.1002/14651858.CD010071.pub2).
12. Van Dijk CN, Kort N. Tendoscopy of the peroneal tendons. *Arthroscopy.* 1998;14:471–8.
13. Grasset W, Mercier N, Chaussard C, Carpentier E, Aldridge A, Saragalia D. The surgical treatment of peroneal tendinopathy (excluding subluxations): a series of 17 patients. *J Foot Ankle Surg.* 2012;51:13–9.
14. Cychosz CC, Phisitkul P, Barg A, Nickisch F, van Dijk CN, Glazebrook MA. Foot and ankle tendoscopy: evidence-based recommendations. *Arthroscopy.* 2014;30(6):755–65.
15. Redfern D, Myerson M. The management of concomitant tears of the peroneus longus and brevis tendons. *Foot Ankle Int.* 2004;25:695–707.
16. Krause JO, Brodsky JW. Peroneus brevis tendon tears: pathophysiology, surgical reconstruction and clinical results. *Foot Ankle Int.* 1998;19:271–9.
17. Sobel M, Geppert MJ, Olson EJ, Bohne WH, Arnoczky SP. The dynamics of peroneus brevis tendon splits: a proposed mechanism, technique of diagnosis, and classification of injury. *Foot Ankle.* 1992;13:413–22.
18. Sobel M, Levy M, Bohne W. Congenital variations of the peroneus quartus muscle: an anatomic study. *Foot Ankle.* 1990;11:81.
19. Sammarco G, Brainard B. A symptomatic anomalous peroneus brevis in a high jumper: a case report. *J Bone Joint Surg.* 1991;73A:131–3.
20. Sobel M, Pavlov H, Geppert MJ, Thompson FM, DeCarlo EF, Davis WH. Painful os peroneum syndrome: a spectrum of conditions responsible for plantar lateral foot pain. *Foot Ankle Int.* 1994;15:112–24.
21. Bravo-Giménez B, García-Lamas L, Jiménez-Díaz V, Llanos-Alcázar LF, Vilá-Rico J. Peroneal tendoscopy: our experience. *Rev Esp Cir Ortop Traumatol.* 2013;57(4):268–75.
22. Mattos E, Dinato MC, de Faria-Freitas M, Pereira-Filho MV. Peroneal tenodesis with the use of tendoscopy: surgical technique and report of 1 case. *Arthrosc Tech.* 2014;3(1):e107–10.
23. Steel MW, DeOrio JK. Peroneal tendon tears: return to sports after operative treatment. *Foot Ankle Int.* 2007;28(1):49–54.
24. Raikin SM, Elias I, Nazarian LN. Intrasheath subluxation of the peroneal tendons. *J Bone Joint Surg Am.* 2008;90:992–9.
25. Vega J, Golanó P, Dalmau A, Viladot R. Tendoscopic treatment of intrasheath subluxation of the peroneal tendons. *Foot Ankle Int.* 2011;32(12):1147–51.
26. Porter D, McCarroll J, Knapp E, Torma J. Peroneal tendon subluxation in athletes: fibular groove deepening and retinacular reconstruction. *Foot Ankle Int.* 2005;26(6):436–41.

Ahmed Nabil Abdulazim, Victor Valderrabano,  
and Jochen Paul

### Abstract

Ruptures of the anterior tibial tendon are quite rare but unfortunately still often missed. Most cases are caused by a degenerative process and only a small number of cases is caused by direct trauma or laceration. The main symptoms of an anterior tibial tendon rupture are swelling, mild pain, a weakened dorsiflexion, equinovalgus foot deformity and as an end-stage even a complete drop foot. However, in most cases the specific history in combination with the physical examination will lead to the diagnosis of anterior tibial tendon rupture. The treatment is dependent on several factors as aetiology, period of time to the onset of symptoms, age and level of activity. Surgical treatment is performed in selected cases, as described in the article, and a direct reinsertion or end-to-end suture of the tendon should be performed when possible. In difficult cases with delayed diagnosis, or initially misdiagnosed cases, a gracilis autologous tendon graft with tunnel-fixation to the medial cuneiform might be necessary to restore physiological biomechanics and gait patterns.

### Keywords

Anterior tibial tendon • Tendon ruptur • Drop foot • Tendon reconstruction

## Anatomy

The anterior tibial tendon originates from the proximal half of the anterior tibia, the lower lateral tibial condyle and the interosseus membrane and inserts on the medial plantar aspect of the first cuneiform and the base of the first metatarsal. Three different patterns of insertion are reported [1]: (1) the tendon inserts into the medial cuneiform and the base of

the first metatarsal (68%). (2) the tendon inserts only into the medial aspect of the first cuneiform (25%). (3) the tendon inserts into the cuneiform and the first metatarsal with an additional accessory tendon that inserts into the base of the first metatarsal (7%). The anterior tibial tendon is innervated by the deep peroneal nerve and serves as the main dorsiflexor of the ankle but also aids in inversion and controlled plantar flexion of the foot [2]. The anterior tibial tendon passes beneath the superior and the inferior extensor retinaculum. An avascular zone can be found where the tendon passes the retinacula which explains why the risk of tendon rupture is highest in this area about 2 cm proximally of insertion [3].

A.N. Abdulazim

Orthopaedic Department, University Hospital, University of Basel,  
Basel, Switzerland  
e-mail: [ahmed.abdulazim@usb.ch](mailto:ahmed.abdulazim@usb.ch)

V. Valderrabano, MD, PhD

Orthopaedic Department, SWISS ORTHO CENTER,  
Schmerzlinik Basel, Swiss Medical Network, Basel, Switzerland  
e-mail: [vvalderrabano@gsmn.ch](mailto:vvalderrabano@gsmn.ch)

J. Paul, MD (✉)

Rennbahnklinik,  
Kriegackerstrasse 100, MuttENZ, Basel CH-4132, Switzerland  
e-mail: [jochen.paul@rennbahnklinik.ch](mailto:jochen.paul@rennbahnklinik.ch)

## Etiology and Pathomechanism

Spontaneous ruptures of the anterior tibial tendon are very rare and often associated with a long history of systemic disease such as polyarthritis, diabetes or gout [4]. They usually occur in men between the fifth and seventh decade [5].

Histological examination showed that in these cases tendons were already degeneratively altered supporting an acute-on-chronic rupture mechanism [6]. Other causes of spontaneous rupture are local steroid injections [6], chronic wear from an underlying exostosis or rubbing against the edge of the inferior extensor retinaculum [7] or in association with a decompensated flatfoot deformity [8]. Traumatic ruptures of the anterior tibial tendon are more common in younger and physically more active patients. A forced or excessive plantar flexion against a contracted anterior tibial muscle can cause rupture even in a healthy tendon [5]. Because of its proximity to the distal tibia the anterior tibial tendon is also susceptible to rupture due to fracture of the distal tibia or laceration in this area. Therefore in cases of open fractures or laceration a surgical exploration of the wounds should be performed to assess the integrity of the anterior tibial tendon.

---

## Symptoms

The main symptoms of an anterior tibial tendon rupture are swelling, mild pain, a weakened dorsiflexion, equinovalgus foot deformity and as an end-stage even a mechanical complete drop foot. Patients may report of difficulties walking on uneven ground.

---

## Diagnostics

In most cases history and physical examination will lead to the diagnosis of anterior tibial tendon rupture. The examination in a pre-rupture phase does not always show a palpable defect at the rupture site due to the swelling. In a rupture phase the retracted ruptured tendon can often be palpated as a prominent stump at the level of or above the ankle joint. Dorsiflexion is weakened compared to the contralateral side. The patients are usually unable to walk on the heel. During gait patients try to compensate the lack of dorsiflexion in the swing phase by using greater knee flexion, the extensor hallucis longus and the extensor digitorum longus [9]. Following a direct trauma or laceration X-rays should be done to assess possible fractures or osseous avulsion of the tendon. Magnetic resonance imaging (MRI) is the first choice of imaging in ruptures of the anterior tibial tendon [10].

---

## Therapy

Ruptures of the anterior tibial tendon are very rare but can cause substantial functional deficits. If not treated, they can result in gait disturbance and flatfoot deformity. Generally, ruptures can be treated conservatively or surgically. Common recommendations support the surgical treatment for younger

patients as well as athletes and physically active patients. Older patients with low physical demands might be treated conservatively. However, recent studies show that age is not a valid factor for choosing different types of treatment [11]. But still to date there is no evidence whether surgical or conservative treatment should be preferred.

---

## Conservative Treatment

Conservative treatment is suggested in older patients with low physical demands and in delayed diagnosis of more than 3 months [1]. Also patients with severe comorbidities (e.g. peripheral arterial disease) should be treated conservatively [12].

Treatment includes the use of ankle foot orthoses that prevent over-plantarflexion and allow active dorsiflexion. With time the retracted tendon becomes adherent around the ankle which leads to variable loss of dorsiflexion motion and strength. Depending on the compliance of the patient relatively good results can be achieved. Although not obtaining full functional recovery the results may be acceptable in older patients. Markarian et al. found no significant difference in the conservative and surgical treatment of older patients [13].

---

## Surgery

Several techniques for the surgical treatment of anterior tibial tendon ruptures are described. Choosing the right technique depends on several factors: acute vs. chronic rupture, retraction of the tendon, site of rupture and amount of defect of the tendon.

In cases of acute rupture a direct repair is recommended. As most ruptures occur 2–3 cm proximally of the insertion in most cases an end-to-end suture of the tendon edges can be performed. In cases of avulsion or detachment from its insertion a bony reattachment in the cuneiform using a suture anchor or transosseous attachment is recommended. In cases of delayed diagnosis with a retracted anterior tibial tendon an end-to-end repair might not be possible. Treatment options then include reconstruction with a sliding graft or a free allograft. When using a sliding graft the proximal end of the ruptured tendon is found and half of the width of the anterior tibial tendon is harvested and transferred distally. In cases of severe retraction or when bridging a larger gap a free allograft should be used. Preferred tendons are the plantaris longus tendon, the gracilis tendon (preferred autologous graft of the authors of this chapter) or the semitendinosus tendon. The desired graft is harvested. A 4.5 mm drill hole is placed in the medial cuneiform and the graft is passed through the drill hole. Both ends of the graft are then sutured to the proximal

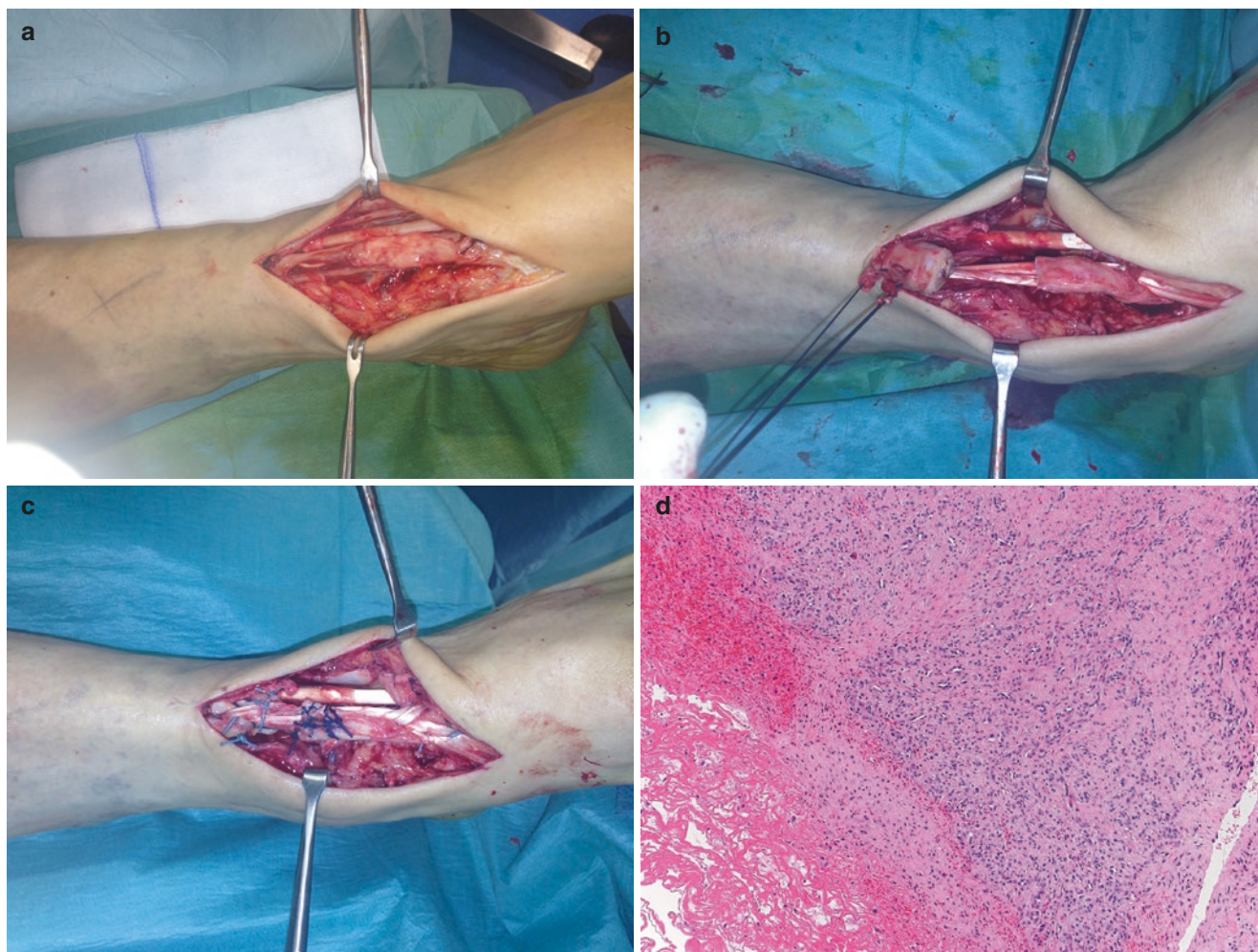
end of the anterior tibial tendon by placing the ankle in dorsiflexion and inversion. The distal stump rest of the ruptured anterior tibial tendon is over-sutured to the graft.

### Authors' Preferred Surgical Technique

We chose to perform the reconstruction of a ruptured and retracted anterior tibial tendon using a free gracilis autograft (Fig. 25.1):

1. The patient is placed in a supine position. A thigh tourniquet is applied.

2. A longitudinal incision is made over the ruptured tendon medially from the level of tendon retraction to the medial aspect of the cuneiform.
3. The ruptured tendon is identified proximally and distally (Fig. 25.1a) and both stumps are debrided.
4. The gracilis tendon is harvested using a tendon stripper.
5. A 4.5 mm drill hole is placed through the medial cuneiform dorsoplantar.
6. The gracilis tendon is pulled through the medial cuneiform drill hole and, with the ankle in dorsiflexion and inversion, sutured to itself and to the debrided proximal anterior tibial tendon stump (Fig. 25.1b).



**Fig. 25.1** Anterior tibial tendon reconstruction with gracilis tendon graft. A 59 year old patient reported to our outpatient clinic after receiving a direct hit to his left ankle 2 months ago during windsurfing. Physical examination showed a steppage gait due to restricted dorsiflexion. The anterior tibial tendon could not be palpated. The MRI of the left foot/ankle revealed a complete rupture of the anterior tibial tendon. The patient was assigned to surgical treatment. We performed a reconstruction of the anterior tibial tendon using a free autologous gracilis tendon graft. The tendon was very much retracted (a). The gracilis graft was passed through a drill hole in the cuneiform and sutured to the

proximal end of the ruptured anterior tibial tendon (b, c). The histological analysis of the stump biopsy shows mucoid degeneration and inflammation infiltration of the ruptured tendon (d). Postoperatively the patient was placed in a below knee walker with partial weight bearing allowed. After 6 weeks weight bearing was gradually increased and physiotherapy was initiated. At 6 months after surgery walking was almost back to normal. A slightly restricted as well as a weakened dorsiflexion remained. At 1 year after surgery the patient was pain free and was able to go hiking and perform low impact sports without restriction

7. The distal stump of the ruptured anterior tibial tendon is then sutured to the gracilis graft as augmentation (Fig. 25.1c).
8. Subcutaneous and cutaneous sutures; dressing; intraoperative cast in neutral dorsi-/plantarflexion.

## Rehabilitation and Back to Sports

Two days postoperative the dressing is changed and the patient is mobilized with a walker or a below knee cast for 6 weeks with the ankle in neutral position with partial weight bearing allowed. Important is also a neutral dorsi-/plantarflexion night splint or cast. After 6 weeks a special protective shoe is used and weight bearing is gradually increased. Physiotherapy is started with mild exercises to restore ankle mobility. After 12 weeks full weight bearing is allowed. Physiotherapy is intensified to further improve ankle mobility and strengthen the crural muscles. Returning to sports is not allowed until 4–6 months after surgery.

## Evidence

To date there are no long term prospective studies on the surgical vs conservative treatment of anterior tibial tendon ruptures. However, recent studies seem to favour the surgical treatment in acute as well as chronic ruptures ([11], Level IV). Conservative treatment is mostly recommended in older patients with a low level of activity. Non traumatic ruptures in these patients are often misdiagnosed causing a delay in treatment and making surgery even more difficult ([9], Level IV). Even with early diagnosis conservative treatment can result in long term pain and flat foot deformity ([14], Level IV). In the largest series of surgical treatment to date 19 anterior tibial tendon ruptures were examined ([5], Level IV). The authors found that the average AOFAS improved from 55.5 points preoperatively to 93.6 points postoperatively supporting the surgical treatment. However, reported complications after surgical treatment include adhesions, wound healing problems, bowstringing and infections. Even with surgical treatment there might remain a weakened dorsiflexion and restricted ankle mobility.

## Summary

- Ruptures of the anterior tibial tendon are rare and often missed.
- In most cases the rupture occurs due to a degenerative process and only in few cases because of direct trauma or laceration.

- Treatment is dependent on age and level of activity: low-demand elderly patients conservatively; younger patients, athletes and physically active patients surgically.
- When surgical treatment is chosen a direct reinsertion or end-to-end suture of the tendon should be performed when possible.
- In cases with delayed diagnosis and retracted anterior tibial tendon a gracilis autologous tendon graft with tunnel-fixation to the medial cuneiform is the authors' treatment of choice.

## References

1. Anagnostakos K, Bachelier F, Fürst OA, Kelm J. Rupture of the anterior tibial tendon: three clinical cases, anatomical study, and literature review. *Foot Ankle Int.* 2006;27(5):330–9.
2. Fennell CW, Phillips P. Redefining the anatomy of the anterior tibialis tendon. *Foot Ankle Int.* 1994;15(7):396–9.
3. Petersen W, Stein V, Bobka T. Structure of the human tibialis anterior tendon. *J Anat.* 2000;197(Pt 4):617–25.
4. Jerome JTJ, Varghese M, Sankaran B, Thomas S, Thirumagal SK. Tibialis anterior tendon rupture in gout--case report and literature review. *Foot Ankle Surg Off J Eur Soc Foot Ankle Surg.* 2008;14(3):166–9.
5. Sammarco VJ, Sammarco GJ, Henning C, Chaim S. Surgical repair of acute and chronic tibialis anterior tendon ruptures. *J Bone Joint Surg Am.* 2009;91(2):325–32.
6. Velan GJ, Hendel D. Degenerative tear of the tibialis anterior tendon after corticosteroid injection--augmentation with the extensor hallucis longus tendon, case report. *Acta Orthop Scand.* 1997;68(3):308–9.
7. Grundy JRB, O'Sullivan RM, Beischer AD. Operative management of distal tibialis anterior tendinopathy. *Foot Ankle Int.* 2010;31(3):212–9.
8. Frigg AM, Valderrabano V, Kundert H-P, Hintermann B. Combined anterior tibial tendon rupture and posterior tibial tendon dysfunction in advanced flatfoot. *J Foot Ankle Surg Off Publ Am Coll Foot Ankle Surg.* 2006;45(6):431–5.
9. Ellington JK, McCormick J, Marion C, Cohen BE, Anderson RB, Davis WH, et al. Surgical outcome following tibialis anterior tendon repair. *Foot Ankle Int.* 2010;31(5):412–7.
10. Mengiardi B, Pfirrmann CWA, Zanetti M. MR imaging of tendons and ligaments of the midfoot. *Semin Musculoskelet Radiol.* 2005;9(3):187–98.
11. Michels F, Van Der Bauwhede J, Oosterlinck D, Thomas S, Guillo S. Minimally invasive repair of the tibialis anterior tendon using a semitendinosus autograft. *Foot Ankle Int.* 2014;35(3):264–71.
12. Petersen W, Hohmann G, Stein V, Tillmann B. The blood supply of the posterior tibial tendon. *J Bone Joint Surg Br.* 2002;84(1):141–4.
13. Markarian GG, Kelikian AS, Brage M, Trainor T, Dias L. Anterior tibialis tendon ruptures: an outcome analysis of operative versus nonoperative treatment. *Foot Ankle Int.* 1998;19(12):792–802.
14. DiDomenico LA, Williams K, Petrolla AF. Spontaneous rupture of the anterior tibial tendon in a diabetic patient: results of operative treatment. *J Foot Ankle Surg Off Publ Am Coll Foot Ankle Surg.* 2008;47(5):463–7.



May Fong Mak and Mathieu Assal

**Abstract**

Athletic disorders of the flexor hallucis longus (FHL) tendon primarily afflict dancers and tend to be overuse injuries. The commonest symptom is posteromedial ankle pain, arising from FHL disease in the unyielding fibro-osseous tunnel at the posterior talus. Less common sites are the knot of Henry in the midfoot and intersesamoid area in the forefoot. A high degree of suspicion is necessary to distinguish FHL pathology from co-existing clinical entities, since the FHL is intimately related with various other structures in its course. Conservative treatment focuses on FHL stretching exercises. Surgery is reserved for athletes with debilitating symptoms refractory to nonoperative measures. Open and arthroscopic approaches each have advantages and disadvantages, however no consensus exists regarding the superiority of one over the other. Rehabilitation cornerstones are neuromuscular re-education, rectifying faulty sport technique, and physical conditioning to minimize recurrence.

**Keywords**

Flexor hallucis longus tendon disorders in athletes • FHL tendinitis athlete • FHL tenosynovitis athlete • FHL injury athlete • FHL injury dancer • FHL injury ballet • FHL management athlete • FHL rehabilitation athlete • FHL prevention athlete • FHL evidence athlete

**Introduction**

Disorders of the FHL tendon most commonly afflict athletes who repeatedly and forcefully axial load their ankles in hyperplantarflexion or pushoff with their forefoot [1, 2]. Athletic injury of the FHL is primarily seen in dancers, hence the term, “dancer’s tendonitis” [3]. Classical ballet dancers who perform in the “demi pointe” (standing high on the ball of the foot) and “en pointe” (standing on the tips of the toes)

positions form the typical athlete population at risk of FHL disorders [4–13]. FHL injuries are less common in non-dancers, for example, long-distance runners [14–17], soccer players [15, 18], platform divers [19], tennis players [15, 20], gymnasts [2], ice skaters [2], and swimmers [2].

**Anatomy**

The FHL originates from the posterior border of the distal two-thirds of the fibula and interosseous membrane. From its location in the deep posterior compartment of the leg, the FHL courses distally and becomes myotendinous just before it enters the fibro-osseous tunnel between the medial and lateral tubercles in the posterior part of the talus. The FHL then abruptly changes direction medially and passes under the sustentaculum tali. Next, it enters the midfoot where it crosses and binds to the flexor digitorum longus (FDL) at the

M.F. Mak, FRCSEd (Ortho) (✉)  
Department of Orthopaedic Surgery, Khoo Teck Puat Hospital,  
90 Yishun Central, Singapore 768828, Singapore  
e-mail: [makmayfong@gmail.com](mailto:makmayfong@gmail.com)

M. Assal, MD  
Center for Surgery of the Foot & Ankle, Clinique La Colline,  
Av. Beau-Séjour 6, Geneva 1206, Switzerland  
e-mail: [mathieu.assal@bluewin.ch](mailto:mathieu.assal@bluewin.ch)

master knot of Henry [21]. The FHL then continues deep to the soft tissue pulley formed by the intersesamoid ligament connecting the sesamoids, and finally inserts into the plantar aspect of the base of the distal phalanx of the hallux.

The posterior tibial and medial plantar arteries supply the FHL. Two avascular zones exist where the tendon glides around the talus and runs between the sesamoids. The poor peri- and intratendinous local blood supply have been implicated in degeneration and rupture of these segments of tendon [22].

---

## Biomechanics and Function

The FHL, innervated by the tibial nerve, primarily functions to plantar flex the first metatarsalphalangeal (MTP) and interphalangeal (IP) joints. It is also a secondary torque producer at the ankle-subtalar joint [23–25]. In the normal terminal stance phase of gait, active plantar flexion of the hallux by the FHL resists the dorsiflexion force exerted on the forefoot by the ground. This counteraction moment produced by the FHL serves to stabilize the hallux and medial column of the foot for load to transfer through it during the push-off phase [26–28]. Therefore, the FHL is believed to have a role in determining the extent and pattern of forefoot loading during locomotion [29–31].

---

## Types of Pathology

FHL pathology in ballet dancers are thought to represent an overuse phenomenon whereby repetitive microtrauma leads to micro- and macroscopic tissue damage [3, 7]. It is less commonly due to a single traumatic event [32]. A continuum of disease that includes tendinitis, tenosynovitis, pseudocyst, fusiform tendon thickening (nodularity) causing triggering, muscle hypertrophy, mucoid degeneration, fibrosis, partial longitudinal tear, and complete rupture have been described [3, 7, 8, 11, 15, 33].

The fibro-osseous tunnel in the hindfoot, Henry's knot in the midfoot, and intersesamoid space in the forefoot are potential FHL entrapment sites as the tendon is bound at these locations by unyielding retinaculum [2]. Commonest site of FHL tenosynovitis in dancers is at the posteromedial ankle, where the FHL myotendinous junction enters its fibro-osseous tunnel [3]. Hypotheses for the high incidence of problems here include the abrupt change in vector of the tendon at the posterior talus, local avascularity of the tendon segment [22], distal encroachment of the low-lying FHL muscle belly into the constricting fibro-osseous tunnel causing entrapment [2], friction against the os trigonum [32] and structural mismatch between the FHL and its tunnel causing FHL compression when the foot is in accentuated plantar flexion [7, 11]. FHL tenosynovitis at the knot of Henry [3] and the intersesamoid area [34, 35] are uncommon in dancers.

## History and Physical Examination

The dancer with FHL pathology most commonly presents with insidious onset of pain at the posteromedial ankle, often aggravated when rising on the forefoot or standing on the tips of the toes [15]. Rest may relieve pain initially, however it often increases in severity and frequency as training progresses. Depending on the location of FHL disease, the dancer may experience pain under medial longitudinal arch or under the first metatarsal head. Pain, however, may not be the predominant symptom. The dancer may report of difficulty in performing specific routines, for example jumping and landing, and pointing and stretching the leg in a straight line [15]. Other symptoms include swelling posterior to the medial malleolus, snapping and triggering of the great toe if a nodule is present, and tightness of the MTP or IP joints in "pseudo hallux rigidus", a condition caused by reduced distal excursion of the FHL tendon as a result of nodular tenosynovitis occurring at the leading edge of fibro-osseous tunnel [2, 36]. In a study comparing dancers with nondancers with FHL symptoms, dancers were found to experience three times as long duration of symptoms than nondancers [15].

In the clinical examination, the FHL should be directly palpated at four different sites to elicit tenderness, crepitus, or nodular thickening [36]. The sites are medial retro-malleolar, inferior to the ridge of the sustentaculum tali, plantar to the navicular and medial cuneiform in the mid-foot, and between the sesamoids [2]. Maintaining local pressure on a tender point whilst passively moving the great toe helps to isolate the finding to the FHL tendon. If there is reduced dorsiflexion at the first MTP joint, the FHL stretch test should be performed to assess if the FHL is accountable for this restriction. In this test, the great toe is dorsiflexed while the ankle is first positioned in maximal plantar flexion, and then in dorsiflexion. A decrease in MTP joint dorsiflexion to less than 20° with the ankle dorsiflexed is a positive test [2].

The passage of the FHL tendon is intimately related with that of the os trigonum, posterior tibial tendon, FDL tendon, plantar fascia, and MTP joint. It is important to exercise good clinical judgment to accurately diagnose FHL disorders from the other commonly injured structures, particularly when signs and symptoms overlap when the clinical entities coexist.

---

## Imaging

Ankle and foot weight bearing radiographs may reveal bony sources of pain, for example, the presence of os trigonum in posterior impingement syndrome, os navicularis, hallux rigidus, and pes planovalgus in context of posterior tibial tendon dysfunction pain.

Further to radiographs, magnetic resonance imaging (MRI) may be valuable to establish FHL pathology if the clinical diagnosis is unclear, or help to direct surgical intervention. Excess fluid surrounding the FHL tendon in tenosynovitis is recognized as increased signal on T2-weighted MR images [37, 38]. It is important to remember that in asymptomatic individuals, the FHL sheath may contain small amounts of fluid as the sheath is in continuity with the ankle joint [32, 37]. Other MRI findings include tendon swelling, split tears, and complete ruptures.

---

## Management

### Nonoperative

Athletes with FHL tenosynovitis should first undergo a regime of nonoperative treatment lasting for up to 6 months [1]. Flexibility of the FHL may be increased through repetitive and progressive sets of dorsiflexion exercises at the ankle and first MTP joints. If symptoms do not improve after 6 weeks, night splinting is introduced to prevent FHL contracture. Full-time immobilization may be necessary if stretching and night splinting have failed. Oral non-steroidal anti-inflammatory drugs should be introduced from the start of treatment [2]. Local infiltration of corticosteroids into the tendon sheath and oral steroids should be avoided [2, 36]. Every effort should be made to avoid specific pain-exacerbating motions during training. Unfortunately, conservative treatment has been shown to be ineffective in 40–100 % of cases [4, 7, 15, 39].

### Operative

Surgery is indicated when all conservative measures have failed to sufficiently improve the athlete's symptoms for return to full activity. The type of surgery should be based upon FHL pathology, anatomic site, coexisting pathology, and surgeon's familiarity. In general, the components of surgery are tenosynovectomy, tendon debridement and repair, and release of constriction; aimed at accomplishing pain relief and full return to sports.

In terms of technique, open or arthroscopic approaches may be considered.

As FHL tenosynovitis most commonly originate at the fibro-osseous tunnel, different open approaches to this anatomical region have been described. The widely used medial approach enables direct visualization of the contents of the tarsal canal, hence the tibial nerve and posterior tibial artery can be isolated and retracted during FHL decompression at the posteromedial ankle [3, 7–9, 11, 13, 15, 36]. In this approach, it is possible to perform the FHL release very distally to the level of the sustentaculum tali [8]. The posterior

approach is excellent for exposure of the proximal part of the fibro-osseous tunnel, allowing for any impinging structures in the posterior ankle, for example, the os trigonum, to be addressed directly. This safe approach avoids dissection of structures medial to the FHL thereby minimizing their risk of injury, and avoids iatrogenic sural nerve problems since the approach starts medial to the Achilles tendon [2].

In posterior ankle arthroscopy, two portals are created medial and lateral to the border of the Achilles tendon at 1 cm proximal to calcaneal tuberosity. Normally, the lateral portal is for visualization with a 4.0 mm, 30° arthroscope, and the medial portal is the working portal [32]. There is significant risk to the sural nerve and its lateral calcaneal branch when creating the posterolateral portal [1]. Conversely, the medial neurovascular bundle is in danger if instruments are not directed laterally when utilizing the posteromedial portal. One study demonstrated that debridement, os trigonum excision, and FHL decompression can be accomplished with arthroscopy to yield results that are good to excellent in 80 % of patients with posterior ankle impingement [40].

The benefits and risks of open versus arthroscopic surgery continue to be debated. Proponents of arthroscopic treatment of FHL tenosynovitis believe that the minimally invasive technique allows good visualization and is effective in producing good results [32]. Potential benefits include decreased wound complications and earlier return to work and sports [1, 40]. However, hindfoot arthroscopy is technically demanding and associated with operative morbidities that include iatrogenic injury to the FHL tendon and neurovascular structures, and incomplete release of the distal end of the FHL fibro-osseous tunnel [1]. The open approach is generally believed to be reliable and safer. In one series, all patients who underwent open FHL release and synovectomy had significant improvement within the first 6 weeks and no complications [2]. In another study, 23 out of 26 patients who had open FHL release and repair had good to excellent results [15]. Complications of open surgery are usually wound-related, with a rate of nearly 17 % reported in one study [8].

---

## Rehabilitation

Injuries in dancers are believed to stem from a synthesis of incorrect technique, dysfunctional foot and ankle biomechanics, and other specific risk factors [13]. Recognition and correction of these contributing factors are fundamental. Neuromuscular re-training is paramount and ideally delivered by a multi-disciplinary team comprising an orthopaedic surgeon, a sports medicine physician, sports physiotherapists and podiatrists, and with involvement of the athlete's trainer.

The postoperative athlete will benefit from sport-specific rehabilitation to optimize his or her surgical outcome. In general, following FHL release, the patient may bear weight as tolerated in a removable walking boot for 2 weeks. In the

next 3 weeks, the patient should start active and passive motion exercises to increase FHL flexibility, with resistance strength training following on closely. In the subsequent 4 weeks, the patient should begin neuromuscular exercises comprising balance, proprioception, co-ordination, and gait training. By postoperative week 10, the patient may consider gradual return to sports.

## Prevention

The literature is scarce pertaining to prevention of FHL overuse injuries in the athlete. The athlete, trainer, and healthcare professionals should have precise understanding of the sport and awareness of common injuries specific to the sport. Collaborative efforts to address technical pitfalls in the athlete's day-to-day training regime, and adopt correct physical conditioning including maintenance of full range of joint motion, muscle power, balance, coordination, and general fitness, are fundamental in injury prevention [41, 42].

## Summary

FHL disorders tend to be overuse injuries that afflict dancers, but may also occur after trauma, and in non-dance athletes. Posteromedial ankle pain is the commonest symptom, arising from FHL disease within the fibro-osseous tunnel of the hindfoot. A high degree of suspicion will aid in differentiating FHL disease from other co-existing pathology. Conservative treatment focuses on FHL stretching. Surgery is reserved for athletes whose symptoms are disabling and refractory to nonoperative measures. Open and arthroscopic approaches each have advantages and disadvantages, but there is still no consensus regarding the superiority of one over the other. Cornerstones of rehabilitation are neuromuscular re-education, addressing faulty sport technique, and correct physical conditioning to minimize recurrent injuries.

## References

- Keeling JJ, Guyton GP. Endoscopic flexor hallucis longus decompression: a cadaver study. *Foot Ankle Int.* 2007;28:810–4.
- Michelson J, Dunn L. Tenosynovitis of the flexor hallucis longus: a clinical study of the spectrum of presentation and treatment. *Foot Ankle Int.* 2005;26:291–302.
- Hamilton WG. Foot and ankle injuries in dancers. *Clin Sports Med.* 1988;7:143–73.
- De Asla RJ, O'Malley M, Hamilton WG. Flexor hallucis tendonitis and posterior ankle impingement in the athlete. *Tech Foot Ankle Surg.* 2002;1:123–30.
- Garth WP. Flexor hallucis tendonitis in a ballet dancer. *J Bone Joint Surg.* 1981;63A:1489.
- Hamilton WG. Tendonitis about the ankle joint in classical ballet dancers. *Am J Sports Med.* 1977;5:84–8.
- Hamilton WG, Chao W. Posterior ankle pain in athletes and dancers. *Foot Ankle Clin.* 1999;4:811–32.
- Hamilton WG, Geppert MJ, Thompson FM. Pain in the posterior aspect of the ankle in dancers: differential diagnosis and operative treatment. *J Bone Joint Surg.* 1996;78A:1491–500.
- Hardaker Jr WT, Margello S, Goldner JL. Foot and ankle injuries in theatrical dancers. *Foot Ankle.* 1985;6:59–69.
- Howse AJ. Posterior block of the ankle joint in dancers. *Foot Ankle.* 1982;3:81–4.
- Kolletis GJ, Micheli LJ, Klein JD. Release of the flexor hallucis longus tendon in ballet dancers. *J Bone Joint Surg.* 1996;78A:1386–90.
- Marotta JJ, Micheli LJ. Os trigonum impingement in dancers. *Am J Sports Med.* 1992;20:533–6.
- Solomon R, Brown T, Gerbino PG, Micheli LJ. The young dancer. *Clin Sports Med.* 2000;19:717–39.
- Coghlan BA, Clarke NMP. Traumatic rupture of the flexor hallucis longus tendon in a marathon runner. *Am J Sports Med.* 1993;21:617–8.
- Sammarco GJ, Cooper PS. Flexor hallucis longus tendon injury in dancers and nondancers. *Foot Ankle Int.* 1998;19:356–62.
- Holt KWG, Cross MJ. Isolated rupture of the flexor hallucis longus tendon: a case report. *Am J Sports Med.* 1990;18:645–6.
- Romash MM. Closed rupture of the flexor hallucis longus tendon in a long distance runner: report of a case and review of the literature. *Foot Ankle Int.* 1994;15:433–6.
- Inokuchi S, Usami N. Closed complete rupture of flexor hallucis longus tendon at the groove of the talus. *Foot Ankle Int.* 1997;18:47–9.
- Krackow KA. Acute traumatic rupture of a flexor hallucis longus tendon: a case report. *Clin Orthop Relat Res.* 1980;150:261–2.
- Trepman A, Mizel MS, Newberg AH. Partial rupture of the flexor hallucis longus tendon in a tennis player: a case report. *Foot Ankle Int.* 1995;16:227–31.
- Henry AK. *Extensile exposure*. 2nd ed. Edinburgh: Churchill-Livingstone; 1973. p. 300–8.
- Peterson W, Pufe T, Zantop T, Paulsen F. Blood supply of the flexor hallucis longus tendon with regard to dancer's tendinitis: injection and immunohistochemical studies of cadaver tendons. *Foot Ankle Int.* 2003;24:591–6.
- Hintermann B, Nigg BM, Sommer C. Foot movement and tendon excursion: an in vitro study. *Foot Ankle Int.* 1994;15:386–95.
- Klein P, Mattys S, Rooze M. Moment arm length variations of selected muscles acting on talocrural and subtalar joints during movement: an in vitro study. *J Biomech.* 1996;29:21–30.
- Spoor CW, van Leeuwen JL, Meskers CG, Titulaer AF, Huson A. Estimation of instantaneous moment arms of lower-leg muscles. *J Biomech.* 1990;23:1247–59.
- Bojsen-Moller F, Lamoreux L. Significance of free-dorsiflexion of the toes in walking. *Acta Orthop Scand.* 1979;50:471–9.
- Mann RA, Coughlin MJ, DuVries HL. Hallux rigidus: a review of the literature and a method of treatment. *Clin Orthop Relat Res.* 1979;142:57–63.
- Wickiewicz TL, Roy RR, Powell PL, Edgerton VR. Muscle architecture of the human lower limb. *Clin Orthop Relat Res.* 1983;179:275–83.
- Ferris L, Sharkey NA, Smith TS, Matthews DK. Influence of extrinsic plantar flexors on forefoot loading during heel rise. *Foot Ankle Int.* 1995;16:464–73.
- Hamel AJ, Donahue SW, Sharkey NA. Contributions of active and passive toe flexion to forefoot loading. *Clin Orthop Relat Res.* 2001;393:326–34.
- Kirane YM, Michelson JD, Sharkey NA. Contribution of the flexor hallucis longus to loading of the first metatarsal and first metatarsophalangeal joint. *Foot Ankle Int.* 2008;29:367–76.
- Corte-Real NM, Moreira RM, Guerra-Pinto F. Arthroscopic treatment of tenosynovitis of the flexor hallucis longus. *Foot Ankle Int.* 2012;33:1108–12.

33. Boruta PM, Beaperthy GD. Partial tear of the flexor hallucis longus at the knot of Henry: presentation of three cases. *Foot Ankle Int.* 1997;18:243–6.
34. Gould N, Schneider W, Ashikaga T. Epidemiology survey of foot problems in the continental United States, 1978–1979. *Foot Ankle.* 1980;1:8–10.
35. Sanhudo JAV. Stenosing tenosynovitis of the flexor hallucis longus tendon at the sesamoid area. *Foot Ankle Int.* 2002;23:801–3.
36. Hamilton WG. Stenosing tenosynovitis of the flexor hallucis longus tendon and posterior impingement upon the os trigonum in ballet dancers. *Foot Ankle.* 1982;3:74–80.
37. Link SC, Erickson SJ, Timins ME. MR imaging of the ankle and foot: normal structures and anatomic variants that may simulate disease. *Am J Roentgenol.* 1993;161:607–12.
38. Rosenberg ZS, Cheung Y, Jahss MH. Computed tomography scan and magnetic resonance imaging of ankle tendons: an overview. *Foot Ankle.* 1988;8:297–307.
39. Abramowitz Y, Wollstein R, Barzilay Y, London E, Matan Y, Shabat S, Nyska M. Outcome of resection of a symptomatic os trigonum. *J Bone Joint Surg.* 2003;85A:1051–7.
40. van Dijk CN. Anterior and posterior ankle impingement. *Foot Ankle Clin North Am.* 2006;11:663–83.
41. Khan K, Brown J, Way S, Vass N, Crichton K, Alexander R, Baxter A, Butler M, Wark J. Overuse injuries in classical ballet. *Sports Med.* 1995;19(5):341–57.
42. Koutedakis Y, Jamurtas A. The dancer as a performing athlete: physiological considerations. *Sports Med.* 2004;34(10):651–61.

Travis J. Dekker, Alexander J. Lampley,  
Jonathan A. Godin, and Mark E. Easley

## Abstract

Ankle ligament injuries are common amongst athletes. Low ankle sprains typically involve the anterior talofibular ligament and the calcaneofibular ligament while high ankle sprains involve the syndesmosis. These injuries present with focal tenderness, swelling over the involved ligaments, and pain with weight bearing. Specialized physical exam maneuvers and radiographic imaging can aid in the diagnosis of these injuries. Non-operative management includes immobilization followed by stretching, proprioception training and peroneal strengthening. Furthermore, ankle braces and proprioception training prevent repeat injury. Surgical management includes multiple procedures ranging from ankle arthroscopy to anatomic reconstruction.

## Keywords

Acute • Ankle • Instability • Sprain • Treatment

## Introduction

Injuries to the ankle are the most common reason for missed participation in athletics, accounting for 14 % of all sports injuries [1]. Moreover, ankle ligament sprains are the most common injury in college athletes, and soccer, volleyball, basketball and long distance running demonstrate the highest injury prevalence [2]. The most common ankle injury is a sprain to the lateral ankle ligamentous complex, which involves the anterior talofibular ligament (ATFL) and calcaneofibular ligament (CFL) with more severe injuries extending into the posterior talofibular ligament (PTFL). High ankle sprains, those that extend into the syndesmosis, are commonly seen in sports requiring the ankle to be held in a fixed position, such as skiing or hockey [3, 4]. These are encountered less often with stated incidence of 15 high ankle

sprains per 100,000 per year [1]. Financial ramifications exist due to loss of playing or work time along with the extensive associated medical costs [5]. Ankle sprains lead to an array of disability ranging from pain, swelling and ecchymosis to muscle weakness, decreased proprioception, inability to bear weight, and even fracture [6]. Non-operative management consisting of immobilization followed by stretching, proprioception and peroneal strengthening remains the mainstay of care for the majority of patients with acute ankle instability [7]. Insufficient rehabilitation and inadequate treatment can lead to residual symptoms and, potentially, chronic ankle instability (CAI) and permanent disability [8, 9].

## Etiology and Pathomechanism

The lateral ligamentous complex consists of the ATFL, CFL and PTFL. The ATFL extends from the distal anteroinferior border of the fibula to the neck of the talus. The CFL extends from the fibula to the lateral tubercle of the calcaneus, and the PTFL runs from the digital fossa on the posterior aspect of the fibula to the lateral tubercle of the talus [5]. The ATFL

T.J. Dekker, MD • A.J. Lampley, MD • J.A. Godin, MD, MBA  
M.E. Easley, MD (✉)  
Department of Orthopaedic Surgery, Duke University Medical  
Center, 4709 Creekstone Drive, Durham, NC 27703, USA  
e-mail: [travis.dekker@duke.edu](mailto:travis.dekker@duke.edu); [alexander.lampley@dm.duke.edu](mailto:alexander.lampley@dm.duke.edu);  
[jonathan.godin@duke.edu](mailto:jonathan.godin@duke.edu); [mark.e.easley@dm.duke.edu](mailto:mark.e.easley@dm.duke.edu)

ranges from 20 to 25 mm long, 7–10 mm wide and 2 mm thick [10]. The ATFL prevents anterior displacement and internal rotation of the talus, most noticeably when the talocrural joint is plantarflexed [11]. The ATFL is universally the weakest ligament within the lateral ligamentous complex with reported loads to failure ranging from 138.9 to 300 N [12–15]. Meanwhile, the PTFL is twice as strong with failure loads between 261–407 N, while the CFL demonstrates a load to failure between 307 and 345 N [12–15].

Due to its anatomic location and biomechanical characteristics, the ATFL is the most commonly injured ligament in an inversion type ankle injury, which occurs when the foot is placed in a position of supination while the foot is plantar flexed [16]. Fuller further added that the injury occurs due to an increased supination moment at the subtalar joint, often a result of the position and magnitude of the vertically projected ground reaction force at initial foot contact [17]. Positioning of the foot can increase risk of sprain as well. Wright demonstrated that plantar flexion increases the moment arm of the subtalar axis with resultant torque leading to increased risk of inversion injuries placing maximal strain on the lateral ankle ligaments [18]. Another proposed theory for lateral ankle sprains is based on the belief that the peroneal muscles do not have enough time to overcome the vertical ground reaction force, thereby causing the inversion force. Ashton-Miller et al. demonstrated peroneal muscle reaction time to be 50 ms with the vertical ground reaction force occurring within 40 ms [19]. Patients subjected to these forces are likely to be participants in indoor and court sports. Women sustain ankle sprains at nearly twice the rate of men (13.6 per 1000 exposures versus 6.94 per 1000 exposures) [8]. Lastly, children and adolescents are more likely to sustain lateral ankle injury/inversion sprains than their adult counterparts [8].

The ankle syndesmosis consists of the anterior-inferior tibiofibular ligament (AITFL), interosseous ligament (IL), posterior-inferior fibular ligaments (PIFL), and inferior transverse tibiofibular ligament (ITFL) [20]. The lateral ligamentous complex, the ankle syndesmotic structures and the superficial and deep deltoid ligaments act to prevent diastasis of the fibula from the tibia. The AITFL originates at the longitudinal tubercle of the lateral malleolus and attaches to the anterolateral tubercle of the tibia. The PITFL originates on the posterior tubercle of the tibia and attaches to the posterior lateral malleolus. The inferior transverse ligament passes from the posterior tibial margin to the osteochondral junction on the distal fibula, thereby preventing posterior talar translation. The deltoid ligament complex prevents eversion of the subtalar joint. The interosseous membrane stabilizes the tibia and fibula, resisting posterolateral bowing of the fibula [21]. Stability is often difficult to first assess in the patient with a high ankle sprain. If all ligaments of this complex are disrupted, then instability is clear and surgical intervention is required. However, if disruption of individual components of

this complex, namely the anterior ligaments, is not present, then surgical intervention is not often needed [22].

Disruption of the syndesmosis is commonly linked to abrupt eversion injuries about the ankle. Often times, disruption of the syndesmosis occurs concurrently with a fracture of the fibula. The mechanism of injury is based on sudden external rotation of the ankle while the foot is in a locked dorsiflexed position. This commonly occurs in sports requiring the participants' foot to be locked in a boot, such as hockey or skiing. The first ligaments to fail are usually the AITFL and the superficial deltoid ligament, and a syndesmotic requires higher energy compared to a sprain localized to the lateral ligamentous complex [21]. This portends to long term disability, longer rehabilitation and delayed return to play.

---

## Symptoms and Diagnosis

Athletes often report an injury after “rolling” their ankle when taking an awkward step while running or landing after a jump. Acute disability varies from minimal pain about the lateral ankle with continued ability to perform their respective sport to complete disability with inability to bear weight or ambulate. Clinical signs of ecchymosis and edema localized to the lateral malleolus often aid in diagnosis of acute lateral ankle sprains [23]. Specific areas of tenderness may aid in diagnosis of specific ligamentous damage and should be well-documented. In the most severe of inversion ankle sprains, peroneal nerve injury has been seen, often documented as a neuropraxia that resolves with time [24]. Multiple other sports related injuries mimic a routine lateral ankle sprain. A fracture of the anterior process of the calcaneus has been coined the “sprain fracture” as many of the symptoms seen with this injury are similar to those of a lateral ankle sprain. Furthermore, a complete work-up should rule out subtalar dislocation and avulsion fractures of the fifth metatarsal [23].

Numerous authors have documented various approaches to diagnosing and assessing a lateral ankle sprain. Harmon et al. described a set of rules to best assess a lateral ankle sprain: (1) Palpate bony structures, (2) palpate ligamentous structures, (3) assess range of motion, (4) test muscles of ankle and (5) perform special tests [25]. Fracture must first be ruled out; this is done by obtaining ankle x-rays and foot x-rays as guided by the Ottawa Ankle rules. The Ottawa Ankle rules have demonstrated 100% sensitivity, and they state that radiographs should be obtained if: (1) tenderness is elicited about the distal 6 cm of the posterior aspect of the fibula or (2) tenderness is elicited about the distal 6 cm of the posterior aspect of the tibia or (3) there is an inability to bear weight immediately after injury or inability to bear weight for four steps in the Emergency Department [26]. Once fracture has been ruled out, an anterior drawer test can be performed to aid in grading the severity of the sprain.

The anterior drawer is performed with the knee flexed  $\sim 20^\circ$ , the hindfoot in neutral, and the ankle in neutral dorsiflexion while applying an anterior force about the heel to test anterior translation of the talus. Laxity with respect to the contralateral limb suggests ATFL injury. The CFL can be assessed by an inversion test, which determines the amount of talar tilt and pain present with the hindfoot inverted while the talocrural joint is maintained in dorsiflexion [27].

Syndesmotic injuries demonstrate a different pattern of pain compared to a classic ankle sprain. As previously described, the patient will describe a vastly different mechanism of injury with the primary driving force being eversion of the foot and ankle. Pain can be elicited by a squeeze test by compressing the fibula to the tibia in the proximal third of the lower leg. However, swelling is not a common finding in an isolated syndesmotic injury and can often lead to misdiagnosis [28, 29]. Pain can be seen localized to the AITFL along with difficulty in active or passive external rotation of the foot. The external rotation test is performed by using one hand to stabilize the foot and apply an external rotation force while the other hand is used to stabilize the leg. Pain about the syndesmosis and tibiofibular joint separation suggest a positive test. This test has been determined to be more specific than the aforementioned squeeze test [30]. Gait changes occur as well from a typical heel-toe pattern to a heel-raise gait pattern, which prevents dorsiflexion of the ankle and decreases pain with push-off [29].

Injury to the deltoid ligament can be seen frequently in the setting of syndesmotic injury. Pain with valgus stress to the hindfoot or tenderness to palpation of the medial malleolus necessitates stress radiography, on which 1 mm of syndesmosis widening demonstrates instability [31]. Concurrent injury of both structures indicates a highly unstable ankle mortise that will require surgical stabilization. A simple AP radiograph can demonstrate injury to the syndesmosis utilizing certain measurements. With all measurements being obtained 1 cm proximal to the joint line, tibiofibular overlap should be greater than 6 mm or more than 42% of the width of the fibula, and the tibiofibular clear space should be less than 6 mm [32]. An undiagnosed unstable ankle joint has dire consequences predisposing the ankle to arthritis secondary to abnormal joint reactive forces, chronic pain and osteochondral lesions [22]. MRI is not necessary to diagnose acute ankle instability, though advanced imaging can be obtained when history and physical examination do not yield a clear diagnosis or when the physician is concerned about concomitant injury.

---

## Classification

Multiple grading schemes have been described in the setting of acute lateral ligamentous ankle sprains. The Anatomic System divides the injury into three grades

according to the ligaments that have been damaged. The American Medical Association Standard Nomenclature takes into account the severity of the injury to the individual ligaments [33]. Kaikkonen et al. devised a dynamic functional grading scheme. A performance test protocol with an associated scoring scale was based on three subjective responses, two clinical measurements of the ankle, two muscle strength tests, one functional stability test and one balancing test. This scheme is practical for clinical evaluation of ankle sprains as the total score correlates with isokinetic strength, subjective recovery and subjective functional assessment [34]. Clanton et al. devised a scheme with therapeutic implications. Ankle injuries were first divided into stable and unstable injuries. The unstable group was then divided into non-athletes and older patients and those who were considered young active athletes. The athlete group was further divided into three patient groups: injuries with negative stress radiographs, injuries demonstrating positive tibiotalar instability and injuries with subtalar instability [33]. Clanton et al. recommended patients with tibiotalar instability pursue surgical repair of the disrupted ligamentous complex [33].

Porter et al. has classified syndesmotic ankle injuries and further described the complications associated with untreated or undertreated unstable syndesmosis injuries [35]. This group categorized syndesmotic injuries into three gradations and their associated instability. Grade I involves injury to the anterior deltoid ligament and the distal interosseous ligament without tearing of the more proximal syndesmosis or the deep deltoid ligament. The AITFL is typically tender to palpation and may have a higher-grade injury. There is no diastasis, and, by definition, Grade I injuries are stable. Grade II injuries include disruption of the anterior and deep deltoid ligaments along with a tear throughout a significant portion of the syndesmosis. This results in an unstable ankle that is well-aligned on non-stress radiographs. Grade II injuries are difficult to assess because of the lack of injury extent and occult instability. Last, a Grade III syndesmotic injury occurs due to severe external rotation and abduction with complete disruption of the medial ligaments and extensive disruption of the syndesmosis frequently accompanied by fracture of the proximal fibula (Maisonneuve fracture). Grade III injuries are typically obvious on plain film x-rays [35].

Sikka et al. described an imaging classification utilizing MRI for syndesmotic injuries [36]. Grade I injuries are isolated to the AITFL; Grade II injuries include the AITFL and the interosseous ligament; Grade III injuries include the AITFL, interosseous ligament and PITFL; Grade IV injuries include the AITFL, interosseous ligament, PITFL and the deltoid ligament. This classification scheme was used to evaluate high ankle sprains in American football players with increased grade of injury correlating with increased number of missed games and practices [36].



## Therapy

### Non-operative Management

Many treatment options for ankle sprains have been suggested, including surgery, immobilization, functional treatment with bandages, tape or different braces, and balance training. Most authors recommend non-surgical treatment for lateral ankle sprains, and treatment should begin with restricted activity and physical therapy. Physical therapy should focus on stretching, proprioception and peroneal strengthening [37]. The most common non-operative treatment modalities used to manage grade III acute lateral ankle ligament injury are immobilization and functional management [37]. Immobilization usually entails a brief period ( $\leq 3$  weeks) in a below knee walking cast, splint or CAM boot. This immobilization period is followed by up to 12 weeks of proprioceptive rehabilitation. Although immobilization may not be advantageous in the high-performance athlete, it may have a limited role in the low demand patient who is unable to bear weight through the affected limb immediately following a severe acute sprain.

Functional management includes early mobilization with external support (i.e. bracing), as well as a period of rest, ice, compression and elevation. After immediate management, a rehabilitation program comprising ROM exercises, strengthening, proprioception, and activity-specific training should be initiated. Proprioception training, which is essential for the recovery of balance control, consists of a series of progressive drills on devices such as wobble boards and trampolines. In addition to providing mechanical stability, external supports also provide proprioceptive feedback and thus aid in rehabilitation. Ardèvol et al. conducted a randomized controlled trial comparing cast immobilization with functional management in patients engaged in regular sports activity [38]. Functional management allowed earlier resumption of sports training, with fewer symptoms at 3 and 6 months post-injury [38]. A greater reduction in objective radiographic laxity with functional management was noted, but there was no difference in re-injury rates found between the two management groups [38].

Functionally managed patients also have been shown to have a higher rate of satisfaction than patients treated with cast immobilization [39]. In a systematic review of nine studies on functional management, Kerkhoffs et al. concluded that lace-up supports were most effective, tapes were associated with skin irritation and were no better than semi-rigid supports, and that elastic bandages were the least effective form of management [40].

One study examined the role of platelet-rich plasma (PRP) in the treatment of high ankle sprains in 16 competitive athletes [41]. This report demonstrated that patients

injected with PRP were able to return to sport at a mean 40.8 days, while those in the control group returned to sport at a mean 59.6 days [41]. Patients in the PRP group also had statistically significantly lower pain levels upon returning to athletic activity.

### Surgical Management

Surgical intervention in acute ligament injuries remains controversial. Relative contraindications for operative management include the following: pain without instability, neuropathy, peripheral vascular disease, and noncompliance.

#### Preoperative Planning

Patients should be evaluated for a tarsal coalition. Moreover, hindfoot alignment should be assessed, as varus alignment predisposes to inversion injuries. Therefore, a Dwyer calcaneal osteotomy may need to be performed in addition to a lateral ligament repair. Concomitant peroneal tendon injuries should also be evaluated and addressed at the time of operative intervention.

#### Positioning

Patient positioning for lateral ankle ligament repair or reconstruction is based on the chosen procedure. For anatomic ligament repair, the patient may be placed supine with a bump under the operative hip, or the patient may be placed in the semi-lateral or lateral decubitus positions. Patient undergoing concomitant ankle arthroscopy should be placed supine with a bump placed under the ipsilateral hip following the arthroscopic portion of the procedure.

#### Approach

The incision for the Brostrom-Gould procedure is J-shaped and based just anterior to the fibula to allow easy access to both the ATFL and the CFL. Another option is a larger curvilinear incision from proximal-posterior (4–5 cm proximal to the fibular tip) to anterior-distal along the course of the peroneal tendons allows for access to both the lateral ankle ligaments and the peroneal tendons.

### Techniques

#### Modified Brostrom Lateral Ligament Repair

In 1966, Brostrom reported a series of patients who underwent a direct repair of the ATFL and CFL by mid-substance suturing of the torn ligaments [42]. In 1980, Gould modified this procedure by advancing the lateral aspect of the inferior extensor retinaculum to the fibula, thereby reinforcing the repair of the ATFL [42]. Upon incision, care should be made to avoid the superficial peroneal and sural nerves. Anterior

and posterior flaps are created as dissection is carried down to the layer of the fibular periosteum. The anterolateral ankle capsule, peroneal tendons and inferior extensor retinaculum are identified. The peroneal sheath can be opened at this time to address peroneal tendon pathology as needed, though care should be made to maintain the integrity of the superior peroneal retinaculum. The lateral ankle gutter is then identified before the capsule is divided. A cuff of tissue is maintained on the fibula for later advancement and imbrication. The arthrotomy is then extended from the level of the tibiotalar joint to the peroneal tendons, as this will divide both the ATFL and the CFL and allow for evaluation of the ankle joint [42]. The ligaments are then imbricated in a pants-over-vest fashion with 0-vicryl. The ankle should be held in eversion and dorsiflexion as the sutures are tied down [42]. The extensor retinaculum is then advanced to the fibular periosteum, covering the ligament and capsular repair. The subcutaneous and skin layers are then closed before splinting the ankle in slight eversion.

### **Modified Brostrom Lateral Ligament Repair with Suture Anchors**

Upon incision, care should be made to avoid the superficial peroneal and sural nerves. Anterior and posterior flaps are created as dissection is carried down to the layer of the fibular periosteum. The anterolateral ankle capsule, peroneal tendons and inferior extensor retinaculum are identified. The peroneal sheath can be opened at this time to address peroneal tendon pathology as needed, though care should be made to maintain the integrity of the superior peroneal retinaculum. The lateral ankle gutter is then identified before the capsule is divided. A cuff of tissue is maintained on the fibula for later advancement and imbrication. The arthrotomy is then extended from the level of the tibiotalar joint to the peroneal tendons, as this will divide both the ATFL and the CFL and allow for evaluation of the ankle joint. The anterior inferior tibiofibular ligament (Bassett's ligament) is then excised, as this may lead to anterolateral impingement. After any intra-articular work is completed, attention is turned to creating a trough with a rongeur in the anterior distal fibula. Suture anchors are then placed at the anatomic footprints for the ATFL and CFL [42]. Care should be taken to avoid violation of the joint and the posterior fibular cortex, as this may irritate the peroneal tendons. Suture limbs are then passed through the anterior capsule, ATFL, and CFL [42]. The ankle mortise is then reduced with care taken to avoid anterior subluxation of the talus. Moreover, the ankle is placed in neutral dorsiflexion and slight hindfoot valgus. The sutures are then tied down. Next, pass the sutures through the distal fibula periosteal flap for additional reinforcement before advancing the inferior extensor retinaculum (Gould modification) while protecting the peroneal tendons.

### **Modified Brostrom-Evans Procedure**

This procedure is a combination of the modified Brostrom lateral ligament repair described above and the Evans procedure, which involves tenodesing the anterior 50% of the peroneus brevis to the fibula. This procedure is indicated for patients in whom greater resistance against inversion is desired. For example, this procedure is appropriate for patients with lateral ankle instability and concomitant peroneus brevis tendon tear. The initial portions of the modified Brostrom are the same as described above. After releasing the ATFL, the CFL and the capsule, the peroneus brevis tendon is isolated proximal and distal to the superior peroneal retinaculum [42]. After splitting the peroneus brevis tendon in a longitudinal fashion, the anterior 50% of the tendon is passed beneath the superior peroneal retinaculum distally [42]. The tendon can be split with suture replicating a saw to cleanly divide the tendon fibers. Next, a bone tunnel is drilled obliquely through the distal fibula, and the anterior tendon limb is passed through the tunnel in a distal to proximal fashion [42]. Thereafter, the modified Brostrom procedure is completed. When closing the fibular periosteum, the anterior limb of the peroneus brevis tendon should be sutured to the periosteum. The subcutaneous and skin layers are then closed.

### **The Role of Ankle Arthroscopy in Acute Ankle Instability**

Acute arthroscopic assessment of the ankle joint may be performed in conjunction with acute lateral ligament repair or reconstruction. It is well-documented that intra-articular joint surface damage is common in the sprained ankle, and much of this damage is confined to the chondral surface and does not involve underlying bone [43]. Ankle arthroscopy is indicated in patients with talar osteochondral lesions, anterior impingement lesions and exostoses. In acute ankle instability, MRI does not always show tibiotalar lesions. Komenda and Ferkel reported an incidence of up to 93% of intra-articular disease in patients undergoing arthroscopy immediately before lateral ligament repair, whereas others have shown as little as 40% sensitivity of MRI to coexistent intra-articular lesions after lateral ligament injury [44, 45]. Although most of these studies relate to the chronic situation, it is reasonable to assume that a percentage of acutely diagnosed lateral ligament injuries undergoing early surgery have combined intra-articular disease either not visible on MRI or obscured by acute edema/hemorrhage. Therefore, acute ankle arthroscopy in these cases has potentially significant benefits in detecting lesions that may prevent or hamper recovery after an otherwise successful ligament repair, but more work is necessitated to clarify this. Recently, some investigators have described novel techniques to achieve lateral ligament stabilization by entirely arthroscopic means, but these procedures remain experimental in the acute setting [44].

## Post-operative Care

The patient is to remain non-weightbearing for 10–14 days until their first post-operative clinic visit. At the first post-operative clinic visit, the splint is taken down, and the wound is carefully inspected. If no signs of wound dehiscence or infection are present, then the sutures are removed, and a short leg cast is applied for the next 4–5 weeks. At the next post-operative visit, the cast is removed, and a physical therapy program is initiated that stresses range of motion, proprioception, peroneal strengthening and resistive strength training.

## Risks/Complications

Be sure to evaluate hindfoot alignment, as failure to address varus alignment can lead to failure of ligament repair or reconstruction. Secondary pathology should be assessed, as instability can lead to OCD lesions, peroneal tendinopathy, subtalar instability or other lesions. The most common complications pertain to nerve injury, with an incidence between 7 and 19% [42]. Other complications include the following: infection, wound dehiscence, failure of repair, peroneal weakness, and tibiotalar malreduction.

## Rehabilitation and Back-to-Sports

### Rehabilitation

Post-operative rehabilitation protocols vary, but the following is a general set of guidelines. During the first post-operative phase, physical therapy focuses on proximal lower extremity, upper extremity and core strengthening [46]. Independent transfers and ambulation, as well as pain and edema control, are a focus during the immediate post-operative period. After the initial 6 week immobilization period, progressive weight-bearing is initiated as tolerated, first in a pneumatic walking boot and then transitioned to a semi-rigid ankle stirrup orthotic. Proprioception and eversion against gravity are initiated around 6 weeks post-operatively [47]. Gentle AROM exercises, including inversion, are begun approximately 8–9 weeks following surgery. Proprioception activities involving unilateral stance with eyes open, eyes closed, external perturbations, foam block, rocker board, and ball toss are initiated along the same timeframe. Plyometrics can be worked into the rehabilitation protocol roughly 11–12 weeks following surgery [47]. At 3 months, patients may progress to running as tolerated and return to sports functional progression and testing [46, 47].

## Return to Sport

Gradual return to sport is allowed between 12 and 16 weeks after surgery.

## Prevention

Prevention methods utilize external bracing or proprioceptive/balance training to reduce the likelihood of ankle ligamentous injury. Orthotic devices (i.e. semi-rigid ankle orthosis, laced ankle stabilizer) provide a compressive force that maintains the anatomical alignment of the ankle by holding the talus within the tibiofibular mortise; therefore, the initiation of an ankle inversion injury is prevented [48]. The results of multiple trials [49–51] evaluating ankle orthotics consistently demonstrate a statistically significant reduction in ligamentous ankle injuries in the athletes wearing these devices, especially in athletes with prior ankle sprains. Orthotics are a cost effective method to reduce ankle injuries in athletes; however, ankle braces can cause discomfort and reduce athletic performance in some patients.

Ankle taping is thought to prevent ankle sprains by reducing plantar flexion and inversion of the ankle [52] while also improving ankle proprioception [53]. While in theory this should prevent ankle injury, there is a lack of data demonstrating the effectiveness of ankle taping in reducing injury. Furthermore, ankle taping has been criticized for loosening with physical activity, and loosening has been shown to reduce the original support of the taping by 50% after only 10–30 min of exercise [54].

Proprioceptive/coordination balance board training has been advocated as an alternative to ankle bracing in ankle injury prevention. Many studies [49, 55, 56] have shown this method to decrease the incidence of re-injury in athletes with a history of ligamentous ankle injury. However, these studies have not shown a statistically significant reduction in ankle injuries in athletes without prior history of ankle sprains. While prevention of re-injury in athletes with a history of ankle injury is well supported in the literature, further evidence is required prior to drawing any conclusions regarding the effectiveness of proprioceptive training in the prevention of primary ligamentous ankle injuries.

## Outcomes (Evidence)

Treatment options for grade III acute lateral ankle ligamentous injuries include cast immobilization, functional management and surgical repair. A meta-analysis reviewing 21

randomized controlled trials comparing immobilization versus functional treatment found statistically significant differences in outcomes favoring functional treatment for acute ankle sprains [39]. When compared to immobilization, the functional treatment group showed that a greater number of patients returned to sport in the long term (RR 1.86), a greater number of patients returned to work in the short term (RR 5.75), fewer patients experienced persistent swelling of the ankle (RR 1.74), and more patients were satisfied with their treatment (RR 1.83). No significant difference was found between the two groups in regards to recurrent sprain, long term impaired range of motion, and ankle pain. Thus, multiple randomized controlled trials (Level 1 evidence) support functional treatment rather than immobilization for the treatment of acute lateral ligamentous ankle injuries.

Another meta-analysis compared surgical versus nonsurgical management of acute ankle injuries by reviewing 17 trials [57]. However, all reviewed studies were found to have methodological flaws; and the authors were unable to determine the effectiveness of surgical and conservative management for acute ankle injuries. A more recent randomized controlled trial found statistically significant outcomes favoring surgical intervention [43]. The study followed 317 patients with a mean follow up of 8 years. When compared to functional treatment, the surgically managed group revealed fewer complaints of residual pain (16% versus 25%, RR 0.64), fewer complaints of their ankle 'giving way' (20% versus 32%, RR 0.62) and less recurrent sprains (22% vs. 34%, RR 0.66). In addition, the anterior drawer test was less frequently positive in the surgically treated patients (30% versus 54%, RR 0.54). Overall, this randomized controlled trial (Level 1 evidence) supports better outcomes in the surgically managed group when compared to functional treatment.

In regards to preventing ankle ligament injuries, a meta-analysis [58] reviewing 14 randomized controlled studies found a statistically significant reduction in ankle sprains for patients wearing external ankle orthotics when compared to a control group (RR 0.53). The prevention of ankle sprains was greater in patients with previous history of ankle sprains (RR 0.33). Similarly, coordination/proprioception training in patients with a history of ankle sprain resulted in a significant reduction in ankle sprains when compared to a control group (RR 0.28). Overall, there are multiple randomized controlled studies (Level 1 evidence) supporting the use of external ankle support devices, specifically semi-rigid ankle orthotics to prevent ligamentous ankle injuries during high risk athletic activities. Additionally, athletes with a history of ankle sprain would benefit from coordination/proprioceptive training or external ankle bracing.

## Summary

- Acute lateral ankle sprain is a common sports-related injury.
- The evidence suggests that functional rehabilitation should be the treatment of choice for acute injuries, with acute anatomic repair reserved for high-demand athletes.
- Cast immobilization for a brief period may have a limited role in the low demand individual who is unable to bear weight immediately following injury.
- External ankle bracing with orthotics can reduce the risk of ligamentous ankle injury during sporting events, especially in athletes with a history of ankle sprains. Additionally, proprioceptive training can reduce the risk of recurrent ankle sprains in athletes with history of ankle ligamentous injury.
- Gradual return to sport is usually allowed between 12 and 16 weeks after surgery.

## References

1. Fong DT, Chan YY, Mok KM, Yung PS, Chan KM. Understanding acute ankle ligamentous sprain injury in sports. *Sports Med Arthrosc Rehabil Ther Technol*. 2009;1:14. doi:10.1186/1758-2555-1-14.
2. Hootman JM, Dick R, Agel J. Epidemiology of collegiate injuries for 15 sports: summary and recommendations for injury prevention initiatives. *J Athl Train*. 2007;42:311–9.
3. Wright RW, Barile RJ, Surprenant DA, Matava MJ. Ankle syndesmosis sprains in national hockey league players. *Am J Sports Med*. 2004;32:1941–5.
4. Fritschy D. An unusual ankle injury in top skiers. *Am J Sports Med*. 1989;17:282–5.
5. Ferran NA, Mauffulli N. Epidemiology of sprains of the lateral ankle ligament complex. *Foot Ankle Clin*. 2006;11:659–62.
6. Gerber JP, Williams GN, Scoville CR, Arciero RA, Taylor DC. Persistent disability associated with ankle sprains: a prospective examination of an athletic population. *Foot Ankle Int*. 1998;19:653–60.
7. Tanen L, Docherty CL, Van Der Pol B, Simon J, Schrader J. Prevalence of chronic ankle instability in high school and division I athletes. *Foot Ankle Spec*. 2014;7:37–44.
8. Doherty C, Delahunt E, Caulfield B, Hertel J, Ryan J, Bleakley C. The incidence and prevalence of ankle sprain injury: a systematic review and meta-analysis of prospective epidemiological studies. *Sports Med*. 2014;44:123–40.
9. van den Bekerom MP, Lamme B, Hogervorst M, Bolhuis HW. Which ankle fractures require syndesmotic stabilization? *J Foot Ankle Surg*. 2007;46:456–63.
10. Burks RT, Morgan J. Anatomy of the lateral ankle ligaments. *Am J Sports Med*. 1994;22:72–7.
11. Stephens MM, Sammarco GJ. The stabilizing role of the lateral ligament complex around the ankle and subtalar joints. *Foot Ankle*. 1992;13:130–6.
12. Funk JR, Hall GW, Crandall JR, Pilkey WD. Linear and quasi-linear viscoelastic characterization of ankle ligaments. *J Biomech Eng*. 2000;122:15–22.

13. Attarian DE, McCrackin HJ, DeVito DP, McElhaney JE, Garrett WE. Biomechanical characteristics of human ankle ligaments. *Foot Ankle*. 1985;6:54–8.
14. Siegler S, Block J, Schneck CD. The mechanical characteristics of the collateral ligaments of the human ankle joint. *Foot Ankle*. 1988;8:234–42.
15. Parenteau CS, Viano DC, Petit PY. Biomechanical properties of human cadaveric ankle-subtalar joints in quasi-static loading. *ASME J Biomech Eng*. 1998;120:105–11.
16. Cass JR, Settles H. Ankle instability: in vitro kinematics in response to axial load. *Foot Ankle Int*. 1994;15:134–40.
17. Fuller EA. Center of pressure and its theoretical relationship to foot pathology. *J Am Podiatr Med Assoc*. 1999;89:278–91.
18. Wright IC, Neptune RR, van den Bogert AJ, Nigg BM. The influence of foot positioning on ankle sprains. *J Biomech*. 2000;33:513–9.
19. Ashton-Miller JA, Ottaviani RA, Hutchinson C, Wojtys EM. What best protects the inverted weightbearing ankle against further inversion? Evertor muscle strength compares favorably with shoe height, athletic tape, and three orthoses. *Am J Sports Med*. 1996;24:800–9.
20. Snedden MH, Shea JP. Diastasis with low distal fibula fractures: an anatomic rationale. *Clin Orthop Relat Res*. 2001;382:197–205.
21. Lin CF, Gross ML, Weinhold P. Ankle syndesmosis injuries: anatomy, biomechanics, mechanism of injury, and clinical guidelines for diagnosis and intervention. *J Orthop Sports Phys Ther*. 2006;36:372–84.
22. Zalavras C, Thordarson D. Ankle syndesmotom injury. *J Am Acad Orthop Surg*. 2007;15:330–9.
23. Anderson R, Hunt K, McCormick J. Management of common sports related injuries about the foot and ankle. *J Am Acad Orthop Surg*. 2010;18:546–56.
24. Nitz AJ, Dobner JJ, Kersey D. Nerve injury and grades II and III ankle sprains. *Am J Sports Med*. 1985;13:177–82.
25. Harmon KG. The ankle examination. *Primary Care Clin Office Pract*. 2004;31:1025–37.
26. Stiell I. Ottawa ankle rules. *Can Fam Physician*. 1996;42:478–80.
27. Hertel J. Functional anatomy, pathomechanics and pathophysiology of lateral ankle instability. *J Athl Train*. 2002;37:364–75.
28. Fincher AL. Early recognition of syndesmotom ankle sprains. *Athl Ther Today*. 1999;4:42–3.
29. Brosky T, Nyland J, Nitz A, Caborn DN. The ankle ligaments: consideration of syndesmotom injury and implications for rehabilitation. *J Orthop Sports Phys Ther*. 1995;21:197–205.
30. Ebraheim NA, Lu J, Yang H, Mekhail AO, Yeasting RA. Radiographic and CT evaluation of tibiofibular syndesmotom diastasis: a cadaver study. *Foot Ankle Int*. 1997;18:693–8.
31. Miller CD, Shelton WR, Barrett GR, Savoie FH, Dukes AD. Deltoid and syndesmosis ligament injury of the ankle without fracture. *Am J Sports Med*. 1995;23:746–50.
32. Harper MC, Keller TS. A radiographic evaluation of the tibiofibular syndesmosis. *Foot Ankle*. 1989;10:156–60.
33. Clanton TO. Athletic injuries to the soft tissues of the foot and ankle. In: Coughlin MJ, Mann RA, editors. *Surgery of the foot and ankle*. Mosby: St Louis; 1999. p. 1090–209.
34. Kaikkonen A, Kannus P, Jarvinen M. A performance test protocol and scoring scale for the evaluation of ankle injuries. *Am J Sports Med*. 1994;22:462–9.
35. Porter DA. Ligamentous injuries of the foot and ankle. In: Fitzgerald RH, Kaufer H, Malkani AL, editors. *Orthopaedics*. St. Louis: Mosby; 2002.
36. Sikka RS, Fetzer GB, Sugarman E, Wright RW, Fritts H, Boyd JL, et al. Correlating MRI findings with disability in syndesmotom sprains of NFL players. *Foot Ankle Int*. 2012;33:371–8.
37. Maffulli N, Ferran N. Management of acute and chronic ankle instability. *J Am Acad Orthop Surg*. 2008;16:608–15.
38. Ardèvol J, Bolívar I, Belda V, Argilaga S. Treatment of complete rupture of the lateral ligaments of the ankle: a randomized clinical trial comparing cast immobilization with functional treatment. *Knee Surg Sports Traumatol Arthrosc*. 2002;10:371–7.
39. Kerkhoffs GM, Rowe BH, Assendelft WJ, Kelly K, Struijs PA, van Dijk CN. Immobilisation and functional treatment for acute lateral ankle ligament injuries in adults. *Cochrane Database Syst Rev* 2002;3:CD003762.
40. Kerkhoffs GM, Struijs PA, Marti RK, Blankevoort L, Assendelft WJ, van Dijk CN. Functional treatments for acute ruptures of the lateral ankle ligament: a systematic review. *Acta Orthop Scand*. 2003;74:69–77.
41. Laver L, Carmont MR, McConkey MO, Palmanovich E, Yaacobi E, Mann G, Nyska M, Kots E, Mei-Dan O. Plasma rich in growth factors (PRGF) as a treatment for high ankle sprain in elite athletes: a randomized control trial. *Knee Surg Sports Traumatol Arthrosc*. 2014. doi:10.1007/s00167-014-3119-x.
42. Berlet GC, Landis GS, Hyer CF, Philbin TM. Anatomic repair of lateral ankle instability. In: Wiesel SW, editor. *Operative techniques in orthopaedic surgery*. Philadelphia: Lippincott Williams & Wilkins; 2010. p. 4314–21. Print.
43. Pijnenburg AC, Bogaard K, Krips R, Marti RK, Bossuyt PM, van Dijk CN. Operative and functional treatment of rupture of the lateral ligament of the ankle: a randomised, prospective trial. *J Bone Joint Surg Br*. 2003;85:525–30.
44. Hepple S, Guha A. The role of ankle arthroscopy in acute ankle injuries of the athlete. *Foot Ankle Clin*. 2013;18:185–94. doi:10.1016/j.fcl.2013.02.001.
45. Kemonda GA, Ferkel RD. Arthroscopic findings associated with the unstable ankle. *Foot Ankle Int*. 1999;20:708–13.
46. Mattacola CG, Dwyer MK. Rehabilitation of the ankle after acute sprain or chronic instability. *J Athl Train*. 2002;37:413–29.
47. Edelstein J, Noonan D. Lateral ankle reconstruction. In: Mosca JC, Cahill J, Young C, editors. *Postsurgical rehabilitation guidelines for the orthopedic clinician*. Philadelphia: Mosby, Inc; 2006. Print.
48. Thonnard JL, Bragard D, Willems PA, Plaghki L. Stability of the braced ankle: a biomechanical investigation. *Am J Sports Med*. 1996;24:356–61.
49. Tropp H, Askling C, Gillquist J. Prevention of ankle sprains. *Am J Sports Med*. 1985;13:259–62.
50. Sitler M, Ryan J, Wheeler B, McBride J, Arciero R, Anderson J, Horodyski M. The efficacy of a semirigid ankle stabilizer to reduce acute ankle injuries in basketball: A randomized clinical study at West Point. *Am J Sports Med*. 1994;22:454–61.
51. Surve I, Schwellnus MP, Noakes T, Lombard C. A fivefold reduction in the incidence of recurrent ankle sprains in soccer players using the Sport-Stirrup orthosis. *Am J Sports Med*. 1994;22:601–6.
52. Laughman RK, Carr TA, Chao EY, Youdas JW, Sim FH. Three-dimensional kinematics of the taped ankle before and after exercise. *Am J Sports Med*. 1980;8:425–31.
53. Robbins S, Waked E, Rappel R. Ankle taping improves proprioception before and after exercise in young men. *Br J Sports Med*. 1995;29:242–7.
54. Fumich RM, Ellison AE, Guerin GJ, Grace PD. The measured effect of taping on combined foot and ankle motion before and after exercise. *Am J Sports Med*. 1981;9:165–70.
55. Bahr R, Lian O, Bahr IA. A twofold reduction in the incidence of acute ankle sprains in volleyball after the introduction of an injury prevention program: a prospective cohort study. *Scand J Med Sci Sports*. 1997;7:172–7.
56. Verhagen EA, van Mechelen W, de Vente W. The effect of preventive measures on the incidence of ankle sprains. *Clin J Sport Med*. 2000;10:291–6.
57. Kerkhoffs GM, Handoll HH, de Bie R, Rowe BH, Struijs PA. Surgical versus conservative treatment for acute injuries of the lateral ligament complex of the ankle in adults. *Cochrane Database Syst Rev*. 2002; Issue 3:CD000380.
58. Handoll HHG, Rowe BH, Quinn KM, de Bie R. Interventions for preventing ankle ligament injuries. *Cochrane Database of Syst Rev*. 2001; Issue 3: CD000018.

Jochen Paul, Christian Stelzenbach,  
and Victor Valderrabano

## Abstract

Acute ankle instability is a frequent injury in young and active folks and can unfortunately quite often (in 20–40 %) proceed to chronic ankle instability. Fifty-five percent of the injuries leading to ankle instability are due to sporting accidents. Symptomatic chronic ankle instability is considered as a pre-osteoarthritic condition and long-term results without adequate therapy are poor. There are different types of chronic ankle instability and consequently functional ankle instabilities are mainly treated conservatively while mechanical ankle instabilities often need a surgical treatment after failed conservative treatment. Ankle arthroscopy seems to be a valuable tool for ankle instability diagnostics and can be used for direct treatment of adjunctant injuries as well. Anatomical ligament reconstruction has been proven to be superior to non-anatomical surgical techniques in clinical investigations. Relevant concomitant injuries (hindfoot malalignment, tendon problems, osteochondral lesions, etc.) should be treated simultaneously to ligament surgery.

## Keywords

Acute ankle instability • Chronic ankle instability • Ankle arthroscopy • Anatomical reconstruction • Ankle ligaments

## Introduction

Acute injuries of the ankle with following acute ankle instability (AAI) are common, especially in young and active people. These acute injuries are often associated

with an acute trauma and therefore rarely misdiagnosed. Even though these injuries can be treated effectively by conservative treatment (Table 28.1), transitions into chronic ankle instability (CAI) are still high and noticed in 20–40 % of these cases [1, 2]. This high number might be due to insufficient primary diagnosis or initial maltreatment (e.g., inadequate physiotherapy and/or unstable immobilization resulting in recurrent trauma). Injuries of the lateral or medial ligamentous complex are often trivialized and the consequence might be a disadvantageous healing with elongated ligaments. In particular the medial instability of the ankle and accompanying lesions can negatively influence the prognosis of AAI. Congenital co-morbidities (e.g., hyper laxity or hindfoot deformities as Varus/Valgus malalignment) can possibly influence the progress of AAI to CAI. In addition proprioceptive and neurological deficits can have negative effects on the healing process.

---

J. Paul, MD (✉)  
RENNBAHNKLINIK,  
Kriegackerstrasse 100, MuttENZ, Basel CH-4132, Switzerland  
e-mail: [jochen.paul@rennbahnklinik.ch](mailto:jochen.paul@rennbahnklinik.ch)

C. Stelzenbach  
Orthopaedic Department, University Hospital,  
University of Basel, Basel, Switzerland  
e-mail: [christian.stelzenbach@usb.ch](mailto:christian.stelzenbach@usb.ch)

V. Valderrabano  
Orthopaedic Department, SWISS ORTHO CENTER,  
Schmerzklinik Basel, Swiss Medical Network, Basel, Switzerland  
e-mail: [vvalderrabano@gsmn.ch](mailto:vvalderrabano@gsmn.ch)

**Table 28.1** Basal acute ankle instability grades and treatment protocol

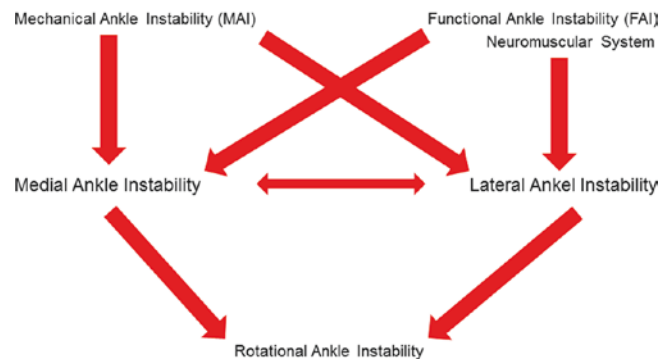
Grade	Symptoms	Ligament lesion	Therapy
<b>I</b>	<b>Swelling lateral</b> <b>Full weight bearing possible</b>	Partial ATFL ligament rupture, capsule sprain	PRICE-scheme, taping, stabilizing ankle orthotics (4–6 weeks)
<b>II</b>	<b>Hematoma lateral</b> Partial weight bearing possible	Complete rupture lateral ligaments (ATFL&CFL)	PRICE-scheme, stabilizing ankle orthotics (6 weeks), weight bearing as possible, functional treatment: physiotherapy, lymphatic drainage, proprioceptive training
<b>III</b>	<b>Hematoma lateral &amp; medial</b> <b>No weight bearing possible</b>	Complete rupture lateral ligaments (ATFL&CFL) AND Lesion medial ligaments (Deltoid/Spring)	PRICE-scheme, walker or cast with partial weight bearing for 6 weeks, functional treatment: physiotherapy, lymphatic drainage, proprioceptive training
<b>IV</b>	Hematoma lateral & medial <b>Concomitant lesions</b> <b>Grade III in professional athlete</b>	Complete rupture ligaments lateral & medial AND Syndesmotic lesion, OCL, fractures, etc. ...	PRICE-Scheme, operative treatment, postoperative protection (walker or cast) and partial weight bearing, supportive physiotherapy

PRICE Protection, Rest, Ice, Compression/Taping/Casting, Elevation, ATFL anterior talofibular ligament, CFL calcaneofibular ligament, OCL osteochondral lesion

## Etiology and Pathomechanism

Regarding etiology of CAI, Valderrabano et al. found that most of the pathologies are due to sporting activities (55%) and everyday living activities (36%) and only 9% are related to work injuries [2]. CAI can be anatomically classified into an isolated lateral or isolated medial or a combined instability, called rotational ankle instability [1] and pathomechanistically classified into mechanical CAI (MAI) or functional CAI (FAI) (Fig. 28.1) [1, 2]. The mechanical instability is caused by structural lesions or weaknesses of the stabilizing ligaments, which might be ruptured or elongated or having a general ligament laxities ending in kinematic changes of ankle motion [3]. The functional instability is frequently caused by deficient neuromuscular ankle stabilization or distortions in proprioception. These are often due to inhibition of peripheral neuronal conduction. Patients suffer from loss of strength at the lower extremity or balance insufficiency, with additional coordination problems [3, 4]. For treatment decision making the type of instability has to be identified and the co-pathomechanical problems addressed. In current literature CAI is even regarded as pre-osteoarthritis at the ankle and is believed to have a negative effect of the long-term outcome regarding development of osteoarthritis [2, 5]. Valderrabano et al. could show in 247 patients that quite a big number of patients (13%) development OA at the ankle due to persistent instability [2]. The average length to OA development was 34 years in their study group and the mechanisms of instability were crucial for OA progression. 55% of their patients were injured during sports, 36% were injured during activities of every day living and 9% during work. The authors concluded that instability at the ankle is an determining factor of developing ankle OA and that biomechanical stabilization of the ankle is necessary to protect the ankle [2]. In the authors opinion the biomechanical stability of the ankle is crucial to avoid OA.

## Chronic Ankle Instability

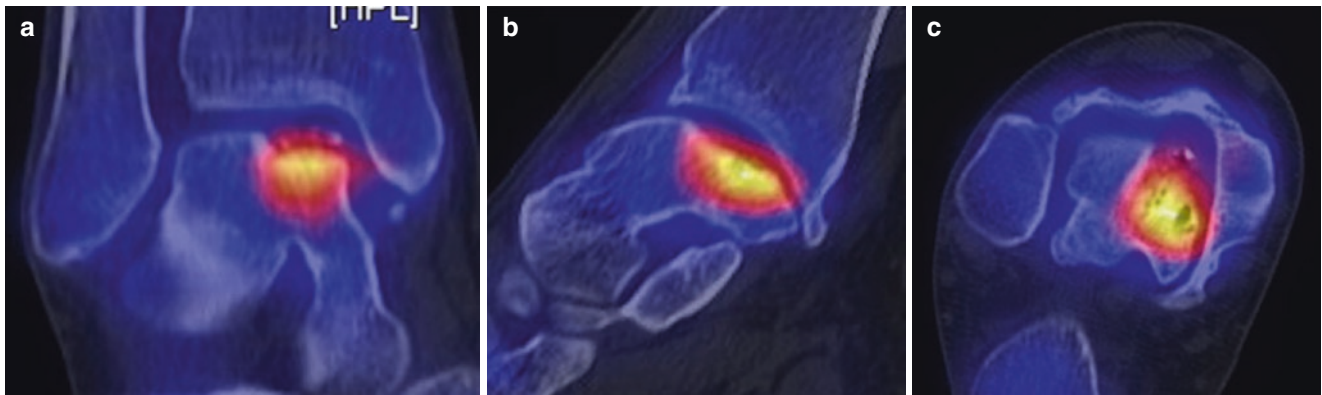


**Fig. 28.1** Types of chronic ankle instability. Chronic Ankle Instability can be distinguished *pathomechanistically* between mechanical and functional ankle instability (MAI vs FAI) as well as anatomically between lateral, medial or combined (rotational). All types do influence each other

According to Halasi et al. the highest rate of AAI in sports occur in american football, basketball, gymnastics, handball, rugby and soccer [6]. As a consequence in these mentioned sports there is a high potential for a progression to CAI.

## Diagnostics, Classification and Graduation

The clinical examination in mechanical CAI includes inspection, palpation and examination of the lateral and medial ligamentous ankle complex and is essential for the treatment decision making. In lateral ankle instability tenderness over the origin of the anterior talofibular ligament (ATFL) and calcaneofibular ligament (CFL) are common. With hanging and relaxed lower-leg the lateral ankle joint instability is examined: anterior drawer stress test, inversion stress



**Fig. 28.2** (a–c). Osteochondral lesion – a typical co-entity in chronic ankle instability. SPECT-CT of an 26 year old chronic ankle instability patient with a symptomatic postero-medial osteochondral lesion at the

talus on the right foot. SPECT-CT shows osseus lesion as well as tracer uptake in this area (a: coronal view, b: sagittal view, c: transverse view)

test; and compared to the contralateral side. The examination of the healthy contralateral side helps to distinguish a pathological from normal stability. In cases with suspected joint hypermobility the Brighton Score can help to verify the diagnosis [7]. Further examination aims the documentation of a possible co-malalignment, typically a hindfoot varus in lateral CAI, together with a peroneal muscle weakness or peroneal tendon tears. Accompanying osteochondral lesions (OCL) or Achilles tendinopathies shall be co-investigated, as they may aggravate the clinical problem.

In medial ankle instability tenderness is found over the deltoid, especially the anterior portion, and the functionally associated Spring ligament. As in lateral CAI, in medial CAI stress tests are also done with hanging and relaxed lower-leg performing the anterior drawer stress test and mainly the eversion stress test. Standing examination might reveal a flat-foot deformity and concomitant Posterior tibial tendon insufficiency.

Stress radiography seems not to be useful in the diagnosis of CAI and the obtained results cannot provide a sufficient therapy regime because there is no reliable algorithm of treatment for this radiographs [8, 9]. Concerning CAI ultrasonography has a high value as it enables dynamic testings and detection of structural lesions [10]. In comparison to magnetic resonance imaging (MRI) ultrasonography analysis showed a similar sensitivity [11]. In addition Cory et al. described an excellent verification of ATFL-injuries and objective assessment of the ligament structure and length by ultrasonography [10]. In CAI cases MRI represents an important additional imaging modality [12]. Especially the medial and lateral ankle ligaments, possible concomitant OCLs or tendon tears, and ankle syndesmosis, which contribute to the direct stability of the ankle can be evaluated by MRI [13, 14]. However, according to Cha et al. compared to arthroscopy MRI is a worse diagnostic method because the sensitivity and inter-observer reliability is lower in MRI than

in direct ankle arthroscopy [15]. Thus, diagnostic ankle arthroscopy, as last diagnostic tool prior to ligament surgery, is strongly recommended by the authors and Cha et al. [15].

Single photon emission computed tomography-CT (SPECT-CT) is another valuable diagnostic tool for detection of concomitant pathologies in cases with CAI and persistent pain. Especially in cases with OCL at the talus and OA at the subtalar or adjunctant joints SPECT-CT seems to be helpful in identifying osteochondral pathologies (Fig. 28.2) [16]. In recent literature Leumann et al. showed that SPECT-CT can influence the decision regarding the therapy of OCL at the talus [16]. Thus it represents a good additional imaging in some elected cases.

The functional instability might be documented by the deficient neuromuscular ankle stabilization/proprioception in tip-toes-standing or unstable ground. Further, patients suffer from strength loss of the peri-ankle muscles, especially of the peroneal and posterior tibial muscle. Imaging of functional CAI is limited to functional biomechanical diagnostic tools, as proprioceptive balance systems or other sports-biomechanics tools.

## Therapy: Conservative Treatment and Surgery

In many cases of CAI the patients have a long-term history of unsuccessful conservative treatment. Nevertheless a systematic conservative treatment with proprioceptive training and structured physiotherapy should be performed for 3–6 month after CAI diagnosis. In literature the functional therapy for CAI shows good response to conservative therapy in many cases [17, 18]. Particularly the neuromuscular stabilization of the ankle joint and the improvement of proprioception seems to be successful. In their prospective randomized study Hoiness et al. showed the superiority of a



special proprioception training in contrast to a normal physiotherapy program with an ergometer [18]. In their patient group a short and intensive training of proprioception and strength was successful to stabilize CAI [18]. In addition, for prevention of further ankle sprains these conservative procedures are suitable as well [19]. Different taping techniques can be used alternatively or in addition for stabilization of the ankle [20]. Especially in high demanding athletes with the need of individual stabilization of the ankle taping techniques seem to have advantages in comparison with standard orthotics.

Irrespectively of the severity of CAI an insole can help to correct the axis of the hindfoot and correct whole foot alignment. Rotational CAI of the ankle is often increased by a posterior tibial tendon insufficiency with a consecutive pes planovalgus et abductus position of the foot. In these cases a sufficient insole can lead to relief of the medial ligament complex as well as the correction of the hindfoot malalignment [21]. These hindfoot correcting advices (e.g., orthopedic technical insoles) have shown a positive effect in CAI and can be combined with the above mentioned conservative treatment [22]. The insoles should be controlled regularly and adapted if necessary. Good individually manufactured insoles include the complete sole, provide a good hindfoot frame and a sufficient medial support for valgus deformities or a lateral support for a varus deformities, respectively.

## Operative Treatment

In CAI operative treatment is much more often necessary than in cases with AAI. Primary goals of operative treatment are an increase in stability of the ankle, reduction of pain, avoidance of recurrences of ankle sprains and a return to activities of daily living at work and sports. Especially the delay of osteoarthritis development in instable ankle joints is a long term goal. Indications for CAI surgery are: failed conservative treatment for 3–6 months, recurrent ankle sprains weekly or daily, chronic ankle pain, with instability disability for sports or work. A contraindication for CAI surgery is the general ligament laxity (as in Down syndrome, Ehlers-Danlos syndrome) or obesity.

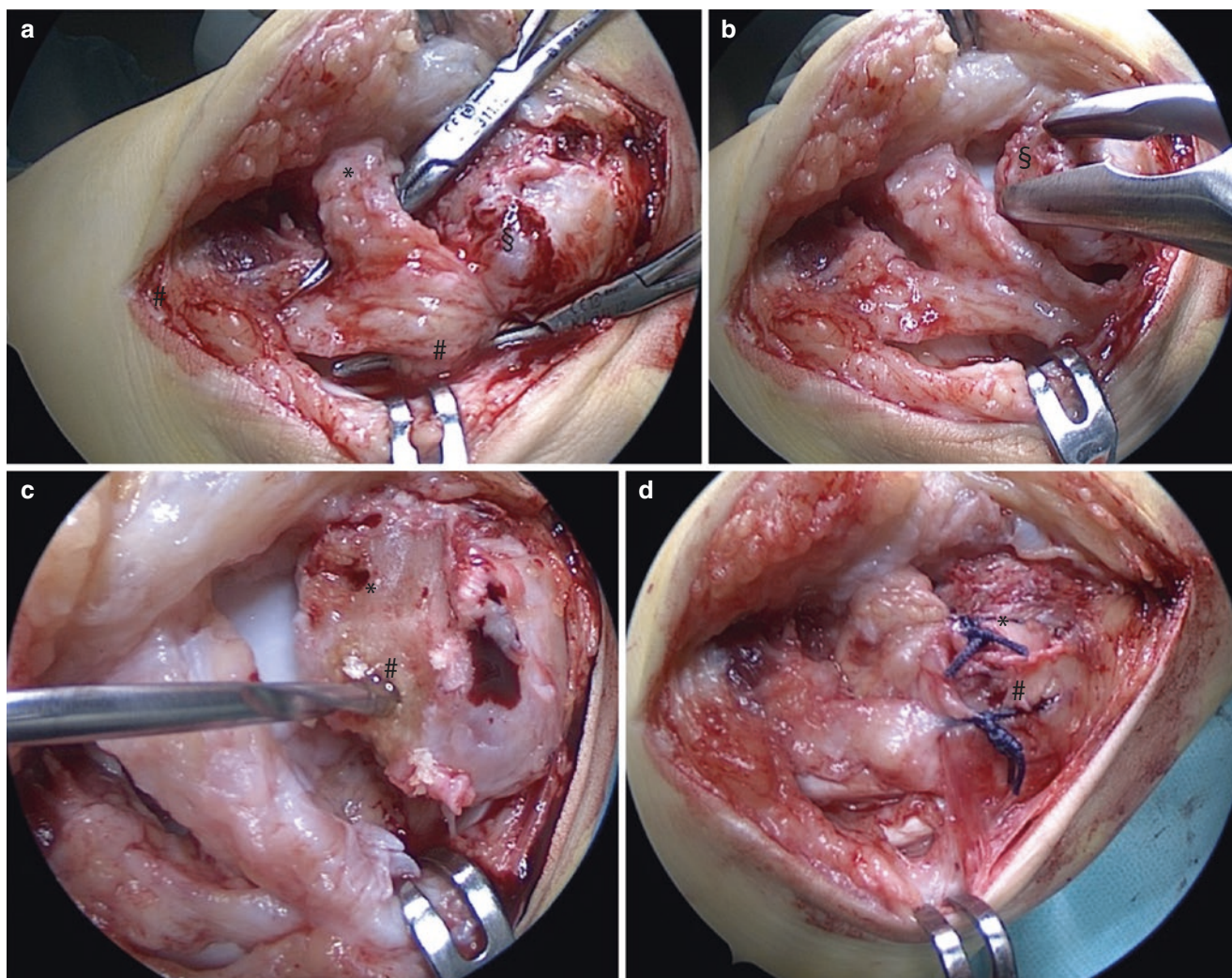
The first step of ligament CAI surgery is ankle arthroscopy, as arthroscopy is – in combination with clinical investigation – the gold standard for diagnostics [1, 23]. It confirms the instability pattern by judging the ligaments from articular and shows concomitant injuries, as: anterior osteophytes, a symptomatic Basset ligament, chondral degeneration, OCLs, and other pathologies [24, 25]. Subsequently to arthroscopy is open surgical treatment. There are different options for surgical reconstruction of the ligaments: anatomical and non-anatomical ligament recon-

structions. Non-anatomical reconstructions, as through tenodeses (Watson-Jones, Chrisman-Snook,...), have been described excessively in literature with multiple variations, regarding tendon transfers/graft, fixation devices, etc. However, such tenodeses cause a substantial pathobiomechanical situation for the ankle and subtalar joint and long-term secondary osteoarthritis [26, 27]. Therefore, anatomical reconstructions of the ligaments, with the idea of stabilizing the ankle joint and preserving its biomechanical properties, have evolved and established in the surgical community. Of the anatomical reconstructions the Brostrom-Gould technique is widely accepted and has been used in different variations [28–30]. When comparing the anatomical with the non-anatomical techniques the mid-to-long term results regarding ankle stability, patient satisfaction and degenerative changes favor the anatomical reconstruction [31]. Even in high-demand athletes anatomical reconstruction of ankle ligaments using a variant of the Gould-modified Brostrom procedure (suture anchors) was successful [32]. The athletes were able to return to their preinjury functional level [32]. In conclusion an anatomic reconstruction of the ligaments is preferred to a non-anatomic reconstruction and current literature shows better clinical results and less likelihood of OA development [31, 33].

The authors use a direct anatomical reconstruction with a modification of the modified Brostrom-Gould technique as shown in Fig. 28.3. Hereby, the lateral ligaments are identified and, after “refreshing” the anterior part of the malleolus, the ligaments are shortening and tensioned by transosseous sutures. The same concept is performed on the medial side, where the superficial and anterior part of the Deltoid ligament is tensioned towards the malleolus and possible Spring ligament tears also addressed.

Unfortunately in CAI cases with defect situation of the ligaments and insufficient residual structures a direct anatomic reconstruction is sometimes not feasible. In these cases anatomical reconstructions with the plantaris [34] or gracilis [35] tendon are suitable options for an anatomic ligament reconstruction which have been described with good clinical results. Takao et al. found several advantages with the reconstruction including the gracilis tendon [35]. The anatomical reconstruction with normal stability, restored range of motion a small incision at the knee were key features [35]. In addition the comparison of the tension strength of the lateral ligaments and the strength at the tendon graft-bone tunnel junction was a major advantage [35].

In addition to the ligament surgery laterally and/or medially, the surgeon might need to address the other possible issues, as: hindfoot varus by a lateral sliding/Dwyer osteotomy, peroneal tendon tears by a tendon surgery, hindfoot valgus by a medial sliding or lateral calcaneal lengthening osteotomy, Posterior tibial tendon insufficiency by a



**Fig. 28.3** Lateral chronic ankle instability – ligament surgery. (a): Left ankle with lateral approach over the tip of the fibula (§). Clamps are holding mobilized scar tissue rests of ATFL (\*) and CFL (#). (b): Preparation of tip of fibula (§) with the rongeur for following insertion

of the anatomical reconstructed lateral ligaments. (c): Transosseous drilling for ligament fixation of the ATFL (\*) and the CFL (#). (d): Anatomical direct fixation of ATFL (\*) and the CFL (#) with transosseous sutures shortening and tensioning the ATFL and CFL

flexor digitorum longus tendon transfer. Only by correcting all pathobiomechanical factors the CAI recurrence is neutralized.

### Rehabilitation and Back-to-Sports

Cases with isolated ligamentous reconstruction can perform full weight bearing postoperative, if additional surgeries (osteotomies, tendon reconstruction) are performed then partial-weight bearing is recommended. For the first 6 weeks a walker and a splint during the night for protection of the ankle ligaments are recommended. During this time physiotherapy with lymphatic drainage can be applied but range of motion (ROM) is limited to a maximum of dorsalexension/plantarflexion 15°/0°/10° [36]. Proprioceptive training for sta-

bilization and an advancement of muscular innervation are advised in a well monitored physiotherapy program. After 6 weeks increasing ROM at the ankle, proprioception training and strengthening of the muscles are promoted.

Back to sports is a hot topic – in competitive sports as well as recreational sports. The time to return to sports is dependent on the kind of sports, the stress on the ankle and the level of activity before the injury. An individual therapy regime has to be developed for every athlete respecting the before mentioned key points. A possible general recommendation can be: 4–6 weeks after surgery: slight sports under walker protection possible, as biking, upper-body-training; 8–12 weeks after surgery under ankle stabilizing orthotic support: slight controlled jogging, swimming. Elite athletes need an individual planning with team physiotherapist and coach.

In literature the return to sports after ATFL reconstruction for CAI in athletes was investigated by Miyamoto et al. [37]. They found an advantage of accelerated rehabilitation after surgery in comparison to traditional rehabilitation with a 5 weeks earlier return to sports [37]. Li et al. found surgical treatment effective in their study group of high-demand athletes and they could reach their preinjury functional level again [32]. Even in their group of high-demand athletes the anatomical ligament reconstruction for CAI with a variant of the modified Gould-Broström procedure showed good return to sports results with a low complication rate [32]. Krips et al. investigated in a retrospective multicenter study the results in athletic patients with lateral ankle instability [33]. The study group compared anatomic reconstructions with tenodesis. Regarding the sports activity level they found that the anatomic reconstruction was superior to tenodesis techniques in all of their outcome measures [33].

## Evidence

Table 28.2 summarizes an overview about the current evidence for ankle instability. The treatment of choice of the AAI is conservative therapy (Level of evidence II) if there are no accompanying injuries [38]. Functional treatment proved to be very valuable for prevention of long-term complications and seems to be superior to ankle immobilization alone (Level of evidence II) [39]. In chronic ankle instability, anatomical reconstructions of the ankle ligaments seem to be superior to tenodesis techniques [27, 33] and symptomatic chronic instability of the ankle without appropriate treatment probably presents a pre-osteoarthritis condition with an associated poor long-term outcome (Level of evidence IV) [2].

**Table 28.2** Summary of current evidence for ankle instability

Topic	Level of evidence	Literature
In AAI surgical treatment is not superior to conservative treatment	Level II	[38]
Functional treatment superior to immobilization	Level II	[39]
Doubtful value of ankle stress X-ray	Level II–IV	[8, 9]
Functional physiotherapy is valuable for outcome	Level II–IV	[17, 18]
Anatomical reconstruction superior to tenodesis	Level IV	[27, 33]
CAI is a pre-osteoarthritis condition	Level IV	[2]

## Summary

- Acute ankle instability can in 20–40 % of the patients proceed to chronic ankle instability (CAI).
- A symptomatic CAI is a pre-osteoarthritis and long-term results without adequate therapy are devastating.
- Functional CAI are mainly treated conservatively while mechanical CAI often need a surgical treatment after failed conservative treatment.
- Ankle arthroscopy seems are valuable tool for diagnosis of CAI as well as treatment option for adjunctant injuries.
- Anatomical reconstruction of the ankle ligaments are superior to non-anatomical surgical techniques and should be applied to preserve the kinematics and mechanics of the ankle and subtalar joint. Concomitant injuries (hindfoot malalignment, tendon problems, osteochondral lesions, etc.) should be treated simultaneously to ligament surgery.

## References

1. Valderrabano V, et al. Chronic ankle instability. *Unfallchirurg*. 2007;110(8):691–9; quiz 700.
2. Valderrabano V, et al. Ligamentous posttraumatic ankle osteoarthritis. *Am J Sports Med*. 2006;34(4):612–20.
3. Hertel J. Functional anatomy, pathomechanics, and pathophysiology of lateral ankle instability. *J Athl Train*. 2002;37(4):364–75.
4. Tropp H, Odenrick P, Gillquist J. Stabilometry recordings in functional and mechanical instability of the ankle joint. *Int J Sports Med*. 1985;6(3):180–2.
5. Golditz T, et al. Functional ankle instability as a risk factor for osteoarthritis: using T2-mapping to analyze early cartilage degeneration in the ankle joint of young athletes. *Osteoarthritis Cartilage*. 2014;22(10):1377–85.
6. Halasi T, et al. Development of a new activity score for the evaluation of ankle instability. *Am J Sports Med*. 2004;32(4):899–908.
7. Grahame R, Bird HA, Child A. The revised (Brighton 1998) criteria for the diagnosis of benign joint hypermobility syndrome (BJHS). *J Rheumatol*. 2000;27(7):1777–9.
8. Senall JA, Kile TA. Stress radiography. *Foot Ankle Clin*. 2000;5(1):165–84.
9. Frost SC, Amendola A. Is stress radiography necessary in the diagnosis of acute or chronic ankle instability? *Clin J Sport Med*. 1999;9(1):40–5.
10. Croy T, et al. Differences in lateral ankle laxity measured via stress ultrasonography in individuals with chronic ankle instability, ankle sprain copers, and healthy individuals. *J Orthop Sports Phys Ther*. 2012;42(7):593–600.
11. Guillodo Y, Varache S, Saraux A. Value of ultrasonography for detecting ligament damage in athletes with chronic ankle instability compared to computed arthrotopography. *Foot Ankle Spec*. 2010;3(6):331–4.
12. Ende D, et al. Value of MRI in diagnosing injuries after ankle sprains in children. *Foot Ankle Int*. 2012;33(12):1063–8.
13. Hermans JJ, et al. Correlation between radiological assessment of acute ankle fractures and syndesmotic injury on MRI. *Skeletal Radiol*. 2012;41(7):787–801.

14. Crim JR, et al. Deltoid ligament abnormalities in chronic lateral ankle instability. *Foot Ankle Int.* 2011;32(9):873–8.
15. Cha SD. Intra-articular lesions in chronic lateral ankle instability: Comparison of arthroscopy with magnetic resonance imaging findings. *Clin orthop Surg.* 2012;4(4):293–9.
16. Leumann A, et al. A novel imaging method for osteochondral lesions of the talus—comparison of SPECT-CT with MRI. *Am J Sports Med.* 2011;39(5):1095–101.
17. Eils E, Rosenbaum D. A multi-station proprioceptive exercise program in patients with ankle instability. *Med Sci Sports Exerc.* 2001;33(12):1991–8.
18. Hoiness P, Glott T, Ingjer F. High-intensity training with a bidirectional bicycle pedal improves performance in mechanically unstable ankles—a prospective randomized study of 19 subjects. *Scand J Med Sci Sports.* 2003;13(4):266–71.
19. Eils E, et al. Comprehensive testing of 10 different ankle braces. Evaluation of passive and rapidly induced stability in subjects with chronic ankle instability. *Clin Biomech (Bristol, Avon).* 2002;17(7):526–35.
20. Janssen KW, Kamper SJ. Ankle taping and bracing for proprioception. *Br J Sports Med.* 2013;47:527–8.
21. Chao W, et al. Nonoperative management of posterior tibial tendon dysfunction. *Foot Ankle Int.* 1996;17(12):736–41.
22. Augustin JF, et al. Nonoperative treatment of adult acquired flat foot with the Arizona brace. *Foot Ankle Clin.* 2003;8(3):491–502.
23. Coughlin MJ, Mann RA, Saltzman CL. *Surgery of the foot and ankle.* 8th ed. Philadelphia: Mosby Elsevier; 2007.
24. Paul J, et al. Treatment of osteochondral lesions of the ankle joint. *Arthroscopie.* 2009;22:102–8.
25. Miska M, Wiewiorski M, Valderrabano V. Reconstruction of a large osteochondral lesion of the distal tibia with an iliac crest graft and autologous matrix-induced chondrogenesis (AMIC): a case report. *J Foot Ankle Surg.* 2012;51(5):680–3.
26. Schmidt R, et al. Reconstruction of the lateral ligaments: do the anatomical procedures restore physiologic ankle kinematics? *Foot Ankle Int.* 2004;25(1):31–6.
27. Krips R, et al. Anatomical reconstruction versus tenodesis for the treatment of chronic anterolateral instability of the ankle joint: a 2- to 10-year follow-up, multicenter study. *Knee Surg Sports Traumatol Arthrosc.* 2000;8(3):173–9.
28. Hamilton WG, Thompson FM, Snow SW. The modified Brostrom procedure for lateral ankle instability. *Foot Ankle.* 1993;14(1):1–7.
29. Messer TM, et al. Outcome of the modified Brostrom procedure for chronic lateral ankle instability using suture anchors. *Foot Ankle Int.* 2000;21(12):996–1003.
30. Valderrabano V, et al. Direct anatomic repair of the lateral ankle ligaments in chronic lateral ankle instability. *Unfallchirurg.* 2007;110(8):701–4.
31. Krips R, et al. Anatomical reconstruction and Evans tenodesis of the lateral ligaments of the ankle. Clinical and radiological findings after follow-up for 15 to 30 years. *J Bone Joint Surg Br.* 2002;84(2):232–6.
32. Li X, et al. Anatomical reconstruction for chronic lateral ankle instability in the high-demand athlete: functional outcomes after the modified Brostrom repair using suture anchors. *Am J Sports Med.* 2009;37(3):488–94.
33. Krips R, et al. Sports activity level after surgical treatment for chronic anterolateral ankle instability. A multicenter study. *Am J Sports Med.* 2002;30(1):13–9.
34. Hintermann B, Renggli P. Anatomic reconstruction of the lateral ligaments of the ankle using a plantaris tendon graft in the treatment of chronic ankle joint instability. *Orthopade.* 1999;28(9):778–84.
35. Takao M, et al. Anatomical reconstruction of the lateral ligaments of the ankle with a gracilis autograft: a new technique using an interference fit anchoring system. *Am J Sports Med.* 2005;33(6):814–23.
36. Karlsson J, et al. Early range of motion training after ligament reconstruction of the ankle joint. *Knee Surg Sports Traumatol Arthrosc.* 1995;3(3):173–7.
37. Miyamoto W, et al. Accelerated versus traditional rehabilitation after anterior talofibular ligament reconstruction for chronic lateral instability of the ankle in athletes. *Am J Sports Med.* 2014;42(6):1441–7.
38. Kerkhoffs GM, et al. Surgical versus conservative treatment for acute injuries of the lateral ligament complex of the ankle in adults. *Cochrane Database Syst Rev.* 2007;(2):CD000380.
39. Kerkhoffs GM, et al. Immobilisation and functional treatment for acute lateral ankle ligament injuries in adults. *Cochrane Database Syst Rev.* 2002;(3):CD003762.

Craig R. Lareau, Andrew R. Hsu, and Bruce E. Cohen

**Abstract**

Ligamentous ankle injuries remain one of the most common injuries sustained by athletes. Syndesmotic, often termed high ankle, sprains are being diagnosed more frequently due to a heightened awareness and improved diagnostic techniques. These injuries result in pain, disability, and a significant delay in return to play. Most operative syndesmotic injuries can be diagnosed on plain radiographs, including stress views; however, ultrasound, CT, and MRI have improved the detection of more subtle injuries. Arthroscopic evaluation can be used to confirm the diagnosis while assessing other intra-articular structures. There has been an abundance of recent research exploring the treatment of syndesmotic injuries in athletes, focusing on surgical intervention and rehabilitation protocols. The literature remains conflicted regarding return to play in athletes.

**Keywords**

Ankle syndesmosis • Syndesmotic injury • High ankle sprain • Distal tibiofibular joint

**Introduction**

It is well-established that ankle sprains and fractures are among the most common sports-related injuries [1]. Isolated syndesmotic disruptions without fracture occur more commonly in athletes than non-athletes [2]. Syndesmotic injuries comprise 1–18% of ankle sprains but are probably underreported since the diagnosis is not always made on initial physical examination and non-weight-bearing radiographs [3–5]. Return to play after syndesmotic injury can take twice as long when compared to isolated lateral ankle ligamentous injury [6]. Return to play after syndesmotic disruption is dependent on the sport, degree of injury, and

variable time to healing for each injury [7]. In addition to the short-term disability caused by these injuries, chronic ankle dysfunction can persist for months following the injury [8, 9]. Unrecognized injuries that are not properly treated can result in chronic ankle instability and post-traumatic ankle arthritis [10].

Syndesmotic disruption can occur in isolation or with concomitant adjacent bony, cartilaginous, or ligamentous injuries. Therefore, the importance of a focused and comprehensive exam of the entire foot and ankle cannot be overemphasized [9]. Considerable discrepancies exist in the literature regarding the reporting of these injuries, the use of advanced imaging and arthroscopy to aid in diagnosis, and the appropriate treatment for different degrees of injury [2]. The diagnosis and management of these injuries, especially in elite athletes, remains controversial since there is a lack of level I evidence surrounding this topic. This chapter will review relevant anatomy, etiology, pathophysiology, symptomatology, diagnosis, treatment, rehabilitation, and prevention, and summarize the highest level of existing evidence.

C.R. Lareau, MD (✉)  
Department of Orthopaedic Surgery,  
OrthoCarolina Foot & Ankle Institute,  
2001 Vail Avenue, Suite 200B, Charlotte, NC 28207, USA  
e-mail: [craig.lareau@gmail.com](mailto:craig.lareau@gmail.com)

A.R. Hsu, MD • B.E. Cohen, MD  
OrthoCarolina Foot & Ankle Institute, Charlotte, NC, USA  
e-mail: [andyhsu1@gmail.com](mailto:andyhsu1@gmail.com); [Bruce.Cohen@orthocarolina.com](mailto:Bruce.Cohen@orthocarolina.com)

## Etiology and Pathomechanism

The distal tibiofibular syndesmosis is located between the convex distal fibula and the concave incisura fibularis of the tibia. A synovial-lined joint cavity, contiguous with the ankle joint, extends proximally 12–15 mm and includes articulating facets between the tibia and fibula 75 % of the time [11]. The syndesmosis is stabilized by the anterior inferior tibiofibular ligament (AITFL), interosseous ligament (IOL), and posterior ligamentous complex consisting of the posterior inferior tibiofibular ligament (PITFL) and fibrocartilaginous inferior transverse ligament (ITL) [12]. The posterior ligaments are confluent with the posterior ankle capsule. The AITFL, PITFL, and IOL limit fibular external rotation, posterior translation, and lateral translation, respectively [13]. Within the AITFL, the inferior band is most important for stability.

Normal fibular motion within the syndesmosis, albeit slight in comparison to other joints, is important in preserving ankle joint congruity [14, 15]. When the ankle is dorsiflexed, the fibula externally rotates and migrates proximally and posterolaterally to accommodate the wider anterior talar dome [16]. With plantarflexion, the fibula internally rotates and translates distally and anteromedially. External rotation of the foot results in fibular external rotation and posteromedial translation [17].

The syndesmosis functions synergistically with the deltoid ligament complex to maintain stability and congruency of the ankle mortise. Congruity of the ankle joint is essential in order to maintain normal contact characteristics and thereby prevent the development of post-traumatic arthritis [18, 19]. For this reason, syndesmotic instability and malreduction is associated with poor functional outcome and secondary arthritis.

Together with the ITL, the PITFL provides 42 % of syndesmotic stability, compared to 35 % for the AITFL and 22 % for the IOL [20]. When all syndesmotic ligaments are sectioned, fibular motion relative to the tibia increases 8.8 mm in the sagittal plane and 1.5 mm in the coronal plane with stress examination [21]. In contrast, disruption of the AITFL alone results in only 0.5 mm of translation in the coronal and sagittal planes. With sectioning of the syndesmosis 4–6 cm proximal to the ankle joint with a concomitant deep deltoid ligament rupture can result in tibiotalar dislocation when the ankle is loaded in 20° of dorsiflexion [22].

Syndesmotic injuries most often result from an external rotation force applied to a hyperdorsiflexed ankle while the foot is planted [12]. This mechanism causes the fibula to externally rotate while translating posteriorly and laterally, resulting in rupture of the AITFL first, followed by the deep deltoid ligament, IOL, and lastly PITFL [23]. Most syndesmotic injuries involve rupture of only the AITFL and IOL [2]. Other described mechanisms of syndesmotic injury

include inversion, inversion with external rotation, and eversion [24, 25]. More significant syndesmotic injuries are more likely to occur with external rotation compared with other mechanisms. Syndesmotic widening of at least 2 mm more than the contralateral ankle has been shown to correlate with injury to two or more syndesmotic ligaments [26].

## History and Physical Examination

Athletic syndesmotic injuries occur most commonly in high-impact sports, including ice hockey and football [6, 27]. An attempt to define the mechanism of injury should be made while taking the history. Treatment varies dramatically between acute (less than 3 weeks) and chronic (greater than 3 months) injuries so it is important to establish the date of injury [28]. One should inquire about a history of previous injury or treatment. As with all foot and ankle injuries, the patient should be asked to localize the area of maximum pain. Patients may report a sense of ankle instability.

Physical examination should begin with inspection for swelling, ecchymosis and deformity. In addition, it should focus reproduction of pain along the anterolateral border of the leg during ankle dorsiflexion, instability testing, and pain with weight-bearing or during push-off [29]. Tenderness localized to the AITFL is non-specific since this can occur with many other ankle injuries in the acute period [30]. Forty percent of patients with ATFL rupture resulting from a supination injury had tenderness over the AITFL without rupture of this ligament [30].

Multiple specific stress tests have been designed to assess syndesmotic stability, including the squeeze test, external rotation stress test, Cotton test, crossed-leg test and fibula-translation test [4, 26, 29, 31]. The squeeze test involves compression of the tibia and fibula proximally in the calf and is considered positive when this causes pain at the ankle joint. The external rotation stress test involves an external rotation stress applied to the foot while the ankle is dorsiflexed, with reproducible pain signifying a positive result. This test has been shown to have the lowest inter-observer error and highest sensitivity [32]. A positive Cotton test occurs with pain when the talus is translated laterally within the mortise. The fibula-translation test is performed by stabilizing the tibia and trying to displace the fibula anteriorly or posteriorly. A positive crossed-leg test occurs when pain is elicited by pushing the affected fibula downwards against the knee.

Physical examination alone has been shown to detect only 75 % of injuries [32]. With MRI-confirmed syndesmotic injuries, the external rotation stress test had a sensitivity of 20 % and specificity of 84.8 % versus 30 and 93.5 % for the squeeze test [33]. While the presence of a positive test should heighten suspicion for a syndesmotic injury, no physical exam maneuver has been shown to reliably predict the pres-

ence or degree of syndesmotom injury and therefore imaging studies are necessary [34].

## Imaging

Initial imaging for evaluation of syndesmotom injuries should include anteroposterior (AP), mortise, and lateral radiographs of the affected ankle. If possible, weight-bearing radiographs are beneficial in identifying more subtle injuries but are not necessary when incongruence of the mortise is evident on non-weight-bearing views. An increase in the medial clear space between the medial malleolus and talus can occur with a combined syndesmotom and deltoid disruption (Fig. 29.1) [33]. In the case of supination-external rotation (SER) IV injuries, the integrity of the syndesmotom can only be assessed after anatomic fixation of the fibula (Fig. 29.2). While it is well-established that syndesmotom disruption is more common with pronation-type (Weber C) fibula fractures, it occurs in 20–30% of supination-type (Weber B) fibula fractures [35, 36]. A tibiofibular clear space 1 cm proximal to the tibial plafond of greater than 6 mm on AP and mortise radiographs indicates a syndesmotom injury [37]. Since the appearance of the tibiofibular clear space does not change over an arc of 5° of external rotation to 25° of internal rotation, it has been demonstrated to be the more reliable radiographic measurement [38].

In the case of subtle injuries (Fig. 29.3a, b), contralateral comparison views can be helpful since a difference in tibiofibular clear space by at least 2 mm indicates syndesmotom injury. Fluoroscopic stress evaluation is beneficial but has a high false-negative rate in low-grade injuries and may not detect partial rupture of the AITFL and IOL [39]. In fact, it has been shown that malrotation of as much as 30° of external rotation can occur if relying on intraoperative fluoroscopy alone [40].

CT scan with fine axial cuts is most useful to evaluate the location of the distal fibula in relation to the incisura fibularis of the tibia [41]. This is especially true in cases of tibial AITFL and PITFL avulsion fractures, which can occur in as many as 50% of syndesmotom injuries [42]. Again, with more subtle injuries, the contralateral ankle should be included in the field of view to allow comparison. While CT has its advantages, one must remember that it remains a static test, unless weight-bearing CT is used. Further study is necessary to explore the utility of weight-bearing CT.

MRI is beneficial in identifying high and low-grade syndesmotom injuries and has high inter-observer reliability [43]. It has been shown to have 100% sensitivity and 93% specificity for AITFL injuries and 100% sensitivity and specificity for PITFL ruptures in cases of high-grade syndesmotom injury [44]. Since MRI, like CT, is not a dynamic test and therefore is not ideal for detecting syndesmotom instability in low-grade injuries.

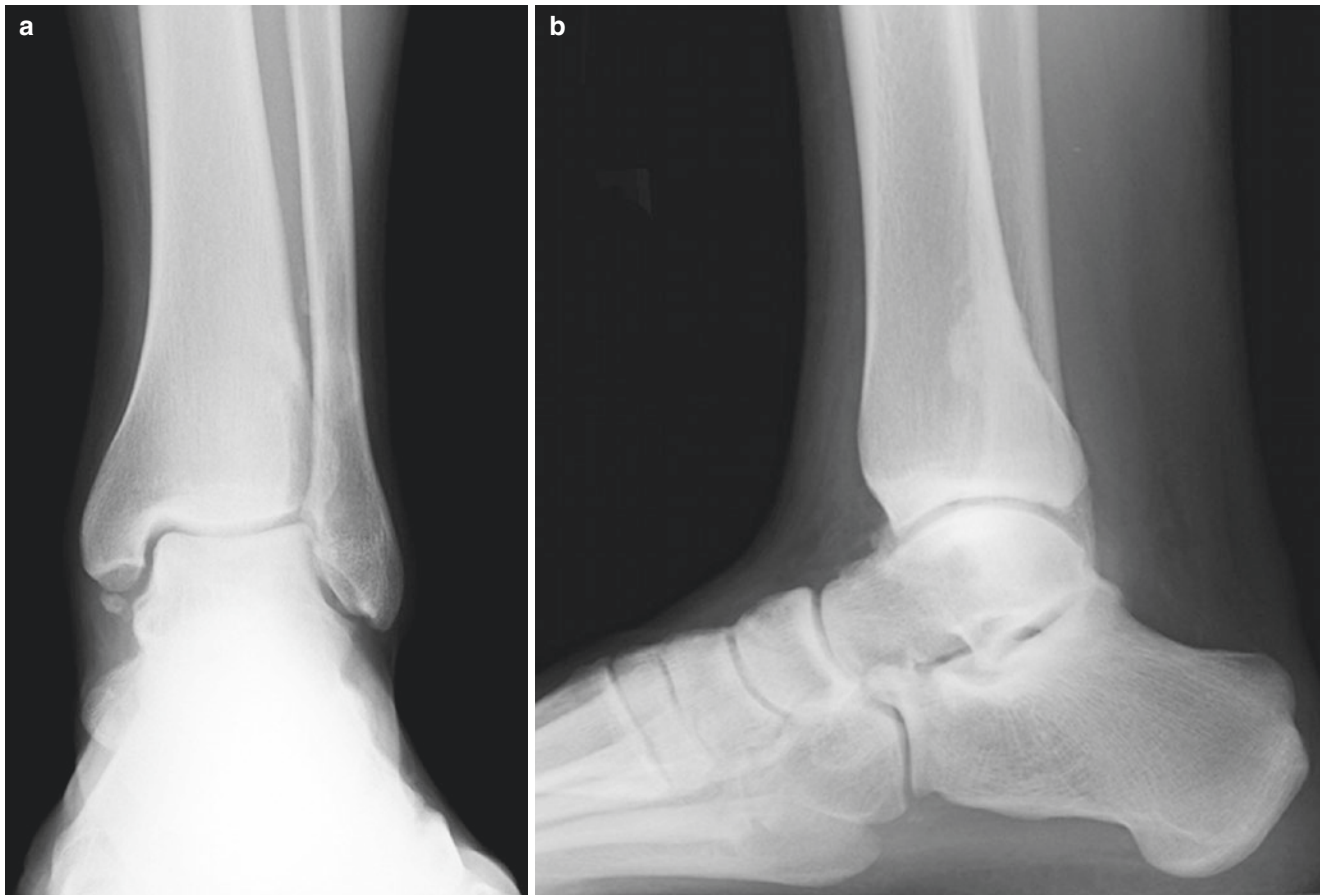


**Fig. 29.1** Preoperative AP radiograph during external rotation stress test demonstrating widening of the medial clear space and syndesmotom



**Fig. 29.2** Intraoperative AP radiograph showing syndesmotom instability during stress testing after fibula ORIF

Ultrasound is a quick, safe, and inexpensive imaging modality that allows dynamic testing. It can depict widening of the tibiofibular clear space and AITFL disruption when applying external rotation stress [45]. One must be cognizant that ultrasound is highly operator-dependent and diagnostic



**Fig. 29.3** (a, b) Preoperative AP (a) and lateral (b) radiographs showing medial malleolus avulsion fracture with suspicion for possible syndesmotic injury

utility is likely to vary based on the skill and experience of the ultrasonographer. Compared to MRI, ultrasound is not effective in detecting osteochondral lesions and bone bruising, which are present in 28 % of cases [46].

If imaging modalities fail to provide a conclusive diagnosis, ankle arthroscopy can be used to directly visualize the syndesmosis and articular cartilage [47]. Although invasive, it can have both diagnostic and therapeutic utility. It is superior to any imaging modality in terms of its ability to definitively diagnose syndesmotic and osteochondral injuries, and allows dynamic instability to be surgically treated in the same setting [48].

## Classification

Syndesmotic injuries can be classified based on physical examination as described by the West Point Ankle grading system [8]. Grade I (low-grade) injuries include mild sprains and AITFL ruptures without ankle instability. Grade II injuries involve rupture of the AITFL and IOL with slight instability. Grade III (high-grade) injuries are grossly unstable and result from disruption of all

syndesmotic ligaments. It is difficult to differentiate between grade I and II injuries based on physical examination alone, so it is necessary to employ advanced imaging or arthroscopy to make this distinction.

Edwards and DeLee developed another classification of acute syndesmotic injury without fracture [49]. First, they broadly defined these injuries as sprains without diastasis, sprains with frank diastasis (evident on initial radiographs), and sprains with latent diastasis (diagnosed only on stress radiographs). Second, they categorized injuries as type I, lateral fibular translation without fracture; type II, lateral fibular translation due to plastic deformation of the fibula; type III, posterior subluxation or dislocation of the fibula; and type IV, proximal dislocation of the talus causing divergence of the distal tibia and fibula. Currently, no classification system exists that clearly defines treatment guidelines, injury severity, or prognosis.

## Conservative Treatment

Nonoperative treatment is indicated for stable grade I syndesmotic injuries. This involves rest and immobilization followed by a progressive rehabilitation program consisting of



stretching, strengthening, and proprioceptive exercises [50]. After a 1-week period of protected weight-bearing in a cast or CAM boot, progression to full weight-bearing should occur over the following week. Additionally, active-assisted ankle range of motion exercises and light proprioceptive training should commence with the guidance of a physical therapist. During the following week (14–21 days post-injury), athletes may begin strength training and sports-specific exercises. Return to play is permitted when the athlete is able to single-leg hop for at least 30 s (or 15 repetitions) without pain, which usually occurs between 4 and 6 weeks post-injury [2].

The appropriate initial treatment of grade II injuries is nonoperative. There is a spectrum of injury within this grade and patients exhibit variable recovery times. In patients with persistent symptoms and protracted rehabilitation course, dynamic stress examination as well as arthroscopic evaluation can be helpful to evaluate ligamentous stability. If dynamic testing reveals diastasis of more than 2 mm, surgical intervention should be considered [26]. Although conservative management leads to good to excellent outcomes in 86–100% of patients, the resultant prolonged rehabilitation course (average return to play of 13.4 weeks) may be problematic, particularly in elite athletes [8, 29, 34]. Not surprisingly, patient outcomes are inferior when tibiofibular diastasis persists and/or calcification develops above the syndesmotom ligaments [51].

## Operative Treatment

### Reduction Technique

There is some debate as to the appropriate clamp position for reduction of syndesmotom injuries. While it seems logical that an oblique clamp position should be used since the fibula is posterior in relation to the tibia, a cadaver study demonstrated that clamp orientation along the neutral anatomic axis yields an optimal reduction [52]. With regard to ankle position during clamp reduction and screw placement, it has been postulated that the ankle should be dorsiflexed to prevent overtightening of the syndesmotom since the talar dome is wider anteriorly. Yet, no difference in ankle range of motion was noted in a recent study between screws tightened in plantarflexion and dorsiflexion [53]. In fact, excessive ankle dorsiflexion can posteriorly displace the fibula out of the incisura. Therefore, we recommend clamp and screw tightening with the ankle in resting position to prevent malreduction. It is prudent to have a low threshold to dissect anteriorly to the fibula to achieve an anatomic reduction by direct visualization. Some surgeons advocate manual reduction of the syndesmotom to avoid overcompression and facilitate an anatomic reduction. The use of a smaller clamp may result in fibular translation.

## Radiographic Assessment of Syndesmotom Reduction

It is advisable to obtain comparison fluoroscopic AP and lateral views of the contralateral ankle to ascertain the normal relationship of the patient's distal tibia and fibula. Even using direct visualization and fluoroscopic guidance, anatomic reduction of the syndesmotom can be difficult. In patients with associated malleolar fractures, high rates of syndesmotom malreduction have been observed on postoperative imaging, up to 52% using CT [54–57]. The distance between the fibula and posterior facet of the incisura was greater in 77% of malreductions, representing iatrogenic internal rotation or anterior translation of the fibula [56]. Syndesmotom widening beyond 1.5 mm after open reduction internal fixation results in poor functional outcomes [54, 58].

In a case report, intraoperative O-arm 3-D cone-beam CT was found to be fast and effective in assessing fibular length and rotation as well as mortise congruency [59]. Another study reported the use of 3-D imaging with mobilized C-arms in 251 syndesmotom injuries [60]. This technology identified malreductions in 33% of cases that were corrected during the same procedure. Although intraoperative 3-D imaging is appealing, it is too expensive for many institutions and it is questionable as to whether its benefit outweighs the risk of increased radiation exposure and longer surgery duration.

## Diagnostic Arthroscopy

Stress radiographs and MRI have been found to be less effective than direct arthroscopic visualization in demonstrating the true degree of syndesmotom disruption (Fig. 29.3) [44, 48]. While MRI is very sensitive for detecting syndesmotom injuries with flexor hallucis longus edema being a clue to posterior tibiofibular ligament injury [61] and CT can detect minor (2–3 mm) diastasis [41], neither advanced imaging modality is predictive of syndesmotom instability. In addition, many patients with syndesmotom injury are at high risk of associated traumatic talar osteochondral lesions which can be managed arthroscopically at the time of syndesmotom evaluation [62]. Syndesmotom injuries can vary widely in the degree of severity, with some patients being appropriate candidates for non-operative management and others requiring arthroscopic debridement and/or ORIF [10].

Arthroscopy can be a particularly valuable diagnostic tool in the setting of a subtle syndesmotom injury with negative radiographs, positive MRI for edema, and a protracted recovery course with vague pain. In these situations, an exam under anesthesia is performed, the syndesmotom is probed under direct arthroscopic visualization, and distal tibiofibular instability is evaluated while performing an external rotation stress test (Fig. 29.4a, b). In a cadaveric study, the distance between the tibia and fibula at the distal tibiofibular joint

averaged 1.3 mm in the anterior syndesmosis, 3.7 mm at the midsection, and 3.3 mm at the posterior aspect [63]. Therefore, physiologic opening of the syndesmosis depends on the level of testing, and an opening of more than 2 mm anteriorly is pathologic. When instability is present, arthroscopic syndesmotomy with percutaneous fixation provides good or excellent results [64, 65].

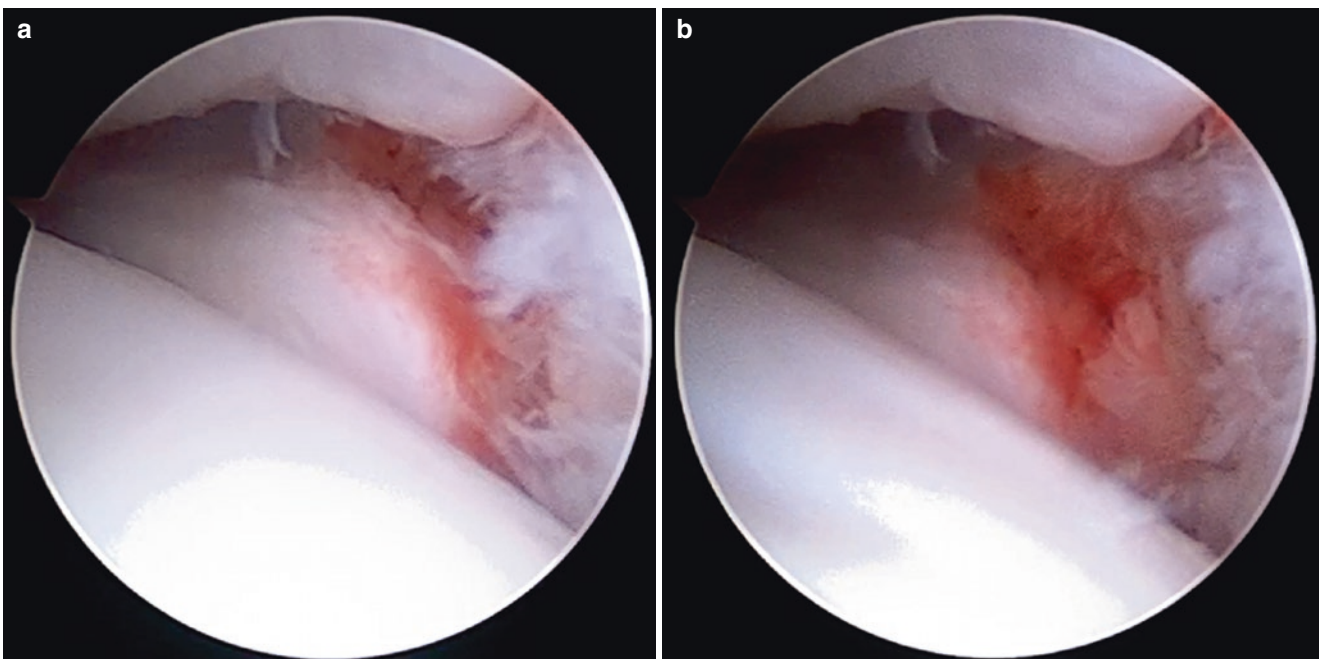


**Fig. 29.4** Arthroscopic image of complete syndesmotomy

## Treatment of Acute Injuries

Surgical intervention of acute syndesmotomy injuries consists of screw fixation or dynamic stabilization with a suture-button construct or hybrid fixation. A widened mortise on weight-bearing radiographs (Fig. 29.5) necessitates surgical reduction and stabilization to reestablish normal ankle biomechanics and optimize patient outcomes by preventing post-traumatic arthritis and chronic instability [66, 67]. The only significant predictor of function after syndesmotomy injury is maintenance of reduction [36]. Other indications for syndesmotomy stabilization include a fibula fracture at least 4.5 cm proximal to the tibiotalar joint coupled with deltoid ligament injury and intraoperative fluoroscopic evidence of gross syndesmotomy instability following malleolar fixation [68, 69].

The literature is less definitive regarding the management of low-grade injuries in athletes with more subtle radiographic abnormalities. Surgical stabilization coupled with arthroscopic treatment of concomitant intra-articular pathology has been recommended for grade II injuries [34]. Since grade III injuries are commonly associated with osteochondral defects as well as deltoid and lateral ligamentous ruptures, preoperative advanced imaging and/or intraoperative arthroscopy can be beneficial. Return to play in as early as 6 weeks has been demonstrated with early surgical intervention for grade III syndesmotomy sprains paired with an aggressive rehabilitation protocol [70].



**Fig. 29.5** (a, b) Arthroscopic images of a patient with a subtle syndesmotomy injury before (a) and after (b) stress examination

### Screw Fixation

Despite an abundance of research on the topic, there remains no clear consensus regarding the ideal technique of syndesmotom stabilization. In general, most agree that implants should be placed at least 1.5 cm proximal to the tibial plafond (Fig. 29.6) to avoid the true syndesmotom joint, parallel to the joint and directed from posterolateral to anteromedial at a 25–30° angle from the coronal plane while an external clamp or other reduction aid is maintaining the reduction [71, 72]. It is well-established that screws should not be placed in lag fashion as this would overcompress the syndesmosis. Ongoing debate exists with respect to screw number and type, number of cortices penetrated, and the need for reoperation for hardware removal. Most commonly, either 3.5 or 4.5-mm fully threaded cortical screws are utilized depending on surgeon preference, patient size, and degree of instability. When using two syndesmotom screws, a cadaveric study showed that screw size (3.5 or 4.5-mm) and number of engaged cortices (tricortical or quadricortical) had no significant impact on mechanical stability [73].

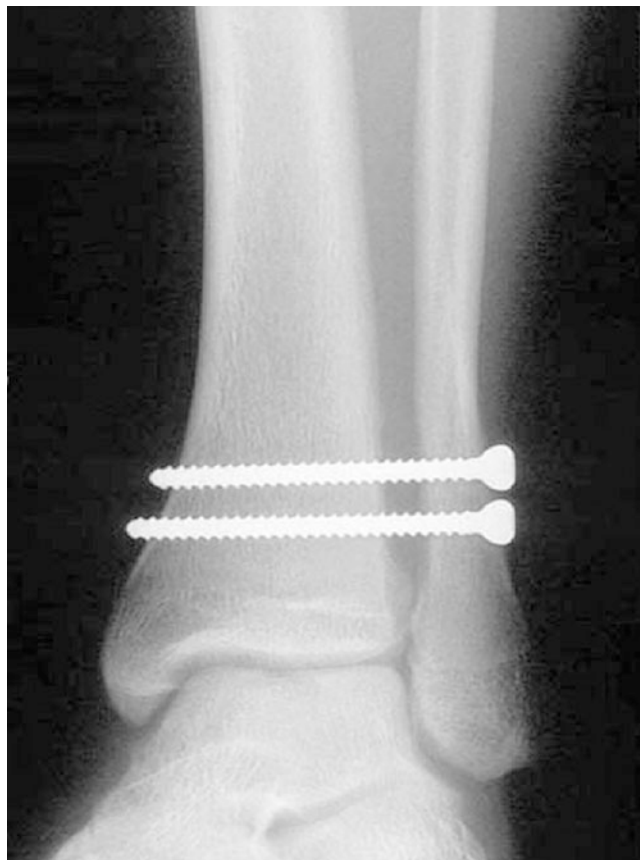
Other studies have demonstrated that 4.5-mm screws have higher resistance to shear stress while 3.5-mm screws are more likely to break [74, 75]. Proponents of tricortical screws argue that more physiologic motion is preserved,

while those in favor of quadricortical screws assert that more robust fixation is achieved and complete hardware removal is more easily performed after screw breakage [75]. One study showed that one quadricortical 4.5-mm screw had equivalent functional outcome, range of motion, and pain level as two 3.5-mm screws placed tricortically [76]. Bioabsorbable screws have been shown to function as effectively as conventional stainless steel screws at 1-year follow-up; [77] however, they are not commonly used because granuloma formation and foreign body reaction are potential complications [78].

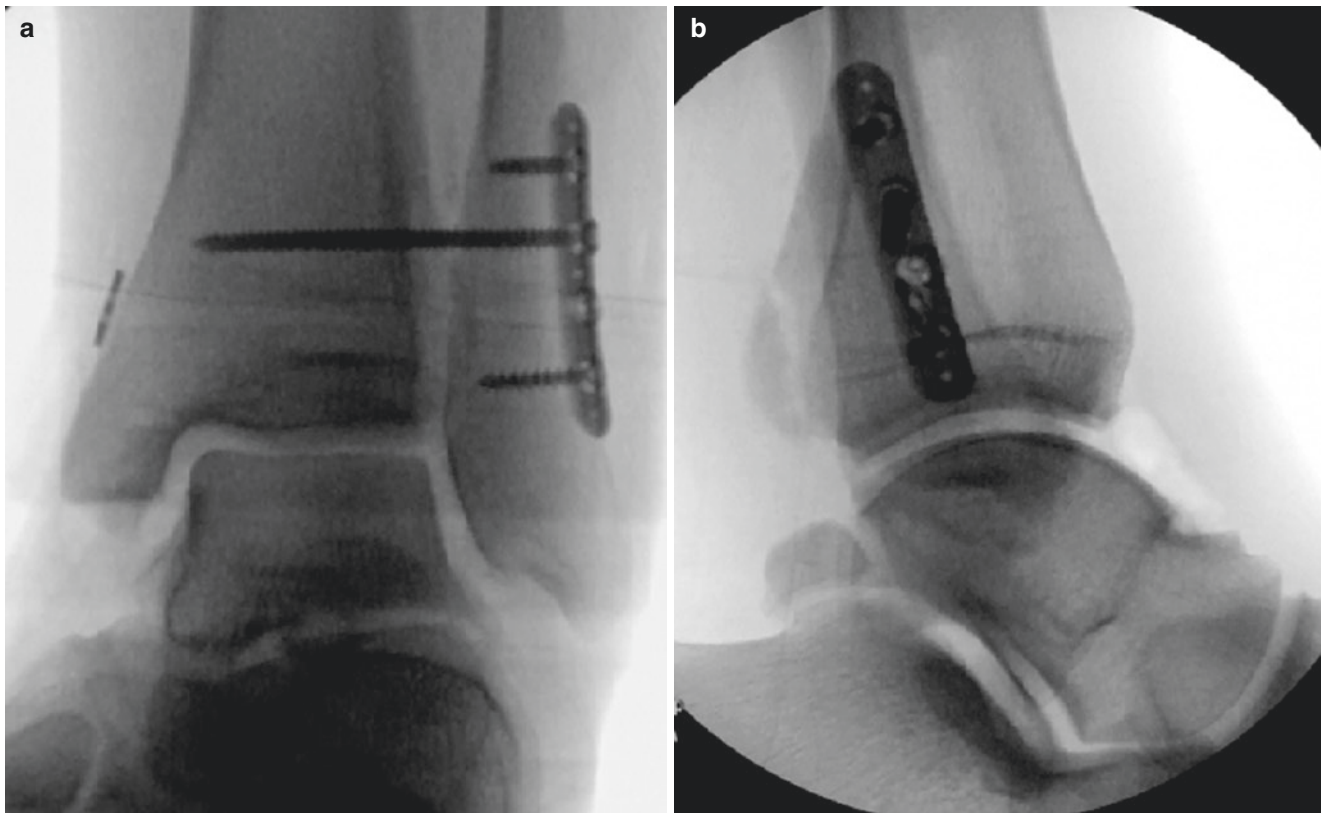
Even in the absence of fibular fracture, a one-third tubular plate with fibula-only screws placed proximally and distally can be used in conjunction with syndesmotom screws. This plate functions both as a washer to distribute forces on the fibula and to optimize syndesmotom screw position within the fibula (Fig. 29.7a, b). The plate also improves stiffness and decreases the likelihood of stress fracture following syndesmotom screw removal. In a recent cadaveric study, retention of a supplementary one-third tubular plate following syndesmotom screws removal increased fibular torsional stiffness in comparison to syndesmotom screw-only specimens [79].



**Fig. 29.6** Weight-bearing mortise radiograph demonstrating gross syndesmotom widening



**Fig. 29.7** Postoperative mortise radiograph showing syndesmotom fixation with two fully-threaded quadricortical screws



**Fig. 29.8** (a, b) Intraoperative AP (a) and lateral (b) radiographs showing syndesmotic fixation with one-third tubular plate to distribute forces on the fibula

### Dynamic Suture-Button Fixation

Dynamic stabilization of the syndesmosis using a suture-button fixation (TightRope, Arthrex, Inc., Naples, FL, USA) has become increasingly popular particularly in the treatment of athletes because it theoretically allows more physiologic motion at the syndesmosis and does not require an additional surgery for implant removal (Fig. 29.8) [80, 81]. Some authors favor the use of a one-third tubular plate with a single 4.5-mm tricortical syndesmotic screw and one suture button in elite athletes to obtain the benefits of both implants (Fig. 29.9) [25]. It is important to mention that there are several reports of skin irritation and granuloma formation from suture knots as well as osteolysis and osteomyelitis requiring surgical removal [82, 83]. Since these reports, the manufacturer has developed a knotless suture button that is currently in use but limited data exists at this time regarding long-term outcomes with this implant.

Although biomechanical studies have demonstrated marginally lower stability with suture buttons compared to screw fixation, it is this decrease in multidirectional rigidity that allows more anatomic micromotion (Fig. 29.10a, b) [84, 85]. In a cadaver model with deliberate syndesmotic malreduction,



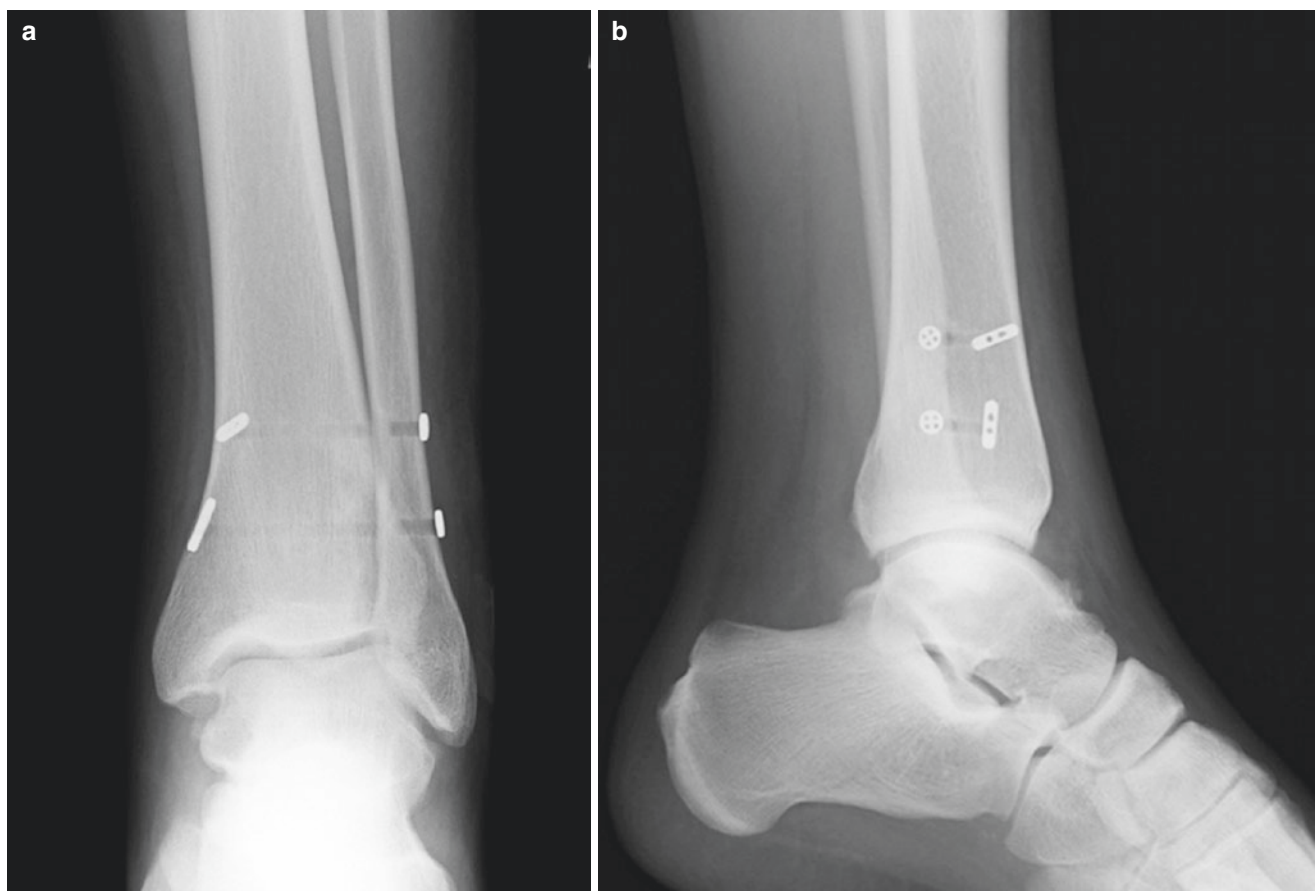
**Fig. 29.9** Intraoperative AP radiograph of suture-button fixation with one-third tubular plate-screw construct



**Fig. 29.10** Intraoperative AP radiograph of hybrid syndesmotom fixation

suture-button stabilization resulted in decreased post-operative displacement as opposed to conventional screw fixation [86]. Therefore, dynamic syndesmotom fixation may help to decrease the negative sequelae of iatrogenic clamp malreduction. Biomechanical studies are limited in their ability to replicate the actual forces of physiologic loading endured by the ankle during ambulation and athletic participation.

Multiple clinical studies have directly compared suture buttons and screws. In a series of 46 patients with average follow-up of 2.5 years, no malreductions were observed with the use of suture-button compared with a 21.7% malreduction rate in the screw group [87]. Additionally, this study reported no significant differences in clinical outcome scores or time to full weight bearing. In a recent prospective randomized multicenter trial, dynamic suture-button fixation was shown to provide adequate syndesmotom stabilization without failure or loss of reduction with a significantly lower reoperation rate compared with single 3.5-mm quadricortical screw fixation [88]. In addition, better clinical outcome scores were observed in the dynamic fixation group but this difference did not reach statistical significance.



**Fig. 29.11** (a, b) Postoperative AP (a) and lateral (b) radiographs showing syndesmotom fixation with two suture buttons alone

## Screw Removal

We do not recommend routine removal of syndesmotic screws placed proximal to the true syndesmotic joint. Exceptions include hardware infection, symptomatic hardware, syndesmotic malreduction, and the need for revision surgery. Screws removed prematurely (median of 9 weeks after surgery) resulted in a 10% loss of reduction [36]. Multiple studies have demonstrated that retention of syndesmotic screws, including those that are broken, does not pose a clinical problem [89, 90]. In the case of postoperative syndesmotic malreduction, the first step is to remove the syndesmotic screws. In a recent case series, 36% of syndesmosis were malreduced after screw placement and 89% of these reduced after screw removal [91].

## Rehabilitation and Back-to-Sports

Postoperatively, patients are made non-weight-bearing in a splint for 2 weeks to allow soft tissue healing and subsidence of swelling. Range of motion exercises commence 2–3 weeks after surgery if wound healing is appropriate. Partial weight bearing in a CAM boot can begin at 3–4 weeks postoperatively in a compliant, highly motivated patient. Full weight bearing as tolerated is initiated at 4 weeks after surgery along with proprioceptive and strength exercises. The return to running and high-impact sports is guided by patient symptoms and progression with a graduated rehabilitation protocol but generally is permitted at 8–10 weeks.

Again, if screws or suture-buttons are placed above the true syndesmotic joint, we do not recommend routine removal. In the case of intolerable symptomatic hardware, the surgeon should wait a minimum of 12 weeks before removing hardware to prevent recurrent diastasis. Screws placed across the true syndesmotic joint should not be removed before 12 weeks. After screw removal, a period of protected weight bearing is recommended to prevent fracture at the level of the screw holes. Leaving a one-third tubular plate in place has been shown to improve torsional strength of the fibula [79]. As previously mentioned, multiple studies have shown no difference in clinical outcome between patients with retained and removed syndesmotic screws [89, 90, 92]. Some evidence supports the contention that screw removal can facilitate improved ankle motion and patient function but generally retained screws will break to allow restoration of motion [90].

Less definitive information exists regarding suture buttons but theoretically these implants allow more physiologic syndesmotic multidirectional motion. In a recent series of 25 patients, the average reported time to full-weight-bearing was 5.5 weeks using a suture-button technique without any recurrent diastasis at 10.9 months after surgery [93]. The majority of patients in this series were treated with a single suture button, while four had two devices placed. Patients had significant improvement in SF-12 and AOFAS-Hindfoot scores.

## Evidence

The vast majority of literature surrounding treatment of syndesmotic injuries is based on level IV and V data, as well as anecdotal experience. No level I studies exist on this topic to our knowledge. With regard to our references, there are 12 level II studies and one level III study. The remainder are level IV and V studies.

## Summary

1. In the case of subtle syndesmotic injuries, contralateral comparison weight-bearing radiographs are recommended. While CT and MRI can provide additional information, these are static tests that will not identify instability.
2. Arthroscopy is a very sensitive and accurate means of diagnosing syndesmotic instability and allows the surgeon to identify and treat other intra-articular injuries. Physiologic syndesmotic opening varies among individuals, but an anterior opening of more than 2 mm is pathologic.
3. Syndesmotic implants, whether screws or suture buttons, should be placed above the true syndesmotic joint (more than 15 mm above the plafond) and therefore do not require routine removal. Premature screw removal can lead to recurrent diastasis.
4. Anatomic syndesmotic reduction is the most important predictor of functional outcome. To avoid iatrogenic malreduction, a large clamp should be placed in a transverse orientation with the ankle in resting position while using intraoperative fluoroscopy with comparison views.
5. In cases of concomitant syndesmotic and deltoid injury, one should strongly consider visualizing and repairing the deltoid ligament. The superficial deltoid can avulse off the medial malleolus and become entrapped in the medial gutter obstructing anatomic reduction.
6. The optimal treatment of syndesmotic injuries remains controversial and discrepancies exist in the literature regarding return to play for elite athletes. Future level I studies with long-term follow-up are necessary to provide these answers.

## References

1. Brosky T, Nyland J, Nitz A, Caborn DN. The ankle ligaments: consideration of syndesmotic injury and implications for rehabilitation. *J Orthop Sports Phys Ther.* 1995;21(4):197–205.
2. McCollum GA, van den Bekerom MP, Kerkhoffs GM, Calder JD, van Dijk CN. Syndesmosis and deltoid ligament injuries in the athlete. *Knee Surg Sports Traumatol Arthrosc.* 2013;21(6):1328–37.
3. Fallat L, Grimm DJ, Saracco JA. Sprained ankle syndrome: prevalence and analysis of 639 acute injuries. *J Foot Ankle Surg.* 1998;37(4):280–5.

4. Boytim MJ, Fischer DA, Neumann L. SyndesmotiC ankle sprains. *Am J Sports Med.* 1991;19(3):294–8.
5. Waterman BR, Belmont Jr PJ, Cameron KL, Svoboda SJ, Alitz CJ, Owens BD. Risk factors for syndesmotiC and medial ankle sprain: role of sex, sport, and level of competition. *Am J Sports Med.* 2011;39(5):992–8.
6. Wright RW, Barile RJ, Surprenant DA, Matava MJ. Ankle syndesmotiC sprains in national hockey league players. *Am J Sports Med.* 2004;32(8):1941–5.
7. Jones MH, Amendola A. SyndesmotiC sprains of the ankle: a systematic review. *Clin Orthop Relat Res.* 2007;455:173–5.
8. Gerber JP, Williams GN, Scoville CR, Arciero RA, Taylor DC. Persistent disability associated with ankle sprains: a prospective examination of an athletic population. *Foot Ankle Int.* 1998;19(10):653–60.
9. Hopkinson WJ, St Pierre P, Ryan JB, Wheeler JH. SyndesmotiC sprains of the ankle. *Foot Ankle.* 1990;10(6):325–30.
10. Williams GN, Jones MH, Amendola A. SyndesmotiC ankle sprains in athletes. *Am J Sports Med.* 2007;35(7):1197–207.
11. Bartonicek J. Anatomy of the tibiofibular syndesmotiC and its clinical relevance. *Surg Radiol Anat.* 2003;25(5–6):379–86.
12. Xenos JS, Hopkinson WJ, Mulligan ME, Olson EJ, Popovic NA. The tibiofibular syndesmotiC. Evaluation of the ligamentous structures, methods of fixation, and radiographic assessment. *J Bone Joint Surg Am.* 1995;77(6):847–56.
13. Close JR. Some applications of the functional anatomy of the ankle joint. *J Bone Joint Surg Am.* 1956;38-A(4):761–81.
14. Michelson JD, Helgemo Jr SL. Kinematics of the axially loaded ankle. *Foot Ankle Int.* 1995;16(9):577–82.
15. Lundberg A. Kinematics of the ankle and foot. In vivo roentgen stereophotogrammetry. *Acta Orthop Scand Suppl.* 1989;233:1–24.
16. Beumer A, van Hemert WL, Niesing R, Entius CA, Ginai AZ, Mulder PG, et al. Radiographic measurement of the distal tibiofibular syndesmotiC has limited use. *Clin Orthop Relat Res.* 2004;423:227–34.
17. Beumer A, Valstar ER, Garling EH, Niesing R, Ranstam J, Lofvenberg R, et al. Kinematics of the distal tibiofibular syndesmotiC: radiostereometry in 11 normal ankles. *Acta Orthop Scand.* 2003;74(3):337–43.
18. Ramsey PL, Hamilton W. Changes in tibiotalar area of contact caused by lateral talar shift. *J Bone Joint Surg Am.* 1976;58(3):356–7.
19. Calhoun JH, Li F, Ledbetter BR, Viegas SF. A comprehensive study of pressure distribution in the ankle joint with inversion and eversion. *Foot Ankle Int.* 1994;15(3):125–33.
20. Ogilvie-Harris DJ, Reed SC, Hedman TP. Disruption of the ankle syndesmotiC: biomechanical study of the ligamentous restraints. *Arthroscopy.* 1994;10(5):558–60.
21. Candal-Couto JJ, Burrow D, Bromage S, Briggs PJ. Instability of the tibio-fibular syndesmotiC: have we been pulling in the wrong direction? *Injury.* 2004;35(8):814–8.
22. Michelson JD, Waldman B. An axially loaded model of the ankle after pronation external rotation injury. *Clin Orthop Relat Res.* 1996;328:285–93.
23. Beumer A, Valstar ER, Garling EH, Niesing R, Ginai AZ, Ranstam J, et al. Effects of ligament sectioning on the kinematics of the distal tibiofibular syndesmotiC: a radiostereometric study of 10 cadaveric specimens based on presumed trauma mechanisms with suggestions for treatment. *Acta Orthop.* 2006;77(3):531–40.
24. Lauge-Hansen N. Fractures of the ankle. II. Combined experimental-surgical and experimental-roentgenologic investigations. *Arch Surg.* 1950;60(5):957–85.
25. Anderson RB, Hunt KJ, McCormick JJ. Management of common sports-related injuries about the foot and ankle. *J Am Acad Orthop Surg.* 2010;18(9):546–56.
26. Beumer A, van Hemert WL, Swierstra BA, Jasper LE, Belkoff SM. A biomechanical evaluation of clinical stress tests for syndesmotiC ankle instability. *Foot Ankle Int.* 2003;24(4):358–63.
27. Hunt KJ, George E, Harris AH, Dragoo JL. Epidemiology of syndesmotiC injuries in intercollegiate football: incidence and risk factors from National Collegiate Athletic Association injury surveillance system data from 2004–2005 to 2008–2009. *Clin J Sport Med.* 2013;23(4):278–82.
28. Mak MF, Gartner L, Pearce CJ. Management of syndesmotiC injuries in the elite athlete. *Foot Ankle Clin.* 2013;18(2):195–214.
29. Nussbaum ED, Hosea TM, Sieler SD, Incremona BR, Kessler DE. Prospective evaluation of syndesmotiC ankle sprains without diastasis. *Am J Sports Med.* 2001;29(1):31–5.
30. van Dijk CN, Mol BW, Lim LS, Marti RK, Bossuyt PM. 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.* 1996;67(6):566–70.
31. Kiter E, Bozkurt M. The crossed-leg test for examination of ankle syndesmotiC injuries. *Foot Ankle Int.* 2005;26(2):187–8.
32. Beumer A, Swierstra BA, Mulder PG. Clinical diagnosis of syndesmotiC ankle instability: evaluation of stress tests behind the curtains. *Acta Orthop Scand.* 2002;73(6):667–9.
33. de Cesar PC, Avila EM, de Abreu MR. Comparison of magnetic resonance imaging to physical examination for syndesmotiC injury after lateral ankle sprain. *Foot Ankle Int.* 2011;32(12):1110–4.
34. Amendola A, Williams G, Foster D. Evidence-based approach to treatment of acute traumatic syndesmotiC (high ankle) sprains. *Sports Med Arthrosc.* 2006;14(4):232–6.
35. Ebraheim NA, Elgafy H, Padanilam T. SyndesmotiC disruption in low fibular fractures associated with deltoid ligament injury. *Clin Orthop Relat Res.* 2003;409:260–7.
36. Weening B, Bhandari M. Predictors of functional outcome following transsyndesmotiC screw fixation of ankle fractures. *J Orthop Trauma.* 2005;19(2):102–8.
37. Harper MC, Keller TS. A radiographic evaluation of the tibiofibular syndesmotiC. *Foot Ankle.* 1989;10(3):156–60.
38. Ostrum RF, De Meo P, Subramanian R. A critical analysis of the anterior-posterior radiographic anatomy of the ankle syndesmotiC. *Foot Ankle Int.* 1995;16(3):128–31.
39. Beumer A, Valstar ER, Garling EH, van Leeuwen WJ, Sikma W, Niesing R, et al. External rotation stress imaging in syndesmotiC injuries of the ankle: comparison of lateral radiography and radiostereometry in a cadaveric model. *Acta Orthop Scand.* 2003;74(2):201–5.
40. Marmor M, Hansen E, Han HK, Buckley J, Matityahu A. Limitations of standard fluoroscopy in detecting rotational malreduction of the syndesmotiC in an ankle fracture model. *Foot Ankle Int.* 2011;32(6):616–22.
41. Ebraheim NA, Lu J, Yang H, Mekhail AO, Yeasting RA. Radiographic and CT evaluation of tibiofibular syndesmotiC diastasis: a cadaver study. *Foot Ankle Int.* 1997;18(11):693–8.
42. Sclafani SJ. Ligamentous injury of the lower tibiofibular syndesmotiC: radiographic evidence. *Radiology.* 1985;156(1):21–7.
43. Muhle C, Frank LR, Rand T, Ahn JM, Yeh LR, Trudell D, et al. Tibiofibular syndesmotiC: high-resolution MRI using a local gradient coil. *J Comput Assist Tomogr.* 1998;22(6):938–44.
44. Takao M, Ochi M, Oae K, Naito K, Uchio Y. Diagnosis of a tear of the tibiofibular syndesmotiC. The role of arthroscopy of the ankle. *J Bone Joint Surg Br.* 2003;85(3):324–9.
45. Mei-Dan O, Kots E, Barchilon V, Massarwe S, Nyska M, Mann G. A dynamic ultrasound examination for the diagnosis of ankle syndesmotiC injury in professional athletes: a preliminary study. *Am J Sports Med.* 2009;37(5):1009–16.
46. Brown KW, Morrison WB, Schweitzer ME, Parellada JA, Nothnagel H. MRI findings associated with distal tibiofibular syndesmotiC injury. *AJR Am J Roentgenol.* 2004;182(1):131–6.
47. Hepple S, Guha A. The role of ankle arthroscopy in acute ankle injuries of the athlete. *Foot Ankle Clin.* 2013;18(2):185–94.
48. Ogilvie-Harris DJ, Gilbert MK, Chorney K. Chronic pain following ankle sprains in athletes: the role of arthroscopic surgery. *Arthroscopy.* 1997;13(5):564–74.

49. Edwards Jr GS, DeLee JC. Ankle diastasis without fracture. *Foot Ankle*. 1984;4(6):305–12.
50. Williams GN, Allen EJ. Rehabilitation of syndesmotom (high) ankle sprains. *Sports Health*. 2010;2(6):460–70.
51. Amendola A. Controversies in diagnosis and management of syndesmosis injuries of the ankle. *Foot Ankle*. 1992;13(1):44–50.
52. Phisitkul P, Ebinger T, Goetz J, Vaseenon T, Marsh JL. Forceps reduction of the syndesmosis in rotational ankle fractures: a cadaveric study. *J Bone Joint Surg Am*. 2012;94(24):2256–61.
53. Tornetta 3rd P, Spoo JE, Reynolds FA, Lee C. Overtightening of the ankle syndesmosis: is it really possible? *J Bone Joint Surg Am*. 2001;83-A(4):489–92.
54. Sagi HC, Shah AR, Sanders RW. The functional consequence of syndesmotom joint malreduction at a minimum 2-year follow-up. *J Orthop Trauma*. 2012;26(7):439–43.
55. Miller AN, Barei DP, Iaquinto JM, Ledoux WR, Beingessner DM. Iatrogenic syndesmosis malreduction via clamp and screw placement. *J Orthop Trauma*. 2013;27(2):100–6.
56. Gardner MJ, Demetrakopoulos D, Briggs SM, Helfet DL, Lorch DG. Malreduction of the tibiofibular syndesmosis in ankle fractures. *Foot Ankle Int*. 2006;27(10):788–92.
57. Miller AN, Carroll EA, Parker RJ, Boraiah S, Helfet DL, Lorch DG. Direct visualization for syndesmotom stabilization of ankle fractures. *Foot Ankle Int*. 2009;30(5):419–26.
58. Chissell HR, Jones J. The influence of a diastasis screw on the outcome of Weber type-C ankle fractures. *J Bone Joint Surg Br*. 1995;77(3):435–8.
59. Hsu AR, Gross CE, Lee S. Intraoperative O-arm computed tomography evaluation of syndesmotom reduction: case report. *Foot Ankle Int*. 2013;34(5):753–9.
60. Franke J, von Recum J, Suda AJ, Grutzner PA, Wendl K. Intraoperative three-dimensional imaging in the treatment of acute unstable syndesmotom injuries. *J Bone Joint Surg Am*. 2012;94(15):1386–90.
61. Oae K, Takao M, Naito K, Uchio Y, Kono T, Ishida J, et al. Injury of the tibiofibular syndesmosis: value of MR imaging for diagnosis. *Radiology*. 2003;227(1):155–61.
62. Loren GJ, Ferkel RD. Arthroscopic assessment of occult intra-articular injury in acute ankle fractures. *Arthroscopy*. 2002;18(4):412–21.
63. Dijk CN, SpringerLink (Online service). Ankle arthroscopy techniques developed by the Amsterdam Foot and Ankle School. Available from: <http://dx.doi.org/10.1007/978-3-642-35989-7>.
64. Ogilvie-Harris DJ, Reed SC. Disruption of the ankle syndesmosis: diagnosis and treatment by arthroscopic surgery. *Arthroscopy*. 1994;10(5):561–8.
65. Wolf BR, Amendola A. Syndesmosis injuries in the athlete: when and how to operate. *Curr Opin Orthop*. 2002;13(2):151–4.
66. Miller SD. Controversies in ankle fracture treatment. Indications for fixation of stable Weber type B fractures and indications for syndesmosis stabilization. *Foot Ankle Clin*. 2000;5(4):841–51. vi.
67. Stufkens SA, van den Bekerom MP, Doornberg JN, van Dijk CN, Kloen P. Evidence-based treatment of maisonneuve fractures. *J Foot Ankle Surg*. 2011;50(1):62–7.
68. Yamaguchi K, Martin CH, Boden SD, Labropoulos PA. Operative treatment of syndesmotom disruptions without use of a syndesmotom screw: a prospective clinical study. *Foot Ankle Int*. 1994;15(8):407–14.
69. Boden SD, Labropoulos PA, McCowin P, Lestini WF, Hurwitz SR. Mechanical considerations for the syndesmosis screw. A cadaver study. *J Bone Joint Surg Am*. 1989;71(10):1548–55.
70. Taylor DC, Tenuta JJ, Uhorchak JM, Arciero RA. Aggressive surgical treatment and early return to sports in athletes with grade III syndesmosis sprains. *Am J Sports Med*. 2007;35(11):1833–8.
71. Kukreti S, Faraj A, Miles JN. Does position of syndesmotom screw affect functional and radiological outcome in ankle fractures? *Injury*. 2005;36(9):1121–4.
72. McBryde A, Chiasson B, Wilhelm A, Donovan F, Ray T, Bacilla P. Syndesmotom screw placement: a biomechanical analysis. *Foot Ankle Int*. 1997;18(5):262–6.
73. Markolf KL, Jackson SR, McAllister DR. Syndesmosis fixation using dual 3.5 mm and 4.5 mm screws with tricortical and quadricortical purchase: a biomechanical study. *Foot Ankle Int*. 2013;34(5):734–9.
74. Hansen M, Le L, Wertheimer S, Meyer E, Haut R. Syndesmosis fixation in pronation-lateral rotation ankle fractures on 3.5-mm and 4.5-mm quadricortical syndesmotom screws. *J Foot Ankle Surg*. 2006;45(2):65–9.
75. Thompson MC, Gesink DS. Biomechanical comparison of syndesmosis fixation with 3.5- and 4.5-millimeter stainless steel screws. *Foot Ankle Int*. 2000;21(9):736–41.
76. Hoiness P, Stromsoe K. Tricortical versus quadricortical syndesmosis fixation in ankle fractures: a prospective, randomized study comparing two methods of syndesmosis fixation. *J Orthop Trauma*. 2004;18(6):331–7.
77. Thordarson DB, Samuelson M, Shepherd LE, Merkle PF, Lee J. Bioabsorbable versus stainless steel screw fixation of the syndesmosis in pronation-lateral rotation ankle fractures: a prospective randomized trial. *Foot Ankle Int*. 2001;22(4):335–8.
78. Bostman OM, Pihlajamaki HK. Late foreign-body reaction to an intraosseous bioabsorbable polylactic acid screw. A case report. *J Bone Joint Surg Am*. 1998;80(12):1791–4.
79. Clanton TO, Matheny LM, Jarvis HC, Lewis EV, Ambrose CG. Quantitative analysis of torsional stiffness in supplemental one-third tubular plate fixation in the management of isolated syndesmosis injuries: a biomechanical study. *Foot Ankle Int*. 2013;34(2):267–72.
80. Seitz Jr WH, Bachner EJ, Abram LJ, Postak P, Polando G, Brooks DB, et al. Repair of the tibiofibular syndesmosis with a flexible implant. *J Orthop Trauma*. 1991;5(1):78–82.
81. Thornes B, Walsh A, Hislop M, Murray P, O'Brien M. Suture-endobutton fixation of ankle tibio-fibular diastasis: a cadaver study. *Foot Ankle Int*. 2003;24(2):142–6.
82. Willmott HJ, Singh B, David LA. Outcome and complications of treatment of ankle diastasis with tightrope fixation. *Injury*. 2009;40(11):1204–6.
83. Storey P, Gadd RJ, Blundell C, Davies MB. Complications of suture button ankle syndesmosis stabilization with modifications of surgical technique. *Foot Ankle Int*. 2012;33(9):717–21.
84. Soin SP, Knight TA, Dinah AF, Mears SC, Swierstra BA, Belkoff SM. Suture-button versus screw fixation in a syndesmosis rupture model: a biomechanical comparison. *Foot Ankle Int*. 2009;30(4):346–52.
85. Teramoto A, Suzuki D, Kamiya T, Chikenji T, Watanabe K, Yamashita T. Comparison of different fixation methods of the suture-button implant for tibiofibular syndesmosis injuries. *Am J Sports Med*. 2011;39(10):2226–32.
86. Westermann RW, Rungprai C, Goetz JE, Femino J, Amendola A, Phisitkul P. The effect of suture-button fixation on simulated syndesmotom malreduction: a cadaveric study. *J Bone Joint Surg Am*. 2014;96(20):1732–8.
87. Naqvi GA, Cunningham P, Lynch B, Galvin R, Awan N. Fixation of ankle syndesmotom injuries: comparison of tightrope fixation and syndesmotom screw fixation for accuracy of syndesmotom reduction. *Am J Sports Med*. 2012;40(12):2828–35.
88. Laflamme M, Belzile EL, Bedard L, van den Bekerom MP, Glazebrook M, Pelet S. A prospective randomized multicenter trial comparing clinical outcomes of patients treated surgically with a static or dynamic implant for acute ankle syndesmosis rupture. *J Orthop Trauma*. 2014;29(5):216–23.
89. Moore Jr JA, Shank JR, Morgan SJ, Smith WR. Syndesmosis fixation: a comparison of three and four cortices of screw fixation without hardware removal. *Foot Ankle Int*. 2006;27(8):567–72.



90. Hamid N, Loeffler BJ, Braddy W, Kellam JF, Cohen BE, Bosse MJ. Outcome after fixation of ankle fractures with an injury to the syndesmosis: the effect of the syndesmosis screw. *J Bone Joint Surg Br.* 2009;91(8):1069–73.
91. Song DJ, Lanzi JT, Groth AT, Drake M, Orchowski JR, Shaha SH, et al. The effect of syndesmosis screw removal on the reduction of the distal tibiofibular joint: a Prospective Radiographic Study. *Foot Ankle Int.* 2014;35(6):543–8.
92. Schepers T. To retain or remove the syndesmotic screw: a review of literature. *Arch Orthop Trauma Surg.* 2011;131(7):879–83.
93. Cottom JM, Hyer CF, Philbin TM, Berlet GC. Treatment of syndesmotic disruptions with the Arthrex Tightrope: a report of 25 cases. *Foot Ankle Int.* 2008;29(8):773–80.

Sergio Tejero

## Abstract

In athletes the repetitive sprain of foot and ankle could lead to acute or chronic hindfoot and midfoot instability. Mechanical instability is defined as laxity and excessive joint motion of the joints caused by structural damage of the supporting ligamentous tissues, which could need operative treatment. Functional instability does not exhibit any physical laxity of the joints because it may be caused by specific insufficiencies in proprioception, neuromuscular control, postural control, or muscle strength, which were mainly treated by physiotherapeutic rehabilitation if there were any neuromuscular disorders previously. Early diagnosis and appropriate treatment are considered vital by the athlete to return to the best sport level as soon as possible.

## Keywords

Subtalar Instability • Sports • Athletes • Lisfranc Instability • Chopart Instability

## Subtalar Instability

### Etiology and Pathomechanism

Isolated subtalar instability and complex subtalar and ankle instability are disorders that still remain controversial actually. The posterior component of the subtalar joint is made up by the thalamus of the calcaneus, which forms a convex cylinder jointed on an equivalently concave talar surface. Great individual variations have been reported in the architecture of the anterior component [1]. It is formed by the anterior and middle facets of the calcaneus, the concave surface of the navicular bone and the spring ligament (acetabulum pedis) [2]. The subtalar joint acts as a switch, with two positions controlling the movement of the other foot joints: *inversion* or midtarsal joint locking, with both columns arranged in a divergent position and *eversion* or release of the midtarsal joint, with both columns in a parallel position.

A forced inversion leads to rotational incongruence of the posterior and anterior components, compromising the rest of the joints proximally and distally, potentially leading to the long-term degeneration of the joints involved.

The typical acute injury in athletes that could lead to chronic instability of the subtalar joint is a severe supination-inversion applied to the hindfoot. Chronic instability is produced by these repetitive lateral sprains that injure the passive soft-tissue stabilizers. This results in a progressive injury of the three stabilizing layers [3]. The most superficial and lateral layer is made up by the lateral root of the inferior extensor retinaculum, the lateral talocalcaneal ligament and calcaneofibular ligament. The intermediate layer consists of the intermediate root of the inferior extensor retinaculum and the cervical ligament. The deep layer consists of the medial root of the inferior extensor retinaculum and the interosseus talocalcaneal ligament (ITCL), recognized as the primary restraint of the subtalar joint. A disruption of this ligament causes an unphysiological anterolateral rotational talar displacement. When due to an inversion trauma the anterior talofibular ligament (ATFL) is also affected, and since presumably the ITCL contributes to the stability to the ankle joint, the instability of the ankle-subtalar complex could be considered as a chronic disease [4].

S. Tejero, MD, PhD  
Foot and Ankle Unit, Virgen del Rocío, Sevilla (Spain),  
Narciso Monturiol 17 C/Manuel Siurot s/n, Bormujos 41930,  
Sevilla, Spain  
e-mail: [sertejar@us.es](mailto:sertejar@us.es)



**Fig. 30.1** Acute posteromedial subtalar dislocation without fractures

On the other hand, acute subtalar dislocation is a relatively uncommon injury (1–2% of all joint dislocations). Low- and high-energy mechanisms create two subtypes of subtalar dislocations. High-injuries are more likely to be open (20–40% of all), more likely to be lateral, and have a higher incidence of associated fracture with worse long-term prognosis [5]. Nevertheless, it may result from a twisting athletic injury (Fig. 30.1). The dislocation involves the talocalcaneal and talonavicular joints. Forced inversion of the foot results in a medial subtalar dislocation whereas eversion causes a lateral dislocation. The acute subtalar dislocation does not usually result in chronic subtalar instability. Rather, some patients present later with painful stiffness of the subtalar joint. The progression of posttraumatic degenerative changes is common in patients who have experienced a subtalar dislocation [4].

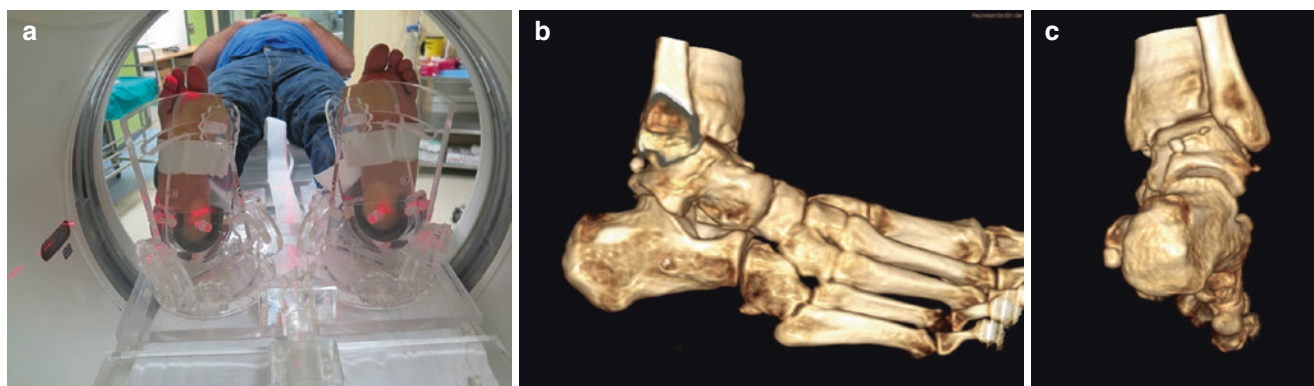
### Symptoms

In the acute phase after subtalar sprain clinical findings are similar to those in patients with severe ankle sprain: lateral ecchymosis/hematoma, swelling, and tenderness, often localized laterally in the area of the sinus tarsi. In the chronic disease due to repetitive sprains, the athletes complain about

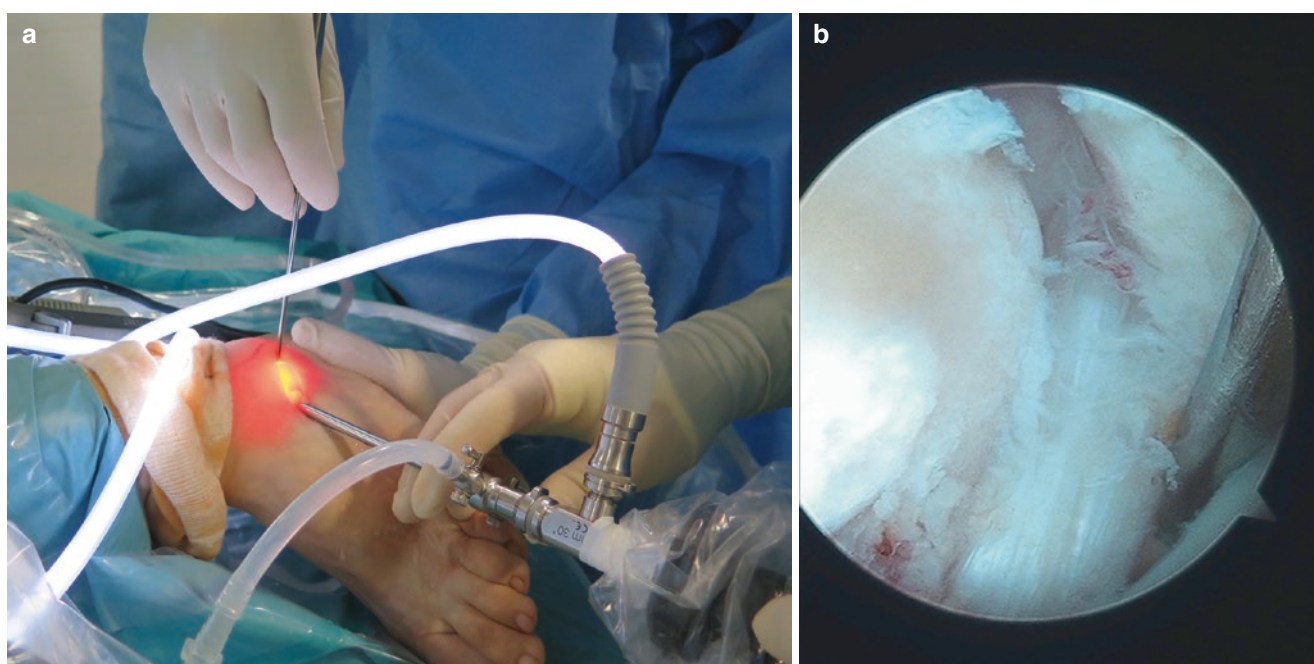
permanent or recurrent feeling of instability of the ankle like “given away” [6]. Other symptoms include recurrent swelling, painful stiffness of the subtalar joint and diffuse pain in the hindfoot, which significantly aggravates during athletic activities. Additionally, uneven surfaces may cause pain and feeling of instability. Athletic activities can exacerbate the symptoms and therefore, athletes prefer to wear subtalar stabilizing orthoses or functional tappings.

### Diagnosis

On the physical examination, lower limb alignment should be assessed, especially regarding cavovarus deformity and the function of the peroneal tendons. Chronic subtalar instability has signs and symptoms comparable with those of ankle instability [7]. When the pain level allows stressing of the hindfoot, it usually reveals an increased amount of inversion, although other times anesthesia injection into sinus tarsi is needed to explore it [6]. A separation of the posterior facet of the calcaneus and talus greater than 7 mm may indicate chronic subtalar instability for some authors [8]. Nevertheless, it has been documented that Borden’s view technique shows certain limitations [9]. We use an original device which allows for axial load while maintaining both hindfeet in supination and



**Fig. 30.2** (a) Rotatory subtalar instability confirmed by CT Scan through an original device for stress of the hindfoot in inversion and internal rotation while an axial load is applied. (b, c) Posterior subtalar subluxation in this stress position



**Fig. 30.3** (a) Ligaments of the Sinus tarsi may be explored by direct endoscopic view. (b) Interosseus talocalcaneal ligament

maximum internal rotation while performing the CT scan. Later another CT scan is performed but with both feet in maximum pronation and external rotation. Divergence of the talocalcaneal angle is measured in these stress positions. This method assesses excess joint mobility between the symptomatic and the contralateral subtalar joint (Fig. 30.2) [4, 6, 10]. Injury of the ligaments can be confirmed by means of an endoscopy prior to surgical treatment (Fig. 30.3).

### Conservative Treatment

In an **acute sprain** the usual treatment for lateral ankle injuries will suffice for subtalar ligamentous injuries, as well.

RICE (rest, ice, compression and elevation) is part of a good protocol as well as immobilization and physical therapy, when necessary. As in **chronic** ankle instability physical therapy directed at the soft tissue envelope and dynamic stabilizers will include the same muscle groups that are emphasized in ankle rehabilitation, not just the peroneals, but global strengthening around the ankle and hindfoot. Achilles tendon stretching and flexibility will allow better hindfoot position and prevent rigid hindfoot positioning from a tight gastrocnemius complex. Balance and proprioception exercises will work for both the ankle and subtalar joints. Bracing to reduce joint motion of the ankle and subtalar is important although a subtalar sling may impede performance of certain activities [11]. High top footwear may also help to reduce

pain. Hindfoot malalignment should be evaluated and first treated with orthotics if possible [12].

## Surgery

In case of failure of conservative care, **surgical treatment** is indicated [13, 14]. When there are other symptoms (anterolateral, posterior ankle impingement, tenderness of peroneal tendons) in addition to instability, an arthroscopic or tendoscopic evaluation may be recommendable [15–17]. When MR imaging or arthroscopic evaluation reveal a relatively normal anatomy with an intact CFL and ITCL, but clinically there remains a suspicion of instability, modified extensor retinaculum advancement onto the fibula is suggested as a means ensuring ST joint stability [18]. When a clear ST joint instability is suspected, reconstruction of the lateral ligament complex, including the CFL and ATFL should be performed (modified Brostrom) [19, 20]. Several surgical procedures have been described in the recent literature: anatomic repairs as well as tendon transfer procedures [4, 6]. Nevertheless, removing the peroneus brevis would cancel the role of the dynamic stabilizers of hindfoot inversion; therefore, anatomic repair is preferable in athletes. An augmentation with a free plantaris tendon graft or semitendinous allograft tendon with anatomic placement may be performed and fixed with interferential screws for reconstruction of the ATFL and CFL [21]. However, other researchers have stressed that if the ITCL is injured, a triligamentous reconstruction, including anatomic reconstruction of the CFL, ATFL and cervical ligament should be performed [22, 23]. Others advocate that taking into account the importance of the ITCL as a primary stabilizer of the subtalar joint, ITCL reconstruction appears reasonable and promising even by arthroscopic techniques [24–26]. Nevertheless, when many procedures are described to correct one condition, this means that none is perfect. Therefore, further research is required to recommend an ideal technique to stabilize the ankle-hindfoot complex without causing subtalar stiffness.

The **risk** of lateral stabilization techniques is causing nerve lesions (intermediate branch of the superficial peroneal nerve proximally and the sural nerve distally), which may result in a symptomatic neuroma. On the other hand, excessive tension of the plasty causes subtalar stiffness, preventing a correct adaptation to the ground. Persistent postoperative pain is further potential complication.

In general, **after surgical treatment** subjects should be casted for four-six weeks postoperatively, followed by a course of bracing and rehabilitation for an additional six weeks.

## Rehabilitation and Back-to-Sports

Non-weightbearing should be maintained for 1–2 weeks. The rehabilitation program is based on increasing ankle joint movement range and strength, and stretching Achilles

tendon after 4–6 weeks Proprioceptive training is initiated with strengthening after closed chain exercises are started [27]. In general, subjects should return to training at week 16 if possible and start to compete at week 20–24 after surgery. Before that, readaptation to physical activity by means of simulation of the movements specific to the relevant sport.

## Prevention

A well-regimented program of isometric and eccentric peroneal strengthening exercises, before and during the season in athletes is mandatory. This may prevent hindfoot instability and thus loss of playing time [28].

## Evidence

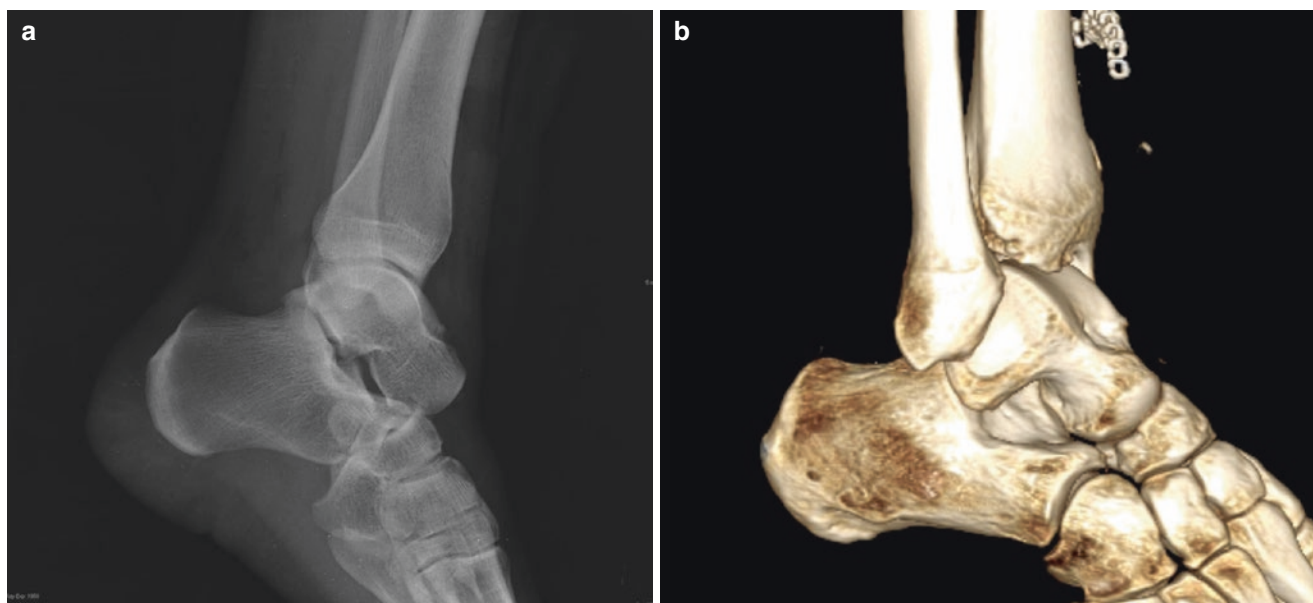
The authors compared MRI and arthroscopy for evaluation the injuries of intrinsic ligamentous of sinus tarsi and they concluded that MRI is useful for detecting CL tears, sinus tarsi fat alterations, and synovial thickening, but is inadequate for correctly detecting ITCL tears.

*Lee KB, Bai LB, Park JG, Song EK, Lee JJ. Efficacy of MRI Arthroscopy for Evaluation of Sinus Tarsi Syndrome. Foot Ank Int 2008. 29(11): 1111–1116.*

**Level II, Prospective Comparative Study. Grade of Recommendation: B**

## Summary

- Subtalar joint pain and instability is a problem encountered in athletes who suffered repetitive supination of the hindfoot, and all seem to attenuate the lateral ligaments of the ankle and deep ligamentous layers of the subtalar joint.
- The diagnosis must be inferred from an accurate history, physical examination, conferring radiographic and CT scan studies, and failure of non-surgical management (often, for ankle instability).
- Post-injury measures include early (ice and immobilization) and late (bracing and proprioceptive training).
- When conservative treatment fails, surgical procedures attempt to recreate the lateral ligament structures (CFL, ATFL, inferior retinaculum retension) should be performed
- The ideal treatment seems to reconstruct the cervical and the interosseous talocalcaneal ligaments to control the rotational movement and to avoid excessive constraint subtalar joint although further work is needed.



**Fig. 30.4** (a) Talonavicular and calcaneocuboid luxation without fractures; (b) Subluxation of the Chopart joint is also present before the closed reduction (an open reduction was needed)

## Chopart Instability

### Etiology and Pathomechanism

Isolated soft tissue injuries of the Chopart joint are scarcely described in literature because they are uncommon (10–25%) owing to the close proximity of the adjacent subtalar and Lisfranc joints. The amount of force required to cause ligamentous disruption often results in multiple fracture and/or dislocations, and the Lisfranc or subtalar joints usually fail first [29, 30]. Notwithstanding, sometimes high-energy accidents in sports practice may result in Chopart joint luxation without bony injuries (Fig. 30.4).

The spring ligament complex helps maintaining the medial longitudinal arch by serving as a fibrocartilaginous sling for the head of the talus at the talonavicular joint. The cause of spring ligament incompetence remains unclear although it seems to be secondary to acute trauma or repetitive microtrauma in eversion. A low-energy plantarflexion injury while running with subluxation of the Chopart joint may injure the anterior talonavicular ligament dorsally. On the other hand, classic dancers who have to perform extreme positions as pointe and demi-pointe experience many acute and overuse injuries (Fig. 30.5) [31–37]. In such position, the anterior talonavicular ligament plays an important role in stabilizing the talar head. When the foot is in plantar flexion, the talus is also stabilized by the anterior talofibular ligament. However, this ligament is often lax in dancers because of multiple previous ankle sprains, which lead to anterior laxity, or because of the natural ligamentous laxity commonly found in dancers. Contraction of the tibialis posterior



**Fig. 30.5** Professional ballet dancer performing a grand plie on pointes

muscle pulls the navicular down and medially. Thus, the talar head is partially subluxated anteriorly and laterally [38].

The calcaneocuboid joint is very resistant to dorsal flexion due to the bony roof of the anterior part of the calcaneus and the strong plantar ligament complex. However in sports, many cases of inversion sprain when the foot is more plantarflexed and is twisted over the fifth metatarsal head can injure the dorsolateral calcaneocuboid ligament without bony involvement [39, 40].

### Symptoms

The athletes affected by talonavicular instability feel the occurrence of rolling over on the midfoot in pronation or

internal rotation and tenderness on the spring ligament and tibial posterior tendon [41]. Chopart instability, the chronic phase, load-induced pain during sports activities and fear of giving-way or recurrent giving-way are the leading symptoms [40].

## Diagnosis

On the physical examination, lower limb alignment should be assessed, especially regarding valgus deformity and the function of the PTT tendons. To assess the radiographic alignment weight-bearing plain radiography is mandatory. Oblique view of the foot can advocate irregularity of the S-shaped Cyma line (Fig. 30.4) [42]. Stress radiography could reveal instability to varus stress testing of the transverse tarsal joints with diastasis of the calcaneocuboid joint and associated medial translation of the navicular [43]. Calcaneocuboid joint injuries that are inconspicuous in a dorsoplantar overview x-ray may be differentiated based on the lateral calcaneocuboid angle in a stress x-ray, where values below  $10^\circ$  are thought to represent a stable joint, whereas values  $>10^\circ$  may identify an unstable calcaneocuboid joint [40, 43]. CT scan is important in acute cases to identify the presence of associated fractures or bony osteochondral injury that may require concomitant treatment. MRI may be helpful in mainly ligamentous injuries. Sonographic and endoscopic view may be used to assess PTT and superomedial part of spring ligament and others medial structures [44].

## Classification

The medial injuries are classified as grade I tears, longitudinal tear of the spring ligament; grade II, spring ligament laxity without gross tear; and grade III, complete rupture of the spring ligament [41, 45].

## Conservative Treatment

In an acute sprain the standard RICE treatment is part of the protocol as well as immobilization and physical therapy, when necessary. In cases of acute Chopart dislocation, midfoot should be reduced as soon as possible under regional anesthesia or/and sedation. In subluxation in dancers, reduction should be performed in a prone position, with the knee flexed at  $90^\circ$ , opening the subtalar joint by first applying a vertical upward force with the hand holding the calcaneus and applying a posterior stress on the head of the talus. Reduction may be secured by casting in cases of dislocation or by tape around the midtarsal joint and the ankle in cases of subluxation.

In chronic midtarsal instability, Achilles tendon stretching and flexibility will allow better hindfoot position and

prevent rigid hindfoot positioning from a tight gastrocnemius complex. Balance and proprioception exercises should be the base of this treatment in athletes, together with plantar orthotics to sustain the medial longitudinal arch and protective taping. In case of failure of conservative care, surgical treatment should be considered.

## Surgery

Although some surgeons only repair the spring ligament directly, an augmentation through flexor digitorum longus (FDL) transfer may be recommended in athletes [46]. In cases of injured isolated dorsolateral calcaneocuboid ligament, the surgical procedure involves a reefing (tensioning) of the stretched original dorsal calcaneocuboid ligament and an augmentation by a local periosteal flap. A sharp U-shaped incision through the periosteum is performed at the anterolateral calcaneus with its open end adjacent to the calcaneocuboid joint line. Then the periosteal flap is slightly tightened back to its original position with the foot passively held in abduction [43].

The main **risk** in surgical stabilization techniques is nerve lesions (sural nerve).

In general, **after surgical treatment** the subjects should be casted for four-six weeks postoperatively, followed by a course of bracing and rehabilitation for an additional six weeks.

## Rehabilitation and Back-to-Sports

After the first dislocation episode immobilization should be maintained for 4–6 weeks. In cases of subluxation in dancers, specific proprioceptive training of the anteroposterior balance of the feet is started at 3–4 weeks. Later, dancers are allowed to resume dancing with protective taping that locks the anterior opening of the talonavicular joint [38].

After calcaneocuboid ligament reconstruction load bearing is allowed with an orthotic protection and a night splint until the week 6 postoperatively. At the week 2 postoperatively, intrinsic foot muscle training (physiotherapeutically guided toe strengthening of the PTT and peroneal tendons), active as well as passive supination and adduction of the foot should be restricted for 6 weeks postoperatively. Jogging activities can be allowed starting from week 8 postoperatively with a standard jogging shoe equipped with an individually constructed orthotic insert in order to align the calcaneocuboid joint and therefore protect it from non-physiological loads. Return to training at 16–20 weeks if possible.

## Prevention

A well-regimented program of isometric and eccentric peroneal strengthening exercises, before and during the season in athletes is mandatory. Stretching of the Achilles tendon is mandatory.

Orthotics should be assessed every season. This could prevent hindfoot instability and thus loss of playing time [28].

## Evidence

In a retrospective review, 110 Chopart joint dislocations were treated and evaluated in the authors's institution. A closed reduction yielded good results only in pure dislocations. An anatomical open reduction is recommended to avoid functional restrictions especially in Chopart fracture-dislocations.

*Richter M, Thermann H, Huefner T, Schmidt U, Goesling T, Krettek C. Chopart Joint Fracture-Dislocation: Initial Open Reduction Provides Better Outcome Than Closed Reduction. FAI, 25 (5):340–348, 2004.*

*Level IV. Grade of recommendation: C*

## Summary

- Although it is quite infrequent, a low-energy plantarflexion injury while running with subluxation of the Chopart joint may injure the calcaneonavicular ligament or the dorsolateral calcaneocuboid ligament.
- There is a strong association of deltoid ligament sprain with tibiofibular syndesmosis injury, PTT injury, and injury to the subtalar and transverse tarsal joints; therefore, the clinician should keep in mind that there is a high likelihood of associated injury [26].
- Oblique view of the foot can advocate irregularity of the S-shaped Cyma line. CT scan is necessary if there is a dislocation and possible fractures should be ruled out.
- In dancers, Chopart subluxation could occur after a grand plie on pointes or at the landing of a jump on demi-pointes, without any mechanism of ankle sprain.
- Before repairing only the spring ligament it is necessary to exclude misalignment and PPT dysfunction by tendoscopic view or through sonographic exploration. The dorsolateral calcaneocuboid ligament may be tensioned by augmentation of a local periosteal flap.

## Lisfranc Instability

### Etiology and Pathomechanism

Midfoot sprains are the second most common athletic foot injury (after injuries to the metatarsophalangeal joint). Every year 4% of American football players suffer this injury, which is most common among offensive linemen (29.2%) [47]. These sprains occur after an indirect low-energy force and present in a more subtle manner. In **acute injuries**, two main mechanisms of injury have been described in athletes. The first occurs in athletes that have their feet held in a strap,

such as equestrians and windsurfers, who sustain a hyperplantarflexion force while the body falls backward. The second mechanism is when the foot is in hyperplantarflexion with the metatarsophalangeal joint maximally dorsiflexed; in this position it they sustain a direct force onto their heel by a falling player that leads to hyperplantarflexion [48]. These injuries have been also described in other sporting disciplines such as football, basketball and running but they also appear to have a high incidence in gymnastics; in this case they may be caused by direct forces that occur when the foot hits the balance beam [49]. This type of injuries have been also described but they are much less frequent in comparison with other disciplines: This may be due to the stability of the pointe shoes, the physiologic control of the dancer, and possibly a degree of increased stability of the joint during the end-pointe position [50].

On the other hand, repetitive and missed midfoot sprains can lead to chronic pain and functional loss due to arthritis, deformity, or residual and **chronic** ligamentous instability.

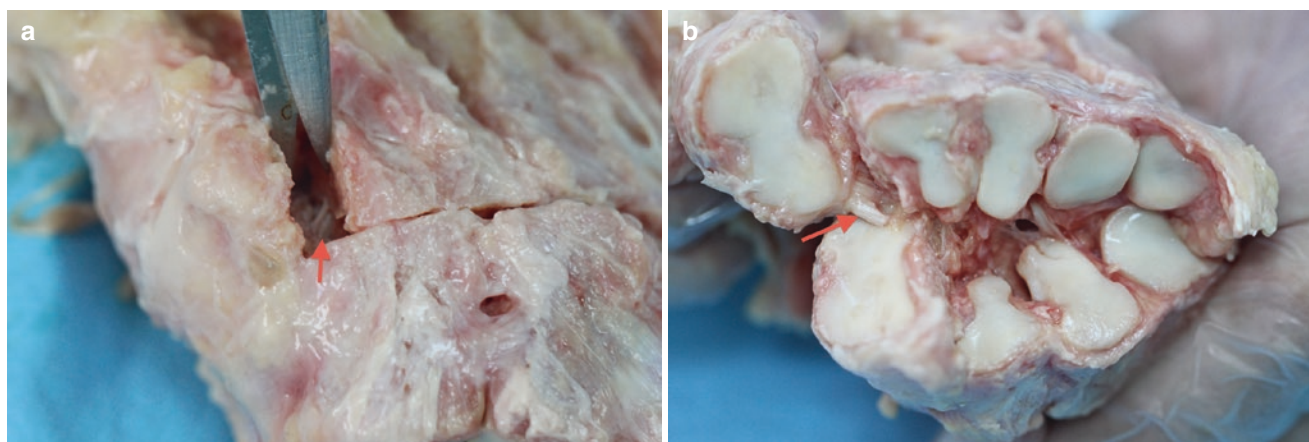
### Symptoms

Lisfranc injuries from high-energy mechanisms are associated to more prominent examination findings such as significant bruising, swelling, pain and instability; however, a proportion is still missed. In the low-energy injuries with more subtle signs and a lower index of suspicion they may be even more difficult to detect. Such difficulty is increased by the fact that athletes often tend to underestimate the injury and try to walk it off [49].

### Diagnosis

The history of the mechanism of injury provides important clues. Passive dorsiflexion and abduction of the forefoot cause pain, while dorsal bruising over the forefoot may appear later. Bilateral injuries can be found in windsurfers and parachutists [51]. Anteroposterior, 30° oblique and lateral radiographs are mandatory in these cases. Presence of one of the five following elements will support the diagnosis: (1) Loss of alignment of the medial border of the second metatarsal with the medial border of the medial cuneiform; (2) Diastasis greater than 2 mm between the base of first and second metatarsals on AP view, or a difference greater than 1 mm with the uninjured contralateral foot; (3) Loss of alignment between the medial border of fourth metatarsal and the medial border of the cuboid on oblique view (4) Presence of the fleck sign on the AP view representing an avulsion of the Lisfranc ligament; (5) Loss of alignment between the plantar aspect of the fifth metatarsal and the medial cuneiform on the lateral view. Weight-bearing views or stress views (an ankle block with local anesthesia could allow this) to compare with





**Fig. 30.6** Anatomical dissection to the Lisfranc joint. (a) The *arrow* points at the intact Lisfranc ligament. (b) The first ray may be separated from the second ray easily when the Lisfranc ligament is sectioned

the contralateral uninjured foot can be helpful [52]. Computed tomography scanning provides a more accurate assessment of minor displacement, showing occult fractures and helping preoperative planning [53–56]. Magnetic resonance imaging (MRI) scans, on the other hand, may be more helpful in identifying Stage I injuries [57–60]. Disruption of the plantar ligament between first cuneiform and the bases of the second and third metatarsals on MRI has been shown to be the strongest predictor of intraoperative instability with sensitivity and predictive values of 94% [61] (Fig. 30.6).

### Classification

Nunley and Vertullo have put forward a classification system that addresses injuries in sport, based on clinical examination, comparative weight-bearing radiographs, and bone scans (Fig. 30.7) [62].

### Conservative Treatment

Although a plaster cast may initially hold an anatomic closed reduction, as the swelling subsides this is often lost [63]. **Nonoperative treatment** should be reserved for athletes with stable Stage I injuries, and good results have been reported both when athletes are kept in a non-weight-bearing cast for 6 weeks and when they are allowed to bear weight immediately in an orthotic [62, 64]. Despite the variation existing in nonoperative treatment for Stage I injuries, the evidence shows that athletes with this type of injuries will recover and are usually able to return to sport [48, 62, 64].

### Surgery

Patients with diastasis of more than 2 mm, especially among the elite athletes, should still be operatively treated if

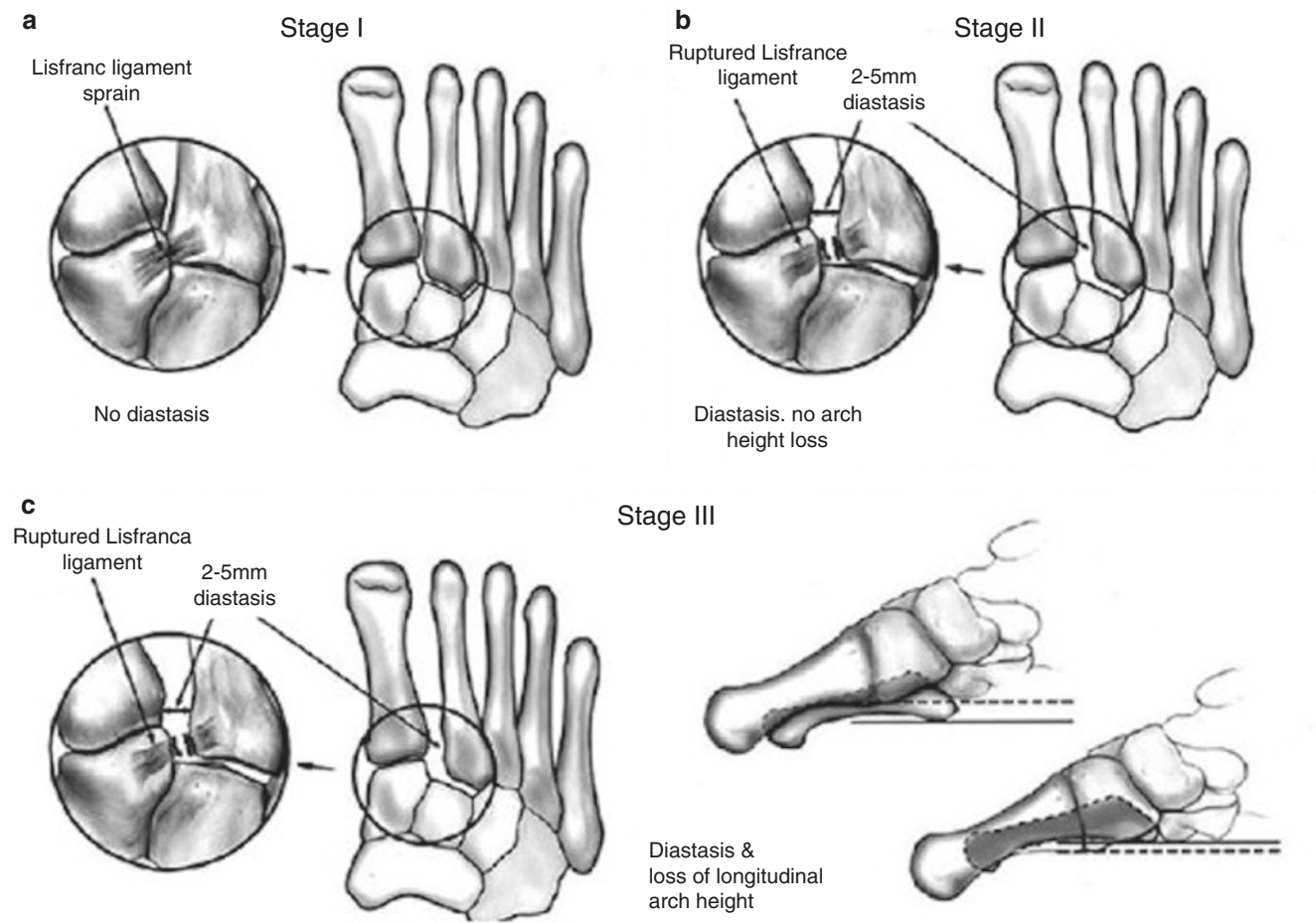
identified. Closed reduction may be possible, although some argue that an open reduction may be the best option to restore joint congruency [65]. It can be achieved by traction with finger traps and applying plantarflexion and supination of the forefoot, followed by dorsiflexion and pronation [66]. Percutaneous fixation may then be used, but if we do not achieve a reduction of the displacement in a closed manner, open reduction through two longitudinal incisions should be undertaken. After open reduction screw fixation is the preferred option, usually with 3.5- or 4.0-mm [67] (Fig. 30.8). K-wires may be used to stabilize the fourth and fifth TMT articulations if necessary. In order to avoid compromising the joint locking plates may be used to avoid. The trend to provide a more physiologic fixation to the Lisfranc articulation and to reduce the need for screw removal has led to using a suture-button fixation. This technique is promising; however, it requires further research in the athlete population [68]. On the other hand, when symptomatic **chronic lisfranc instability** is detected a fusion of the first-third TMT joint is needed.

The **risks** of this surgery are complications related to nerves and vascular elements (deep peroneal nerve and *arteria pedis*).

**After surgical treatment** a protection for at least 6 weeks is recommended, but this may be extended depending on the degree of injury [69]. It is common to have problems with screws arising after fixation, and screws have to be removed in approximately 16% of patients [49]. Some surgeons prefer to remove the screws only when patients are symptomatic [60, 70].

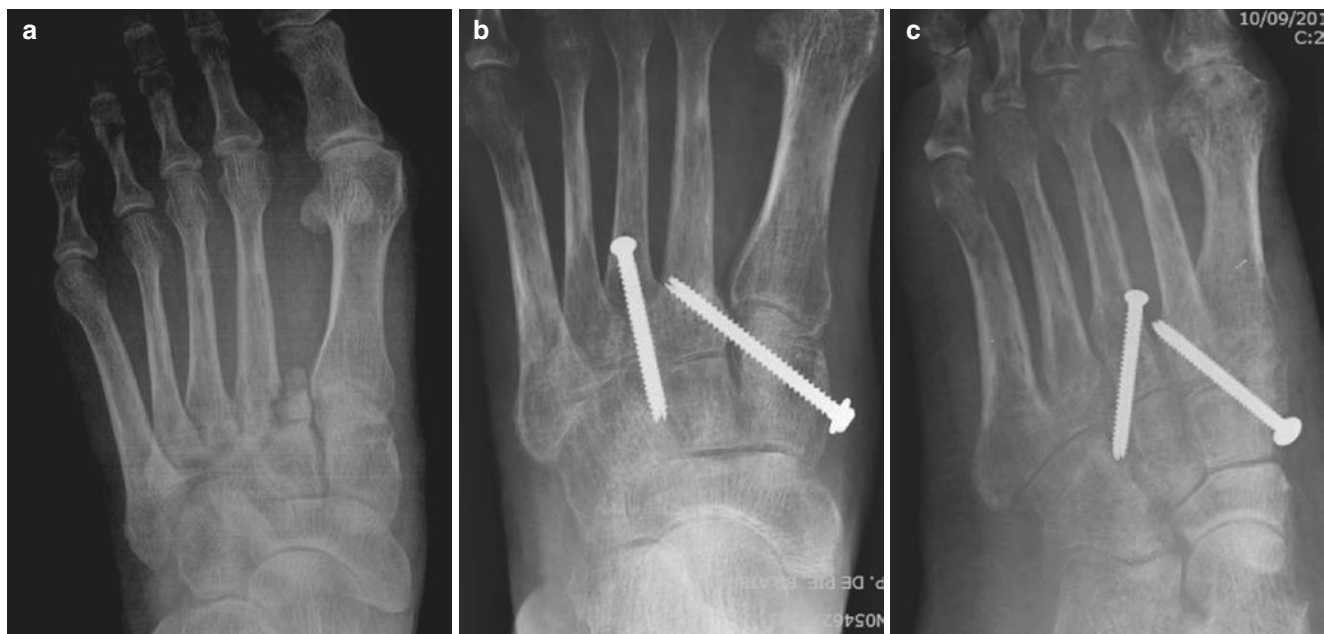
### Rehabilitation and Back-to-Sports

At week 3 postoperatively, intrinsic foot muscle training (physiotherapeutically guided toe strengthening of PTT and peroneal tendons) should be started. Weight-bearing exercises should be restricted for 6 weeks postoperatively.



**Fig. 30.7** Nunley and Vertullo's Classification of Lisfranc subtle injuries. (a) Stage I is a sprain of the Lisfranc ligament with no diastasis or arch height loss seen on radiographs but increased uptake on bone scintigrams; (b) Stage II sprains have a first to second intermetatarsal diastasis of 1–5 mm because of failure of the Lisfranc ligament but no arch height

loss. (c) Stage III sprains display first to second intermetatarsal diastasis and loss of arch height, as represented by a decrease in or inversion of the distance between the plantar aspect of the fifth metatarsal bone and the plantar aspect of the medial cuneiform bone on an erect lateral radiograph (Reprinted with permission from Nunley and Vertullo [62])



**Fig. 30.8** Screw fixation for a Lisfranc injury (a) Lisfranc injury with basal fracture of the second metatarsal (b) and (c) after reduction and fixation

Athletes with Stage I and Stage II injuries have been reported to be able to return to sport activity after just 12–14 weeks, and there is no evidence that operative treatment may will shorten this time [48, 49, 62]. Stage III injuries (or greater) have a worse outcome.

## Prevention

It is recommended to use of a well-molded arch support orthotic for athletic activities for 3–4 months after injury [53]. A well-regimented program of intrinsic and extrinsic muscles strengthening exercises, before and during the season in athletes is mandatory. Stretching of the Achilles tendon remains important.

## Evidence

A systematic review of the literature was performed to evaluate the role of surgical treatment of Lisfranc joint fracture-dislocation. The authors concluded that open reduction and internal fixation of the first three metatarsal rays with screws is a reliable method for the management of Lisfranc injuries.

*Stavlas P, Roberts CS, Xypnitos FN, et al. The role of reduction and internal fixation of Lisfranc fracture-dislocations: a systematic review of the literature. Int Orthop 2010;34:1083–91.*

**Level III. Grade of Recommendation: B**

## Summary

- Midfoot sprains are common in sports orthopaedics. Accurate diagnosis of Lisfranc injuries in athletes is essential in preventing career-ending injury.
- Undisplaced injuries have an excellent result with nonoperative treatment.
- The presence of any displacement required open reduction and anatomic fixation. The most recent available evidence mostly supports screw fixation. Plate fixation may avoid joint intrusion.
- Athletes with significantly displaced injuries should be warned of the risk of a poor outcome. Recent evidence, however, suggests that patients can return to elite competitive sports if they are treated surgically.
- Posttraumatic arthritis is the commonest complication after Lisfranc injuries. A delay of operative treatment of more than 6 months and a concomitant compensation claim are associated with a poorer outcome [43].

## References

1. Viladot A, Lorenzo JC, Salazar J, Rodriguez A. The subtalar joint: embryology and morphology. *Foot Ankle*. 1984;5:54–66.
2. Taniguchi A, Tanaka Y, Takakura Y, Kadono K, Maeda M, Yamamoto H. Anatomy of the Spring Ligament. *J Bone Joint Surg Am*. 2003;85(11):2174–8.
3. Harper MC. The lateral ligamentous support of the subtalar joint. *Foot Ankle Int*. 1991;11:354–8.
4. Barg A, Tochigi Y, Amendola A, Phisitkul P, Hintermann B, Saltzman CL. Subtalar instability: diagnosis and treatment. *Foot Ankle Int*. 2012;33(2):151–60.
5. Goldner JL, Poletti SC, Gates HS, Richardson WJ. Severe open subtalar dislocation: long-term results. *J Bone Joint Surg*. 1995;77A:1075–9.
6. Keefe DT, Haddad SL. Subtalar instability. Etiology, diagnosis and management. *Foot Ankle Clin*. 2002;7:577–609.
7. Zwipp H, Rammelt S, Grass R. Ligamentous injuries about the ankle and subtalar joints. *Clin Podiatr Med Surg*. 2002;19:195–229.
8. Heilman AE, Braly WG, Bishop JO, Noble PC, Tullos HS. An anatomic study of subtalar instability. *Foot Ankle*. 1990;10:224–8.
9. Sijbrandij ES, van Gils AP, van Hellemond FJ, Louwerens JW, de Lange EE. Assessing the subtalar joint: the Broden view revisited. *Foot Ankle Int*. 2001;22:329–34.
10. Tejero García S, Lirola Criado JF, Giráldez Sánchez MA, Cano Luis P, Navarro Robles A, Carranza Bencano A. Analysis of unstable-painful subtalar joint syndrome using dynamic stress system CT scan. *Rev S Traum y Ort*. 2013;30(1/2):10–8.
11. Wilkerson GB. Comparative biomechanical effects of the standard method of ankle taping and a taping method designed to enhance subtalar stability. *Am J Sport Med*. 1991;19(6):588–95.
12. LoPiccolo M, Chilvers M, Graham B, Manoli A. Effectiveness of the cavus foot orthosis. *J Surg Orthop Adv*. 2010;19:166–9.
13. Knupp M, Stufkens S, Bolliger L, Barg A, Hintermann B. Classification and treatment of supramalleolar deformities. *Foot Ankle Int*. 2011;32:1023–31.
14. Saltzman CL, el Khoury GY. The hindfoot alignment view. *Foot Ankle Int*. 1995;16:572–6.
15. Lee KB, Saltzman CL, Suh JS, Wasserman L, Amendola A. A posterior 3-portal arthroscopic approach for isolated subtalar arthrodesis. *Arthroscopy*. 2008;24:1306–10.
16. Lee KB, Bai LB, Park JG, Song EK, Lee JJ. Efficacy of MRI Versus Arthroscopy for evaluation of Sinus Tarsi syndrome. *Foot Ankle Int*. 2008;29(11):1111–6.
17. Oloff LM, Schulhofer SD, Bocko AP. Subtalar joint arthroscopy for Sinus Tarsi Syndrome: a review of 29 cases. *J Foot Ankle Surg*. 2001;40(3):152–7.
18. Gould N, Seligson D, Gassman J. Early and late repair of lateral ligament of the ankle. *Foot Ankle*. 1980;1:84–9.
19. Brostrom L. Sprained ankles VI. Surgical treatment of “chronic” ligaments ruptures. *Acta Chir Scand*. 1966;132:551–65.
20. Hamilton WG, Thompson FM, Snow SW. Modified Brostrom procedure for lateral ankle instability. *Foot Ankle*. 1980;1:84–9.
21. Pagenstert GL, Valderrabano V, Hintermann B. Lateral ankle ligament reconstruction with free plantaris tendon graft. *Tech Foot Ankle*. 2005;4:104–12.
22. Schon LC, Clanton TO, Baxter DE. Reconstruction for subtalar instability: a review. *Foot Ankle*. 1991;11:319–25.
23. Pisani G. Chronic laxity of the subtalar joint. *Orthop*. 1996;19:431–7.
24. Kato T. The diagnosis and treatment of instability of the subtalar joint. *J Bone Joint Surg*. 1995;77B:400–6.
25. Mann RA, Coughlin MJ. *Surgery of the foot and ankle*. 7th ed. St. Louis: Mosby; 1999. p. 1154.

26. Liu C, Jiao C, Hu Y, Guo QW, Wand C, Ao YF. Interosseous talocalcaneal ligament reconstruction with hamstring autograft under subtalar arthroscopy: case report. *Foot Ankle Int.* 2011;32:1089–94.
27. Sammarco VJ. Principles and techniques in rehabilitation of the Athlete's foot. *Techn Foot Ankle Surg.* 2003;2(3):199–207.
28. Smith RW, Reischl SF. Treatment of ankle sprains in young athletes. *Am J Sports Med.* 1986;14:465–71.
29. Ip KY, Lui TH. Isolated dorsal midtarsal (Chopart) dislocation: a case report. *J Orthop Surg.* 2006;14(3):357–9.
30. Putezhath K, Veluthedath R, Kumaran CM, Patinharayil G. Acute isolated dorsal midtarsal (Chopart's) dislocation: a case report. *J Foot Ankle Surg.* 2009;48(4):462–5.
31. Hamilton WG. Foot and ankle injuries in dancers. *Clin Sports Med.* 1988;7:143–73.
32. Hamilton WG. Sprained ankles in ballet dancers. *Foot Ankle.* 1982;3:99–102.
33. Hardaker WTJ. Foot and ankle injuries in classical ballet dancers. *Orthop Clin North Am.* 1989;20:620–7.
34. Quirk R. Injuries in classical ballet. *Aust Fam Physician.* 1984;13:802–4.
35. Quirk R. Ballet injuries: the Australian experience. *Clin Sports Med.* 1983;2:507–14.
36. Sammarco GJ. The foot and ankle in classical ballet and modern dance. In: Jahss MH, editor. *Disorders of the foot.* Philadelphia: WB Saunders Company; 1982. p. 1626–59.
37. Sammarco GJ, Miller EH. Forefoot conditions in dancers. Part 1. *Foot Ankle.* 1982;3:85–92.
38. Menetrey J, Fritschy D. Subtalar subluxation in ballet dancers. *Am J Sports Med.* 1999;27(2):143–9.
39. Andermahr J, Helling HJ, Maintz D, Mönig S, Koebke J, Rehm KE. The injury of the calcaneocuboid ligaments. *Foot Ankle Int.* 2000;21:379–84.
40. Leland RH, Marymont JV, Trevino SG, Varner KE, Noble PC. Calcaneocuboid stability: a clinical and anatomic study. *Foot Ankle Int.* 2001;22:880–4.
41. Orr JD, Nunley II J. Isolated spring ligament failure as a cause of adult-acquired flatfoot deformity. *Foot Ankle Int.* 2004;25(5):349–56.
42. Schmitt JW, Werner CML, Ossendorf C, Wanner GA, Simmen P. Avulsion fracture of the dorsal talonavicular ligament: a subtle radiographic sign of possible chopart joint dislocation. *Foot Ankle Int.* 2011;32:722–6.
43. Lohrer H, Arentz S. Calcaneocuboid joint instability: a novel operative technique for anatomic reconstruction. *Foot Ankle Int.* 2004;25:349–56.
44. Harish S, Jan E, Finlay K, Petrisor B, Popowich T, Friedman L, Jurriaans E. Sonography of the superomedial part of the spring ligament complex of the foot: a study of cadavers and asymptomatic volunteers. *Skeletal Radiol.* 2007;36(3):221–8.
45. Gazdag AR, Cracchiolo A. Rupture of the posterior tibial tendon: evaluation of injury of the spring ligament and clinical assessment of tendon transfer and ligament repair. *J Bone Joint Surg.* 1997;79-A:675–81.
46. Borton DC, Saxby TS. Tear of the plantar calcaneonavicular (spring) ligament causing flatfoot: a case report. *J Bone Joint Surg.* 1997;79-B:641–3.
47. Meyer SA, Callaghan JJ, Albright JP, Crowley ET, Powell JW. Midfoot sprains in collegiate football players. *Am J Sports Med.* 1994;22:392–401.
48. Shapiro MS, Wascher DC, Finerman GA. Rupture of Lisfranc's ligament in athletes. *Am J Sports Med.* 1994;22:687–91.
49. Eleftheriou KI, Rosenfeld PF. Lisfranc injury in the athlete evidence supporting management from sprain to fracture dislocation. *Foot Ankle Clin N Am.* 2013;18:219–36.
50. Kadel N, Boenisch M, Teitz C, et al. Stability of Lisfranc joints in ballet pointe position. *Foot Ankle Int.* 2005;26:394–400.
51. Lattermann C, Goldstein JL, Wukich DK, et al. Practical management of Lisfranc injuries in athletes. *Clin J Sport Med.* 2007;17:311–5.
52. Panchbhavi VK, Andersen CR, Vallurupalli S, et al. A minimally disruptive model and three-dimensional evaluation of Lisfranc joint diastasis. *J Bone Joint Surg Am.* 2008;90:2707–13.
53. Haapamaki V, Kiuru M, Koskinen S. Lisfranc fracture-dislocation in patients with multiple trauma: diagnosis with multidetector computed tomography. *Foot Ankle Int.* 2004;25:614–9.
54. Kalia V, Fishman EK, Carrino JA, et al. Epidemiology, imaging, and treatment of Lisfranc fracture-dislocations revisited. *Skeletal Radiol.* 2012;41:129–36.
55. Richter M, Wippermann B, Krettek C, et al. Fractures and fracture dislocations of the midfoot: occurrence, causes and long-term results. *Foot Ankle Int.* 2001;22:392–8.
56. Hawkes NC, Flemming DJ, Ho VB. Radiology corner. Answer to last month's radiology case and image: subtle Lisfranc injury: low energy midfoot sprain. *Mil Med.* 2007;172:xii–xiii.
57. Kalia V, Fishman EK, Carrino JA, et al. Epidemiology, imaging, and treatment of Lisfranc fracture-dislocations revisited. *Skeletal Radiol.* 2012;41:129–36.
58. Hatem SF, Davis A, Sundaram M. Your diagnosis? Midfoot sprain: Lisfranc ligament disruption. *Orthopedics.* 2005;28(2):75–7.
59. Potter HG, Deland JT, Gusmer PB, et al. Magnetic resonance imaging of the Lisfranc ligament of the foot. *Foot Ankle Int.* 1998;19:438–46.
60. Preidler KW, Wang YC, Brossmann J, et al. Tarsometatarsal joint: anatomic details on MR images. *Radiology.* 1996;199:733–6.
61. Raikin SM, Elias I, Dheer S, et al. Prediction of midfoot instability in the subtle Lisfranc injury. Comparison of magnetic resonance imaging with intraoperative findings. *J Bone Joint Surg Am.* 2009;91:892–9.
62. Nunley JA, Vertullo CJ. Classification, investigation, and management of midfoot sprains: Lisfranc injuries in the athlete. *Am J Sports Med.* 2002;30:871–8.
63. Myerson MS, Fisher RT, Burgess AR, et al. Fracture dislocations of the tarsometatarsal joints: end results correlated with pathology and treatment. *Foot Ankle.* 1986;6:225–42.
64. Meyer SA, Callaghan JJ, Albright JP, et al. Midfoot sprains in collegiate football players. *Am J Sports Med.* 1994;22:392–401.
65. Arntz CT, Veith RG, Hansen Jr ST. Fractures and fracture-dislocations of the tarsometatarsal joint. *J Bone Joint Surg Am.* 1988;70:173–81.
66. van Rijn J, Dorleijn DM, Boetes B, Wiersma-Tuinstra S, Moonen S. Missing the Lisfranc fracture: a case report and review of the literature. *J Foot Ankle Surg.* 2012;51:270–4.
67. Lee CA, Birkedal JP, Dickerson EA, et al. Stabilization of Lisfranc joint injuries: a biomechanical study. *Foot Ankle Int.* 2004;25:365–70.
68. Ahmed S, Bolt B, McBryde A. Comparison of standard screw fixation versus suture button fixation in Lisfranc ligament injuries. *Foot Ankle Int.* 2010;31:892–6.
69. Kuo RS, Tejjwani NC, Digiovanni CW, et al. Outcome after open reduction and internal fixation of Lisfranc joint injuries. *J Bone Joint Surg Am.* 2000;82:1609–18.
70. Ly TV, Coetzee JC. Treatment of primarily ligamentous Lisfranc joint injuries: primary arthrodesis compared with open reduction and internal fixation. A prospective, randomized study. *J Bone Joint Surg Am.* 2006;88:514–20.

Martin Wiewiorski, Alexej Barg, Beat Hintermann,  
and Victor Valderrabano

## Abstract

Osteochondral and chondral lesions of the talus are commonly found following sport related trauma to the ankle joint. Conservative treatment frequently fails, and most patients undergo surgery. Several surgical treatment techniques are available, showing good short and mid-term clinical and radiological results.

## Keywords

Talus • Ankle • OCLT • Cartilage

## Introduction

Varying terminology has been used to describe the entity of osteochondral and chondral lesions of the talus: Osteochondritis dissecans, flake fracture, osteochondral fracture, transchondral fracture or osteochondral defect. Recent expert consensus and literature have use the term osteochondral lesion of the talus (OCLT) to encompass all previously used terminology (regardless of etiology, morphology; including purely cartilaginous or combined cartilage- bone lesions) [1]. The incidence of cartilaginous lesions (0–77.5%) and combined osteo-chondral lesions (12.5–100%) varies strongly in the literature [2, 3].

M. Wiewiorski, MD (✉)  
Osteoarthritis Research Center Basel, University Hospital of Basel,  
Hebelstrasse 32, Basel 4031, Switzerland  
e-mail: [wiewiorskim@gmail.com](mailto:wiewiorskim@gmail.com)

A. Barg, MD  
Department of Orthopaedics, University of Utah,  
Salt Lake City, USA  
e-mail: [alexejbarg@mail.ru](mailto:alexejbarg@mail.ru)

B. Hintermann, MD  
Department of Orthopaedics, Kantonsspital Baselland,  
Liestal, Switzerland  
e-mail: [b.hintermann@bluewin.ch](mailto:b.hintermann@bluewin.ch)

V. Valderrabano, MD, PhD  
Orthopaedic Department, SWISS ORTHO CENTER,  
Schmerzklinik Basel, Swiss Medical Network,  
Basel, Switzerland  
e-mail: [vvalderrabano@gsmn.ch](mailto:vvalderrabano@gsmn.ch)

## Etiology and Pathomechanism

Chronic OCLT are often seen in sports active people [4], particularly in sports in which participants frequently jump and land on one foot or are expected to make sharp cutting maneuvers (for example, basketball, soccer, football, and volleyball) [5]. Most adult patients suffering from OCLT receive treatment, therefore the natural history of OCLT remains unclear. Daily clinical experience and literature suggest OCLT to be primarily caused by trauma to the ankle joint. Talar tilt in the malleolar mortise during ankle sprains and ankle fractures has been shown to induce cartilage damage and osteochondral fractures in ex- as well as in-vivo studies [6–8]. Laboratory investigations by Bernd and Harty in 1959 on cadaver ankle joints showed trauma to the upper talar surface, when exposed to inversion or eversion forces. Based on those experiments, a radiological classification was developed, which described a chain of consecutive morphological changes induced by traumatic forces to the ankle joint. These ranged from compression of subchondral bone to occurrence and displacement of an osteochondral fragment. The original classification was later modified by Loomer et al. and amended by an additional stage describing the presence of subchondral cysts [9]. These cysts are frequently found, but their pathoetiology is still unclear. Van Diejk et al. theorize that due to ultra-high congruency of the ankle joint, the synovial fluid forces its way into small cracks in the subchondral bone, creating caverns in the spongiosa

[10]. It is possible that repetitive high fluid pressure in the subchondral bone could explain the pain perceived by the patients [11, 12].

Flick and Gould found reports of osteochondral lesions of the talar dome in over 500 patients. They observed that 98 % of lateral talar dome lesions and 70 % of medial talar dome lesions were associated with trauma [13].

The authors of this chapter made experience seeing significant numbers of medial OCLT in patients and athletes with chronic ankle instability as well as in those with pes planovalgus. Pathomechanistically, in these cases with medial ankle instability and valgus tilt the medial talus edge does an impingement against the medial malleolus causing at the beginning a chondral softening, then a chondral split and damage, and at the end an OCLT.

---

## Symptoms

Patients present with pain, swelling and occasional locking of the joint. History frequently reveals past traumatic events to the ankle joint (sprains/fractures), a chronic ankle instability or a flatfoot deformity, or combinations of these. Sport activities are reduced or halted. Sports competition level is often not possible.

---

## Diagnostics, Classification

Clinical examination assessment should include assessment of ankle joint stability and hindfoot alignment. Weight-bearing radiographs are indicated as a primary imaging modality (namely: ankle mortise view, foot view dp and lateral, Saltzman), but should be complemented by modern imaging techniques such as computertomography based imaging (CT, arthro-CT, SPECT-CT) and/or magnetic resonance imaging (MRI). The defect volume and location can be precisely determined by the CT, which allows planning for osseous reconstruction and intra-operative access to the defect (open vs. arthroscopically) [14]. Routine MRI sequences add information about cartilage morphology, soft tissue pathologies and activity of subchondral bone (bone marrow edema) and are sensitive during early stages of the disease. A superiority of MRI over CT could not be determined [15] and its use is subject to surgeon experience and approach. Other available techniques scintigraphy/SPECT-CT [16, 17], ultrasound [18] may add further additional information. SPECT-CT has been shown to give information about the biological activity of the OCLT [17] and be able to improve the decision making in OCLT treatment [16].

Although several advanced classification systems describing the morphological aspect of the lesion exist for CT/MRI, the most recognized 'gold standard' classification is still the classification by Berndt/Harty (Loomer) for planar radiographs [19]. A list of all currently used classification systems can be found in a review by O'Loughlin et al [4].

Ankle arthroscopy is useful in cases of unclear ankle pain and should be performed before any open procedure. Lesion location and extent of cartilage degeneration can be easily assessed and graded according to Outerbridge [20]. However it is more difficult to exactly assess the extent of the osseous lesion. An excellent correlation between arthroscopic and MRI detection of cartilage damage has been shown in several studies [2, 21].

---

## Therapy: Conservative Treatment and Surgery

### Conservative Treatment

The first treatment of choice in chronic OCLT is the conservative treatment.

Hereby, the athlete is treated based on the individual risk OCLT factors:

- Chronic ankle instability: physiotherapy to improve the functional and mechanical ankle stability
- Mal-alignment: custom-made insoles to correct the mal-alignment
- Poor biology: hyaluronic acid or platelet-reached-plasma (PRP) infiltration

Mei-Dan et al. evaluated the use of PRP and hyaluronic acid injections as a first line of treatment for patients with primary and revision OCLT [22]. They noticed a significant improvement in pain and function for both substances over the pre-operative scores after 6 months, but no significant superiority of PRP over hyaluronic acid in terms of VAS pain improvement [22]. No clinical or radiologic results have been reported regarding the outcomes after the use of PRP in conjunction with surgical articular cartilage reparative or restorative procedures.

In general, conservative treatment shall be offered to the patient and athlete, however, it is often not always successful.

### Surgery

#### General Considerations

Lesion size is recognized as an important parameter for outcome after OCLT repair, with 1.5 cm<sup>2</sup> cited by several authors as a useful cut-off for determining prognosis and choosing among treatment options [23, 24]. Other factors that have been shown to correlate with treatment outcome in OCLT include arthroscopic appearance [25], the presence of associated lesions, whether the OCLT were contained within peripheral cartilage borders [26], symptom duration [4], and history of trauma [27]. Choi et al. found age to have a negative effect on outcome, once confounding factors such as duration of symptoms and history of trauma had been taken into consideration [28].

### Debridement Followed by Bone Marrow Stimulation

This readily available, cheap, single step technique is regarded as the gold standard for initial surgical treatment for most lesions. It usually involves arthroscopic removal of unstable cartilage and the underlying fibrotic bone (debridement/curettage/excision) and bone marrow stimulation (microfracturing/microdrilling). Penetration of the subchondral plate releases growth factors and mesenchymal stem cells (MSCs) into the defect site, and fibrocartilage develops. Several case series have demonstrated this method it provides short-term symptomatic relief [29–31]. However, lesion size seems to be the decisive factor for outcome. Chuckpaiwong et al. reported no failures after microfracturing with lesions smaller than 15 mm (n=73) regardless of location, but only one successful outcome in lesions greater than 15 mm (n=32) (level 4 study) [32, 33].

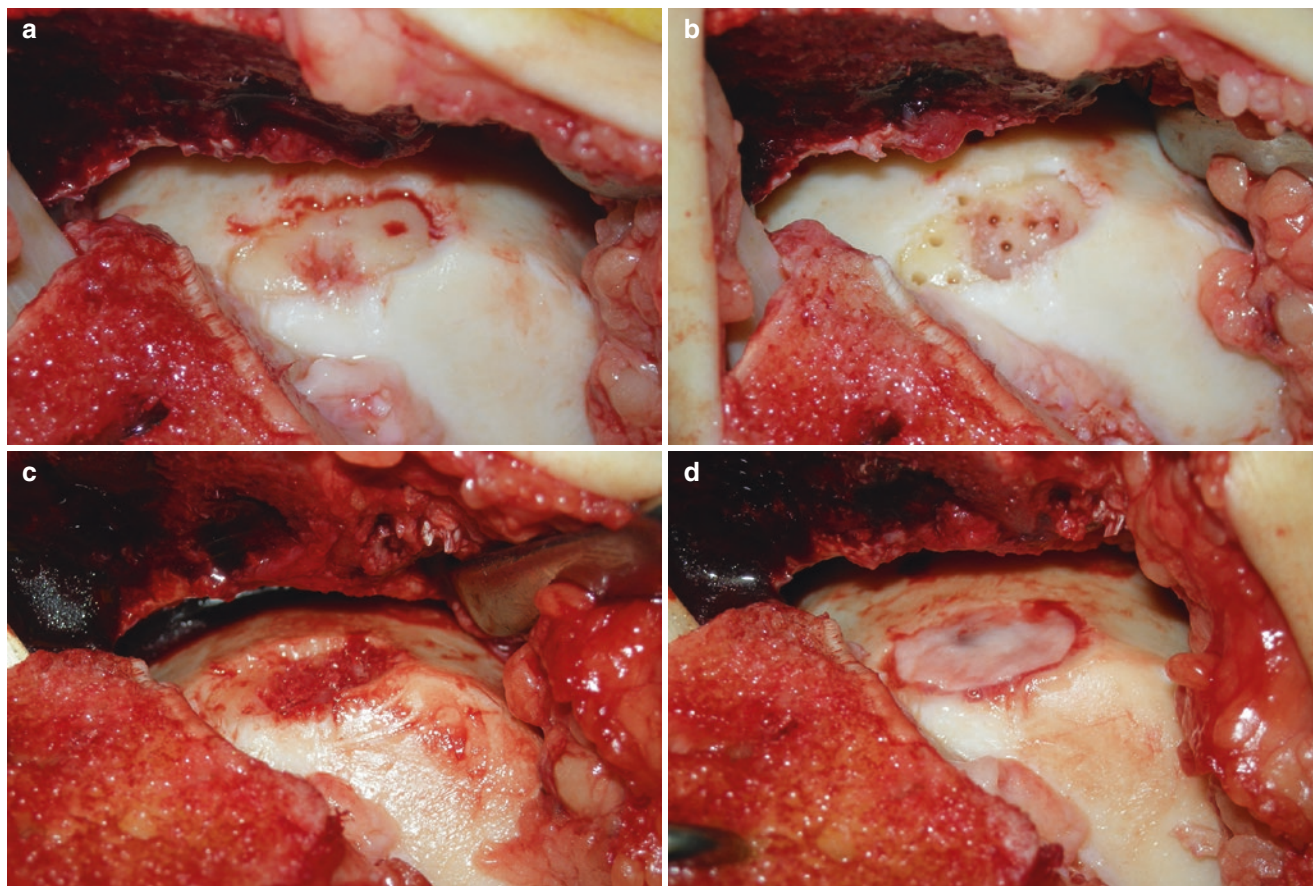
### Autologous Bone Grafting

Autologous cancellous bone (rich in growth factors and MSCs) is harvested from a suitable donor site (e.g. iliac crest, proximal/distal tibia, and calcaneus) and impacted into the debrided defect. The technique can be performed antegrade or retrograde. Kolker and al. retrospectively reviewed

a group of 13 patients treated with open antegrade bone grafting [34]. Six patients (46%) were clinical failures requiring further surgery. Of the remaining seven, postoperative functional outcome results were obtained at a mean of 51.9 months with the AOFAS hindfoot scale improving from 55 to 84.3 after surgery. Another way of bone grafting as a treatment of cystic lesions with intact cartilage covering is retrograde drilling [35]. The cartilage is first inspected arthroscopically for lesions. If healthy, a drill guide is inserted through of the portals and a guide wire is advanced through the sinus tarsi in the cystic region under fluoroscopic control. The cystic material is drilled out and the remaining cavity is filled with cancellous bone. In the largest case series available, Taranow et al. performed retrograde drilling in 16 patients and found good results after 24 months (range, 19–38) with the AOFAS hindfoot scale increasing from 53.9 to 83.6 points [36].

### Autologous Matrix-Induced Chondrogenesis (AMIC)

The AMIC procedure can be seen as an extension of antegrade bone grafting (Fig. 31.1) [37, 38]. Following open or arthroscopic debridement and microfracturing, the bone defect is filled with autologous bone graft, and then sealed



**Fig. 31.1** Open treatment of OCLT with AMIC. The medial OCLT was exposed using medial malleolar osteotomy (a). All defective cartilage and fibrotic bone was debrided followed by microdrilling (b).

Autologous bone was used to reconstruct the bony defect (c). The defect was sealed with a collagen matrix (Chondro-Gide, Geistlich Pharma AG, Wolhusen, Switzerland) (d)

with a collagen matrix (Chondro-Gide, Geistlich Pharma AG, Wolhusen, Switzerland) [39]. Valderrabano et al. recently described their experience with 26 cases of OCLT with a mean size of 1.7 cm<sup>3</sup> [3]. At mean followup of 31 months (range, 24–83) the patients showed an improved AOFAS hindfoot scale from 60 to 89 points and a decrease in VAS from 5 to 1.6 points. The advantages of the AMIC procedure are the off-the shelf availability of the matrix, a one step procedure, and only slightly higher costs than microfracturing alone.

### ACI/MACI

In this 2-stage procedure, the cartilage is initially arthroscopically harvested from the knee or ankle joint. Following culturing, chondrocyte rich is seeded on the damaged area underneath a periosteal patch (1st generation ACI). The periosteum can be replaced with a collagen matrix which can be glued onto the defect instead of suturing [40]. In the most recent development of this technique, the chondrocytes are imbedded on the collagen matrix immediately after culturing (matrix-associated ACI [MACI]; 2nd generation ACI). The currently longest available followup has recently been published by Anders et. al [41]. Twenty-two patients undergoing MACI were followed up to 63.5 months. The AOFAS hindfoot score and the VAS score remained significantly lower over the course of 5 years (95.3 and 0.9, respectively) than preoperatively (70.1 and 5.7, respectively). In two cases a biopsy taken after 12 months showed hyaline-like repair cartilage.

One study suggests that decreased postoperative pain may be an advantage of ACI in comparison to other techniques. Gobbi et al. compared surgical outcomes in 33 patients treated with chondroplasty (11 cases), microfracture (10 cases), and OATS (12 cases) [30]. No significant difference was detected between the groups at 53 months followup time (range, 24–119 months) regarding the AOFAS hindfoot scale.

Certain disadvantages of ACI/MACI remain, including high costs for culturing and the need for two surgical procedures.

### Autologous Osteochondral Transplantation (AOT)

One large or multiple smaller sized cylindrical plugs can be harvested from the non-weightbearing area of the ipsilateral knee and impacted into the debrided OCLT [42, 43]. The intervening spaces fill up with fibrocartilage but hyaline transplanted cartilage populates the bulk remainder. Advantages for AOT include the use of autograft tissue with good fixation and a single stage procedure that can be performed open or arthroscopically depending on lesion location. Surgical exposure of the posterior talar dome, large defect size, and matching cartilage shape, curvature and depth of resection are all technically demanding. Donor site

morbidity at the knee joint has been reported [44]. The longest followup for this procedure has been published by Imhoff et al [45]. Twenty-five patient (9 revision AOT) were followed up to 84 months (range, 53–124). The authors found significant increases for the AOFAS score (50–78 points) and a significant decrease for the VAS (7.8–1.5 points) from preoperative to postoperative [45].

### Allograft Transplantation

Bulk allograft transplantation is reserved for very large-volume cystic OCLT (<3 cm<sup>3</sup>). A fresh or fresh frozen allograft talus is obtained and cut-to-shape to match the receiving defect according to intraoperative radiological and direct measurements [46–48]. The transplanted allograft is fixed with screws. Raikin et al. performed fresh-allograft transplantation in 15 patients with large OCLT (mean size 6 cm<sup>3</sup>) (level 4 study) [49]. At followup after 54 months (range, 26–88), the AOFAS hindfoot scale and VAS improved from a mean of 30 points to 83 points, and 8.5–3.3, respectively. Only two patients required a subsequent arthrodesis. Some of the drawbacks of allografts are high costs and limited availability.

### Novel Techniques

A novel allograft transplantation technique involving the use of cartilage from juvenile donors (<13 years) was recently described [50]. After debridement of the OCLT, the defect is filled with particulated cartilage mixed with fibrin glue to keep it in place. This can be done in an open [51] or arthroscopic [52] procedure. So far only case reports are available [50–53].

Van Bergen et al. describe a procedure, where the gap of the debrided lesion is replaced by a metallic inlay resembling the articular talar surface [54]. Early results at 1-year followup in 15 patients are promising showing a significant decrease of pain [55].

### Supplemental Procedures

Many authors regard concomitant treatment of posttraumatic deformities (mal-alignment), ligamentous instabilities, and the reconstruction of bony defects as mandatory for successful OCLT repair [3, 56]. The restoration of correct alignment and stability of the hindfoot is essential for successful outcome after OCLT surgery. Remaining pathological stress on the repair tissue can lead to graft failure and recurrence of pain. However, the role of instability and mal-alignment in treatment of OCLT is not fully understood and evidence is sparse. Ligament repair for chronic ankle joint instability accompanying OCLT repair has been mentioned successfully in a few case series [3, 57, 58]. Valderrabano et al. performed corrective calcaneal osteotomies to correct hindfoot valgus in 16 of their 26 reported OCLT cases undergoing AMIC repair [3]. In cases of severe malalignment at the level



of the ankle joint, a supramalleolar osteotomy (SMOT) can be considered [59, 60]. In a recent comparison of CT studies before/after SMOT for partial ankle osteoarthritis, Egloff et al. could show a redistribution of subchondral bone density from the degenerated site of the talus to the opposite talar shoulder [61]. This suggests that the talar shoulder can be successfully unloaded by this procedure.

### Rehabilitation and Back-to-Sports

Immediate postoperative care consists of immobilization using an ankle walker boot (e.g. Aircast Walker, DJO Global, Vista, USA) and functional physiotherapy with 15 kg partial weight bearing starting on the second postoperative day for 8 weeks. The range of motion is restricted to 20° with use of a continuous passive motion machine, and lymphatic drainage massage for the first eight weeks. Competition level is reached in average after 6 months. For complete rehabilitation algorithm please see Table 31.1.

### Prevention

Sport specific protection from ankle sprains and ankle fractures can help reduce the incidence of chronic ankle instability and mal-alignment and therefore OCLT.

### Evidence

For literature and evidence see Table 31.2. Several techniques show satisfactory clinical outcome in case series, however, based on level of evidence criteria, there is no evidence of superiority of any surgical technique over

debridement and bone marrow stimulation. The overall level of evidence in the existing literature is low. No randomized controlled studies exist, and recommendations for OCLT surgery can only be made upon level 4 studies or expert consensus. Available studies are mostly retrospective in nature, include all OCLT sizes and morphologic types, wide ranges of followup time points, mix primary surgeries with revision surgeries, and rarely note joint alignment, stability and additional procedures. Commonly used outcome scales like the AOFAS hindfoot scale show clustering of results, poor responsiveness and ceiling effects [1]. OCLT surgery is often performed due to loss of sports activity, which is not reflected in the AOFAS hindfoot scale. A multi-center randomized controlled trial would be favorable.

### Summary

- OCLT is often seen in sports active people and correlates with chronic ankle instability (ankle sprains) and mal-alignment
- Conservative treatment of OCLT fails frequently.
- Several surgical techniques are available to reconstruct the OCLT.
- Most techniques show good mid-term results, however overall level of evidence of available studies is low.
- Restoration of correct alignment and joint stability is important for overall outcome after OCLT surgery.

**Table 31.1** Postoperative rehabilitation algorithm, the Basel approach [62]

0	1–8	9–10	11–12	13–16	17–20	21–25	26
Surgery	Partial weight bearing 15 kg in walker boot	Achievement of full-weight bearing	Ankle muscles strength/force training	See week 11–12	See week 13–16	See week 17–20	1st competition/match
	Ankle always in neutral, i.e 0° PF/DF	Achievement of full ankle ROM	Proprioceptive training	Start sport specific training including plyometric drills	Start of sport specific training with team	Consider hardware removal	
	CPM 2 × 30 min/d with ROM: DF 10°, PF 10°	Insoles for arch protection	Aqua jogging	Isokinetic training	Isokinetic training		
	Upper body ergometer for cardio training	Start of proprioceptive training	Treadmill jogging with controlled set-up				
	Upper body/hip/knee muscles strength training	Core stability training	Core stability training				
	Cryotherapy in elevation	Stationary bike for cardio training	Maintain cardiac fitness				
	Lymphatic drainage	Aquatic training					

Number of postoperative weeks

DF dorsiflexion, PF plantarflexion, ROM range of motion

**Table 31.2** Results of operative techniques for osteochondral lesions of the talus

Author	Operative technique	Patient number	Mean followup (mo)	AOFAS pre	AOFAS post	Level of evidence
Giannini et al. (2001) [63]	ACI	8	12	32.1	90	4
Taranow et. al (1999) [36]	Retrograde drilling	16	24	48	82.6	4
Schneider et al. (2009) [64]	MACI	20	21.1	60	87	4
Aurich et al. (2011) [65]	MACI	18	24.5	58.6	80.4	4
Assenmacher et al. (2001) [66]	OATS	9	9.3	n.a.	80.2	4
Valderrabano et al. (2009) [44]	OATS	12	72	45.9	80.2	4
Imhoff et al. (2011) [45]	OATS	26	84	50	78	4
Giannini et al. (2009) [67]	Bone marrow concentrate + Hylauronic acid matrix	48	29	64.4	91.4	4
Valderrabano et al. (2013) [3]	AMIC	26	31	60	89	4

*N.a.* not available

## References

- Ferkel RD, Van Dijk CN, Younger A. Instructional course lecture: osteochondral lesions of the talus, current treatment dilemmas. In: American Association of Orthopaedic Surgeons Annual Meeting. Chicago; 2013.
- Mintz DN, Tashjian GS, Connell DA, Deland JT, O'Malley M, Potter HG. Osteochondral lesions of the talus: a new magnetic resonance grading system with arthroscopic correlation. *Arthroscopy*. 2003;19:353–9.
- Valderrabano V, Miska M, Leumann A, Wiewiorski M. Reconstruction of osteochondral lesions of the talus with autologous spongiosa grafts and autologous matrix-induced chondrogenesis. *Am J Sports Med*. 2013;41:519–27.
- O'Loughlin PF, Heyworth BE, Kennedy JG. Current concepts in the diagnosis and treatment of osteochondral lesions of the ankle. *Am J Sports Med*. 2010;38:392–404.
- Fong DT-P, Hong Y, Chan L-K, Yung PS-H, Chan K-M. A systematic review on ankle injury and ankle sprain in sports. *Sports Med*. 2007;37:73–94.
- Alanen V, Taimela S, Kinnunen J, Koskinen SK, Karaharju E. Incidence and clinical significance of bone bruises after supination injury of the ankle. A double-blind, prospective study. *J Bone Joint Surg Br*. 1998;80:513–5.
- DiGiovanni BF, Fraga CJ, Cohen BE, Shereff MJ. Associated injuries found in chronic lateral ankle instability. *Foot Ankle Int Am Orthopaedic Foot Ankle Soc Swiss Foot Ankle Soc*. 2000;21:809–15.
- Hintermann B, Regazzoni P, Lampert C, Stutz G, Gachter A. Arthroscopic findings in acute fractures of the ankle. *J Bone Joint Surg Br*. 2000;82:345–51.
- Loomer R, Fisher C, Lloyd-Smith R, Sisler J, Cooney T. Osteochondral lesions of the talus. *Am J Sports Med*. 1993;21:13–9.
- van Dijk CN, Reilingh ML, Zengerink M, van Bergen CJ. Osteochondral defects in the ankle: why painful? *Knee Surg Sports Traumatol Arthrosc*. 2010;18:570–80.
- Wojtys EM, Beaman DN, Glover RA, Janda D. Innervation of the human knee joint by substance-P fibers. *Arthroscopy*. 1990;6:254–63.
- Bjurholm A, Kreicbergs A, Brodin E, Schultzberg M. Substance P and CGRP-immunoreactive nerves in bone. *Peptides*. 1988;9:165–71.
- Flick AB, Gould N. Osteochondritis dissecans of the talus (transchondral fractures of the talus): review of the literature and new surgical approach for medial dome lesions. *Foot Ankle*. 1985;5:165–85.
- van Bergen CJ, Tuijthof GJ, Blankevoort L, Maas M, Kerkhoffs GM, van Dijk CN. Computed tomography of the ankle in full plantar flexion: a reliable method for preoperative planning of arthroscopic access to osteochondral defects of the talus. *Arthroscopy*. 2012;28:985–92.
- Verhagen RA, Maas M, Dijkgraaf MG, Tol JL, Krips R, van Dijk CN. Prospective study on diagnostic strategies in osteochondral lesions of the talus. Is MRI superior to helical CT? *J Bone Joint Surg Br*. 2005;87:41–6.
- Leumann A, Valderrabano V, Plaass C, Rasch H, Studler U, Hintermann B, Pagenstert GI. A novel imaging method for osteochondral lesions of the talus – comparison of SPECT-CT with MRI. *Am J Sports Med*. 2011;39:1095–101.
- Wiewiorski M, Pagenstert G, Rasch H, Jacob AL, Valderrabano V. Pain in osteochondral lesions. *Foot Ankle Spec*. 2011;4:92–9.
- McCarthy CL, Wilson DJ, Colman TP. Anterolateral ankle impingement: findings and diagnostic accuracy with ultrasound imaging. *Skeletal Radiol*. 2008;37:209–16.
- Berndt AL, Harty M. Transchondral fractures (osteochondritis dissecans) of the talus. *J Bone Joint Surg Am*. 1959;41-A:988–1020.
- Outerbridge RE. The etiology of chondromalacia patellae. *J Bone Joint Surg Br*. 1961;43-B:752–7.
- Ferkel RD, Flannigan BD, Elkins BS. Magnetic resonance imaging of the foot and ankle: correlation of normal anatomy with pathologic conditions. *Foot Ankle*. 1991;11:289–305.
- Mei-Dan O, Carmont MR, Laver L, Mann G, Maffulli N, Nyska M. Platelet-rich plasma or hyaluronate in the management of osteochondral lesions of the talus. *Am J Sports Med*. 2012;40:534–41.
- Choi WJ, Park KK, Kim BS, Lee JW. Osteochondral lesion of the talus: is there a critical defect size for poor outcome? *Am J Sports Med*. 2009;37:1974–80.
- Cuttica DJ, Smith WB, Hyer CF, Philbin TM, Berlet GC. Osteochondral lesions of the talus: predictors of clinical outcome. *Foot Ankle Int Am Orthopaedic Foot Ankle Soc Swiss Foot Ankle Soc*. 2011;32:1045–51.
- Choi GW, Choi WJ, Youn HK, Park YJ, Lee JW. Osteochondral lesions of the talus: are there any differences between osteochondral and chondral types? *Am J Sports Med*. 2013;41:504–10.
- Choi WJ, Choi GW, Kim JS, Lee JW. Prognostic significance of the containment and location of osteochondral lesions of the talus: independent adverse outcomes associated with uncontained lesions of the talar shoulder. *Am J Sports Med*. 2013;41:126–33.
- Takao M, Ochi M, Uchio Y, Naito K, Kono T, Oae K. Osteochondral lesions of the talar dome associated with trauma. *Arthroscopy*. 2003;19:1061–7.

28. Choi WJ, Kim BS, Lee JW. Osteochondral lesion of the talus: could age be an indication for arthroscopic treatment? *Am J Sports Med.* 2012;40:419–24.
29. Becher C, Thermann H. Results of microfracture in the treatment of articular cartilage defects of the talus. *Foot Ankle Int Am Orthopaedic Foot Ankle Soc Swiss Foot Ankle Soc.* 2005;26:583–9.
30. Gobbi A, Francisco RA, Lubowitz JH, Allegra F, Canata G. Osteochondral lesions of the talus: randomized controlled trial comparing chondroplasty, microfracture, and osteochondral autograft transplantation. *Arthroscopy.* 2006;22:1085–92.
31. Thermann H, Becher C. Microfracture technique for treatment of osteochondral and degenerative chondral lesions of the talus. 2-year results of a prospective study. *Unfallchirurg.* 2004;107:27–32.
32. Chuckpaiwong B, Berkson EM, Theodore GH. Microfracture for osteochondral lesions of the ankle: outcome analysis and outcome predictors of 105 cases. *Arthroscopy.* 2008;24:106–12.
33. Wright JG, Swiontkowski MF, Heckman JD. Introducing levels of evidence to the journal. *J Bone Joint Surg Am.* 2003;85-A:1–3.
34. Kolker D, Murray M, Wilson M. Osteochondral defects of the talus treated with autologous bone grafting. *J Bone Joint Surg Br.* 2004;86:521–6.
35. Lee CK, Mercurio C. Operative treatment of osteochondritis dissecans in situ by retrograde drilling and cancellous bone graft: a preliminary report. *Clin Orthop Relat Res.* 1981;158:129–126.
36. Taranow WS, Bisignani GA, Towers JD, Conti SF. Retrograde drilling of osteochondral lesions of the medial talar dome. *Foot Ankle Int Am Orthopaedic Foot Ankle Soc Swiss Foot Ankle Soc.* 1999;20:474–80.
37. Wiewiorski M, Leumann A, Buettner O, Pagenstert G, Horisberger M, Valderrabano V. Autologous matrix-induced chondrogenesis aided reconstruction of a large focal osteochondral lesion of the talus. *Arch Orthop Trauma Surg.* 2011;131:293–6.
38. Wiewiorski M, Miska M, Nicolas G, Valderrabano V. Revision of failed osteochondral autologous Transplantation procedure for chronic talus osteochondral lesion with iliac crest graft and autologous matrix-induced chondrogenesis: a case report. *Foot Ankle Spec.* 2012;5(2):115–20.
39. Valderrabano V, Leumann A, Frigg A, Pagenstert G, Wiewiorski M. Autologous matrix-induced chondrogenesis-aided repair of osteochondral lesions of the talus. *Tech Foot Ankle Surg.* 2011;10:159–62. [10.1097/BTF.1090b1013e318237c318231b318230](https://doi.org/10.1097/BTF.1090b1013e318237c318231b318230).
40. Drobnic M, Radosavljevic D, Ravnik D, Pavlovic V, Hribernik M. Comparison of four techniques for the fixation of a collagen scaffold in the human cadaveric knee. *Osteoarthritis Cartilage.* 2006;14:337–44.
41. Anders S, Goetz J, Schubert T, Grifka J, Schaumburger J. Treatment of deep articular talus lesions by matrix associated autologous chondrocyte implantation – results at five years. *Int Orthop.* 2012;36:2279–85.
42. Hangody L. The mosaicplasty technique for osteochondral lesions of the talus. *Foot Ankle Clin.* 2003;8:259–73.
43. Hangody L, Kish G, Modis L, Szerb I, Gaspar L, Dioszegi Z, Kendik Z. Mosaicplasty for the treatment of osteochondritis dissecans of the talus: two to seven year results in 36 patients. *Foot Ankle Int Am Orthopaedic Foot Ankle Soc Swiss Foot Ankle Soc.* 2001;22:552–8.
44. Valderrabano V, Leumann A, Rasch H, Egelhof T, Hintermann B, Pagenstert G. Knee-to-ankle mosaicplasty for the treatment of osteochondral lesions of the ankle joint. *Am J Sports Med.* 2009;37 Suppl 1:105S–11.
45. Imhoff AB, Paul J, Ottinger B, Wortler K, Lammle L, Spang J, Hinterwimmer S. Osteochondral transplantation of the talus: long-term clinical and magnetic resonance imaging evaluation. *Am J Sports Med.* 2011;39:1487–93.
46. Stevenson S, Li XQ, Martin B. The fate of cancellous and cortical bone after transplantation of fresh and frozen tissue-antigen-matched and mismatched osteochondral allografts in dogs. *J Bone Joint Surg Am.* 1991;73:1143–56.
47. Williams SK, Amiel D, Ball ST, Allen RT, Wong VW, Chen AC, Sah RL, Bugbee WD. Prolonged storage effects on the articular cartilage of fresh human osteochondral allografts. *J Bone Joint Surg Am.* 2003;85-A:2111–20.
48. Raikin SM. Stage VI: massive osteochondral defects of the talus. *Foot Ankle Clin.* 2004;9:737–44, vi.
49. Raikin SM. Fresh osteochondral allografts for large-volume cystic osteochondral defects of the talus. *J Bone Joint Surg Am.* 2009;91:2818–26.
50. Cerrato R. Particulated juvenile articular cartilage allograft transplantation for osteochondral lesions of the talus. *Foot Ankle Clin.* 2013;18:79–87.
51. Hatic 2nd SO, Berlet GC. Particulated juvenile articular cartilage graft (DeNovo NT Graft) for treatment of osteochondral lesions of the talus. *Foot Ankle Spec.* 2010;3:361–4.
52. Giza E, Howell S. Allograft juvenile articular cartilage transplantation for treatment of talus osteochondral defects. *Foot Ankle Spec.* 2013;6:141–4.
53. Kruse DL, Ng A, Paden M, Stone PA. Arthroscopic De Novo NT((R)) juvenile allograft cartilage implantation in the talus: a case presentation. *J Foot Ankle Surg.* 2012;51:218–21.
54. van Bergen CJ, Zengerink M, Blankevoort L, van Sterkenburg MN, van Oldenrijk J, van Dijk CN. Novel metallic implantation technique for osteochondral defects of the medial talar dome. A cadaver study. *Acta Orthop.* 2010;81:495–502.
55. van Bergen CJA, Reilingh ML, Dijk CNV. Novel metal implantation technique for secondary osteochondral defects of the medial talar dome – one-year results of a prospective study. *Fuß Sprunggelenk.* 2012;10:130–7.
56. Aurich M, Venbrocks RA, Fuhrmann RA. Autologous chondrocyte transplantation in the ankle joint. Rational or irrational?. *Orthopade.* 2008;37:188, 190–185.
57. Kono M, Takao M, Naito K, Uchio Y, Ochi M. Retrograde drilling for osteochondral lesions of the talar dome. *Am J Sports Med.* 2006;34:1450–6.
58. Giannini S, Buda R, Faldini C, Vannini F, Bevoni R, Grandi G, Grigolo B, Berti L. Surgical treatment of osteochondral lesions of the talus in young active patients. *J Bone Joint Surg Am.* 2005;87 Suppl 2:28–41.
59. Pagenstert G, Leumann A, Hintermann B, Valderrabano V. Sports and recreation activity of varus and valgus ankle osteoarthritis before and after realignment surgery. *Foot Ankle Int Am Orthopaedic Foot Ankle Soc Swiss Foot Ankle Soc.* 2008;29:985–93.
60. Pagenstert GI, Hintermann B, Barg A, Leumann A, Valderrabano V. Realignment surgery as alternative treatment of varus and valgus ankle osteoarthritis. *Clin Orthop Relat Res.* 2007;462:156–68.
61. Egloff C, Paul J, Pagenstert G, Vavken P, Hintermann B, Valderrabano V, Muller-Gerbl M. Changes of density distribution of the subchondral bone plate after supramalleolar osteotomy for valgus ankle osteoarthritis. *J Orthopaedic Res Off Pub Orthopaedic Res Soc.* 2014;32:1356–61.
62. Valderrabano V, Barg A, Alattar A, Wiewiorski M. Osteochondral lesions of the ankle joint in professional soccer players: treatment with autologous matrix-induced chondrogenesis. *Foot Ankle Spec.* 2014;7:522–8.
63. Giannini S, Buda R, Grigolo B, Vannini F. Autologous chondrocyte transplantation in osteochondral lesions of the ankle joint. *Foot Ankle Int Am Orthopaedic Foot Ankle Soc Swiss Foot Ankle Soc.* 2001;22:513–7.
64. Schneider TE, Karaikudi S. Matrix-Induced Autologous Chondrocyte Implantation (MACI) grafting for osteochondral

- lesions of the talus. *Foot Ankle Int Am Orthopaedic Foot Ankle Soc Swiss Foot Ankle Soc.* 2009;30:810–4.
65. Aurich M, Bedi HS, Smith PJ, Rolaufts B, Muckley T, Clayton J, Blackney M. Arthroscopic treatment of osteochondral lesions of the ankle with matrix-associated chondrocyte implantation: early clinical and magnetic resonance imaging results. *Am J Sports Med.* 2011;39:311–9.
66. Assenmacher JA, Kelikian AS, Gottlob C, Kodros S. Arthroscopically assisted autologous osteochondral transplantation for osteochondral lesions of the talar dome: an MRI and clinical follow-up study. *Foot Ankle Int Am Orthopaedic Foot Ankle Soc Swiss Foot Ankle Soc.* 2001;22:544–51.
67. Giannini S, Battaglia M, Buda R, Cavallo M, Ruffilli A, Vannini F. Surgical treatment of osteochondral lesions of the talus by open-field autologous chondrocyte implantation: a 10-year follow-up clinical and magnetic resonance imaging T2-mapping evaluation. *Am J Sports Med.* 2009;37 Suppl 1: 112S–8.

Norman Espinosa, Ana Fajardo-Ruiz, and Anita Hasler

## Abstract

Anterior and posterior ankle impingement may become debilitating and needs specific attention when dealing with painful syndromes around the ankle. There are multiple causes and conditions that may provoke either an anterior or posterior ankle impingement.

A proper knowledge of the underlying problems and physical examination will lead to the right diagnosis. The correct diagnosis helps to formulate an individual treatment plan that ensures good-to-excellent outcome while attempting to bring patients back to sports.

## Keywords

Anterior • Posterior • Ankle • Impingement • Treatment

## Introduction

Bones and soft-tissues that are squeezed during certain sports activities and positions of the ankle can result in a painful impingement syndrome with variable clinical presentations. Usually, impingement syndromes can be classified into anterior and posterior syndromes.

Historically, Baetzner in 1927 was first to publish a report on this pathology [1]. Anatomical alterations on the dorsal aspect of the talus, i.e. spurs, were observed, that came into close contact with the anterior, distal tibia and caused pain. Baetzner thought that local tears within the capsule and reactive apposition of bone could promote those spurs [1]. At that time it was postulated that a forced plantarflexion would lead to the assumed capsular tears and indeed most of those problems were found in soccer player and high jumpers. This is the reason why impingement syndromes at the ankle were

called “athlete’s ankle” or “soccer’s ankle” [2]. However, the fact that other athletes (e.g. ballet dancers, runners, volleyball etc.) also suffered from those impairing pathologies has led to a change of terminology and therefore the entity has been named “impingement syndrome”. The term is far better because it does also include the mechanism of the problem.

Anterior ankle impingement is a common clinical condition characterized by chronic anterior ankle pain, which is aggravated by forced dorsiflexion. Chronic anterior ankle pain is frequently caused by tibiotalar osteophytes, which develop secondary to ankle arthrosis or recurrent micro-trauma. Chronic posterior ankle pain is frequently caused by an os trigonum, hypertrophic posterior process of the talus or other articular causes.

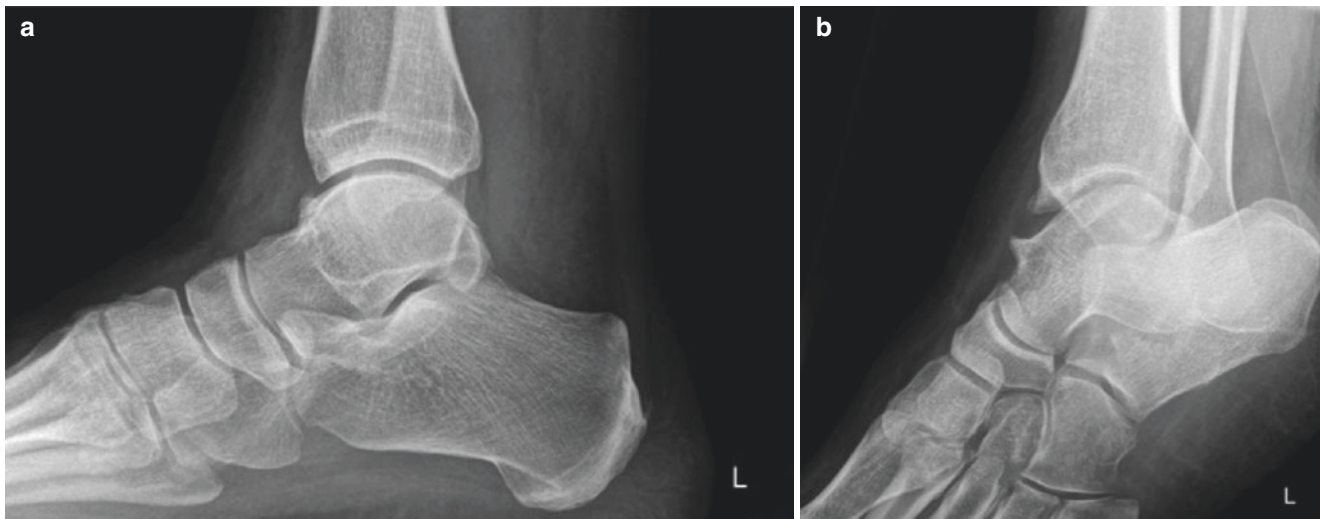
## Etiology and Pathophysiology

### Anterior Ankle Impingement

Direct and/or repetitive mechanical force effects between the anterior distal tibial rim and the talar neck while holding the ankle joint in maximal dorsiflexion are seen as primary causes with regard of development of anterior ankle osteophytes [3, 4].

N. Espinosa, MD (✉) • A. Fajardo-Ruiz (✉) • A. Hasler, MD (✉)  
Institute for Foot and Ankle Reconstruction,  
Kappelstrasse 7, Zurich 8002, Switzerland

Department of Orthopaedics, University of Zurich, Balgrist  
Hospital, Forchstrasse 340, Zurich 8008, Switzerland  
e-mail: [espinosa@fussinstitut.ch](mailto:espinosa@fussinstitut.ch); [anafajadoruiz@gmail.com](mailto:anafajadoruiz@gmail.com);  
[anitahasler@hotmail.com](mailto:anitahasler@hotmail.com)



**Fig. 32.1** (a) Depicted is the lateral view of an ankle under weight bearing conditions. An anterior bony spur can be identified at the talar neck. (b) The image demonstrates the AMI (Antero-Medial

Impingement) view of the same patient as shown in (a). The radiograph reveals a large talar and anteromedial osteophyte

Predisposed individuals with a higher risk include runners, dancers and high jumpers.

Another type of mechanism is a direct force impact onto the anterior part of the ankle joint. Predisposed individuals are soccer players.

Anatomically there is an unloaded and cartilaginous zone found, located at the distal anterior tibial rim, extending up to 3 mm proximally. As shown by Tol et al., this zone becomes frequently injured during ankle sprains [5]. Depending on the extent of lesions a variable stimulation of chondral and bony reparative processes is induced that ultimately results in cartilaginous proliferation, scarring and calcification. An osteophyte is created. The surface layer of the osteophyte is made up of mesenchymal, fibrous connective tissue. Underneath there is an intermediate layer found, which consists of fibrocartilage and hyaline cartilage. At the base of the osteophyte there is hypertrophic cartilage and bone. Frequently, enchondral ossification of the deep layers is found in osteophytes [6].

Osteophytes can develop everywhere. They can appear at the distal tibia, the talar neck, the medial and lateral gutters of the ankle joint. In patients suffering from chronic ankle instability aggravated formation of osteophytes can be seen. The pain occurring in patients with anterior ankle impingement may only partly be explained by the presence of osteophytes alone (i.e. in case of fracture or massive irritation). It is thought that pain is predominantly generated by soft-tissues that become squeezed between the osteophytes and which get secondary inflamed. This hypothesis sounds quite logic and has been confirmed by histo-pathological analyses of arthroscopically harvested specimens during surgery [7]. In order to understand the squeezing mechanism anatomical studies were done. Those studies revealed a triangular,

synovial plica and subsynovial fat and collagen tissue located at the distal, anterior tibial rim. During maximum dorsiflexion those soft-tissues are forced into the ankle joint and get clutched. As a result of the repetitive contusions the synovial layers undergo a hypertrophy, scarring, inducing an inflammation of the adjacent soft-tissues. The anterior space at the ankle joint compartment gets smaller while increasing the risk for consecutive symptomatic impingement [8].

Young athletes with recurrent ankle sprains are at risk for anterior ankle impingement [9–13]. Often they present with diffuse joint swellings after longstanding exercises, impaired dorsiflexion and pain within the anterior ankle joint compartment. Simple palpation may allow identification of prominent osteophytes and provokes pain at the anterior joint. Palpation also facilitates the distinction between anteromedial and anterolateral pain. Sometimes the combined compression of the anterior ankle joint compartment and forced dorsiflexion can provoke or increase pain [14]. However, there is a high rate of false-negative results obtained by simple palpation alone. Therefore, although palpation is necessary during clinical examination there are additional diagnostics that need to be performed to confirm the suspected impingement syndrome.

An anterior and lateral radiograph of the ankle joint (Fig. 32.1a) under weight bearing conditions is not always sufficient to assess osteophytes around the ankle joint [12, 15].

While prominent osteophytes of the distal tibial rim may easily be identified on a lateral view of the ankle, anteromedial or medial osteophytes often go mis- or undiagnosed [16]. This is due to the overlapping phenomenon of the talus and tibia. But the identification of those osteophytes is necessary when planning adequate treatment strategy. In case of suspicious anteromedial or medial ankle impingement, van

Dijk et al. suggested a new diagnostic radiographic view [15]. To obtain that specific view the leg is externally rotated by 30° and the foot held in maximum plantarflexion. The beam runs 45° angulated in a cranio-caudal direction. van Dijk and coworkers were able to demonstrate that the so-called AMI (anteromedial impingement) view (Fig. 32.1b) reached sensitivities of 85 % for tibial osteophytes and 73 % for talar osteophytes when used in conjunction with the simple lateral view of the ankle joint [15].

CT scans can be useful to assess fractures or to define possible differential diagnoses. The extent of lesions; the locations of fragments or loose bodies and calcifications can be assessed [8, 17, 18].

MRI provides useful information on involved soft-tissues and about osseous abnormalities. One of the most intriguing aspects of MRI is the three dimensional depiction of the joint and the surrounding soft-tissues. This can be of importance in the preoperative setting. The MR-arthrography has been shown to be more accurate than the native investigation alone [19–26].

## Posterior Ankle Impingement

Posterior ankle impingement syndrome reflects a clinical diagnosis. Generally, it should be distinguished between traumatic causes and those impingement syndromes that are the result of chronic overuse. According to van Dijk this distinction is essential because the latter one shows better results with regard to the surgical treatment [11].

Posterior ankle impingement is predominantly found in ballet dancers and runners [11, 27–29]. The highly trained ballet dancers and runners may reveal increased range of movement at the ankle joint. Therefore, in ballet dancers, forced plantarflexion (en pointe or demi-pointe) causes compression of the posterior ankle compartment. In contrast, runners exert repetitive traction and compression stresses on the osseous and soft tissues of the posterior ankle compartment. Both mechanisms lead to overuse of tissues between tibia and calcaneus. The following structures can be compressed in case of maximum plantarflexion: The os trigonum; a hypertrophic posterior talar process; a thickened joint capsule; posttraumatic scar formations; posttraumatic calcifications of the posterior capsule; a loose body or osteophyte at the posterior inferior tibial rim [21, 22, 30–32].

### Specific Aspects of Posterior Ankle Impingement

Very rarely, an isolated prominent posterior process of the talus or os trigonum may result in posterior ankle impingement (Figs. 32.2a–d). However, if there is an external force

that hits those structures they may become injured and painful. Ankle sprains, dancing on hard surfaces or chronic overuse with fatigue of tissues can affect the os trigonum or posterior process of the talus [22, 23, 30].

The os trigonum becomes hypermobile in relation to the talus. This abnormal movement can be painful. Another possibility is pain, which is generated by squeezing of the thickened capsular or scarred tissue either between the os trigonum and the distal tibial rim or between os trigonum and calcaneus. This condition is also called the “dancer’s heel”.

In contrast to the above-mentioned mechanisms the posterior process of the talus can fracture due to extreme plantarflexion resulting in crepitation and pain [33–35].

Both forms of posterior ankle impingement can lead to repetitive compression of the posterior joint capsule, which in turns can calcify. Those calcifications increase the stresses and reduce the space in the posterior ankle compartment, while promoting posterior ankle impingement syndrome.

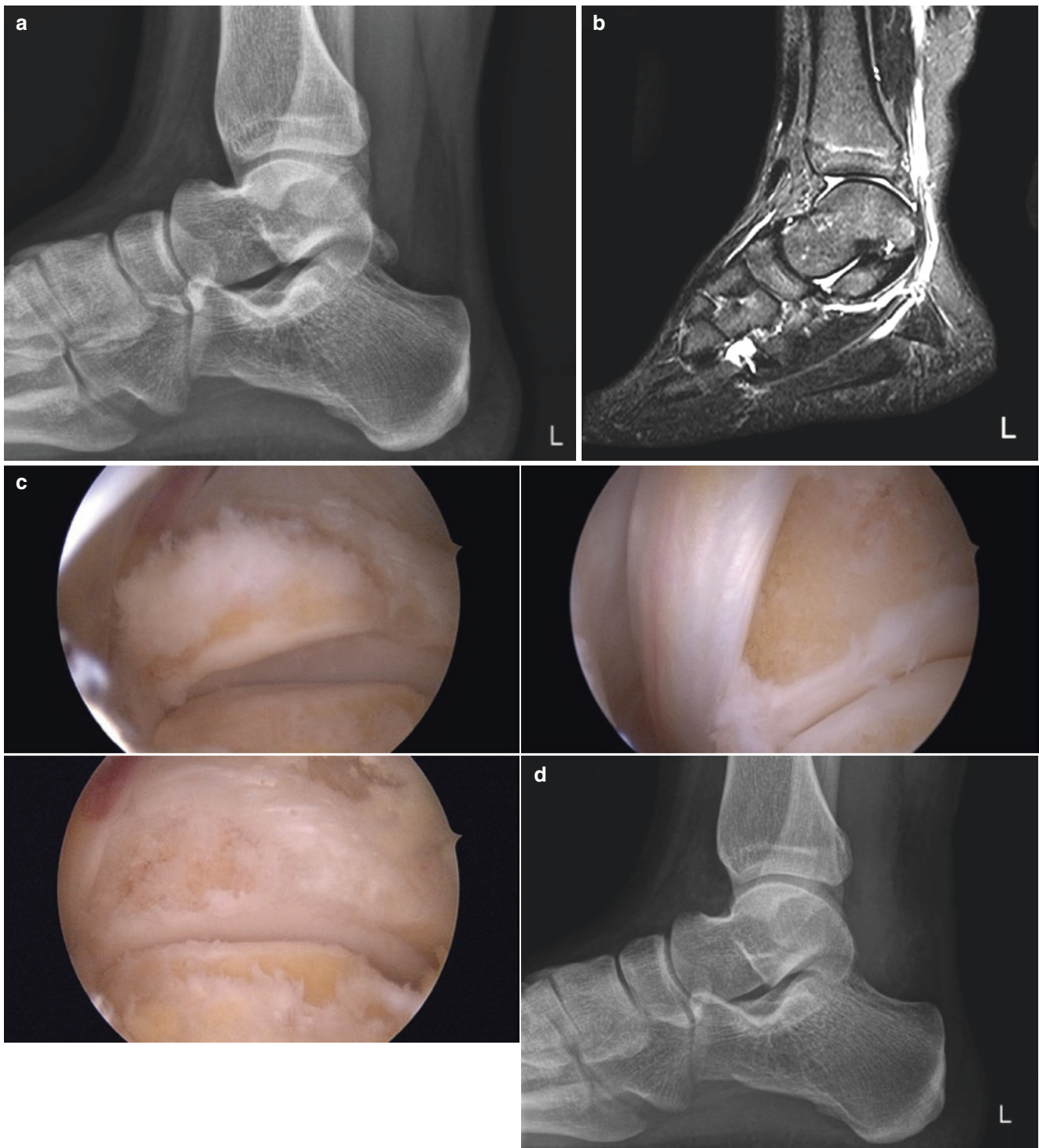
In certain cases the combined trauma mechanism of supination and plantarflexion can lead to entrapment of the posteromedial joint structures resulting in posteromedial ankle impingement syndrome.

Patients suffering from posterior ankle impingement syndrome report on pain in the posterior ankle compartment during maximum plantarflexion. Palpation can help to localize the most tender points in the posterior ankle compartment and pain can be aggravated when adding repetitive and forced plantarflexion during examination while compressing the posterior structures [28, 32, 33, 36–39].

The posterior process of the talus is best-felt posterolaterally between the Achilles tendon and the peroneal tendons. The posteromedial region is far more difficult to examine because of the flexor tendons and neurovascular bundle may mimic a posteromedial impingement syndrome.

For manual testing of any posterior impingement the patient is sitting with the knee joints in 90° of flexion [36]. During examination the rotation of the foot and tibia can be changed. By so doing a grinding moment is initiated between the posterior process of the talus or os trigonum and the tibia and calcaneus. A negative test confirms absence of a posterior impingement syndrome. In case of a positive test in conjunction with posterolateral pain during palpitation an additive, diagnostic infiltration between posterior distal tibial rim and the posterior process of the talus should be done. If after injection the pain is reduced or completely relieved the diagnosis is assured.

On conventional radiography the lateral view of the ankle allows the identification of a hypertrophic posterior process of the talus or a dislocated os trigonum. Because of projection phenomena may blur any correct diagnosis, van Dijk et al. proposed a new lateral view of the ankle joint with 25° of external rotation [11].



**Fig. 32.2** (a) Lateral foot radiograph of a 16-years old female patient. The patient complained about posterior ankle pain. (b) MRI of the same patient as shown in (a) It can clearly be seen how the soft-tissues are irritated in the posterior compartment of the ankle joint. (c) Intraoperative view while performing the 2-portal posterior hindfoot

endoscopy according to van Dijk. The photographs show the larger and hypermobile os trigonum, which subsequently has been removed. Note, the flexor hallucis longus tendon is free and does not infringe against the os trigonum anymore. (d) Postoperative radiograph of the young female patient as presented in (a–c)



## Epidemiology

There is only a paucity of reports available regarding the epidemiology of ankle impingement. The incidence of anterior impingement syndromes in soccer players is 45 % and in dancers 59 % [40]. The overall incidence in the U.S. population is thought to range about 15 %.

## Rehabilitation and Back to Sports

### Nonoperative Treatment

In early stages of disease a nonoperative treatment could be helpful to reduce symptoms and to improve quality of life [3, 28].

The treatment modalities include temporary immobilization (e.g. fractures of the os trigonum or posterior process of the talus), local and guided steroid injections, physical therapy and nonsteroidal anti-inflammatory drugs. Shock-absorbing shoes have been advocated and represent reasonable options in the nonsurgical treatment of ankle impingement syndromes [41]. In case of chronic ankle instability training of the peroneal musculature is recommended to improve dynamic stabilization of the ankle. In addition, proprioceptive training needs to be continued to maintain stability [42].

Anterior ankle impingement can be addressed by applying a heel wedge in order to open the anterior ankle compartment and to reduce the pressure.

### Operative Treatment

When nonoperative treatment fails surgery may be warranted. McMurray was first to publish a series of successfully treated patients who suffered from ankle impingement [43]. In the beginnings of treatment open surgery has been applied and different authors reported good results after open resections. However, in the meantime arthroscopic treatment has become one of the standards in the treatment of either anterior or posterior ankle impingement.

Contraindications encompass local soft tissue infection and severe osteoarthritis. Diabetic vascular disease and edema are seen as relative contraindications for surgery [44].

### Anterior Impingement

Anterior osteophytes can best be removed using a small osteotome during open surgery or by means of a shaver

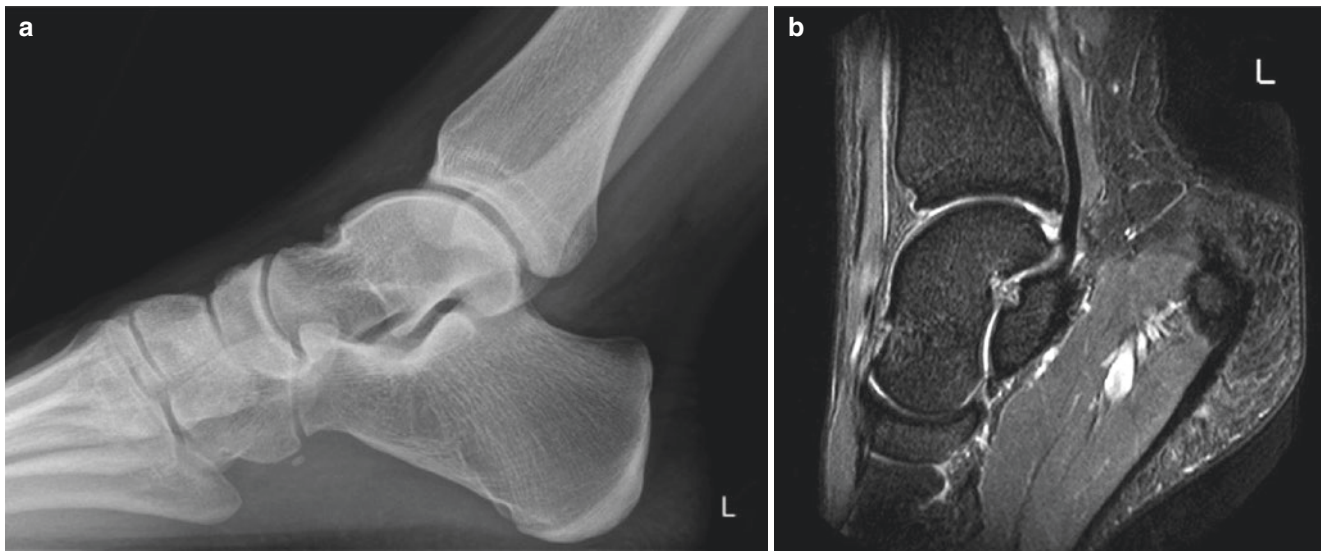
when arthroscopically addressed. In case of open surgery the author approaches the joint through a anteromedial and/or anterolateral skin incision and prefers a slightly curved osteotome to remove the osteophyte. A gouge is a good tool to shape the talar neck in case of a flat talar neck or when removing a talar osteophyte. Postoperatively, the patients are mobilized under partial weight bearing (15–20 kg) for 5 days. During the first five postoperative days the patients are counseled to start with active and passive dorsal extension of the ankle joint. One week postoperatively patients start with full weight bearing while sports activities are avoided for further 4 weeks. The rate of complications after open resections of anterior ankle osteophytes ranges about 18 % [14]. Common complications include lesions and irritations of the cutaneous nerves, injuries of the extensor tendons, hypertrophic scars and infections. In a study by Coull et al. 79 % of patients were able to return to their sports and 92 % of patients were satisfied with their clinical outcome [45].

In contrast to open surgery, arthroscopic treatment has become an important treatment option [18, 35, 46–53]. Scranton et al. thought that the size of osteophytes determines the overall outcome after treatment [54]. However, Tol and coworkers published their long-term results after arthroscopic resection of anterior ankle osteophytes. They found an association between the amount of joint space narrowing and overall results. While 83 % of patients without joint space narrowing showed a good-to-excellent result only 53 % of patients with joint space narrowing had a good-to-excellent result [55]. The authors were able to show that the presence of degenerative alterations of the ankle joint had a better prognostic value than the size of osteophytes. Therefore, the assumption of Scranton and Mc Dermott was refuted [54]. More recently, Walsh et al demonstrated significant functional improvement following arthroscopic treatment of anterior ankle impingement 5 years postoperatively [56].

A comparative study showed that patients who underwent arthroscopic surgery needed only half the time to return to their previous sports level than those who underwent open surgery [54].

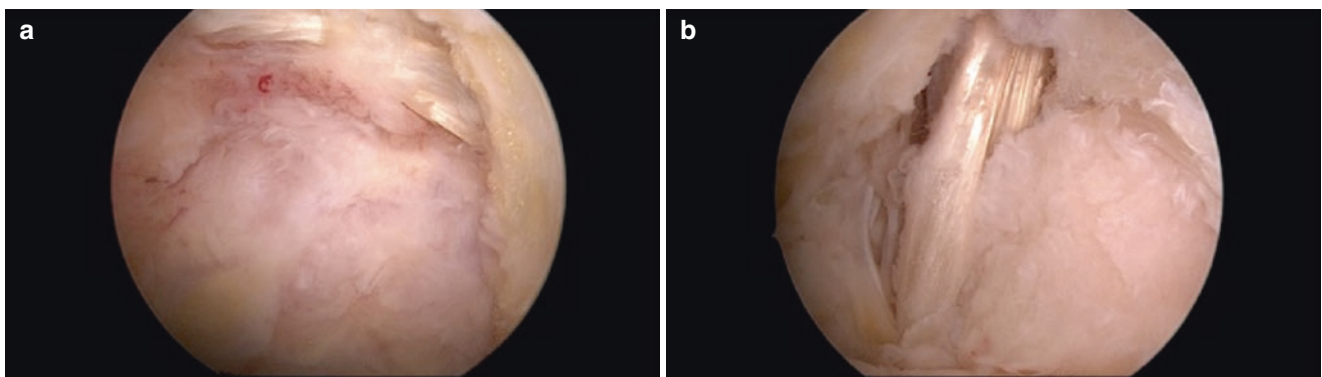
### Posterior Impingement

An open approach allows removal of both a prominent posterior process of the talus and the os trigonum [23, 28, 35, 36]. At the same time the inspection and subsequent debridement of the posterior joint capsule and the treatment



**Fig. 32.3** (a) Lateral ankle view of a 16-years old male patient who suffered from posterior ankle pain during maximum of plantarflexion. The pain was localized posteromedially. (b) The MRI of the same

patient as described in (a) shows the kinking of the flexor hallucis longus tendon in maximum plantarflexion



**Fig. 32.4** (a) Intraoperative view of a completely scarred flexor hallucis longus tendon. (b) Intraoperative view of the flexor hallucis longus tendon after debridement

of a tendinopathy of the flexor hallucis longus can be performed.

The rate of complications after open surgery in the treatment of posterior ankle impingement ranges from 16% up to 24% and patient recovery back to sports takes up to 5 months [28, 30]. With introduction of the two-portal technique described by van Dijk et al. this rate could be dramatically reduced. Hindfoot endoscopy allows the direct approach to the posterior structures and treatment (Figs. 32.3 and 32.4). Depending on the studies published in the literature, arthroscopic treatment reduces the complication rate to 2–11% and shortens the time back to sports to 5 weeks [7, 37]. Complications after arthroscopic treatment include mainly lesions of the sural nerve and wound infections (3%) [57].

## Evidence

Surgery of anterior ankle impingement reveals good-to-excellent results in 57–67% of cases. However, most of those studies are of retrospective nature.

One of the most reliable studies with regard to anterior ankle impingement has been published by van Dijk and coworkers. This prospective study has first been published in 1997. The same patient population has been followed-up a few years later and the results published in 2001 (see Table 32.1) [48, 55].

Surgery of posterior ankle impingement shows limited evidence. Hamilton published a very useful but retrospective study [28]. A prospective study has been published by van Dijk and coworkers (see Table 32.1) [58].

**Table 32.1** Shows the most important scientific articles published in the literature and their evidence level

Authors	Journal, year	Topic	Study design	Kohort	Level of evidence
van Dijk et al.	Am J Sports Med, 1997	Anterior ankle impingement	Prospective and comparative	62 patients	Level II
Walsh et al.	Am J Sports Med, 2014	Anterior ankle impingement	Prospective, case series	46 patients	Level IV
Tol et al.	J Bone Joint Br, 2001	Anterior ankle impingement	Prospective and comparative	62 patients	Level II
Hamilton et al.	J Bone Joint Am, 1996	Posterior ankle impingement	Retrospective cohort study	37 dancer 41 operations	Level III

## Summary

1. Anterior and posterior impingement syndromes are frequent found pathologies in young athletes.
2. Clinical assessment is mandatory but often needs additional imaging work up in order to confirm the diagnosis.
3. Nonoperative measures can be considered as first line treatment strategies to address impingement syndromes.
4. When nonoperative treatment fails or in case of severe presentations of impingement syndromes that may preclude conservative measures surgical treatment strategies are warranted.
5. Arthroscopic treatment has shown to be a very effective approach to address impingement syndromes and accelerates time back to sports while reducing the rate of complications.

## References

1. Baetzner W. Sportschäden am Bewegungsapparat. Wien: Urban & Schwarzenberg; 1927.
2. Maffulli N, et al. Sport injuries: a review of outcomes. *Br Med Bull*. 2011;97:47–80.
3. Biedert R. Anterior ankle pain in sports medicine: aetiology and indications for arthroscopy. *Arch Orthop Trauma Surg*. 1991;110(6):293–7.
4. O'Donoghue D. Impingement exostoses of the talus and tibia. *J Bone Joint Surg Am*. 1957;39:835–52.
5. Tol JL, van Dijk CN. Etiology of the anterior ankle impingement syndrome: a descriptive anatomical study. *Foot Ankle Int*. 2004;25(6):382–6.
6. Williams J, Bandt K. Exercise increases osteophyte formation and diminishes fibrillation following chemically induced articular cartilage injury. *J Anat*. 1984;139(4):599–611.
7. Ferkel RD, et al. Arthroscopic treatment of anterolateral impingement of the ankle. *Am J Sports Med*. 1991;19(5):440–6.
8. Russo A, et al. Ankle impingement: a review of multimodality imaging approach. *Musculoskelet Surg*. 2013;97 Suppl 2:S161–8.
9. Kleiger B. Anterior tibiotalar impingement syndromes in dancers. *Foot Ankle*. 1982;3(2):69–73.
10. Kleiger B. Injuries of the talus and its joints. *Clin Orthop Relat Res*. 1976;121:243–62.
11. Niek van Dijk C. Anterior and posterior ankle impingement. *Foot Ankle Clin*. 2006;11(3):663–83.
12. Robinson P. Impingement syndromes of the ankle. *Eur Radiol*. 2007;17(12):3056–65.
13. Watson AD. Ankle instability and impingement. *Foot Ankle Clin*. 2007;12(1):177–95.
14. Ogilvie-Harris DJ, Mahomed N, Demaziere A. Anterior impingement of the ankle treated by arthroscopic removal of bony spurs. *J Bone Joint Surg Br*. 1993;75(3):437–40.
15. van Dijk CN, et al. Oblique radiograph for the detection of bone spurs in anterior ankle impingement. *Skeletal Radiol*. 2002;31(4):214–21.
16. Vertullo C. Unresolved lateral ankle pain. It's not always 'just a sprain'. *Aust Fam Physician*. 2002;31(3):247–53.
17. Berberian WS, et al. Morphology of tibiotalar osteophytes in anterior ankle impingement. *Foot Ankle Int*. 2001;22(4):313–7.
18. Takao M, et al. Arthroscopic treatment for anterior impingement exostosis of the ankle: application of three-dimensional computed tomography. *Foot Ankle Int*. 2004;25(2):59–62.
19. Huh YM, et al. Synovitis and soft tissue impingement of the ankle: assessment with enhanced three-dimensional FSPGR MR imaging. *J Magn Reson Imaging*. 2004;19(1):108–16.
20. Kirby AB, et al. Magnetic resonance imaging findings of chronic lateral ankle instability. *Curr Probl Diagn Radiol*. 2005;34(5):196–203.
21. Larciprete M, et al. [Ankle impingement syndrome]. *Radiol Med (Torino)*. 2000;99(6):415–9.
22. Masciocchi C, Catalucci A, Barile A. Ankle impingement syndromes. *Eur J Radiol*. 1998;27 Suppl 1:S70–3.
23. Masciocchi C, Maffey MV, Matri F. Overload syndromes of the peritalar region. *Eur J Radiol*. 1997;26(1):46–53.
24. Robinson P, et al. Anteromedial impingement of the ankle: using MR arthrography to assess the anteromedial recess. *AJR Am J Roentgenol*. 2002;178(3):601–4.
25. Robinson P, et al. Anterolateral ankle impingement: MR arthrographic assessment of the anterolateral recess. *Radiology*. 2001;221(1):186–90.
26. Schweitzer ME. Magnetic resonance imaging of the foot and ankle. *Magn Reson Q*. 1993;9(4):214–34.
27. Brown TD, Micheli LJ. Foot and ankle injuries in dance. *Am J Orthop*. 2004;33(6):303–9.
28. Hamilton WG, Geppert MJ, Thompson FM. Pain in the posterior aspect of the ankle in dancers. Differential diagnosis and operative treatment. *J Bone Joint Surg Am*. 1996;78(10):1491–500.
29. Sohl P, Bowling A. Injuries to dancers. Prevalence, treatment and prevention. *Sports Med*. 1990;9(5):317–22.
30. Abramowitz Y, et al. Outcome of resection of a symptomatic os trigonum. *J Bone Joint Surg Am*. 2003;85-A(6):1051–7.
31. Berkowitz MJ, Kim DH. Process and tubercle fractures of the hind-foot. *J Am Acad Orthop Surg*. 2005;13(8):492–502.
32. Billi A, et al. Joint impingement syndrome: clinical features. *Eur J Radiol*. 1998;27 Suppl 1:S39–41.
33. Henderson I, La Valette D. Ankle impingement: combined anterior and posterior impingement syndrome of the ankle. *Foot Ankle Int*. 2004;25(9):632–8.

34. Nasser S, Manoli 2nd A. Fracture of the entire posterior process of the talus: a case report. *Foot Ankle*. 1990;10(4):235–8.
35. Yilmaz C, Eskandari MM. Arthroscopic excision of the talar Stieda's process. *Arthroscopy*. 2006;22(2):225 e1–3.
36. Smyth NA, et al. Posterior hindfoot arthroscopy: a review. *Am J Sports Med*. 2014;42(1):225–34.
37. Zwiers R, et al. Surgical treatment for posterior ankle impingement. *Arthroscopy*. 2013;29(7):1263–70.
38. de Leeuw PA, van Sterkenburg MN, van Dijk CN. Arthroscopy and endoscopy of the ankle and hindfoot. *Sports Med Arthrosc*. 2009;17(3):175–84.
39. Scholten PE, Sierevelt IN, van Dijk CN. Hindfoot endoscopy for posterior ankle impingement. *J Bone Joint Surg Am*. 2008;90(12):2665–72.
40. Stoller SM, Hekmat F, Kleiger B. A comparative study of the frequency of anterior impingement exostoses of the ankle in dancers and nondancers. *Foot Ankle*. 1984;4(4):201–3.
41. Subotnick SI. Anterior impingement exostosis of the ankle: a hazard to the athlete. *J Am Podiatry Assoc*. 1976;66(12):958–63.
42. Rodriguez-Merchan EC. Chronic ankle instability: diagnosis and treatment. *Arch Orthop Trauma Surg*. 2012;132(2):211–9.
43. Mc Murray T. Footballer's ankle. *J Bone Joint Surg Am*. 1950;32:68–9.
44. van Dijk CN, de Leeuw PA, Scholten PE. Hindfoot endoscopy for posterior ankle impingement. *J Bone Joint Surg Am*. 2009;91 Suppl 2:287–98.
45. Coull R, et al. Open treatment of anterior impingement of the ankle. *J Bone Joint Surg Br*. 2003;85(4):550–3.
46. Reynaert P, Gelen G, Geens G. Arthroscopic treatment of anterior impingement of the ankle. *Acta Orthop Belg*. 1994;60(4):384–8.
47. DeBerardino TM, Arciero RA, Taylor DC. Arthroscopic treatment of soft-tissue impingement of the ankle in athletes. *Arthroscopy*. 1997;13(4):492–8.
48. van Dijk CN, Tol JL, Verheyen CC. A prospective study of prognostic factors concerning the outcome of arthroscopic surgery for anterior ankle impingement. *Am J Sports Med*. 1997;25(6):737–45.
49. Kim SH, Ha KI. Arthroscopic treatment for impingement of the anterolateral soft tissues of the ankle. *J Bone Joint Surg Br*. 2000;82(7):1019–21.
50. Philbin TM, Lee TH, Berlet GC. Arthroscopy for athletic foot and ankle injuries. *Clin Sports Med*. 2004;23(1):35–53, vi.
51. Gulish HA, Sullivan RJ, Aronow M. Arthroscopic treatment of soft-tissue impingement lesions of the ankle in adolescents. *Foot Ankle Int*. 2005;26(3):204–7.
52. Nihal A, Rose DJ, Trepman E. Arthroscopic treatment of anterior ankle impingement syndrome in dancers. *Foot Ankle Int*. 2005;26(11):908–12.
53. Urguden M, et al. Arthroscopic treatment of anterolateral soft tissue impingement of the ankle: evaluation of factors affecting outcome. *Arthroscopy*. 2005;21(3):317–22.
54. Scranton Jr PE, McDermott JE. Anterior tibiotalar spurs: a comparison of open versus arthroscopic debridement. *Foot Ankle*. 1992;13(3):125–9.
55. Tol JL, Verheyen CC, van Dijk CN. Arthroscopic treatment of anterior impingement in the ankle. *J Bone Joint Surg Br*. 2001;83:9–13.
56. Walsh SJ, et al. Arthroscopic treatment of anterior ankle impingement: a prospective study of 46 patients with 5-year follow-up. *Am J Sports Med*. 2014;42(11):2722–6.
57. Galla M, Lobenhoffer P. Technique and results of arthroscopic treatment of posterior ankle impingement. *Foot Ankle Surg*. 2011;17(2):79–84.
58. Beimers L, Frey C, van Dijk CN. Arthroscopy of the posterior subtalar joint. *Foot Ankle Clin*. 2006;11(2):369–90, vii.

Alexej Barg, Christian J. Gaffney, and Victor Valderrabano

**Abstract**

Ankle osteoarthritis is a growing problem in health care, with 1 % of the world's adult population being affected. Previous trauma is the most common origin of ankle osteoarthritis. In the current literature, there is no evidence whether sport activities accelerate or prevent the development of ankle degenerative changes. Different surgical approaches have been described for the treatment of ankle osteoarthritis based on osteoarthritis stage. The treatment options range from joint preserving procedures (e.g. open or arthroscopic debridement, joint distraction arthroplasty, and supramalleolar osteotomies) to joint sacrificing procedures (e.g. total ankle replacement and ankle arthrodesis). While numerous studies have addressed clinical and radiographic outcomes in patients who underwent surgery for ankle osteoarthritis, there is scant literature addressing the role of sports participation of patients with ankle osteoarthritis before and after surgical treatment. In general, low impact sports can be recommended postoperatively. However, further prospective clinical studies are needed to identify whether sport activities can be identified as risk factors for treatment failure or for poorer postoperative results.

**Keywords**

Ankle • Ankle osteoarthritis • Sport • Sport activities • Ankle osteoarthritis etiology • Joint-preserving procedure • Joint sacrificing procedures • Supramalleolar osteotomy • Total ankle replacement • Ankle arthrodesis

**Etiology of Ankle Osteoarthritis**

Ankle osteoarthritis (OA) is a constantly growing problem in world health care and should not be underestimated [1]. More than 1 % of the entire world's adult population is affected by ankle OA [2]. The mental and physical disability

in patients suffering from end-stage ankle OA is at least as severe as that associated with end-stage hip OA [2].

In the current literature there are clinical and epidemiologic studies which address the etiology of ankle OA [1, 3]. Valderrabano et al. [4] evaluated the distribution of etiologies in 390 consecutive patients (406 ankles) with end-stage ankle OA. Posttraumatic ankle OA was observed in the majority (78 %) of patients. Malleolar fractures (type AO 44) were the most common injuries (39 %), followed by ankle ligament lesions (16 %), and tibial plafond fractures (type AO 43, 14 %). Secondary, atraumatic OA was observed in 13 % of patients including patients with rheumatoid arthritis, hemochromatosis, hemophilia, clubfoot etc. Primary OA was the rarest etiology, with 9 % of patients affected. Patients with posttraumatic ankle OA were significantly younger than patients with other ankle OA etiologies [4]. Similar findings

A. Barg, MD (✉) • C.J. Gaffney, MD, MSc  
Department of Orthopaedics, University of Utah,  
590 Wakara Way, Salt Lake City, UT 84108, USA  
e-mail: alexej.barg@hsc.utah.edu; christian.gaffney@hsc.utah.edu

V. Valderrabano, MD, PhD  
Orthopaedic Department, SWISS ORTHO CENTER,  
Schmerzklinik Basel, Swiss Medical Network, Basel, Switzerland  
e-mail: vvalderrabano@gsmn.ch

were found in another epidemiologic study by Saltzman et al. [5]. In this study, 639 consecutive patients with Kellgren grade 3 or 4 ankle OA who presented to the University of Iowa Orthopaedic Foot and Ankle Surgery service between 1991 and 2004 were included. The majority of patients (70%) had posttraumatic ankle OA, with rotational ankle fractures and recurrent instability being the most common causes, at 37% and 15%, respectively. Primary and secondary OA was diagnosed in 22% and 8%, respectively [5]. Wang et al. [6] analyzed radiographic hindfoot alignment in 226 consecutive patients with end-stage ankle OA who underwent total ankle replacement (TAR) or ankle arthrodesis. The most common etiology in this cohort was posttraumatic, with 71.2% of patients reporting previous fractures or recurrent ligament injuries. Primary ankle OA was diagnosed in 5.6% of patients. Secondary ankle OA was seen in 23.2%, including OA due to pes planovalgus deformity, rheumatoid arthritis, clubfoot or other congenital foot deformity, postinfectious arthritis, Charcot-Marie-Tooth disease, and haemophilic arthritis [6].

### Sport as Risk Factor for the Development of Ankle Osteoarthritis

In the current literature, there is a controversial discussion whether sport activities play a positive or negative role in the development of ankle OA. Based on the available literature, a causal link between pediatric sports injuries and ankle OA development is possible [7].

In the 1970s, Vincelette et al. [8] compared degenerative changes of the foot and ankle between professional soccer players and controls. Fifty-nine professional football players, with an average age of 23 years (19–27 years), with an average football experience of 9.5 years (2–15 years) were included. The group evaluated 367 radiographs of soccer players and 377 radiographs of control patients. Radiographs were classified as abnormal if they showed signs of OA, periarticular ossifications, dorsal exostosis of the talus, or distal interosseous ligament calcification. Among soccer players, mild findings were found in 10% and severe findings were found in 90% of radiographs. In the control group, 22% had mild findings and 4% had severe findings regarding radiographic degenerative changes [8].

In 1979, Adams [9] examined 56 soccer players and six coaches (who were former professional players) clinically and radiographically at one football club. All 62 patients showed some signs of ankle pathology. However, the overall incidence of radiographic OA was only 1.6% [9].

Hellmann et al. [10] performed radiographic analysis of six amateur athletes who had severe atypical degenerative joint disease. One of the patients presented with right ankle pain. The 20-year-old male participated in cross-country

motorcycling for several years. Radiographs and computed tomography showed degenerative changes of the subtalar joint in this patient, most likely resulting from extensive sport activities [10].

Kujala et al. [11] investigated the cumulative 21 year incidence of admission to a hospital for OA of the hip, knee, and ankle in former elite athletes and control subjects. In total, 2,049 male athletes who had represented Finland in international events from 1920 to 1965 and 1403 controls were included into this national population based study. The cumulative incidence of admission to a hospital among the former athletes was 3.3% (95% CI 2.6–4.1%), 2.4% (95% CI 1.8–3.2%), and 0.4% (95% CI 0.2–0.8%) for hip, knee, and ankle, respectively. These values were substantially higher than values observed in controls: 1.4% (0.9–2.2%), 1.3% (95% CI 0.8–2.0%), and 0% for hip, knee, and ankle, respectively [11].

The potential influence of long-term, high-intensity physical training on early ankle OA has been investigated in a retrospective study by Knobloch et al. [12]. Twenty-seven track and field long-distance runners and orienteers and nine bobsledders with a mean age of 42 years were compared with a control group of 23 healthy men with a mean age of 35 years. The long-distance runners had a higher prevalence of early ankle OA, however, the multivariate analysis demonstrated that not the sport itself, but rather age and ankle instability were significantly correlated with the development of ankle OA [12].

In their comparative study, Gross and Marti [13] addressed the influence of long-term, professional volleyball playing on the development of early ankle OA. A group of 22 former elite volleyball players with a mean age of  $34 \pm 6$  years who had played for at least three years in the highest league in Switzerland was compared to 19 healthy controls with a mean age of  $35 \pm 6$  years. The majority of players (20 of 22) had lateral ligament lesions, and eight of these required surgery. Radiographic evidence of ankle OA was found in 19 of 22 volleyball players, but only in two of 19 controls. The multiple regression analysis revealed that positive anterior drawer test and subjective instability were statistically significant and independent risk factors for radiographic OA [13].

Zinder et al. [14] evaluated the prevalence of ankle OA following ankle sprains in a cohort of retired professional football players. In total, 2,552 retired professional football players completed a general health questionnaire. Of them, 448 (17.69%) reported at least one severe ankle sprain in the past. Forty patients developed significant ankle OA. Compared to players without a history of sprains, the injured cohort had a 2.3 times higher prevalence of ankle OA [14].

Kuijt et al. [15] performed a systematic review of the recent literature to investigate the prevalence of knee and/or ankle OA in former elite soccer players. Only four studies

were included in the final analysis. The prevalence of degenerative joint disease was found to be between 40 and 80% for the knee and between 12 and 17% for the ankle. The prevalence of knee and ankle OA in the former players was substantially higher compared to the general population. However, only the prevalence of OA was measured in this study. Therefore, the authors mentioned the need of further studies based on a health surveillance program to be able to identify players at risk for OA [15].

Recently, Gouttebauge et al. [16] published a systematic review to explore the OA prevalence in former elite athletes from team and individual sports. In total, 15 studies were included in the review. Prevalence of ankle OA was reported only in one study [17]. In this study, by Schmitt et al. [17], which included 40 former elite high jumpers, ankle OA was found in 2.5%, with a mean age of 41.8 years at time of diagnosis.

In 2015, Iosifidis et al. [18] investigated the prevalence of lower extremity clinical and radiographic OA in a comparative study. The study compared 218 former elite male athletes from various sports (soccer, volleyball, martial arts, track and field, basketball, and skiing) with 181 controls. The prevalence of clinical OA was similar in both groups, with 15.6% prevalence for former elite athletes and 14.4% for controls. However, the prevalence of radiographic OA was significantly higher in the former athletes (36.6%) compared to the controls. Therefore, the radiographic signs of OA may precede the clinical onset of OA. In both groups, several risk factors for OA development were identified: age, body mass index, and occupation [18].

### **Ankle Instability as Risk Factor for the Development of Ankle Osteoarthritis**

Lateral ankle sprain (LAS) is one of the most common sports injuries in the world. LAS accounts for up to 30% of all athletic injuries and up to 60% of injuries in certain sports [19, 20]. It is one of the most common injuries in basketball [21–28], soccer [29–41], football [42–47], and tennis [48–51]. One LAS occurs per 10,000 person-days, and an estimated 2 million acute LAS injuries occur each year in the United States [52]. LAS injuries occur most commonly between 15 and 19 years of age [52], and there is no statistically significant difference in LAS incidence between males and females [52–55]. Ankle injuries, including LAS, are among the most common injuries in children and youth sports, accounting for up to one fourth of all injuries sustained by high-school athletes during the 2005–2006 school year [7, 56]. Swenson et al. [57] analyzed the sports injury data for the 2005 through 2008 academic years from a nationally representative sample of 100 United States high schools. The injuries most often involved the ankle (19%),

knee (14.5%) and head/face (10.7%). Notably, the ankle was the most frequently diagnosed site of recurrent injuries at 28.3% [57].

Up to 40% of all patients with LAS develop chronic ankle instability (CAI) [58–60]. Different intrinsic and extrinsic factors have been identified as risk factors for development of CAI after LAS: high body mass index, high height, and severity of initial ankle sprain [61, 62]. As mentioned above, CAI from repetitive ankle sprains is an important etiology of ankle OA – up to 20% of all ankle OA cases and up to 30% of all patients with posttraumatic ankle OA [4, 5, 63]. Different arthroscopic studies demonstrated a high number of chondral injuries in patients with a prolonged history of ankle instability [64–67].

Several studies have investigated the pathomechanism of ankle instability leading to ankle OA [68–71]. McKinley et al. [71] used an ankle cadaveric model to measure incongruity and instability-associated changes in contact stress directional gradients in the tibiotalar joint. An increase of up to 100% was observed in unstable specimens [71].

Caputo et al. [69] used magnetic resonance imaging (MRI) and orthogonal fluoroscopy to perform kinematic measurements in nine patients with lateral ankle instability and, in particular, insufficient anterior talofibular ligaments. Several significant changes were observed as compared with the intact contralateral ankles: increase in anterior translation of  $0.9 \pm 0.5$  mm, increase in internal rotation of  $5.7^\circ \pm 3.6^\circ$ , and increase in superior translation of  $0.2 \pm 0.2$  mm. These findings [69] may explain the degenerative changes often observed on the medial talus in patients with lateral CAI [65, 72, 73].

Bischof et al. [68] used three-dimensional MRI models and biplanar fluoroscopy to evaluate in vivo cartilage contact strains in patients with isolated lateral ankle instability. In total, seven patients were included in this study. The contralateral healthy side was used as a control to measure the magnitude and location of peak cartilage strain. The unstable ankles demonstrated significantly increased peak strain with  $29 \pm 8\%$  vs.  $21 \pm 5\%$ . Furthermore, the location of peak strain in the unstable ankles was translated anteriorly by  $15.5 \pm 7.1$  mm and medially by  $12.9 \pm 4.3$  mm. The authors found a correlation between the translation of peak strain and the location of clinically observed degenerative changes of the tibiotalar joint [68].

Recently, Golditz et al. [70] investigated the impact of functional ankle instability on the development of early cartilage damage using quantitative T2-mapping MRI. In total, 36 patients were included, and they were classified into one of three groups: functional ankle instability, ankle sprain “copers” (persons with initial sprains, but without residual instability), and controls (persons without a history of ankle injuries). The authors performed zonal region-of-interest T2-mapping of the deep and superficial layers of

the talar and tibial cartilage. Significant to highly significant differences in T2-values in 11 of 12 regions were observed, demonstrating that functional instability causes substantially unbalanced loading in the tibiotalar joint. This, in turn, may result in early degenerative changes of the joint cartilage [70].

Valderrabano et al. [63] investigated the data from 30 patients (33 ankles) with ligamentous end-stage ankle OA. There were 23 male and seven female patients with a mean age of 58.6 years (33–78 years). Ligamentous lesions suffered during sports activities were the most frequent cause for ankle OA (55%), followed by ankle sprains that occurred during normal daily activities (36%), and sprains suffered at work (9%). In 33% of patients, the ankle injury occurred while playing soccer. In 28 ankles, the initial injury was a lateral ligament injury, in four ankles the medial ligaments were initially injured, and in one ankle a combined medial-lateral ligament injury was diagnosed. The overall mean latency time between the initial injury and development of ligamentous posttraumatic ankle OA was 34.3 years, with a range between 6 and 57 years. The survivorship rate for single ankle sprains was statistically worse than that for chronic recurrent sprains, with the average latency time of 25.7 and 38.0 years, respectively. Also, the survivorship rate for medial sprains was statistically worse than for lateral sprains with 27.5 and 35.0 years until the development of OA, respectively [63].

### Treatment Options for Ankle Osteoarthritis

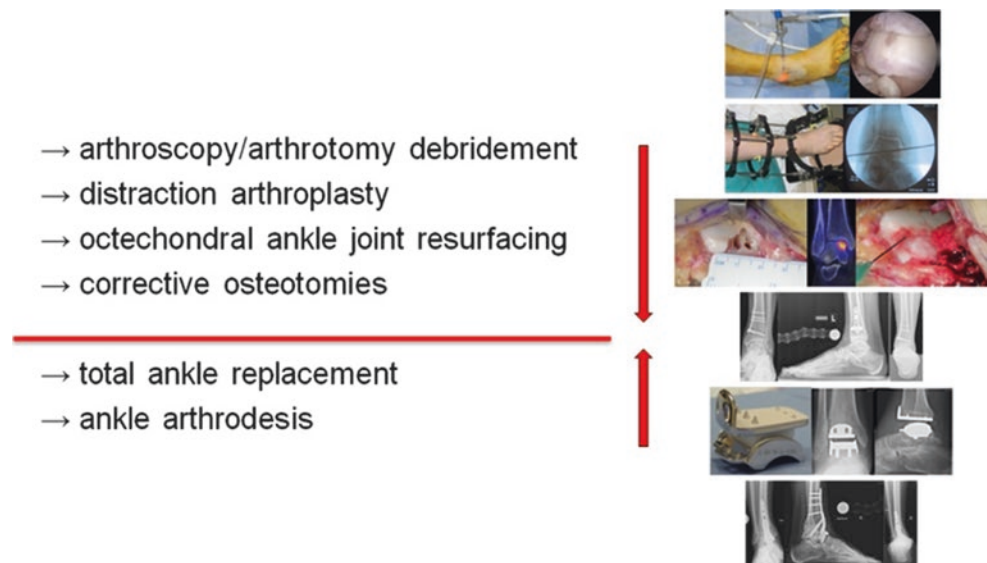
In the current literature, numerous treatment options have been described for ankle OA. In general, surgical treatments can be divided into joint-preserving and joint-sacrificing

procedures (Fig. 33.1). The surgical indications and the choice of treatment for patients with various stages of ankle OA is complex and requires adequate experience in foot and ankle surgery.

### Corrective Osteotomies

As mentioned above, the most common etiology for ankle OA is posttraumatic. Therefore, degenerative changes often develop asymmetrically with a concomitant varus or valgus deformity of the hindfoot [1, 6, 74, 75]. In patients with asymmetric ankle OA, a part of the tibiotalar joint remains preserved and free of degenerative changes. Thus, joint-sacrificing procedures like TAR or ankle arthrodesis may not be the most appropriate treatment options in this patient cohort [76]. In recent decades, realignment surgeries including supramalleolar osteotomies have evolved into valuable treatment options in patients with asymmetric ankle OA [77, 78]. The main indication for supramalleolar osteotomies is asymmetric ankle OA, with concomitant valgus or varus deformity, and a partially (at least 50%) preserved tibiotalar joint surface [77–81] (Fig. 33.2). Recent literature demonstrates that the short- and mid-term results following realignment surgery are promising, with substantial pain relief and functional improvement observed postoperatively in the majority of patients [77, 78]. However, there is limited literature addressing the effect of corrective osteotomies on patients' ability to participate in sports activities.

Takakura et al. [82] performed valgus opening wedge supramalleolar osteotomies in nine patients with a mean age of 35 years (12–61 years) with posttraumatic varus deformity of the ankle. Osseous union occurred within two months in eight patients and after six months in one patient. At the



**Fig. 33.1** Stage-adapted treatment options of ankle osteoarthritis including joint-preserving procedures and joint-sacrificing procedures



mean follow-up of 7.3 years, the range of motion of the ankle decreased in six patients and remained the same in three patients. However, none of the patients reported any limitation in daily activities. Moreover, four adolescent patients were able to participate in sports activities at school [82].

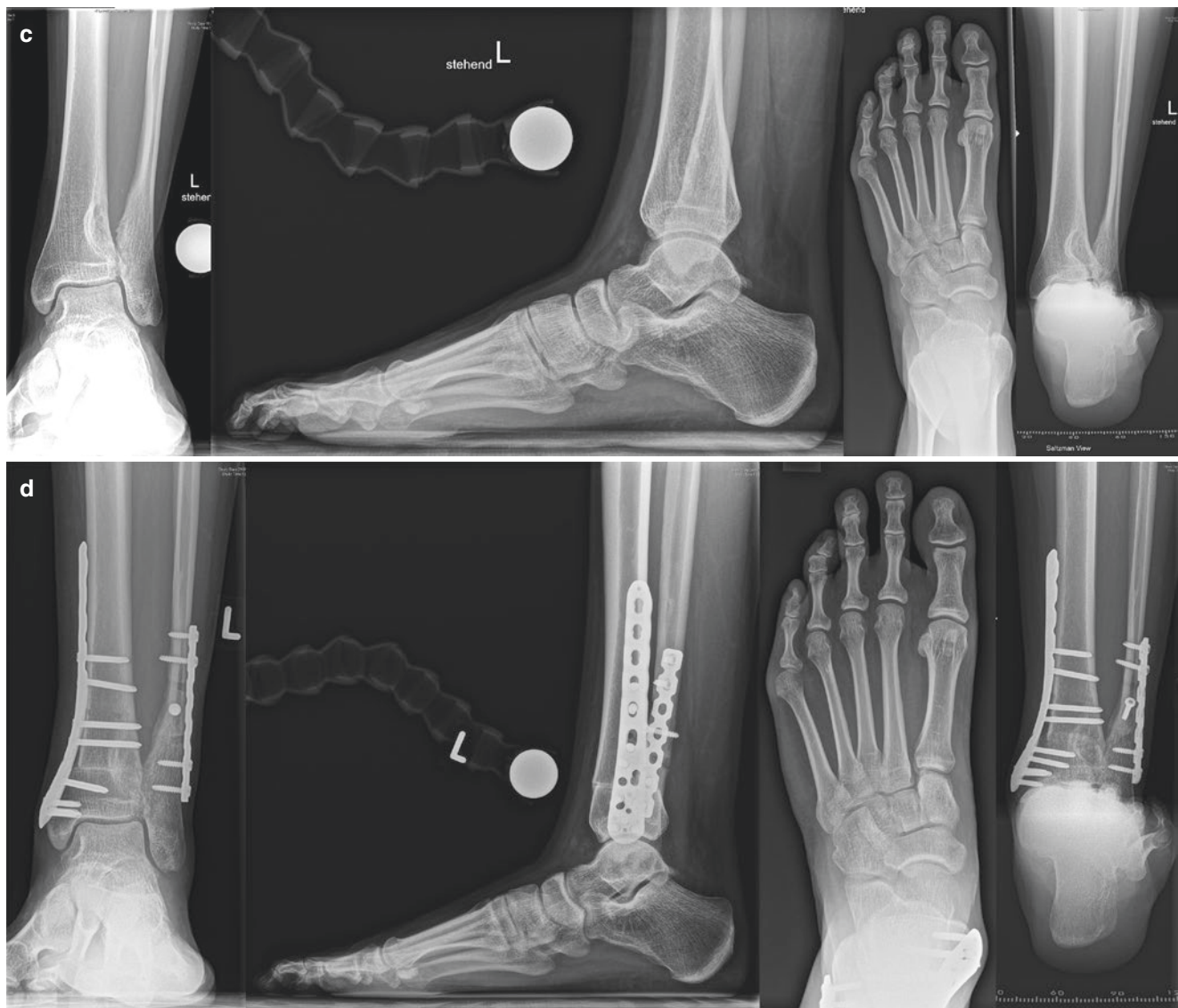
Harstall et al. [83] performed supramalleolar lateral closing wedge osteotomies in nine patients between the ages of 21–59 years, with varus ankle OA. The etiology of disease was

posttraumatic in eight and childhood osteomyelitis in one. At the mean follow-up of 4.7 years, significant pain relief (American Orthopaedic Foot and Ankle Society (AOFAS) pain subscore change from  $16 \pm 8.8$  preoperatively to  $30 \pm 7.1$  postoperatively) and significant functional improvement (AOFAS hindfoot score change from  $48 \pm 16.0$  preoperatively to  $74 \pm 11.7$  postoperatively) was observed. However, only three patients were able to perform sports activities as desired [83].



**Fig. 33.2** Supramalleolar realignment surgery. (a, b) An osteochondroma was diagnosed and resected in a 15 year-old female patient by pediatric orthopaedic surgeons. (c) Three years later, the patient developed a painful supramalleolar valgus deformity with a medial distal tibial angle [180] of  $96^\circ$ , substantial shortening of the fibula according to Weber's criteria [221], and valgus position of the heel on the

Saltzman hindfoot alignment view [222]. The patient complained of 7/10 pain on a visual analog scale and restriction of sports and daily activities. (d) A supramalleolar closing-wedge tibial osteotomy and a corrective lengthening osteotomy of the fibula were performed. At 6-months follow-up, the patient reported substantial pain relief (1–2 on a visual analogue scale)



**Fig. 33.2** (continued)

Pagenstert et al. [84] analyzed a prospective case series of 35 consecutive patients with a mean age of 43 years (26–68 years) who underwent realignment surgery due to varus or valgus ankle OA. Significant pain relief (visual analog scale (VAS) change from  $7 \pm 1.6$  preoperatively to  $2.7 \pm 1.6$  postoperatively) and significant functional improvement (AOFAS hindfoot score change from  $38.5 \pm 17.2$  preoperatively to  $85.4 \pm 12.4$  postoperatively) were observed at the final follow-up of a mean of five years. The authors demonstrated that realignment surgery may increase sports activity (Sports Activity Level change from  $1.3 \pm 1.4$  preoperatively to  $1.8 \pm 1.2$  postoperatively,  $p=0.02$ , and Ankle Activity Scale change from  $25.7 \pm 15$  preoperatively to  $68.1 \pm 21$  postoperatively,  $p=0.0001$ ). The types of sports activities that patients performed after realignment surgery were mostly low impact activities, like hiking, biking,

swimming or golfing. However, jumping and running were reported as well. The rate of sports inactivity decreased from 43% before surgery to 20% at final follow-up. Actually, sports frequency in hours per week showed a weak and statistically insignificant correlation ( $r=0.34$ ,  $p=0.054$ ) with the symptom-related Ankle Activity Scale [84].

Hintermann et al. [85] performed a prospective study to analyze the outcome of 48 malunited pronation-external rotation ankle fractures treated by corrective supramalleolar osteotomy. At the mean follow-up of 7.1 years (2–15 years), patients reported significant pain relief (87.2% of all patients were pain free and 12.8% reported moderate pain with a mean VAS of 2.1 points) and significant functional improvement (AOFAS hindfoot score from 48 points [36–66] preoperatively to 86 points [64–100] postoperatively).

However, the preoperative and postoperative range of motion of the ankle was comparable at  $41.2^\circ$  ( $30^\circ$ – $50^\circ$ ) and  $40.1^\circ$  ( $30^\circ$ – $50^\circ$ ), respectively. In total, 43 patients (89.6%) returned to their former professional activity, and 34 (70.8%) returned to their former sport activities. However, 11 patients did not participate in sport prior to their initial injury [85].

Mann et al. [86] published a retrospective study describing the results of opening medial tibial wedge osteotomy (plafond-plasty) as a novel surgical option for treatment of intra-articular varus ankle OA. Nineteen consecutive patients were assessed clinically and radiographically at a mean follow-up of 4.9 years. In total, the varus ankle deformity significantly decreased from  $18^\circ$  preoperatively to  $10^\circ$  postoperatively. The AOFAS hindfoot score improved significantly from 46 points preoperatively to 78 points postoperatively. During the follow-up, four of 19 patients underwent revision surgery: two ankle arthrodeses at seven and 36 months after the index surgery, and two TARs at 30 and 48 months. At the latest follow-up, 12 patients reported participation in moderate sporting activities like biking, golfing, gym exercising, and skiing. Five of 19 patients, were active and were able to walk up to five miles daily. Two patients did not report any activities; both had procedure failure requiring ankle arthrodesis or TAR [86].

## Conclusion

In conclusion, promising short- and midterm results have been reported in patients who underwent supramalleolar realignment surgery. However, there is only one clinical study prospectively comparing the sport activity level in this patient group preoperatively and postoperatively [84]. It still remains unclear whether postoperative functional improvement positively correlates with sport activity level. Further clinical studies are needed to identify the positive and negative predictors for being active in sports and recreational activities.

## Total Ankle Replacement

While total hip replacement and total knee replacement have evolved to well-established treatment options in patients with end-stage degenerative changes, the indication for TAR remains controversial. TAR using current 3rd-generation prosthesis designs provides substantial postoperative pain relief and good functional outcome, including preserved range of motion [87–91]. However, the overall survivorship can be expected to be approximately 90% at 5 years after initial implantation [92]. The failure rate of TAR is still substantially higher than that of total hip or knee replacement. Labek et al. [93] performed a systematic review, including

national registries and clinical studies, demonstrating that the revision rate after TAR was 3.29 per 100 observed component years. This is significantly higher than after total knee replacement (1.26 revisions), medial unicompartmental replacement (1.53 revisions), or total hip replacement (1.29 revisions). The most common reasons for revision surgery after TAR were aseptic loosening (38%), persisting pain syndrome (12%), and septic loosening (9.8%) [94].

Based on our experience with TAR [87, 88, 95–127], the ideal candidate for TAR is middle-aged or older, is reasonably mobile, has no significant comorbidities, participates in low impact physical activities (e.g. hiking, swimming, biking, golfing), is not obese or overweight, has good bone stock, and has no concomitant instabilities/deformities [88, 105] (Fig. 33.3). In our clinic, we still consider the need for high impact physical activities (e.g. contact sports, jumping) as a contraindication for TAR [88, 105]. However, there is limited evidence whether (1) the patients who underwent TAR are able to return to sport activities postoperatively and (2) postoperative sport activities can be identified as risk factors for TAR failure.

The clinical study by Pipino and Calderale [128] is the first clinical study mentioning sport activities in patients who underwent TAR. In this study from the year 1983, the 1st generation PC ankle prosthesis was used in 15 patients. The authors stated that one of 15 patients had started practicing sports after TAR [128].

The study by Valderrabano et al. [126] was the first study specifically analyzing participation in sports after TAR. In total, 147 patients (152 TARs) with a mean age of 59.6 years (28–86 years) were included in this prospective study. At the mean follow-up of 2.8 years (2–4 years) a significant functional improvement was observed in AOFAS hindfoot score (from 36 preoperatively to 84 postoperatively) and range of motion (from  $21^\circ$  ( $0^\circ$ – $45^\circ$ ) preoperatively to  $35^\circ$  ( $10^\circ$ – $55^\circ$ ) postoperatively). A special sports frequency score was developed to assess sports activity (Table 33.1). TAR resulted in significant increase of sports participation (36% preoperatively to 56% postoperatively). Sports-active patients had a significantly higher AOFAS hindfoot score than patients who did not participate in sports. The most commonly reported sports activities were hiking, biking, swimming, aerobics, downhill skiing, and golfing [126].

Naal et al. [129] analyzed the preoperative and postoperative participation in sports and recreational activities of 101 patients with a mean age of 59.4 years (24–85 years), at a mean of 3.7 years after TAR. The preoperative and postoperative percentage of sports-active patients was 62.4% and 66.3%, respectively. The patients were active in  $3.0 \pm 1.8$  different sports and recreational activities preoperatively, and in  $3.0 \pm 1.6$  activities postoperatively. The sports frequency remained the same, with  $2.0 \pm 1.6$  sessions per week before



**Fig. 33.3** Total ankle replacement. (a) A 45-year old male patient with posttraumatic ligamentous ankle osteoarthritis. (b) At 1-year follow-up, the patient was pain free without any restrictions in sports or daily activities including cycling, golf, hiking, skiing, and tennis

**Table 33.1** Valderrabano's sports frequency score [126]

Score	Definition
0 (none)	no sports activity
1 (moderate)	moderate level of sports activity in leisure time, <1 h/week
2 (normal)	normal level of sports activity in leisure time, 1–5 h/week
3 (high)	high level of sports activity in leisure time, > 5 h/week
4 (elite)	professional level of sports activity, elite athlete

TAR and  $2.3 \pm 1.7$  sessions after TAR. However, 65% of patients stated that the surgery substantially improved their sports ability. The University of California at Los Angeles activity scale improved significantly from  $45.5 \pm 16.6$  to  $84.3 \pm 13.3$ . Neither sports participation nor activity levels

were identified as risk factors for development of periprosthetic radiolucencies [129].

Bonnin et al. [130] evaluated function and return to sports after TAR in 140 patients who underwent TAR with the Salto total ankle prosthesis (noncemented, mobile bearing prosthesis) due to OA (100 patients) or rheumatoid arthritis (40 patients). At the mean follow-up of 4.5 years, the Foot Function Index scores were  $13.7 \pm 17$  for “activity limitations”,  $31.7 \pm 23$  for “disability”, and  $16.9 \pm 19$  for “pain”. The Foot and Ankle Ability Measurement scores were  $74.9 \pm 18$  for activities of daily living and  $48.9 \pm 28$  for sports activities. In the OA subgroup, 38 bicycled, 21 performed recreational gymnastics, 58 swam, 50 gardened, 27 danced, and 43 hiked. Seven patients played tennis, nine cross-country ski, 17 downhill skied, and six regularly ran more than 500 m. Most often, patients limited their sports activity for reasons independent of

their ankle. Of the patients who had regular or intense sports activity, four mentioned substantial pain in their ankle during sports. In conclusion, the authors demonstrated that TAR may improve quality of life, and that return to recreational activities was generally possible. However, the return to high impact sport was rarely possible [130].

A prospective study has been performed by Barg et al. [99] to evaluate the mid-term outcome in eight haemophilia patients treated with TAR (10 TAR). At the mean follow-up of 5.6 years, significant pain relief and functional improvement was observed. Preoperatively, only one patient was able to participate in sport activities. However, at the latest follow-up, five patients had a normal level of sport activity and one patient had a moderate level [99]. Considering the unique hemophilic etiology of OA in this cohort, the postoperative increase in sport activities may have an especially positive effect, as it has been demonstrated that exercise may help reduce further joint destruction in haemophilia patients [131]. Another prospective study by Barg et al. [112] evaluated the feasibility of TAR in 18 patients with von Willebrand disease. Similar to patients with haemophilia, significant pain relief and functional improvement was observed at the mean follow-up of 7.5 years. Preoperatively, seven patients were moderately active in sports. At the latest follow-up, the postoperative sports activity level was moderate and normal in ten and seven patients, respectively [112]. The same group has evaluated the clinical and radiographic outcome in 16 patients with hereditary hemochromatosis who underwent TAR (21 procedures) [100]. At latest follow-up of 5.3 years in this prospective study, four of 19 patients had a normal level of sport activity and four of 19 patients had a moderate level. For comparison, preoperatively only two of 19 patients had a normal level of sport activity and two of 19 had a moderate level [100]. A similar increase in sport activity was observed in another clinical study addressing the outcome of TAR in 16 patients (19 ankles) with gouty arthritis [107]. Preoperatively, only one patient reported a moderate level of sport activity. Postoperatively, at a mean follow-up of 5.1 years, 13 patients reported participating in a moderate level of sport activity [107].

The outcome in 26 patients with simultaneous bilateral TAR has been analyzed by Barg et al. [102]. Significant pain relief and improvement in functional outcome and quality of life were observed at the mean follow-up of five years in this prospective study. Also, sport activity was assessed preoperatively and postoperatively using Valderrabano's score [126]. Preoperatively, five patients had a normal level of sport activity and three patients had a moderate level. At latest postoperative follow-up, eight patients had a normal level of sport activity, six patients had a moderate level, and one patient had a high level. One patient in the cohort had a reduced level of sport activity postoperatively (from normal to moderate) [102].

The component stability, weight change, and functional outcome was analyzed in 118 consecutive obese patients in the International Federation of Foot & Ankle Societies 2011 Award paper [104]. Preoperatively, 23 patients (19.5%) reported a moderate level, 15 patients (12.7%) a normal level, and one patient (0.8%) a high level of sport activity. At the mean follow-up of 5.6 years (2.4–10.5 years), 61 patients (22.9%) reported a moderate level, 29 patients (24.6%) a normal level, and one patient (0.8%) a high level of sport activity. A significant weight loss after one and two years of  $1.6 \pm 3.2$  kg, and  $2.1 \pm 3.6$  kg, respectively. Using a 5% weight loss criterion, 14 patients (11.9%) lost weight at one-year follow-up. Significant weight loss could be predicted by male gender, but not by age or, surprisingly, postoperative sport activity level [104].

Schuh et al. [132] compared the participation in sports and recreational activities in 21 patients with ankle arthrodesis and 20 patients with TAR. At the mean follow-up of 2.9 years of this prospective study, 86% and 76% of all patients were active in sports in the arthrodesis and TAR groups, respectively. Also, clinical and functional outcomes were comparable [132].

Criswell et al. [133] analyzed survival, overall reoperation rate, and functional outcome in 64 patients (65 ankles) who underwent TAR using the Agility prosthesis. Sixteen of the 41 patients (39%) needed revision of at least one prosthesis component. At the median follow-up of 8 years (0.5–11 years), the average Foot and Ankle Ability Measure subscale scores for sports and daily living activities were 33 and 57 points, respectively. Thirty-three patients reported moderate to extreme difficulty with running, jumping, low impact activities, and the ability to participate in sports [133].

Dalat et al. [134] retrospectively evaluated two continuous series of 59 ankle arthroplasties and 46 ankle arthrodeses performed between 1997 and 2009. The mean follow-up was 4.4 and 4.8 years in TAR and ankle arthrodesis groups, respectively. The mean overall sports level was relatively low in both groups. However, patients who underwent TAR had significantly higher Foot Ankle Ability Measure sports scores with  $49.5 \pm 24.4$  points vs.  $29.8 \pm 26.2$  points. The most common sports in both patient cohorts were non-contact sports including cycling, swimming, and hiking [134].

Nodzo et al. [135] performed a retrospective study including 74 consecutive patients (75 ankles), with a mean age of 60.6 years (41–82 years), who underwent TAR using the Salto prosthesis. At the mean follow-up of 3.6 years, average dorsiflexion and plantarflexion significantly improved from  $4.3^\circ \pm 3.3^\circ$  to  $8.7^\circ \pm 5.6^\circ$  and from  $24^\circ \pm 11^\circ$  to  $29^\circ \pm 7^\circ$ , respectively. The subscales of the validated Foot and Ankle Outcome Score for sport activity and activity of daily living also improved significantly from  $20 \pm 12$  to  $55 \pm 35$  and from  $50 \pm 16$  to  $83 \pm 22$ , respectively. Positive correlations between

high patient satisfaction and the pain, activities of daily living, and quality of life scores were identified [135].

Braitto et al. [136] analyzed clinical and radiographic outcomes in 84 patients who underwent HINTEGRA TAR at a mean follow-up of four years. The sports subscale of the Foot and Ankle Outcome Score improved significantly from  $17.2 \pm 15.4$  points preoperatively to  $45.8 \pm 24.2$  points postoperatively [136].

Recently, Kerkhoff et al. [137] analyzed short-term results in 67 patients who underwent primary TAR using the three-component Mobility prosthesis. The percentage of sports active patients was the same before and after surgery at 73%. Six patients stopped their sports activities after surgery and six patients resumed some sports activities following TAR. The most common sports following TAR were cycling, hiking, and swimming. Although 73% of all patients were sport active, 91% were unable to run a short distance [137].

## Conclusion

In the last 25 years, TAR has progressed remarkably as a treatment option in patients with end-stage ankle OA [87, 88]. A prospective controlled trial comparing TAR with ankle arthrodesis demonstrated the superiority of TAR in postoperative pain relief and functional outcome [138]. However, the overall survivorship of TAR is still lower than total hip or knee replacement [93], with aseptic loosening being the most common reason for TAR failure [94]. In the current literature, it remains controversial whether patients with TAR have improved ability to participate in sport activities [126, 129, 130]. Also, the question of whether the patients with TAR are more sports-active than demographically comparable patients who underwent ankle arthrodesis cannot be answered definitively by the current literature. There is only one comparative study addressing participation in sports and recreational activities in patients who underwent either ankle arthrodesis or TAR, and it demonstrated no significant differences between groups [132]. However, both groups were small, with only 41 patients in total, and the mean follow-up was relatively short at 2.9 years [132]. Thus, the results of this study must be interpreted with caution.

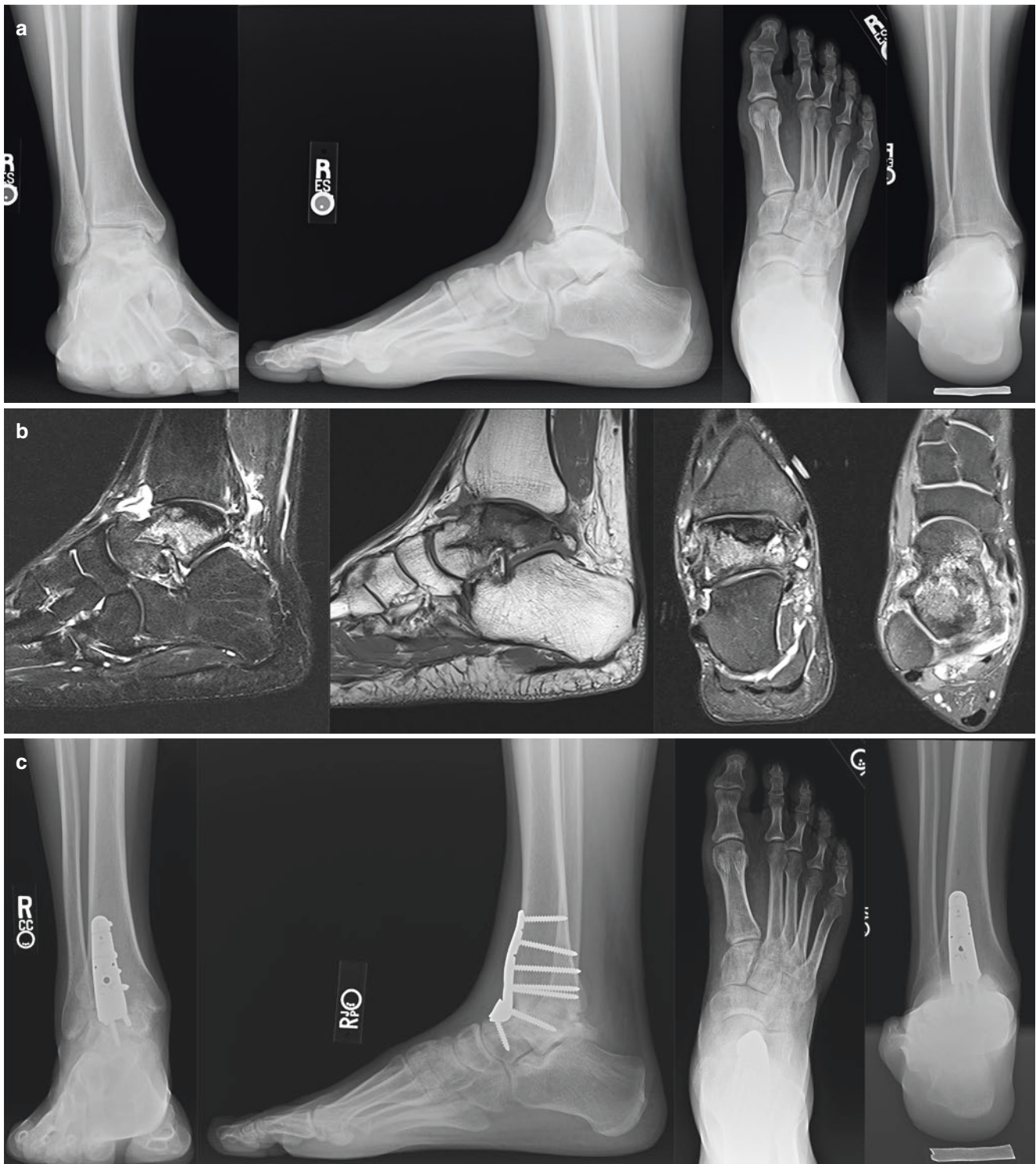
In the current literature, there are no evidence-based findings that high level sport activity is associated with increased failure rate of TAR. Therefore, such activity should not be considered a contraindication for TAR. However, we recommend against performing TAR in patients with high demands or unrealistic expectations for physical activities [88, 105]. This is the most common recommendation in the literature, as it is supported by numerous studies [139–151]. However, we do not support suggestions that patients with any sports activities should be excluded from TAR, as stated by Giannini et al. [152].

## Ankle Arthrodesis

For decades, ankle arthrodesis has been considered the “gold-standard” treatment in patients with end-stage ankle OA, independent of the underlying etiology [153, 154]. Ankle arthrodesis was first described in the nineteenth century [155]. Since then almost 50 different surgical techniques have been described in the literature with good mid-term results: substantial pain relief and acceptable functional outcome [154, 156, 157] (Fig. 33.4). However, many clinical reports have described short-term and long-term problems following ankle arthrodesis in association with daily activities, including climbing stairs, getting out of a chair, walking on uneven surfaces, and running [158–161]. Another major long-term complication following ankle arthrodesis is development of degenerative changes in the adjacent joints, as reported by several authors [157, 160, 162–166]. The development of subsequent OA in adjacent joints may lead to additional fusion surgeries [157, 167]. OA development in adjacent joints can be partially explained by alterations in gait resulting in compensatory motion and overload in the neighboring joints [168]. There are numerous clinical studies investigating gait analysis in patients who underwent ankle arthrodesis. Hahn et al. [169] performed a comparative gait analysis of ankle arthrodesis and TAR. Substantial pain relief and functional improvement was demonstrated one year after both procedures. However, TAR resulted in more natural ankle joint function with increased range of motion [169]. A similar comparative study has been performed by Piriou et al. [170]. Three groups of 12 patients were analyzed: patients with ankle arthrodesis, patients with TAR, and controls. Clinically important differences were demonstrated, but neither TAR nor fusion restored normal movement or walking speed. Patients with ankle arthrodesis showed a faster gait with a longer step length, while patients with TAR had greater ankle range of motion, symmetrical timing of gait, and restored ground reaction force pattern [170]. Recently, Jastifer et al. [171] prospectively compared the performance of TAR and ankle arthrodesis on uneven surfaces, stairs, and inclines. Both groups demonstrated improved performance at one-year follow-up compared to preoperatively. However, TAR patients had higher functional scores than the ankle arthrodesis patients [171].

Regarding the possible development of subsequent OA in adjacent joints and alterations in gait after fusion, it remains unclear whether the patients with ankle arthrodesis are able to regain the ability to participate in sport. Also, there is no data on what effect sport activities may have on long-term outcome.

Mazur et al. [160] analyzed functional outcome and gait in 13 patients with ankle arthrodesis. This was a long-term study with a mean follow-up of 8.3 years. All but one patient were sports active at the latest follow-up, reporting activities



**Fig. 33.4** Ankle arthrodesis. (a) A 29-year old male with ankle osteoarthritis and significant pain due to avascular necrosis of the talus. (b) Magnetic resonance imaging demonstrates avascular necrosis of the talus with more than 50% involvement. (c) At 1-year follow-up after

arthrodesis, the patient had no pain and was able to regain his participation in sport and recreational activities including bowling, cycling, diving, fishing, golf, hiking, ice hockey, and skiing

like hunting, hockey, baseball, golf, swimming, bowling, skiing, and jogging. Ten of 13 patients were able to return to the recreational sports activities they performed before they developed ankle symptoms [160].

Lynch et al. [159] reported their long-term results in 39 patients with a mean age of 50 years who underwent ankle arthrodesis. At the latest follow-up of a mean of seven years, none of the patients were involved in high impact sports, and

running activities were difficult for all but four younger patients who could run limited distances. Three of these four patients were able to participate in ice skating and ice hockey without any restrictions [159].

Thermann et al. [172] reported their results of 225 ankle arthrodeses performed between 1975 and 1995 using external fixation (44 patients) or screw fixation (225 patients). In total, 68% of all patients were able to participate in sports activities (swimming and biking). Younger patients were also active in playing badminton and soccer, climbing (at a low level), and bodybuilding [172].

Vertullo and Nunley [173] used questionnaires sent to members of the American Orthopaedic Foot and Ankle Society and to trainers of professional basketball and American football teams and asked respondents for guidelines for sports participation after an ankle arthrodesis. The most recommended sport activities were golf, skiing, and tennis with 94%, 77%, and 38% of respondents suggesting them. The authors stated that all high-impact sports should be avoided to minimize the development of adjacent joint OA and stress fractures [173].

Goebel et al. [174] performed a short-term prospective study including 29 patients who underwent tibiotalar calcaneal arthrodesis using a retrograde femur nail. At the mean follow-up of 2.1 years, complete osseous union was achieved in 90% of patients. At the latest follow-up one fourth of all patients were sports active, whereas none of the patients were able to participate in their chosen sports before surgery [174].

Akra et al. [175] reported their results of 25 patients (26 ankles) treated with ankle arthrodesis using a transfibular approach over a five-year period. In this patient cohort, all patients who were involved in recreational sporting activities were able to return to their usual sports. However, the authors did not mention the percentage of sports active patients in their study [175].

## Conclusion

Ankle arthrodesis can be used as a safe surgical procedure for numerous indications in patients with end-stage ankle OA [153, 154, 156]. On one hand, it may provide acceptable mid-term results [154]. But, on the other hand, it may be associated with long-term complications including development of painful OA in the adjacent joints [163]. Similar to TAR, there is limited literature addressing patients' participation in sports postoperatively. In general, younger patients with ankle arthrodesis are able to participate in sports. However, there are no studies revealing positive or negative effects of sport activities on long-term clinical outcome and OA status in the neighboring joints. Nevertheless, in the cur-

rent literature there is consensus that high impact sport activities should be avoided in patients who underwent ankle arthrodesis.

---

## Sports Activities in Patients Treated for Ankle Osteoarthritis

















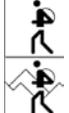



The most common etiology for end-stage ankle OA is post-traumatic [4, 5]. Therefore, it is not surprising that almost half of all patients with posttraumatic ankle OA present with a concomitant hindfoot deformity [6, 74, 75]. A direct correlation between the degree of hindfoot malalignment and degenerative process in the ankle joint has been described in the literature [176, 177]. Patients with substantial varus or valgus hindfoot deformity may develop asymmetric ankle OA [1]. Patients with posttraumatic asymmetric ankle OA are a challenging patient cohort, as they are often young and active. Furthermore, the complex underlying biomechanics of their concomitant deformities needs to be recognized and addressed [178]. The treatment of asymmetric ankle OA is also challenging because a substantial portion of the tibiotalar joint surface is usually preserved, and so joint-sacrificing procedures like TAR or ankle arthrodesis may not be the most appropriate treatment [77]. In the recently published review articles, realignment hindfoot surgeries have been shown to have promising short-term and mid-term results, with substantial pain relief and functional improvement [77, 78].

What can be said about sports participation in this specific patient group, who underwent hindfoot realignment, TAR, or ankle arthrodesis surgery? The literature addressing this specific topic is rare, indeed. As such, our recommendations are based on our experience with these surgical procedures over the last two decades [76–78, 80, 81, 84, 85, 178–193] (Table 33.2).

The next discussion point is represented by patients with complete end-stage ankle OA. Due to improved designs and overall survivorship of newer generation TAR systems, ankle arthrodesis should not be considered as the only “gold-standard” treatment option in patients with end-stage ankle OA. However, ankle arthrodesis still remains a proven procedure with good mid-term results, including substantial pain relief [153, 154, 156, 194–196] and acceptable functional outcome [166, 197]. Considering the survivorship of TAR, which is still lower than knee or hip replacement [93], young patients with end-stage ankle OA are better treated by ankle arthrodesis. In a previous study, we analyzed TAR survivorship in 684 patients and determined that age  $\leq 70$  years is a statistically significant and independent risk factor for TAR failure with an odds ratio of 3.85 (95% CI 1.47–10.00) [111]. Literature addressing outcomes following TAR in younger
























**Table 33.2** Our recommendations regarding sports activities (in alphabetic order) in patients who underwent realignment surgery, total ankle replacement, or ankle arthrodesis due to ankle osteoarthritis

Sports activities		Realignment surgery	Total ankle replacement	Ankle arthrodesis
	Aerobics	Recommended	Recommended	Recommended
	Athletic sports	Possible	Not recommended	Not recommended
	Badminton	Recommended	Possible	Possible
	Basketball	Possible	Not recommended	Not recommended
	Beach volleyball	Possible	Not recommended	Not recommended
	Bowling	Recommended	Recommended	Recommended
	Chess	Recommended	Recommended	Recommended
	Cross-country skiing	Recommended	Possible	Not recommended
	Cycling	Recommended	Recommended	Recommended
	Dancing	Recommended	Recommended	Recommended
	Diving	Possible	Possible	Possible
	Fishing	Recommended	Recommended	Recommended
	Golf	Recommended	Recommended	Recommended
	Gymnastics	Recommended	Possible	Possible
	Handball	Possible	Not recommended	Not recommended
	Hang gliding	Possible	Possible	Possible
	Hiking, hillwalking	Recommended	Recommended	Recommended
	Horseback riding	Possible	Not recommended	Possible
	Hurdle race	Not recommended	Not recommended	Not recommended
	Ice hockey	Possible	Not recommended	Possible

(continued)

**Table 33.2** (continued)

Sports activities	Realignment surgery	Total ankle replacement	Ankle arthrodesis
 Ice skating	Possible	Not recommended	Possible
 Jogging	Possible	Not recommended	Not recommended
 Martial arts	Not recommended	Not recommended	Not recommended
 Mountain biking	Possible	Not recommended	Possible
 Muscle exercises	Recommended	Recommended	Recommended
 Nordic walking	Recommended	Recommended	Recommended
 Oarsmanship	Recommended	Possible	Possible
 Paragliding	Possible	Not recommended	Possible
 Rugby, American football	Not recommended	Not recommended	Not recommended
 Sailing	Recommended	Possible	Possible
 Skiing	Recommend	Possible	Possible
 Sledding	Possible	Not recommended	Possible
 Soccer	Not recommended	Not recommended	Not recommended
 Swimming	Recommended	Recommended	Recommended
 Table tennis	Possible	Possible	Possible
 Tennis	Possible	Possible	Possible
 Volleyball	Possible	Not recommended	Not recommended
 Water aerobics	Recommended	Recommended	Recommended
 Water skiing	Possible	Not recommended	Possible
 Weightlifting	Not recommended	Not recommended	Not recommended
 Windsurfing	Possible	Not recommended	Possible

patients is rare. Kofoed and Lundberg-Jensen [145] compared survivorship and functional outcome of TAR between two groups consisting of 30 patients younger than 50 years and 70 patients aged 70 years or older. The postoperative results were comparable in both groups. However, the mean follow-up in both groups was six years with a wide range between one and 15 years [145]. The results of studies with short follow-up should be interpreted carefully. For example, several studies demonstrated encouraging results following TAR using the STAR prosthesis. These studies showed high prosthesis survivorship: 95% at six years [198], 93.9% at five years [199], and 96.0% at five years [200]. However, a long-term TAR study by Brunner et al. [113] with a mean follow-up of 12.4 years (10.8–14.9 years) revealed the probability of implant survival was 70.7% and 45.6% at 10 and 14 years, respectively. The recent systematic literature reviews by Gougoulias et al. [92] and Zaidi et al. [91] demonstrated a five-year survivorship of 90% and 10-year survivorship of 89%, respectively. Based on results reported in the current literature, a 30 year old patient treated by TAR will most likely have revision surgery due to failure. A salvage procedure of failed TAR – conversion to ankle arthrodesis [201–205] or revision TAR [115, 120, 206, 207] – is a technically demanding procedure and may have worse results than the comparable primary procedure.

Biomechanical cadaver studies demonstrated that TAR may change the natural ankle joint kinematics and biomechanics. However, these changes are less than those observed in fused ankles [208–210]. Still, as mentioned before, patients with high to elite sports activities or unrealistic expectations should not be considered for TAR. Why might excessive sport activities have a negative influence on TAR? First, excessive sport activities and consecutive overload may dramatically change the joint contact pressures. Espinosa et al. [211] analyzed the influence of misalignment of total ankle components on joint contact pressures using finite element models. Two prosthesis types were compared in this in-vitro study: the highly congruent mobile-bearing design of the Mobility TAR and the less congruent two-component Agility TAR. It has been demonstrated, that the congruent mobile-bearing design resulted in more evenly distributed and lower-magnitude joint contact pressure. However, both designs were highly vulnerable to increased contact pressure induced by malalignment of prosthesis components [211]. It can be speculated that excessive sport activities may substantially worsen this problem. Another problem which may occur in excessively sports-active patients may be related to increased polyethylene insert wear or failure. It has been demonstrated that TAR generates wear

particles similar to knee prostheses [212]. Increased polyethylene wear may cause periprosthetic osteolysis as a foreign-body reaction resulting in TAR failure, similar to failures observed in total hip replacement. Harris et al. [213] published a case report describing large wear debris cysts in a 65-year-old man who underwent TAR, and required revision surgery. Another complication is fracture of the mobile bearing, which may occur in up to 14% of patients who undergo TAR using a three-component prosthesis design [87, 113, 214]. Sport activities at high level may increase the rate of this type of complication, although this is only speculation. Finally, periprosthetic fractures may be a specific problem in sports-active patients who undergo TAR. In the current literature, there are only few reports on this complication. The literature consists primarily of case reports [215–217] or short annotations within large TAR studies [218]. Recently, Manegold et al. [219] established a concise classification system of periprosthetic fractures in TAR and described their treatment algorithm based on data from 503 ankle replacements. In total, 21 patients (4.2%) were identified with a periprosthetic fracture. Eleven and ten patients had intraoperative and postoperative fractures, respectively. The postoperative fractures included two traumatic cases and eight stress fractures. The authors did not mention how traumatic periprosthetic fractures occurred. However, high-energy sports, including contact sports, may be responsible for this difficult to treat complication [219].

Can all these problems be easily avoided by choosing ankle arthrodesis as a treatment option for end-stage ankle OA? Problems unique to TAR, like component loosening or failure of polyethylene inserts will not occur in patients who undergo ankle arthrodesis. However, other long-term problems in this patient cohort can occur. In a cadaveric study by Jung et al. [220] it was demonstrated that the tibiotalar joint arthrodesis significantly affected joint pressure distribution in the adjacent tarsal joints. This may explain the high incidence of OA in the adjacent joints following ankle arthrodesis [163, 165]. Further clinical studies need to be performed whether sports activities may accelerate the degenerative changes of the adjacent joints in patients who underwent ankle arthrodesis.

Based on our experience in foot and ankle surgery, we developed guidelines regarding sport activities in patients who underwent surgical treatments due to ankle OA (Table 33.2). We do believe that sports activities are part of an appropriate postoperative rehabilitation process in these patients. We encourage the patients to stay sports active or to regain their previous recreational activities. However, as Paracelsus stated in the sixteenth century, *Dosis sola facit venenum*. “The dose

makes the poison”: that means that even healthy sporting activities can be harmful if taken to excess. Every patient should keep in mind that he or she had ankle surgery in the past which may substantially lower the limits of their personal physical capacity. These limits should be respected, and not exceeded, in order to ensure good long-term results.

## Evidence

- Bonnin MP, Laurent JR, Casillas M. Ankle function and sports activity after total ankle arthroplasty. *Foot Ankle Int* 2009;30:933–44. Level of Evidence: III, Retrospective Case Control Study. *This study included 140 patients who underwent Salto total ankle replacement. Surgery improved their quality of life and return to recreational activities was generally possible, but return to impact sport was rarely possible* [130].
- Iosifidis MI, Tsarouhas A, Fylaktou A. Lower limb clinical and radiographic osteoarthritis in former elite male athletes. *Knee Surg Sports Traumatol Arthrosc* 2015;23:2528–35. Level of Evidence: III, Case–control Prognostic Study. *This case–control study included 218 former elite male athletes and 181 male controls. It showed that former elite athletes may not be at increased risk of developing clinical osteoarthritis. However, radiographic osteoarthritis signs had a significantly higher incidence in the athletes group* [18].
- Naal FD, Impellizzeri FM, Loibl M, Huber M, Rippstein PF. Habitual physical activity and sports participation after total ankle arthroplasty. *Am J Sports Med* 2009; 37:95–102. Level of Evidence: IV, Case Series. *The pre-operative and postoperative percentage of sports-active patients was constant with two-thirds of the 101 patients included in this study. No association between sports participation, increased physical activity, or the appearance of periprosthetic radiolucencies was found at a mean follow-up of 3.7 years after TAR* [129].
- Pagenstert G, Leumann A, Hintermann B, Valderrabano V. Sports and recreation activity of varus and valgus ankle osteoarthritis before and after realignment surgery. *Foot Ankle Int* 2008;29:985–93. Level of Evidence: II, Prospective Comparative Study. *This study demonstrated that hindfoot realignment surgery in patients with varus or valgus deformity substantially increased sports activity postoperatively. Improved ankle pain and function correlated with ability to perform activity without symptoms* [84].
- Saltzman CL, Mann RA, Ahrens JE, Amendola A, Anderson AB, Berlet GC, Brodsky JW, Chou LB, Clanton TO, Deland JT, DeOrto JK, Horton GA, Lee TH, Mann JA, Nunley JA, Thordarson DB, Walling AK, Wapner KL, Coughlin MJ. Prospective controlled trial of STAR total

ankle replacement versus ankle fusion: Initial results. *Foot Ankle Int* 2002; 23:68–74. Level of Evidence: II, Prospective Controlled Comparative Surgical Trial. *The only available prospective controlled trial addressing the safety and efficacy of total ankle replacement versus ankle fusion to treat end-stage ankle osteoarthritis. At 2-years follow-up, patients treated with total ankle replacement had better function and equivalent pain relief as patients treated with fusion* [138].

- Schuh R, Hofstaetter J, Krismer M, Bevoni R, Windhager R, Trnka HJ. Total ankle arthroplasty versus ankle arthrodesis. Comparison of sports, recreational activities and functional outcome. *Int Orthop* 2012; 36:1207–14. Level of Evidence: II, Prospective Comparative Study. *This study included 21 patients with ankle arthrodesis and 20 patients with total ankle replacement. It revealed no significant difference between groups concerning activity levels, participation in sports activities, or functional scores* [132].
- Valderrabano V, Horisberger M, Russell I, Dougall H, Hintermann B. Etiology of ankle osteoarthritis. *Clin Orthop Relat Res* 2009; 467:1800–6. Level of Evidence: IV, Prognostic Study. *In this study, the distribution of etiologies leading to ankle arthritis in 406 ankles was analyzed. Ankle osteoarthritis developed secondary to trauma in 79%. The traumas were mostly malleolar fractures, ligament lesions, and tibial plafond fractures* [4].
- Valderrabano V, Pagenstert G, Horisberger M, Knupp M, Hintermann B. Sports and recreation activity of ankle arthritis patients before and after total ankle replacement. *Am J Sports Med* 2006; 34:993–9. Level of Evidence: IV, Case Series. *This is the first clinical study addressing participation in sports after total ankle replacement. There was a significant increase in sports activity after total ankle replacement, with sports-active patients having better functional outcome than patients who did not participate in sports activities* [126].

## Summary

- Ankle OA is a debilitating disease and a growing problem in health care worldwide.
- Unlike the hip and knee, the ankle is most commonly affected by posttraumatic OA following fractures or ligament injuries.
- In the current literature, there is a controversial discussion whether sport activities play a positive or negative role in the development of ankle OA.
- In patients with ankle OA, numerous treatment options have been described, including joint-preserving and joint-sacrificing procedures.

- There is limited literature addressing participation in sports in patients who underwent surgical procedures due to ankle OA. In general, low impact sports can be recommended postoperatively. Further clinical studies are needed to define the role of sport in postoperative rehabilitation and to determine whether excessive sports activity is a risk factor for failure or worse outcomes.

**Acknowledgements** The authors thank Katharina Nikitina from the University of Freiburg, Germany, for her thorough literature review and her preparation of figures.

## References

- Barg A, Pagenstert GI, Hugle T, Gloyer M, Wiewiorski M, Henninger HB, et al. Ankle osteoarthritis: etiology, diagnostics, and classification. *Foot Ankle Clin.* 2013;18(3):411–26.
- Glazebrook M, Daniels T, Younger A, Foote CJ, Penner M, Wing K, et al. Comparison of health-related quality of life between patients with end-stage ankle and hip arthrosis. *J Bone Joint Surg Am.* 2008;90(3):499–505.
- Egloff C, Gloyer M, Barg K, Hugle T, Pagenstert G, Valderrabano V, et al. Ankle osteoarthritis – etiology and biomechanics. *Fuss Sprungg.* 2013;11(4):179–85.
- Valderrabano V, Horisberger M, Russell I, Dougall H, Hintermann B. Etiology of ankle osteoarthritis. *Clin Orthop Relat Res.* 2009;467(7):1800–6.
- Saltzman CL, Salamon ML, Blanchard GM, Huff T, Hayes A, Buckwalter JA, et al. Epidemiology of ankle arthritis: report of a consecutive series of 639 patients from a tertiary orthopaedic center. *Iowa Orthop J.* 2005;25(1):44–6.
- Wang B, Saltzman CL, Chalayan O, Barg A. Does the subtalar joint compensate for ankle malalignment in end-stage ankle arthritis? *Clin Orthop Relat Res.* 2015;473(1):318–25.
- Caine DJ, Golightly YM. Osteoarthritis as an outcome of paediatric sport: an epidemiological perspective. *Br J Sports Med.* 2011;45(4):298–303.
- Vincelette P, Laurin CA, Levesque HP. The footballer's ankle and foot. *Can Med Assoc J.* 1972;107(9):872–4.
- Adams ID. Osteoarthritis and sport. *J R Soc Med.* 1979;72(3):185–7.
- Hellmann DB, Helms CA, Genant HK. Chronic repetitive trauma: a cause of atypical degenerative joint disease. *Skeletal Radiol.* 1983;10(4):236–42.
- Kujala UM, Kaprio J, Sarna S. Osteoarthritis of weight bearing joints of lower limbs in former elite male athletes. *BMJ (Clin Res ed).* 1994;308(6923):231–4.
- Knobloch M, Marti B, Biedert R, Howald H. Risk of arthrosis of the upper ankle joint in long distance runners: controlled follow-up of former elite athletes. *Sportverletzung Sportschaden.* 1990;4(4):175–9.
- Gross P, Marti B. Risk of degenerative ankle joint disease in volleyball players: study of former elite athletes. *Int J Sports Med.* 1999;20(1):58–63.
- Zinder SM, Guskiewicz KM, Marshall SW. Prevalence of ankle osteoarthritis following a history of ankle sprain in retired professional football players. *Med Sci Sports Exerc.* 2010;42(5):608.
- Kuijt MT, Inklaar H, Gouttebauge V, Frings-Dresen MH. Knee and ankle osteoarthritis in former elite soccer players: a systematic review of the recent literature. *J Sci Med Sport.* 2012;15(6):480–7.
- Gouttebauge V, Inklaar H, Backx F, Kerkhoffs G. Prevalence of osteoarthritis in former elite athletes: a systematic overview of the recent literature. *Rheumatol Int.* 2015;35(3):405–18.
- Schmitt H, Lemke JM, Brocai DR, Parsch D. Degenerative changes in the ankle in former elite high jumpers. *Clin J Sport Med.* 2003;13(1):6–10.
- Iosifidis MI, Tsarouhas A, Fylaktou A. Lower limb clinical and radiographic osteoarthritis in former elite male athletes. *Knee Surg Sports Traumatol Arthrosc.* 2015;23(9):2528–35.
- Fong DT, Hong Y, Chan LK, Yung PS, Chan KM. A systematic review on ankle injury and ankle sprain in sports. *Sports Med.* 2007;37(1):73–94.
- Kobayashi T, Gamada K. Lateral ankle sprain and chronic ankle instability: a critical review. *Foot Ankle Spec.* 2014;7(4):298–326.
- Agel J, Olson DE, Dick R, Arendt EA, Marshall SW, Sikka RS. Descriptive epidemiology of collegiate women's basketball injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 through 2003–2004. *J Athl Train.* 2007;42(2):202–10.
- Borowski LA, Yard EE, Fields SK, Comstock RD. The epidemiology of US high school basketball injuries, 2005–2007. *Am J Sports Med.* 2008;36(12):2328–35.
- Deitch JR, Starkey C, Walters SL, Moseley JB. Injury risk in professional basketball players: a comparison of Women's National Basketball Association and National Basketball Association athletes. *Am J Sports Med.* 2006;34(7):1077–83.
- Dick R, Hertel J, Agel J, Grossman J, Marshall SW. Descriptive epidemiology of collegiate men's basketball injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 through 2003–2004. *J Athl Train.* 2007;42(2):194–201.
- McCarthy MM, Voos JE, Nguyen JT, Callahan L, Hannafin JA. Injury profile in elite female basketball athletes at the Women's National Basketball Association combine. *Am J Sports Med.* 2013;41(3):645–51.
- McKay GD, Goldie PA, Payne WR, Oakes BW. Ankle injuries in basketball: injury rate and risk factors. *Br J Sports Med.* 2001;35(2):103–8.
- Meeuwisse WH, Sellmer R, Hagel BE. Rates and risks of injury during intercollegiate basketball. *Am J Sports Med.* 2003;31(3):379–85.
- Starkey C. Injuries and illnesses in the national basketball association: a 10-year perspective. *J Athl Train.* 2000;35(2):161–7.
- Agel J, Evans TA, Dick R, Putukian M, Marshall SW. Descriptive epidemiology of collegiate men's soccer injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 through 2002–2003. *J Athl Train.* 2007;42(2):270–7.
- Dick R, Putukian M, Agel J, Evans TA, Marshall SW. Descriptive epidemiology of collegiate women's soccer injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 through 2002–2003. *J Athl Train.* 2007;42(2):278–85.
- Dvorak J, Junge A, Derman W, Schweltnus M. Injuries and illnesses of football players during the 2010 FIFA World Cup. *Br J Sports Med.* 2011;45(8):626–30.
- Faude O, Junge A, Kindermann W, Dvorak J. Injuries in female soccer players: a prospective study in the German national league. *Am J Sports Med.* 2005;33(11):1694–700.
- Gaulrapp H, Becker A, Walther M, Hess H. Injuries in women's soccer: a 1-year all players prospective field study of the women's Bundesliga (German premier league). *Clin J Sport Med.* 2010;20(4):264–71.
- Herrero H, Salinero JJ, Del Coso J. Injuries among Spanish male amateur soccer players: a retrospective population study. *Am J Sports Med.* 2014;42(1):78–85.
- Le Gall F, Carling C, Reilly T. Injuries in young elite female soccer players: an 8-season prospective study. *Am J Sports Med.* 2008;36(2):276–84.
- Leininger RE, Knox CL, Comstock RD. Epidemiology of 1.6 million pediatric soccer-related injuries presenting to US emergency departments from 1990 to 2003. *Am J Sports Med.* 2007;35(2):288–93.

37. Smith RW, Reischl SF. Treatment of ankle sprains in young athletes. *Am J Sports Med.* 1986;14(6):465–71.
38. Tegnander A, Olsen OE, Moholdt TT, Engebretsen L, Bahr R. Injuries in Norwegian female elite soccer: a prospective one-season cohort study. *Knee Surg Sports Traumatol Arthrosc.* 2008;16(2):194–8.
39. Valderrabano V, Barg A, Paul J, Pagenstert G, Wiewiorski M. Foot and ankle injuries in professional soccer players. *Sport Orthop Traumatol.* 2014;30(2):98–105.
40. Walden M, Hagglund M, Ekstrand J. Time-trends and circumstances surrounding ankle injuries in men's professional football: an 11-year follow-up of the UEFA Champions League injury study. *Br J Sports Med.* 2013;47(12):748–53.
41. Wong P, Hong Y. Soccer injury in the lower extremities. *Br J Sports Med.* 2005;39(8):473–82.
42. Darrow CJ, Collins CL, Yard EE, Comstock RD. Epidemiology of severe injuries among United States high school athletes: 2005–2007. *Am J Sports Med.* 2009;37(9):1798–805.
43. Iacovelli JN, Yang J, Thomas G, Wu H, Schiltz T, Foster DT. The effect of field condition and shoe type on lower extremity injuries in American Football. *Br J Sports Med.* 2013;47(12):789–93.
44. Kaplan LD, Jost PW, Honkamp N, Norwig J, West R, Bradley JP. Incidence and variance of foot and ankle injuries in elite college football players. *Am J Orthop (Belle Mead, NJ).* 2011;40(1):40–4.
45. Meyers MC. Incidence, mechanisms, and severity of game-related college football injuries on FieldTurf versus natural grass: a 3-year prospective study. *Am J Sports Med.* 2010;38(4):687–97.
46. Olson D, Sikka RS, Labounty A, Christensen T. Injuries in professional football: current concepts. *Curr Sports Med Rep.* 2013;12(6):381–90.
47. Osbahr DC, Drakos MC, O'Loughlin PF, Lyman S, Barnes RP, Kennedy JG, et al. Syndesmosis and lateral ankle sprains in the National Football League. *Orthopedics.* 2013;36(11):e1378–84.
48. Abrams GD, Renstrom PA, Safran MR. Epidemiology of musculoskeletal injury in the tennis player. *Br J Sports Med.* 2012;46(7):492–8.
49. Bylak J, Hutchinson MR. Common sports injuries in young tennis players. *Sports Med.* 1998;26(2):119–32.
50. Hjeltn N, Werner S, Renstrom P. Injury profile in junior tennis players: a prospective two year study. *Knee Surg Sports Traumatol Arthrosc.* 2010;18(6):845–50.
51. Hutchinson MR, Laprade RF, Burnett 2nd QM, Moss R, Terpstra J. Injury surveillance at the USTA Boys' Tennis Championships: a 6-yr study. *Med Sci Sports Exerc.* 1995;27(6):826–30.
52. Waterman BR, Owens BD, Davey S, Zacchilli MA, Belmont Jr PJ. The epidemiology of ankle sprains in the United States. *J Bone Joint Surg Am.* 2010;92(13):2279–84.
53. Beynnon BD, Murphy DF, Alosa DM. Predictive factors for lateral ankle sprains: a literature review. *J Athl Train.* 2002;37(4):376–80.
54. Beynnon BD, Vacek PM, Murphy D, Alosa D, Paller D. First-time inversion ankle ligament trauma: the effects of sex, level of competition, and sport on the incidence of injury. *Am J Sports Med.* 2005;33(10):1485–91.
55. Murphy DF, Connolly DA, Beynnon BD. Risk factors for lower extremity injury: a review of the literature. *Br J Sports Med.* 2003;37(1):13–29.
56. Caine D, Caine C, Maffulli N. Incidence and distribution of pediatric sport-related injuries. *Clin J Sport Med.* 2006;16(6):500–13.
57. Swenson DM, Yard EE, Fields SK, Comstock RD. Patterns of recurrent injuries among US high school athletes, 2005–2008. *Am J Sports Med.* 2009;37(8):1586–93.
58. Chan KW, Ding BC, Mroczek KJ. Acute and chronic lateral ankle instability in the athlete. *Bull NYU Hosp Jt Dis.* 2011;69(1):17–26.
59. de Vries JS, Krips R, Sierevelt IN, Blankevoort L, van Dijk CN. Interventions for treating chronic ankle instability. *Cochrane Database Syst Rev.* 2011(8):CD004124.
60. van Rijn RM, van Os AG, Bernsen RM, Luijsterburg PA, Koes BW, Bierma-Zeinstra SM. What is the clinical course of acute ankle sprains? A systematic literature review. *Am J Med.* 2008;121(4):324–31.
61. Hershkovich O, Tenenbaum S, Gordon B, Bruck N, Thein R, Derazne E, et al. A large-scale study on epidemiology and risk factors for chronic ankle instability in young adults. *J Foot Ankle Surg.* 2015;54(2):183–7.
62. Pourkazemi F, Hiller CE, Raymond J, Nightingale EJ, Refshauge KM. Predictors of chronic ankle instability after an index lateral ankle sprain: a systematic review. *J Sci Med Sport/Sports Med Aust.* 2014;17(6):568–73.
63. Valderrabano V, Hintermann B, Horisberger M, Fung TS. Ligamentous posttraumatic ankle osteoarthritis. *Am J Sports Med.* 2006;34(4):612–20.
64. Cha SD, Kim HS, Chung ST, Yoo JH, Park JH, Kim JH, et al. Intra-articular lesions in chronic lateral ankle instability: comparison of arthroscopy with magnetic resonance imaging findings. *Clin Orthop Surg.* 2012;4(4):293–9.
65. Hintermann B, Boss A, Schafer D. Arthroscopic findings in patients with chronic ankle instability. *Am J Sports Med.* 2002;30(3):402–9.
66. Lee J, Hamilton G, Ford L. Associated intra-articular ankle pathologies in patients with chronic lateral ankle instability: arthroscopic findings at the time of lateral ankle reconstruction. *Foot Ankle Spec.* 2011;4(5):284–9.
67. Sugimoto K, Takakura Y, Okahashi K, Samoto N, Kawate K, Iwai M. Chondral injuries of the ankle with recurrent lateral instability: an arthroscopic study. *J Bone Joint Surg Am.* 2009;91(1):99–106.
68. Bischof JE, Spritzer CE, Caputo AM, Easley ME, DeOrto JK, Nunley 2nd JA, et al. In vivo cartilage contact strains in patients with lateral ankle instability. *J Biomech.* 2010;43(13):2561–6.
69. Caputo AM, Lee JY, Spritzer CE, Easley ME, DeOrto JK, Nunley 2nd JA, et al. In vivo kinematics of the tibiotalar joint after lateral ankle instability. *Am J Sports Med.* 2009;37(11):2241–8.
70. Golditz T, Steib S, Pfeifer K, Uder M, Gelse K, Janka R, et al. Functional ankle instability as a risk factor for osteoarthritis: using T2-mapping to analyze early cartilage degeneration in the ankle joint of young athletes. *Osteoarthritis Cartilage.* 2014;22(10):1377–85.
71. McKinley TO, Tochigi Y, Rudert MJ, Brown TD. The effect of incongruity and instability on contact stress directional gradients in human cadaveric ankles. *Osteoarthritis Cartilage.* 2008;16(11):1363–9.
72. Harrington KD. Degenerative arthritis of the ankle secondary to long-standing lateral ligament instability. *J Bone Joint Surg Am.* 1979;61(3):354–61.
73. Taga I, Shino K, Inoue M, Nakata K, Maeda A. Articular cartilage lesions in ankles with lateral ligament injury. An arthroscopic study. *Am J Sports Med.* 1993;21(1):120–6; discussion 6–7.
74. Horisberger M, Hintermann B, Valderrabano V. Alterations of plantar pressure distribution in posttraumatic end-stage ankle osteoarthritis. *Clin Biomech (Bristol, Avon).* 2009;24(3):303–7.
75. Horisberger M, Valderrabano V, Hintermann B. Posttraumatic ankle osteoarthritis after ankle-related fractures. *J Orthop Trauma.* 2009;23(1):60–7.
76. Barg A, Paul J, Pagenstert GI, Leumann A, Horisberger M, Henninger HB, et al. Supramalleolar osteotomies for ankle osteoarthritis. *Tech Foot Ankle.* 2013;12(2):138–46.
77. Barg A, Pagenstert GI, Horisberger M, Paul J, Gloyer M, Henninger HB, et al. Supramalleolar osteotomies for degenerative joint disease of the ankle joint: indication, technique and results. *Int Orthop.* 2013;37(9):1683–95.

78. Barg A, Saltzman CL. Single-stage supramalleolar osteotomy for coronal plane deformity. *Curr Rev Musculoskelet Med.* 2014;7(4):277–91.
79. Colin F, Gaudot F, Odri G, Judet T. Supramalleolar osteotomy: techniques, indications and outcomes in a series of 83 cases. *Orthop Traumatol Surg Res.* 2014;100(4):413–8.
80. Knupp M, Stufkens SA, Bolliger L, Barg A, Hintermann B. Classification and treatment of supramalleolar deformities. *Foot Ankle Int.* 2011;32(11):1023–31.
81. Pagenstert G, Knupp M, Valderrabano V, Hintermann B. Realignment surgery for valgus ankle osteoarthritis. *Oper Orthop Traumatol.* 2009;21(1):77–87.
82. Takakura Y, Takaoka T, Tanaka Y, Yajima H, Tamai S. Results of opening-wedge osteotomy for the treatment of a post-traumatic varus deformity of the ankle. *J Bone Joint Surg Am.* 1998;80(2):213–8.
83. Harstall R, Lehmann O, Krause F, Weber M. Supramalleolar lateral closing wedge osteotomy for the treatment of varus ankle arthrosis. *Foot Ankle Int.* 2007;28(5):542–8.
84. Pagenstert G, Leumann A, Hintermann B, Valderrabano V. Sports and recreation activity of varus and valgus ankle osteoarthritis before and after realignment surgery. *Foot Ankle Int.* 2008;29(10):985–93.
85. Hintermann B, Barg A, Knupp M. Corrective supramalleolar osteotomy for malunited pronation-external rotation fractures of the ankle. *J Bone Joint Surg.* 2011;93(10):1367–72.
86. Mann HA, Filippi J, Myerson MS. Intra-articular opening medial tibial wedge osteotomy (plafond-plasty) for the treatment of intra-articular varus ankle arthritis and instability. *Foot Ankle Int.* 2012;33(4):255–61.
87. Barg A, Saltzman CL. Ankle replacement. In: Coughlin MJ, Saltzman CL, Anderson RB, editors. *Mann's Surgery of the foot and ankle.* Philadelphia: Elsevier Saunders; 2014. p. 1078–162.
88. Barg A, Wimmer MD, Wiewiorski M, Wirtz DC, Pagenstert GI, Valderrabano V. Total ankle replacement – indications, implant designs, and results. *Dtsch Arztebl Int.* 2015;112(11):177–84.
89. Gougoulias NE, Agathangelidis FG, Parsons SW. Arthroscopic ankle arthrodesis. *Foot Ankle Int.* 2007;28(6):695–706.
90. Bonasia DE, Dettoni F, Femino JE, Phisitkul P, Germano M, Amendola A. Total ankle replacement: why, when and how? *Iowa Orthop J.* 2010;30(1):119–30.
91. Zaidi R, Cro S, Gurusamy K, Siva N, Macgregor A, Henricson A, et al. The outcome of total ankle replacement: a systematic review and meta-analysis. *Bone Joint J.* 2013;95-B(11):1500–7.
92. Gougoulias N, Khanna A, Maffulli N. How successful are current ankle replacements?: a systematic review of the literature. *Clin Orthop Relat Res.* 2010;468(1):199–208.
93. Labek G, Thaler M, Janda W, Agreiter M, Stockl B. Revision rates after total joint replacement: cumulative results from worldwide joint register datasets. *J Bone Joint Surg.* 2011;93(3):293–7.
94. Sadoghi P, Liebensteiner M, Agreiter M, Leithner A, Bohler N, Labek G. Revision surgery after total joint arthroplasty: a complication-based analysis using worldwide arthroplasty registers. *J Arthroplasty.* 2013;28(8):1329–32.
95. Barg A, Barg K, Schneider SW, Pagenstert G, Gloyer M, Henninger HB, et al. Thromboembolic complications after total ankle replacement. *Curr Rev Musculoskelet Med.* 2014;6(4):328–35.
96. Barg A, Barg K, Wiewiorski M, Schneider SW, Wimmer MD, Wirtz DC, et al. Total ankle replacement in patients with bleeding disorders. *Orthopade.* 2015;44(8):623–8.
97. Barg A, Elsner A, Anderson AE, Hintermann B. The effect of three-component total ankle replacement malalignment on clinical outcome: pain relief and functional outcome in 317 consecutive patients. *J Bone Joint Surg Am.* 2011;93(21):1969–78.
98. Barg A, Elsner A, Chuckpaiwong B, Hintermann B. Insert position in three-component total ankle replacement. *Foot Ankle Int.* 2010;31(9):754–9.
99. Barg A, Elsner A, Hefti D, Hintermann B. Haemophilic arthropathy of the ankle treated by total ankle replacement: a case series. *Haemophilia.* 2010;16(4):647–55.
100. Barg A, Elsner A, Hefti D, Hintermann B. Total ankle arthroplasty in patients with hereditary hemochromatosis. *Clin Orthop Relat Res.* 2011;469(5):1427–35.
101. Barg A, Henninger HB, Hintermann B. Risk factors for symptomatic deep-vein thrombosis in patients after total ankle replacement who received routine chemical thromboprophylaxis. *J Bone Joint Surg.* 2011;93(7):921–7.
102. Barg A, Henninger HB, Knupp M, Hintermann B. Simultaneous bilateral total ankle replacement using a 3-component prosthesis: outcome in 26 patients followed for 2–10 years. *Acta Orthop.* 2011;82(6):704–10.
103. Barg A, Hintermann B. Takedown of painful ankle fusion and total ankle replacement using a 3-component ankle prosthesis. *Tech Foot Ankle.* 2010;9(4):190–8.
104. Barg A, Knupp M, Anderson AE, Hintermann B. Total ankle replacement in obese patients: component stability, weight change, and functional outcome in 118 consecutive patients. *Foot Ankle Int.* 2011;32(10):925–32.
105. Barg A, Knupp M, Henninger HB, Zwicky L, Hintermann B. Total ankle replacement using HINTEGRA, an unconstrained, three-component system: surgical technique and pitfalls. *Foot Ankle Clin.* 2012;17(4):607–35.
106. Barg A, Knupp M, Hintermann B. Simultaneous bilateral versus unilateral total ankle replacement: a patient-based comparison of pain relief, quality of life and functional outcome. *J Bone Joint Surg.* 2010;92(12):1659–63.
107. Barg A, Knupp M, Kapron AL, Hintermann B. Total ankle replacement in patients with gouty arthritis. *J Bone Joint Surg Am.* 2011;93(4):357–66.
108. Barg A, Pagenstert GI, Leumann AG, Muller AM, Henninger HB, Valderrabano V. Treatment of the arthritic valgus ankle. *Foot Ankle Clin.* 2012;17(4):647–63.
109. Barg A, Schneider SW, Pagenstert G, Hintermann B, Valderrabano V. Thromboembolic complications following ankle prosthesis implantation. *Orthopade.* 2013;42(11):948–56.
110. Barg A, Suter T, Zwicky L, Knupp M, Hintermann B. Medial pain syndrome in patients with total ankle replacement. *Orthopade.* 2011;40(11):991–2. 4–9.
111. Barg A, Zwicky L, Knupp M, Henninger HB, Hintermann B. HINTEGRA total ankle replacement: survivorship analysis in 684 patients. *J Bone Joint Surg Am.* 2013;95(13):1175–83.
112. Barg K, Wiewiorski M, Anderson AE, Schneider SW, Wimmer MD, Wirtz DC, et al. Total ankle replacement in patients with von Willebrand disease: mid-term results of 18 procedures. *Haemophilia.* 2015;21(5):e389–401.
113. Brunner S, Barg A, Knupp M, Zwicky L, Kapron AL, Valderrabano V, et al. The Scandinavian total ankle replacement: long-term, eleven to fifteen-year, survivorship analysis of the prosthesis in seventy-two consecutive patients. *J Bone Joint Surg Am.* 2013;95(8):711–8.
114. Hintermann B, Barg A. Total ankle replacement in patients with osteoarthritis. *Arthroskopie.* 2011;24(4):274–82.
115. Hintermann B, Barg A, Knupp M. Revision arthroplasty of the ankle joint. *Orthopade.* 2011;40(11):1000–7.
116. Hintermann B, Barg A, Knupp M, Valderrabano V. Conversion of painful ankle arthrodesis to total ankle arthroplasty. *J Bone Joint Surg Am.* 2009;91(4):850–8.
117. Hintermann B, Barg A, Knupp M, Valderrabano V. Conversion of painful ankle arthrodesis to total ankle arthroplasty. Surgical technique. *J Bone Joint Surg Am.* 2010;92(Suppl 1 Pt 1):55–66.
118. Hintermann B, Valderrabano V. Total ankle replacement. *Foot Ankle Clin.* 2003;8(2):375–405.

119. Hintermann B, Valderrabano V, Dereymaeker G, Dick W. The HINTEGRA ankle: rationale and short-term results of 122 consecutive ankles. *Clin Orthop Relat Res.* 2004;424:57–68.
120. Hintermann B, Zwicky L, Knupp M, Henninger HB, Barg A. HINTEGRA revision arthroplasty for failed total ankle prostheses. *J Bone Joint Surg Am.* 2013;95(13):1166–74.
121. Paul J, Barg A, Kretzschmar M, Pagenstert G, Studler U, Hugle T, et al. Increased osseous <sup>99m</sup>Tc-DPD uptake in end-stage ankle osteoarthritis: correlation between SPECT-CT imaging and histologic findings. *Foot Ankle Int.* 2015;36(12):1438–47.
122. Valderrabano V, Frigg A, Leumann A, Horisberger M. Total ankle arthroplasty in valgus ankle osteoarthritis. *Orthopade.* 2011;40(11):971–4. 6–7.
123. Valderrabano V, Hintermann B, Dick W. Scandinavian total ankle replacement: a 3.7-year average followup of 65 patients. *Clin Orthop Related Res.* 2004;424(1):47–56.
124. Valderrabano V, Nigg BM, von Tscharnner V, Frank CB, Hintermann B. J. Leonard Goldner Award 2006. Total ankle replacement in ankle osteoarthritis: an analysis of muscle rehabilitation. *Foot Ankle Int.* 2007;28(2):281–91.
125. Valderrabano V, Nigg BM, von Tscharnner V, Stefanyshyn DJ, Goepfert B, Hintermann B. Gait analysis in ankle osteoarthritis and total ankle replacement. *Clin Biomech (Bristol, Avon).* 2007;22(8):894–904.
126. Valderrabano V, Pagenstert G, Horisberger M, Knupp M, Hintermann B. Sports and recreation activity of ankle arthritis patients before and after total ankle replacement. *Am J Sports Med.* 2006;34(6):993–9.
127. Valderrabano V, Pagenstert GI, Muller AM, Paul J, Henninger HB, Barg A. Mobile- and fixed-bearing total ankle prostheses: is there really a difference? *Foot Ankle Clin.* 2012;17(4):565–85.
128. Pipino F, Caldera PM. PC ankle prosthesis. Five year follow-up. *Acta Orthop Belg.* 1983;49(6):725–35.
129. Naal FD, Impellizzeri FM, Loibl M, Huber M, Rippstein PF. Habitual physical activity and sports participation after total ankle arthroplasty. *Am J Sports Med.* 2009;37(1):95–102.
130. Bonnin MP, Laurent JR, Casillas M. Ankle function and sports activity after total ankle arthroplasty. *Foot Ankle Int.* 2009;30(10):933–44.
131. Harris S, Boggio LN. Exercise may decrease further destruction in the adult haemophilic joint. *Haemophilia.* 2006;12(3):237–40.
132. Schuh R, Hofstaetter J, Krismer M, Bevoni R, Windhager R, Trnka HJ. Total ankle arthroplasty versus ankle arthrodesis. Comparison of sports, recreational activities and functional outcome. *Int Orthop.* 2012;36(6):1207–14.
133. Criswell BJ, Douglas K, Naik R, Thomson AB. High revision and reoperation rates using the Agility Total Ankle System. *Clin Orthop Relat Res.* 2012;470(7):1980–6.
134. Dalat F, Trouillet F, Fessy MH, Bourdin M, Besse JL. Comparison of quality of life following total ankle arthroplasty and ankle arthrodesis: retrospective study of 54 cases. *Orthop Traumatol Surg Res.* 2014;100(7):761–6.
135. Nodzo SR, Miladore MP, Kaplan NB, Ritter CA. Short to midterm clinical and radiographic outcomes of the Salto total ankle prosthesis. *Foot Ankle Int.* 2014;35(1):22–9.
136. Braito M, Dammerer D, Reinthaler A, Kaufmann G, Huber D, Biedermann R. Effect of coronal and sagittal alignment on outcome after mobile-bearing total ankle replacement. *Foot Ankle Int.* 2015;36(9):1029–37.
137. Kerkhoff YRA, Kosse NM, Louwerens JW. Short term results of the Mobility Total Ankle System: Clinical and radiographic outcome. *Foot Ankle Surg.* 2016;22(3):152–7.
138. Saltzman CL, Mann RA, Ahrens JE, Amendola A, Anderson RB, Berlet GC, et al. Prospective controlled trial of STAR total ankle replacement versus ankle fusion: initial results. *Foot Ankle Int.* 2009;30(7):579–96.
139. Colombier JA, Judet T, Bonnin M, Gaudot F. Techniques and pitfalls with the Salto prosthesis: our experience of the first 15 years. *Foot Ankle Clin.* 2012;17(4):587–605.
140. Daniels TR, Younger AS, Penner M, Wing K, Dryden PJ, Wong H, et al. Intermediate-term results of total ankle replacement and ankle arthrodesis: a COFAS multicenter study. *J Bone Joint Surg Am.* 2014;96(2):135–42.
141. Ellis SJ, Moril-Penalver L, Deland JT. The Scandinavian Total Ankle Replacement (STAR) system. *Semin Arthro.* 2010;21(2):275–81.
142. Frigg A, Nigg B, Hinz L, Valderrabano V, Russell I. Clinical relevance of hindfoot alignment view in total ankle replacement. *Foot Ankle Int.* 2010;31(10):871–9.
143. Kofoed H. The evolution of ankle arthroplasty. *Orthopade.* 1999;28(9):804–11.
144. Kofoed H. Scandinavian total ankle replacement: the surgical technique. *Tech Foot Ankle.* 2005;4(2):55–61.
145. Kofoed H, Lundberg-Jensen A. Ankle arthroplasty in patients younger and older than 50 years: a prospective series with long-term follow-up. *Foot Ankle Int.* 1999;20(8):501–6.
146. Krause FG, Schmid T. Ankle arthrodesis versus total ankle replacement: how do I decide? *Foot Ankle Clin.* 2012;17(4):529–43.
147. Lieske S, Schenk K, Neumann HW, John M. Implantation of a Salto 2 total ankle prosthesis. *Oper Orthop Traumatol.* 2014;26(4):401–11.
148. Mittlmeier T. Arthrodesis versus total joint replacement of the ankle. *Unfallchirurg.* 2013;116(6):537–50; quiz 51–2.
149. Rippstein PF, Naal FD. Total ankle replacement in rheumatoid arthritis. *Orthopade.* 2011;40(11):984–6. 8–90.
150. Rzesacz EH, Gosse F. Management of posttraumatic osteoarthritis of the upper ankle joint by implantation of the S.T.A.R. ankle prosthesis. *Oper Orthop Traumatol.* 2007;19(5–6):527–46.
151. Thermann H, Saltzman CL. Prosthetic replacement of the upper ankle joint. *Unfallchirurg.* 2002;105(6):496–510.
152. Giannini S, Romagnoli M, O'Connor JJ, Malerba F, Leardini A. Total ankle replacement compatible with ligament function produces mobility, good clinical scores, and low complication rates: an early clinical assessment. *Clin Orthop Relat Res.* 2010;468(10):2746–53.
153. Ahmad J, Raikin SM. Ankle arthrodesis: the simple and the complex. *Foot Ankle Clin.* 2008;13(3):381–400.
154. Nihal A, Gellman RE, Embil JM, Trepman E. Ankle arthrodesis. *Foot Ankle Surg.* 2008;14(1):1–10.
155. Albert E. Einige Falle kunstlicher Ankylosen: Bildung an paralytischen Gliedmassen. *Wien Med Press.* 1882;23(5):726–8.
156. Chalayon O, Wang B, Blankenhorn B, Jackson 3rd JB, Beals T, Nickisch F, et al. Factors affecting the outcomes of uncomplicated primary open ankle arthrodesis. *Foot Ankle Int.* 2015;36(10):1170–9.
157. Coester LM, Saltzman CL, Leupold J, Pontarelli W. Long-term results following ankle arthrodesis for post-traumatic arthritis. *J Bone Joint Surg Am.* 2001;83(2):219–28.
158. Lance EM, Paval A, Fries I, Larsen I, Patterson Jr RL. Arthrodesis of the ankle joint: a follow-up study. *Clin Orthop Related Res.* 1979;142(1):146–58.
159. Lynch AF, Bourne RB, Rorabeck CH. The long-term results of ankle arthrodesis. *J Bone Joint Surg.* 1988;70(1):113–6.
160. Mazur JM, Schwartz E, Simon SR. Ankle arthrodesis. Long-term follow-up with gait analysis. *J Bone Joint Surg Am.* 1979;61(7):964–75.
161. Morgan CD, Henke JA, Bailey RW, Kaufer H. Long-term results of tibiotalar arthrodesis. *J Bone Joint Surg Am.* 1985;67(4):546–50.
162. Ebalard M, Le Henaff G, Sigonney G, Lopes R, Kerhousse G, Brillhault J, et al. Risk of osteoarthritis secondary to partial or total arthrodesis of the subtalar and midtarsal joints after a minimum



- follow-up of 10 years. *Orthop Traumatol Surg Res.* 2014;100(4 Suppl):S231–7.
163. Fuchs S, Sandmann C, Skwara A, Chylarecki C. Quality of life 20 years after arthrodesis of the ankle. A study of adjacent joints. *J Bone Joint Surg.* 2003;85(7):994–8.
  164. Ling JS, Smyth NA, Fraser EJ, Hogan MV, Seaworth CM, Ross KA, et al. Investigating the relationship between ankle arthrodesis and adjacent-joint arthritis in the hindfoot: a systematic review. *J Bone Joint Surg Am.* 2015;97(6):513–20.
  165. Suckel A, Burger A, Wulker N, Wunschel M. Ankle arthrodesis – clinical, radiological and biomechanical aspects with special regard to the adjacent joints. *Z Orthop Unfall.* 2012;150(6):588–93.
  166. van der Plaats LW, van Engelen SJ, Wajer QE, Hendrickx RP, Doets KH, Houdijk H, et al. Hind- and midfoot motion after ankle arthrodesis. *Foot Ankle Int.* 2015;36(12):1430–7.
  167. Hendrickx RP, Stufkens SA, de Bruijn EE, Sierevelt IN, van Dijk CN, Kerkhoffs GM. Medium- to long-term outcome of ankle arthrodesis. *Foot Ankle Int.* 2011;32(10):940–7.
  168. Fuentes-Sanz A, Moya-Angeler J, Lopez-Oliva F, Forriol F. Clinical outcome and gait analysis of ankle arthrodesis. *Foot Ankle Int.* 2012;33(10):819–27.
  169. Hahn ME, Wright ES, Segal AD, Orendurff MS, Ledoux WR, Sangeorzan BJ. Comparative gait analysis of ankle arthrodesis and arthroplasty: initial findings of a prospective study. *Foot Ankle Int.* 2012;33(4):282–9.
  170. Piriou P, Culpán P, Mullins M, Cardon JN, Pozzi D, Judet T. Ankle replacement versus arthrodesis: a comparative gait analysis study. *Foot Ankle Int.* 2008;29(1):3–9.
  171. Jastifer J, Coughlin MJ, Hirose C. Performance of total ankle arthroplasty and ankle arthrodesis on uneven surfaces, stairs, and inclines: a prospective study. *Foot Ankle Int.* 2015;36(1):11–7.
  172. Thermann H, Huefner T, Schrott HE, von Glinski S, Roehler A, Tscherner H. Schrew fixation for ankle arthrodesis. *Foot Ankle Surg.* 1999;5(3):131–42.
  173. Vertullo CJ, Nunley JA. Participation in sports after arthrodesis of the foot or ankle. *Foot Ankle Int.* 2002;23(7):625–8.
  174. Goebel M, Gerdesmeyer L, Muckley T, Schmitt-Sody M, Diehl P, Stienstra J, et al. Retrograde intramedullary nailing in tibioalocalcaneal arthrodesis: a short-term, prospective study. *J Foot Ankle Surg Off Publ Am Coll Foot Ankle Surg.* 2006;45(2):98–106.
  175. Akra GA, Middleton A, Adedapo AO, Port A, Finn P. Outcome of ankle arthrodesis using a transfibular approach. *J Foot Ankle Surg.* 2010;49(6):508–12.
  176. Puno RM, Vaughan JJ, Stetten ML, Johnson JR. Long-term effects of tibial angular malunion on the knee and ankle joints. *J Orthop Trauma.* 1991;5(3):247–54.
  177. Puno RM, Vaughan JJ, von Fraunhofer JA, Stetten ML, Johnson JR. A method of determining the angular malalignments of the knee and ankle joints resulting from a tibial malunion. *Clin Orthop Relat Res.* 1987;223(3):213–9.
  178. Valderrabano V. Joint-preserving surgery of ankle osteoarthritis. *Foot Ankle Clin.* 2013;18(3):xii–v.
  179. Barg A, Amendola RL, Henninger HB, Kapron AL, Saltzman CL, Anderson AE. Measurement of supramalleolar alignment on the anteroposterior and hindfoot alignment views: Influence of ankle position and radiographic projection angle. *Foot Ankle Int.* 2015;36(11):1352–61.
  180. Barg A, Harris MD, Henninger HB, Amendola RL, Saltzman CL, Hintermann B, et al. Medial distal tibial angle: comparison between weightbearing mortise view and hindfoot alignment view. *Foot Ankle Int.* 2012;33(8):655–61.
  181. Egloff C, Paul J, Pagenstert G, Vavken P, Hintermann B, Valderrabano V, et al. Changes of density distribution of the subchondral bone plate after supramalleolar osteotomy for valgus ankle osteoarthritis. *J Orthop Res.* 2014;32(10):1356–61.
  182. Gloyer M, Barg A, Horisberger M, Paul J, Pagenstert G, Valderrabano V. Joint preserving surgery in patients with valgus- and varus osteoarthritis. *Fuss Sprungg.* 2013;11(4):186–95.
  183. Hintermann B, Knupp M, Barg A. Joint-preserving surgery of asymmetric ankle osteoarthritis with peritalar instability. *Foot Ankle Clin.* 2013;18(3):503–16.
  184. Hintermann B, Knupp M, Barg A. Joint preserving surgery in patients with peritalar instability. *Fuss Sprungg.* 2013;11(4):196–206.
  185. Hintermann B, Knupp M, Barg A. Supramalleolar osteotomies for ankle arthritis. *J Am Acad Orthop Surg.* 2016;24(7):424–32.
  186. Knupp M, Barg A, Bolliger L, Hintermann B. Reconstructive surgery for overcorrected clubfoot in adults. *J Bone Joint Surg Am.* 2012;94(15):e1101–7.
  187. Knupp M, Pagenstert G, Valderrabano V, Hintermann B. Osteotomies in varus malalignment of the ankle. *Oper Orthop Traumatol.* 2008;20(3):262–73.
  188. Nuesch C, Barg A, Pagenstert GI, Valderrabano V. Biomechanics of asymmetric ankle osteoarthritis and its joint-preserving surgery. *Foot Ankle Clin.* 2013;18(3):427–36.
  189. Nuesch C, Huber C, Paul J, Henninger HB, Pagenstert G, Valderrabano V, et al. Mid- to long-term clinical outcome and gait biomechanics after realignment surgery in asymmetric ankle osteoarthritis. *Foot Ankle Int.* 2015;36(8):908–18.
  190. Nuesch C, Valderrabano V, Huber C, Pagenstert G. Effects of supramalleolar osteotomies for ankle osteoarthritis on foot kinematics and lower leg muscle activation during walking. *Clin Biomech (Bristol, Avon).* 2014;29(3):257–64.
  191. Nuesch C, Valderrabano V, Huber C, von Tscharnner V, Pagenstert G. Gait patterns of asymmetric ankle osteoarthritis patients. *Clin Biomech (Bristol, Avon).* 2012;27(6):613–8.
  192. Pagenstert GI, Hintermann B, Barg A, Leumann A, Valderrabano V. Realignment surgery as alternative treatment of varus and valgus ankle osteoarthritis. *Clin Orthop Relat Res.* 2007;462(1):156–68.
  193. Valderrabano V, Paul J, Monika H, Pagenstert GI, Henninger HB, Barg A. Joint-preserving surgery of valgus ankle osteoarthritis. *Foot Ankle Clin.* 2013;18(3):481–502.
  194. DeHeer PA, Catoire SM, Taulman J, Borer B. Ankle arthrodesis: a literature review. *Clin Podiatr Med Surg.* 2012;29(4):509–27.
  195. Muir DC, Amendola A, Saltzman CL. Long-term outcome of ankle arthrodesis. *Foot Ankle Clin.* 2002;7(4):703–8.
  196. Stone JW. Arthroscopic ankle arthrodesis. *Foot Ankle Clin.* 2006;11(2):361–8.
  197. Sealey RJ, Myerson MS, Molloy A, Gamba C, Jeng C, Kalesan B. Sagittal plane motion of the hindfoot following ankle arthrodesis: a prospective analysis. *Foot Ankle Int.* 2009;30(3):187–96.
  198. Wood PL, Sutton C, Mishra V, Suneja R. A randomised, controlled trial of two mobile-bearing total ankle replacements. *J Bone Joint Surg.* 2009;91(1):69–74.
  199. Nunley JA, Caputo AM, Easley ME, Cook C. Intermediate to long-term outcomes of the STAR Total Ankle Replacement: the patient perspective. *J Bone Joint Surg Am.* 2012;94(1):43–8.
  200. Mann JA, Mann RA, Horton E. STAR ankle: long-term results. *Foot Ankle Int.* 2011;32(5):S473–84.
  201. Culpán P, Le Strat V, Piriou P, Judet T. Arthrodesis after failed total ankle replacement. *J Bone Joint Surg.* 2007;89(9):1178–83.
  202. Hopgood P, Kumar R, Wood PL. Ankle arthrodesis for failed total ankle replacement. *J Bone Joint Surg.* 2006;88(8):1032–8.
  203. Kotnis R, Pasapula C, Anwar F, Cooke PH, Sharp RJ. The management of failed ankle replacement. *J Bone Joint Surg.* 2006;88(8):1039–47.
  204. Rahm S, Klammer G, Benninger E, Gerber F, Farshad M, Espinosa N. Inferior results of salvage arthrodesis after failed ankle

- replacement compared to primary arthrodesis. *Foot Ankle Int.* 2015;36(4):349–59.
205. Sagherian BH, Claridge RJ. Salvage of failed total ankle replacement using tantalum trabecular metal: case series. *Foot Ankle Int.* 2015;36(3):318–24.
206. Hsu AR, Haddad SL, Myerson MS. Evaluation and management of the painful total ankle arthroplasty. *J Am Acad Orthop Surg.* 2015;23(5):272–82.
207. Williams JR, Wegner NJ, Sangeorzan BJ, Brage ME. Intraoperative and perioperative complications during revision arthroplasty for salvage of a failed total ankle arthroplasty. *Foot Ankle Int.* 2015;36(2):135–42.
208. Valderrabano V, Hintermann B, Nigg BM, Stefanyshyn D, Stergiou P. Kinematic changes after fusion and total replacement of the ankle: part 3: talar movement. *Foot Ankle Int.* 2003;24(12):897–900.
209. Valderrabano V, Hintermann B, Nigg BM, Stefanyshyn D, Stergiou P. Kinematic changes after fusion and total replacement of the ankle: part 2: movement transfer. *Foot Ankle Int.* 2003;24(12):888–96.
210. Valderrabano V, Hintermann B, Nigg BM, Stefanyshyn D, Stergiou P. Kinematic changes after fusion and total replacement of the ankle: part 1: range of motion. *Foot Ankle Int.* 2003;24(12):881–7.
211. Espinosa N, Walti M, Favre P, Snedeker JG. Misalignment of total ankle components can induce high joint contact pressures. *J Bone Joint Surg Am.* 2010;92(5):1179–87.
212. Kobayashi A, Minoda Y, Kadoya Y, Ohashi H, Takaoka K, Saltzman CL. Ankle arthroplasties generate wear particles similar to knee arthroplasties. *Clin Orthop Relat Res.* 2004;424 (1):69–72.
213. Harris NJ, Brooke BT, Sturdee S. A wear debris cyst following S.T.A.R. Total Ankle Replacement—surgical management. *Foot Ankle Surg.* 2009;15(1):43–5.
214. Scott AT, Nunley JA. Polyethylene fracture following STAR ankle arthroplasty: a report of three cases. *Foot Ankle Int.* 2009;30(4):375–9.
215. Haendlmayer KT, Fazly FM, Harris NJ. Periprosthetic fracture after total ankle replacement: surgical technique. *Foot Ankle Int.* 2009;30(12):1233–4.
216. McGarvey WC, Clanton TO, Lunz D. Malleolar fracture after total ankle arthroplasty: a comparison of two designs. *Clin Orthop Relat Res.* 2004;424:104–10.
217. Yang JH, Kim HJ, Yoon JR, Yoon YC. Minimally invasive plate osteosynthesis (MIPO) for periprosthetic fracture after total ankle arthroplasty: a case report. *Foot Ankle Int.* 2011;32(2):200–4.
218. Barg A, Hintermann B, Manegold S. Periprosthetic fractures in specific anatomical locations: ankle. In: Schuetz M, Perka C, editors. *Periprosthetic fracture management.* Davos: AO Trauma Thieme; 2014. p. 181–9.
219. Manegold S, Haas NP, Tsitsilonis S, Springer A, Mardian S, Schaser KD. Periprosthetic fractures in total ankle replacement: classification system and treatment algorithm. *J Bone Joint Surg Am.* 2013;95(9):815–20.
220. Jung HG, Parks BG, Nguyen A, Schon LC. Effect of tibiotalar joint arthrodesis on adjacent tarsal joint pressure in a cadaver model. *Foot Ankle Int.* 2007;28(1):103–8.
221. Weber BG, Simpson LA. Corrective lengthening osteotomy of the fibula. *Clin Orthop Relat Res.* 1985;199:61–7.
222. Saltzman CL, el-Khoury GY. The hindfoot alignment view. *Foot Ankle Int.* 1995;16(9):572–6.

Jeannie Huh, Christopher E. Gross, and James K. DeOrio

## Abstract

Arthritis of the foot is a frequent source of chronic pain and disability in both recreational and competitive athletes that prompts presentation to the foot and ankle surgeon. Causes of arthritis are multiple and generally include primary degenerative, inflammatory, and post-traumatic conditions. In the athlete, injury during play, either acute or from overuse, contributes to the majority of cases of symptomatic joint degeneration. In the foot, the subtalar, chopart, and Lisfranc joints can be particularly troublesome later in the athlete's life due to the constant forces placed across these joints. The focus of this chapter will be on the evaluation and management of subtalar, chopart, and Lisfranc degenerative joint disease (DJD).

## Keywords

Arthritis • Subtalar joint • Chopart joint • Lisfranc joint

## Abbreviations

AFO	Ankle-foot orthoses
AP	Anteroposterior
DJD	Degenerative joint disease
NSAIDS	Non-steroidal anti-inflammatory drugs
TMT	Tarsometatarsal
UCBL	University of California Biomechanics Laboratory

J. Huh, MD (✉)

Chief, Orthopaedic Foot and Ankle Service,  
Dwight D. Eisenhower Army Medical Center,  
Fort Gordon, GA, USA

Assistant Professor, Uniformed Services University of the Health  
Sciences, Bethesda, MD, USA  
e-mail: [jhandyh@gmail.com](mailto:jhandyh@gmail.com)

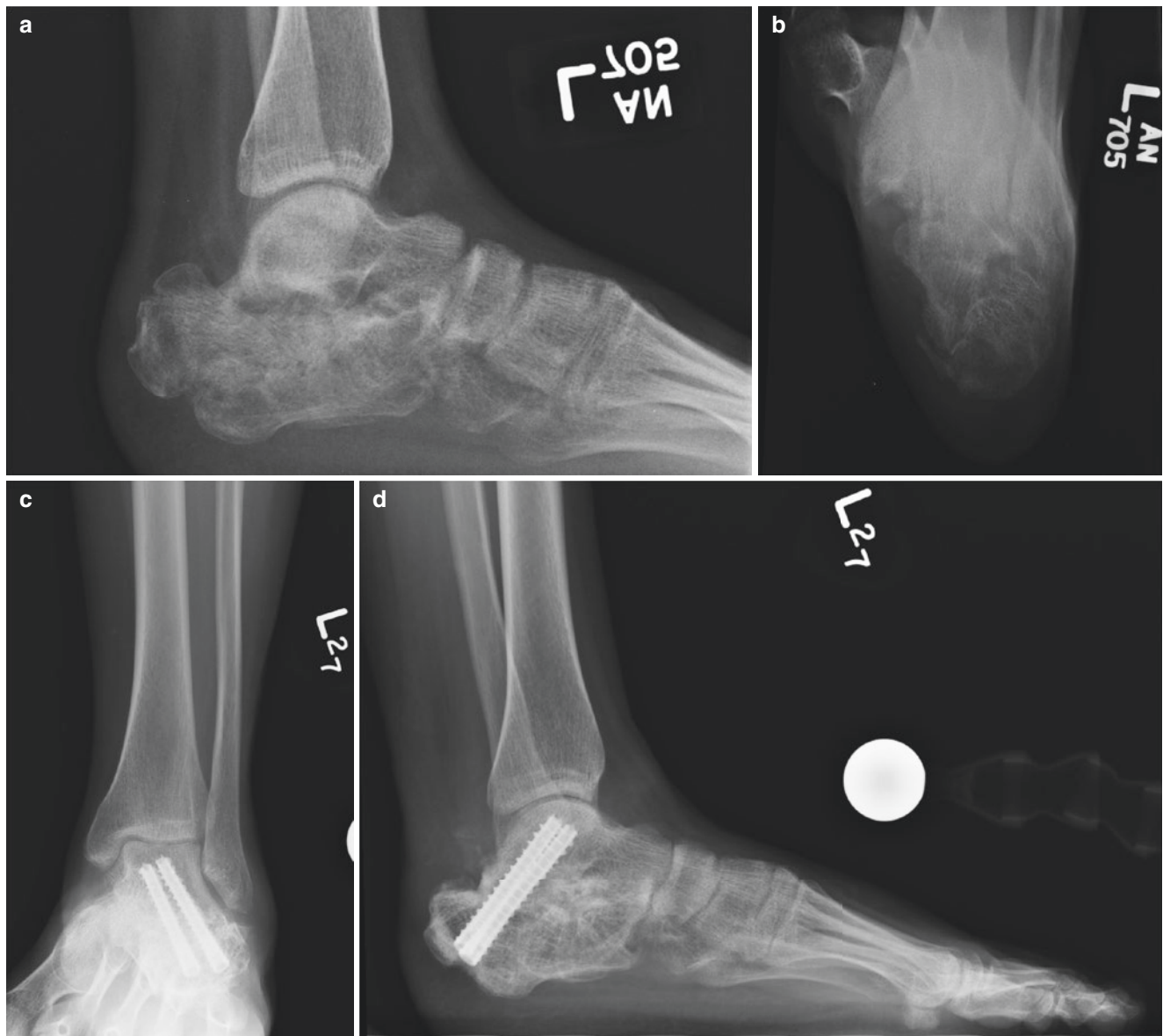
C.E. Gross, MD • J.K. DeOrio, MD  
Department of Orthopaedic Surgery, Duke University Medical  
Center, 4709 Creekstone Drive, Durham, NC 27703, USA  
e-mail: [cgross144@gmail.com](mailto:cgross144@gmail.com); [James.deorio@dm.duke.edu](mailto:James.deorio@dm.duke.edu)

## Section I: Subtalar Joint DJD

### Etiology and Pathomechanism

Isolated subtalar joint arthritis most commonly results from trauma, particularly following fractures of the calcaneus or talus (Fig. 34.1). Subtalar joint instability, which ranges in spectrum from the simple recurrent sprain to subtalar dislocation, can also predispose one to arthritis. Aside from trauma, other causes of subtalar arthritis include primary osteoarthritis, secondary arthritis (adjacent joint disease in the setting of prior ankle fusion), inflammatory arthritis (rheumatoid, psoriatic), residual congenital deformity (talocalcaneal coalition), and deformity from muscular imbalance (posterior tibial tendon dysfunction, Charcot-Marie-Tooth disease). In a review of 95 subtalar arthrodesis, Davies et al. found that 67% were for post-traumatic arthritis, 23% for primary osteoarthritis, 5.3% for tarsal coalition, and 4.2% for inflammatory joint disease [1].

Post-traumatic subtalar arthritis will develop in 50–100% of talar neck fractures [2]. Up to 17% of intraarticular calcaneal fractures will require a second surgery for symptomatic post-traumatic subtalar arthritis [3]. Sanders found that an increasing degree of comminution correlates with need for arthrodesis, with 73% of Sanders type IV fractures requiring subtalar arthrodesis, compared to 23% of type II fractures [4].



**Fig. 34.1** Nineteen year-old female with isolated subtalar arthritis and calcaneus fracture malunion after intraarticular calcaneus fracture sustained 3 years prior. (a) Lateral view shows subtalar arthritis.

(b) Harris heel view demonstrates a short and widened heel with increased hindfoot valgus. The patient ultimately underwent subtalar arthrodesis (c, d)

## Symptoms

Patients with symptomatic subtalar arthritis often report pain, swelling, and stiffness of the lateral hindfoot that is aggravated when walking on uneven ground. They will localize their pain to the sinus tarsi or areas distal to the malleoli. Symptoms may be accompanied by feelings of instability, catching, or locking. The pain often gets better with rest or by wearing high-top shoes. If significant hindfoot deformity is present, patients may experience difficulty with shoe wear, which can predispose them to skin irritation and breakdown.

Physical examination begins with observation of bare-foot gait and stance. Overall alignment of the limb should

be assessed since proximal abnormalities may alter foot biomechanics. Standing heel position is evaluated for the presence of excessive hindfoot varus or valgus. Swelling and tenderness within the sinus tarsi are highly suggestive of subtalar joint pathology. Passive range of motion of the subtalar joint can be difficult to quantify and should be compared with the uninvolved contralateral side. In the painful arthritic subtalar joint, inversion and eversion of the calcaneus will be decreased and painful. The heel cord and gastrocnemius should be evaluated for tightness, which can be seen in cases of chronic deformity. As always, a detailed neurovascular examination should be performed to determine weakness, loss of sensation and the presence of pulses.

## Diagnostics

Imaging of the subtalar joint should start with weight-bearing anteroposterior (AP), oblique, and lateral views of the foot. Weight-bearing radiographs are essential to assess alignment and the degree of degenerative change in the joint. Additionally, weight-bearing AP, lateral, and mortise views of the ankle should be obtained to assess adjacent tibiotalar alignment and joint space. The Broden view is particularly helpful for evaluating the posterior subtalar facet. It is obtained by internally rotating the leg 45° and aiming the X-ray 10–40° cephalad. In the setting of prior calcaneus fracture, a Harris axial view can be helpful in evaluating deformity from fracture malunion, such as varus or valgus malalignment and widening or shortening of the heel (Fig. 34.1b).

While CT is not necessary to diagnose subtalar joint arthritis and is most often performed under non-weight-bearing conditions, it can still be effective for showing subtle arthritic changes. CT also offers the benefit of providing a three-dimensional image that can specify areas of degenerative change and rule out a bony coalition. MRI is rarely indicated in the evaluation for arthritis, unless one is concerned about tumor, avascular necrosis, or a fibrous coalition.

Diagnostic injection of local anesthetic into the subtalar joint may help localize and/or confirm the diagnosis of isolated subtalar joint arthritis as the patient's source of pain. It can be combined with a corticosteroid to provide short term therapeutic relief.

## Classification

Paley and Hall described a classification scheme for subtalar osteoarthritis using plain radiographs. Grade 0 corresponded with no degenerative changes; Grade 1 corresponds with presence of subchondral sclerosis, osteophytes, and/or cyst formation with no narrowing; Grade 2 corresponds with Grade 1 changes plus narrowing; and Grade 3 corresponds with a complete loss of joint space [5].

## Therapy

### Nonoperative Treatment

Conservative management strategies for subtalar joint arthritis include the use of braces and/or orthotics, non-steroidal anti-inflammatory drugs (NSAIDs), subtalar intraarticular corticosteroid injections, and activity modification. Orthotic devices serve to limit painful joint motion, while accommodating a rigid deformity or helping to correct a flexible deformity. Ankle-foot orthoses (AFOs) provide rigid control to both the ankle and hindfoot. The Arizona brace is a lower profile AFO that tends to be less bulky and more comfortable than the standard solid polypropylene AFO. The University

of California Biomechanics Laboratory (UCBL) orthosis is a device worn within the shoe that is used to control hindfoot motion and correct flexible hindfoot deformity.

NSAIDs are used as first-line pharmacologic treatment for patients in whom they are not contraindicated, such as those with a history of gastric ulcers or kidney disease. Intraarticular corticosteroid injections can provide significant and sometimes prolonged symptom relief. However, there is little consensus guiding their therapeutic use specifically in subtalar joint arthritis [6]. The efficacy of intraarticular viscosupplementation with sodium hyaluronate in cases of subtalar arthritis has been studied. Omer, et al. found significant improvement in patient AOFAS hindfoot scores, reported pain, and walking distance tolerance up to 6 months after a 3 week series of subtalar joint viscosupplementation injections for osteoarthritis [7].

### Operative Treatment

Arthrodesis remains the gold standard for treating painful subtalar joint arthritis that is unresponsive to conservative measures (Fig. 34.1c, d) [8]. The primary goals are to achieve pain relief and restore hindfoot alignment. When arthritis resides solely in the talocalcaneal articulation, isolated subtalar arthrodesis is preferred over a double or triple arthrodesis, given its preservation of hindfoot motion, its decreased potential for development of adjacent joint arthritis, and its relative simplicity [9]. Isolated subtalar arthrodesis has been shown to reduce talonavicular joint motion by 74% and calcaneocuboid motion by 44% [10]. Functionally, this leads to complete loss of inversion/eversion; however, dorsiflexion and plantar flexion of the ankle are maintained, as well as some adduction and abduction through the transverse tarsal joints.

### Approach

Surgical approach varies depending on the presence and extent of associated deformity. When deformity correction is not needed, an in situ arthrodesis is indicated. These have traditionally been performed open, however, arthroscopic approaches have also been described and are being used with increasing popularity. The theoretic advantages of an arthroscopic fusion are a more cosmetic approach and fewer wound complications [11]. In experienced hands, union rates and clinical outcomes appear to be comparable to open fusions [12]. Contraindications to arthroscopic arthrodesis include gross malalignment requiring correction, a previously failed subtalar arthrodesis, significant bone loss, previous fracture, and restricted joint access (relative).

When performed open, in situ arthrodesis is most commonly approached laterally, either through an oblique incision over the sinus tarsi, extending proximally to the level of the peroneal tendons, or through a transverse incision extending from approximately 1 cm inferior to the lateral malleolus towards the base of the fourth metatarsal. The origin of the extensor digitorum brevis and sinus tarsi fat pad are elevated as a distally based flap, thereby granting access to the subtalar joint. Improved

access is achieved with removal of the lateral process of the talus. Regardless of approach, successful fusion requires appropriate preparation of the bony surfaces with removal of all cartilage, restoration of foot alignment, and rigid stabilization.

### Preparation of the Arthrodesis Site

Creating bleeding surfaces of the subtalar joint is an important step in obtaining a solid fusion. Due to the complex shape of the joint, this can be difficult. The use of distraction devices, such as lamina spreaders or a pin distractor, can help with exposure. Once the subtalar joint is visualized, cartilage and subchondral bone are removed from both the talar and calcaneal articular surfaces with a chisel, curette or power burr with irrigation. Drill holes are then created in the denuded surfaces to produce vascular channels that will aid in healing. The surfaces are further decorticated by feathering with an osteotome.

### Supplemental Adjuncts

Bone graft is often used to supplement bone healing, fill bone defects, or help with alignment correction, if needed. Options include autograft, allograft, and bone substitutes. Autograft can be harvested from the iliac crest, tibia, or calcaneus. To avoid donor site morbidity, allograft options can be used instead and exist in cancellous, cortical, or corticocancellous forms. In cases of subtalar distraction arthrodesis, structural allografts have been shown to have comparable results to structural autograft for restoration of hindfoot function and alignment [13]. Bioadjuvants, such as bone stimulators, platelet rich plasma, and demineralized bone matrix, are promising for increasing rates of union, especially in challenging cases, but more studies are needed to elucidate their role in hindfoot surgery.

### Position of Arthrodesis

During arthrodesis, the subtalar joint must be fixed in 5° of valgus alignment. Fusing the subtalar joint in varus will lock the transverse tarsal joint, leading to increased lateral forefoot pressures with weight-bearing. Fusing the joint in excessive valgus can lead to subfibular impingement.

### Fixation

Many methods of fixation for isolated subtalar arthrodesis have been described, including staples, dowels, and variable numbers of lag screws introduced either through the talus or the calcaneus. When comparing single-screw to two-screw fixation of isolated subtalar fusion, no significant differences exist in nonunion rate, postoperative complication incidence, or subsequent surgeries [14], however, biomechanically, the double diverging two-screw configuration has been shown to achieve the most compression and torsional stiffness and the least joint rotation [15]. A finite element analysis showed that one screw in the talar neck and another in the posteromedial talar dome provided the best two-screw configuration [16]. In general, the results of isolated subtalar arthrodesis

have been fairly successful, with rates of union ranging from 86 to 100 % for primary procedures [17–19]. While choice of fixation may vary, it is essential that compression of the bony surfaces occurs to achieve fusion across the subtalar joint. This can be achieved with either compression screw fixation or an external pin compressor with neutral screw fixation.

### Postoperative Care

It is standard to limit weight-bearing until evidence of union following subtalar arthrodesis. Usually 6 weeks of non-weight-bearing followed by 2–6 weeks of protected weight-bearing in a cast or walking boot is prescribed. When radiographic union is appreciated, the patient is weaned out of the walking boot and advanced to full weight-bearing.

### Rehabilitation and Back-to-Sports

Clinical and radiographic evidence of complete healing at the fusion site must be present before returning a patient who has undergone subtalar arthrodesis back to sports. Clinical signs include absence of tenderness at the fusion site. It is not uncommon to obtain a CT scan to confirm complete radiographic fusion.

### Prevention

Upon return to sports, consider providing the patient with a lace-up ankle brace to use when engaged in sports and a UCBL orthotic to use whenever in normal shoes as a means to limit early stress across the fusion site.

### Summary

1. Subtalar joint arthritis often occurs following previous calcaneus or talus fracture, despite primary reduction and fixation.
2. Isolated subtalar joint arthrodesis reduces talonavicular joint motion by 74 % and calcaneocuboid motion by 44 %.
3. Successful subtalar joint arthrodesis requires appropriate preparation of the bony surfaces with removal of all cartilage, restoration of foot alignment, and rigid stabilization.
4. When no hindfoot deformity is present, in situ subtalar arthrodesis can be performed arthroscopically as an alternative to the traditional open approach. In experienced hands, rates of fusion and clinical outcomes are similar to open arthrodesis with the benefit of fewer wound complications.
5. Final hindfoot alignment during subtalar arthrodesis should be 5° of valgus.

## Section II: Chopart Joint DJD

### Anatomy

The Chopart joint refers to the midtarsal or transverse tarsal joint, and consists of the talonavicular and calcaneocuboid articulations. Together with the subtalar joint, the talonavicular and calcaneocuboid joints make up the triple joints in the hindfoot. The transverse tarsal joint lies in a plane perpendicular to the longitudinal arch of the foot and functions in a coupled manner to provide stability and shock-absorption to the foot, depending on the position of the subtalar joint [20]. As the subtalar joint inverts, the transverse tarsal joint locks, stiffening the foot to allow for a rigid lever during toe-off. As the subtalar joint everts, the transverse tarsal joint unlocks, making the foot more supple to allow for shock absorption when the foot is planted.

Of the triple joints in the foot, the talonavicular joint has been shown to have the greatest range of motion, and simulated arthrodesis of this joint essentially eliminates motion of the remaining hindfoot joint complex to approximately 2°. In contrast, arthrodesis of the calcaneocuboid joint has little effect on motion of the subtalar joint and reduces motion of the talonavicular joint by a third [11].

### Etiology and Pathomechanism

Like the subtalar joint, arthritis at the transverse tarsal joint can result from multiple etiologies. This section will focus on isolated posttraumatic arthritis of the transverse tarsal joint. Purely ligamentous injuries, including rupture of the strong plantar ligamentous support of the talonavicular joint, particularly the spring ligament, plantar cubonavicular, and plantar calcaneocuboid ligaments, can result in acute or gradual instability and collapse of the talonavicular joint. Intraarticular fractures of the talar head, navicular, cuboid and anterior calcaneal process also contribute to loss of joint congruence and can eventually lead to disabling arthritic changes (Fig. 34.2).

These “transitional zone” injuries are notorious for being missed or misdiagnosed in up to 41% of cases [21], and consequently often present late with degenerative changes to the joint. This is thought to be due to multiple reasons, including their subtle radiographic findings with seemingly benign presentation, relatively low incidence, and a general lack of familiarity with transverse tarsal joint injuries.

### Symptoms

Patients with isolated transverse tarsal arthritis will present with pain, swelling, and stiffness over the talonavicular and/

or calcaneocuboid joints. These symptoms may be accompanied by feelings of instability, catching, or locking. Aggravating factors include ambulating on uneven ground, with subtle inversion and eversion of the hindfoot.

Physical examination of the foot with transverse tarsal arthritis may demonstrate medial arch collapse in the sagittal plane and/or forefoot abduction deformity in the coronal plane. Painful osteophytic dorsal prominences may cause direct irritation with footwear and/or indirectly cause pain, numbness, and paresthesias in the first webspace, from compression of the deep peroneal nerve in the anterior tarsal tunnel. Hindfoot inversion and eversion will be restricted and produce discomfort.

### Diagnostics

Standard AP, lateral, and oblique weight-bearing views of the foot (Fig. 34.2a–c) will show arthritic changes to the involved joints with varying degrees of joint space narrowing, subchondral sclerosis, and spur formation. Talonavicular joint arthritis is often accompanied by midtarsal collapse, as suggested by an abnormal talo-1st metatarsal angle on the lateral view. Calcaneocuboid arthritis is best visualized on the oblique view.

Advanced imaging with CT scan can be useful in identifying subtle arthritic changes, the presence of residual fracture nonunion, and evidence of tarsal coalition. MRI may be obtained if AVN of the navicular or a fibrous coalition is a concern.

### Classification

Injuries to the transverse tarsal joint were classified by Main and Jowett into five broad categories, based on the mechanism of injury, specifically, the direction of the applied force and resultant displacement [21]. In order of frequency, these five categories of transverse tarsal injuries include those that are caused by forces directed longitudinally, medially, laterally, plantarly, or variably during crush injury. Within each of these categories, severity of injury can include sprains, subluxations, dislocations, fractures, or any combination of the above.

### Therapy

#### Nonoperative Treatment

Conservative management of transverse tarsal joint arthritis includes shoe modification, bracing, and orthotics to control hindfoot motion. NSAIDs and selective intraarticular injections may offer temporary pain relief.



**Fig. 34.2** Thirty-nine year-old male presented with a 2 year history of left midfoot pain after sustaining a navicular fracture when he stepped in a hole while running. (a–c) AP, lateral, and oblique radiographs

demonstrate sclerosis and cystic change of the malunited navicular and isolated talonavicular joint space narrowing. The patient ultimately underwent talonavicular arthrodesis

### Operative Treatment

Arthritis of the transverse tarsal joint is addressed by arthrodesis of the involved joints. When only the talonavicular joint is involved, isolated talonavicular arthrodesis can be performed. As stated earlier, fusion of the talonavicular joint alone results in nearly complete elimination of hindfoot motion, and is biomechanically similar to a triple or double arthrodesis that includes the subtalar joint. Isolated talonavicular joint arthrodesis has a high nonunion rate, likely due to the spheric shape of the joint that can make exposure and preparation of the joint surfaces relatively challenging, as well as the relative avascularity of the navicular bone. This isolated arthrodesis is therefore reserved for the low demand patient (i.e., rheumatoid, elderly). For the remainder of patients, the calcaneocuboid joint or subtalar joint may be

added to the fusion to increase stability of the transverse tarsal joint, without significant further loss of hindfoot motion.

Double arthrodesis, as described by DuVries, is performed in patients who have both talonavicular and calcaneocuboid joint arthritis, but sparing of the subtalar joint [22]. It biomechanically results in the same degree of immobilization as a triple arthrodesis, but without the need to include the subtalar portion, therefore, requiring less surgical time and resulting in less patient morbidity and less chance for nonunion across the subtalar joint [23].

### Approach

Both a lateral and medial incision are used to access the calcaneocuboid and talonavicular joints, respectively. For the lateral approach, a similar incision as that used for subtalar



arthrodesis, except more distal, is used. For the medial approach, a longitudinal incision, starting at the tip of the medial malleolus and carried distally 1 cm past the naviculocuneiform joint is used. Once the joints are exposed, the articular surfaces are prepared similarly to any other arthrodesis procedure.

### Position of Arthrodesis

Close attention must be paid during alignment of the foot during double arthrodesis. Because this fusion essentially locks the hindfoot, the subtalar joint must be placed into 5° of valgus and maintained there while the transverse tarsal joint is positioned into the correct abduction or adduction and into a plantigrade position, using the normal foot as a reference.

### Fixation

Typically, 4.0–5.5 mm screws are used for fixation across the talonavicular joint and calcaneocuboid joint. In patients with poor quality bone, additional fixation with staples or plates can be used.

### Rehabilitation and Back-to-Sports

The patient is kept non-weight-bearing for 6 weeks. Radiographs are obtained at that point, and if satisfactory union is occurring, the patient is allowed to bear weight in a cast. Once satisfactory union is demonstrated on radiographs and clinical exam, the patient is progressed to full weight-bearing. This typically occurs at 10–12 weeks.

### Prevention

A high index of suspicion is needed to accurately diagnose injuries involving the Chopart joint early to avoid the late sequelae of posttraumatic arthritis. As in subtalar joint arthrodesis, upon return to sports, consider providing the patient with an orthotic that will limit stress across the hindfoot, such as a lace-up ankle brace or UCBL orthotic.

### Summary

1. “Transitional zone” injuries across the transverse tarsal joint are notorious for missed or misdiagnosis with resultant development of posttraumatic degenerative change.
2. Of the triple joints in the foot, the talonavicular joint has the greatest range of motion, and arthrodesis of this joint results in nearly complete loss of hindfoot motion. In contrast, arthrodesis of the calcaneocuboid joint has little effect on resultant subtalar motion and reduces motion of the talonavicular joint by a third.

3. Double arthrodesis of the talonavicular and calcaneocuboid joints is performed in isolated transverse tarsal arthritis. It biomechanically results in the same degree of hindfoot immobilization as a triple arthrodesis, but requires less surgical time and results in less patient morbidity since the subtalar joint is spared.
4. When positioning the foot during isolated talonavicular or double arthrodesis of the transverse tarsal joints, the subtalar joint must be maintained in 5° of valgus, since this position will be locked with fusion of the talonavicular joint.

---

## Section III: Lisfranc DJD

### Anatomy

The Lisfranc joint refers to the tarsometatarsal (TMT) joint complex of the midfoot and is formed by the five metatarsal bases and their respective cuneiform or cuboid articulations. This complex provides structural support to the transverse arch of the foot through its intrinsic bony architecture and ligamentous anatomy. In coronal cross section, the trapezoidal shape of the middle three metatarsal bases and their associated cuneiforms produce a stable “Roman” arch configuration that limits plantar displacement. Translation in the frontal plane is limited by the recessed position of the 2nd TMT articulation between the medial and lateral cuneiforms [24]. Many robust ligaments, particularly the interosseous and plantar ligaments, further contribute to the static stability of the transverse arch and TMT complex. Dynamic stability comes from the short plantar muscles in addition to the peroneus longus, tibialis anterior, and tibialis posterior tendons.

The midfoot is anatomically divided into three longitudinal columns: the medial column (composed of the 1st metatarso-cuneiform joint), the middle column (composed of the 2nd and 3rd metatarso-cuneiform joints), and the lateral column (composed of the 4th and 5th metatarso-cuboid joints). The lateral column TMT joints are the most mobile, with approximately 10° sagittal plane motion, allowing for balance and accommodation on uneven ground. In general, motion progressively decreases at the TMT joints as you move medially across the midfoot, with the exception of the 2nd TMT joint, which is the least mobile, with <1° sagittal plane motion [25].

### Etiology and Pathomechanism

While TMT joint arthritis can result from primary and inflammatory processes as well as neuropathic conditions, it frequently develops following previous trauma, particularly in the athlete. The differing etiologies of TMT joint degen-



**Fig. 34.3** Thirty-four year-old female with right Lisfranc injury. Patient was initially treated with cast immobilization, but continued to complain of pain. (a) Note on the AP view, increased diastasis between the right 1–2 intermetatarsal space, compared to the left foot. (b, c)

Oblique and lateral views were unremarkable. (d, e) CT scan showed an avulsion fragment of the Lisfranc ligament off the medial cuneiform, necessitating arthrodesis of metatarsals 1–2 and medial and intermediate cuneiform bones (f–h)

eration result in its bimodal presentation. Patients with post-traumatic degeneration become symptomatic earlier and seek care in their 30–40s, while those with primary osteoarthritis present in their 50–60s [26].

The Lisfranc joint complex is the most commonly injured area of the midfoot, and posttraumatic arthritis is its most common complication. Low energy, indirect trauma accounts for approximately 1/3 of all Lisfranc injuries and are most often sustained during athletic activity, including 4% of American football players per season [27]. The classic

mechanism of injury is an axial load applied to the heel of a plantar flexed foot with MTP joint extension, as seen when a football player is tackled from behind. A less common mechanism includes abduction stress to the forefoot around a fixed hindfoot, as seen in sporting activities that require use of foot straps, such as windsurfing and horse-back riding.

Nearly 20% of Lisfranc injuries are misdiagnosed or missed on initial assessment (Fig. 34.3) [28]. Missed, misdiagnosed, and untreated cases of subtle Lisfranc disruption can result in considerable long-term disability characterized

by painful posttraumatic arthritis. Unfortunately, even when an anatomic reduction is achieved after acute injury, radiographic evidence of degenerative change is reported in up to 100% of patients, although not all cases are associated with functional loss [28]. Factors associated with progression of arthritis after Lisfranc injury include severity of initial injury, concomitant articular damage at the time of injury [29], and additional joint damage from the use of transarticular screws.

After injury, the midfoot may become unstable due to a loss of the bony and ligamentous architecture. This, in turn, alters midfoot loading, which may cause subtle incongruent motion at the TMT joints, collapse of the longitudinal arch with development of planovalgus deformity, and compromise of the foot's ability to function as a rigid lever.

## Symptoms

Patients with TMT joint arthritis complain of deep, aching pain localized to the involved midfoot articulations. This pain may be aggravated by activities that require the midfoot to be rigid, such as push off or heel rise as needed during stair climbing, as well as with walking on unlevel surfaces [30]. Patients may also note flattening of their arch or difficulty with shoe wear. Seventy-eight percent of patients complain of difficulty with shoe wear or unusual foot posture [31].

On physical examination, standing position of the hind-, mid-, and forefoot are assessed for any deformity or malalignment. In cases of longitudinal arch collapse from chronic Lisfranc instability, the foot may appear relatively flattened and abducted. The patient's barefoot gait is then assessed. Patients with painful midfoot arthritis will demonstrate a "stiffening strategy" during walking where the patient will try to reduce the range of motion of the 1st metatarsal [32]. However, with increased activity, the midfoot decompensates due to its lack of stability, allowing for an increase in calcaneal eversion and 1st metatarsal dorsiflexion.

Each TMT joint is palpated and passively stressed in a standardized manner starting medially. Any prominent bossing over the joint with associated skin irritation from shoe wear is noted. Abduction of the forefoot and pronation stress can also help identify the location of maximal pain. A useful provocative maneuver is the piano key test, which is performed by applying a plantar directed force to the metatarsal head. If pain is elicited in the same TMT joint, this indicates a symptomatic joint. Range of motion at each TMT joint is subtle and not clinically relevant. However, excessive motion can be present with excessive pronation or midfoot collapse. Presence of gastrocnemius and/or soleus tightness is often seen in the long-standing collapsed midfoot, and should be noted preoperatively if present.

## Diagnostics

Weight-bearing AP, lateral, and oblique radiographs of the foot are indicated in the work up for TMT joint arthritis. In addition to standard arthritic changes of joint space narrowing, subchondral sclerosis, and osteophyte formation, the AP view may also reveal incongruous 1st and 2nd TMT joints in the setting of prior Lisfranc injury (Fig. 34.3a). Lateral radiographs may show flattening or sagging of the longitudinal arch with a lowered medial cuneiform and abnormal talo-1st metatarsal angle.

Because of the varying obliquity of the joint surfaces and their overlapping appearance, the midfoot joints can be difficult to evaluate with plain radiography alone. A CT scan may aid in determining the specific joints that are arthritic and to what extent.

Selective fluoroscopically-guided diagnostic injections with local anesthetic can also be used to identify the symptomatic TMT joints.

## Classification

Several classification systems for Lisfranc injuries have been developed and modified over the years [26, 33]. They are generally based on the congruency of the TMT joints and the direction of displacement of the metatarsal bases. Although these classifications can help guide treatment, most fall short in predicting clinical outcome. Most recently, Chiodo and Myerson [34] suggested classification of TMT joint injuries based on the three mechanical columns of the foot to aid in treatment. This has provided some prognostic value when differences in physiologic motion at each column is taken into account. Komenda et al. [35] reported that posttraumatic arthritis is more common at the base of the 2nd metatarsal, which is a relatively constrained column with the least amount of physiologic motion among the three columns. The lateral column, which has the greatest amount of sagittal plane motion, is the least likely to be involved in symptomatic arthritis.

The above traditional classification systems fail to address subtle Lisfranc diastasis seen in low-energy athletic injuries, whose long term outcomes can be as debilitating as high energy injuries, if not managed appropriately due to missed or misdiagnosis. In 2002, Nunley and Vertullo [36] recognized the importance of the subtle Lisfranc injury specific to the athlete's midfoot and classified them into three groups based on clinical findings, weight-bearing radiographs, and bone scintigraphy. Athletes with stage I injury have pain only at the Lisfranc complex, negative radiographic findings, and increased uptake on bone scan. Those with stage II injuries have diastasis between the first and second metatarsals of 1–5 mm greater than that of the contralateral side, without

loss of midfoot arch height. Diastasis  $>5$  mm and loss of midfoot arch height represents a stage III injury. In their series, nonsurgical treatment of stage I patients and surgical treatment of stage II and III patients led to an excellent result in 93%.

## Therapy

### Nonoperative Treatment

The goal for conservative treatment of midfoot arthritis is to balance the need for midfoot stability and midfoot function. Shoe modification and/or orthoses should be prescribed to alter loading of the TMT joints [37]. Shoes may be modified by switching to rocker-bottom or stiff-soled shoes. Rocker-bottom soles help to facilitate the transfer of load from the hindfoot to the forefoot, bypassing the TMT joints. Stiff-soled shoes attempt to mimic the “stiffening strategy” that a patient with midfoot arthritis develops with normal gait. Stiff carbon fiber, full-length inserts can be used in place of a stiff-soled shoe and are easily transferable between shoes; however, they can be uncomfortable in the shoe. Further motion-limiting orthoses may be used, such as the ankle foot clamshell orthosis coupled with a rocker-bottom sole. However, this increased stability comes at the cost of function.

The first line pharmacologic management of midfoot arthritis is with NSAIDs, when not contraindicated. As with the hindfoot joints, injectable therapy can provide temporary relief and includes corticosteroid and hyaluronic acid. There are few studies that rigorously look at the efficacy of these treatments. However, Drakonaki et al. [38] studied 59 patients with ultrasound-guided steroid injections into the midfoot and found that most patients experienced partial pain relief for up to 3 months.

### Operative Treatment

Patients with persistent pain and functional decline, with or without deformity, who have failed a dedicated course of conservative management are candidates for operative management (Fig. 34.3f–h). The least invasive option involves excision of symptomatic osteophytes, however, in many patients this may not be enough to satisfactorily improve their symptoms. The mainstay definitive treatment of TMT joint arthritis is arthrodesis and can be conceptually separated into treatment for medial and middle column arthritis and treatment for lateral column arthritis.

Pre-operatively, the extent of arthrosis and therefore, which joints should be included in the fusion mass must be determined. This is done via clinical and radiographic exams. If needed, differential intra-articular anesthetic injections into specific midfoot joints can be performed, however, recent cadaver studies have challenged the selectivity of this

tool, showing that the anesthetic leaks into adjacent joints in up to 20% of cases. If there is any doubt, intraoperative assessment of the condition and stability of the individual joints is definitive. The senior author (JKD) always confirms the intended joints for arthrodesis with a CT scan obtained perpendicular and parallel to the midfoot joints. This latter request is done so the surgeon does not have to deal with an infinite number of oblique slices on CT which are sometimes difficult to read.

### Approach

The surgical approach for midfoot fusion varies, depending on which joints are being fused. In general, longitudinal incisions are used and should be long enough to access the joints of interest without placing too much traction on the skin edges. If multiple incisions are used, one should ensure an adequate skin bridge is kept between the incisions.

### Position of Arthrodesis

When arthrodesing the TMT joints, the goal is to fuse the foot in a plantigrade position that matches the opposite foot, if it is normal. This requires careful evaluation of the weight-bearing posture of both feet preoperatively. Most commonly, the associated deformity seen in midfoot arthritis is pes planus and abduction of the forefoot. If deformity is present, concomitant realignment of the foot during arthrodesis is essential for satisfactory result. If a tight heel cord is present after the midfoot is reduced intraoperatively, it should also be addressed, not only to increase dorsiflexion, but also to help reduce forces across the fusion site.

### Medial and Middle Column Arthrodesis

Fusion of the medial and middle columns involves fixation of the 1st, 2nd, and 3rd TMT joints. The intercuneiform and naviculocuneiform joints may need to be included in the fusion based on preoperative and intraoperative assessment as discussed earlier.

Although multiple fixation constructs have been described, including screws alone, and combinations of screws and plates, which themselves can be compression or locking and placed dorsally, plantarly, or medially, no one construct has proven to be the gold standard. Limited evidence suggests that plate fixation may provide increased mechanical stability compared with lag screw fixation alone [39, 40]. The efficacy of bone graft and biologic agents to augment fusion potential is not fully known and has not been widely studied [41]. The senior author (JKD), however, uses allograft or autograft in all patients. The amount of bone required is small and the risk of a nonunion is too great to not use bone graft. Furthermore, when multiple TMT joint are fused, a combination of dorsal plating with medial to lateral cross screw configuration is felt to offer the greatest strength of fixation.

Regardless of fixation and use of augments, the best predictor of good outcomes in midfoot arthrodesis for posttraumatic arthritis is the quality of anatomic reduction of the TMT joint complex [42]. Outcomes studies generally agree that arthrodesis of the medial and middle joints improves stability and decreases pain but does not eliminate pain or normalize function. Fusion rates range from 83 to 97%, with older patients having the highest risk for nonunion [35]. Long-term complications are often related to loss of midfoot flexibility and/or malunion, and can include metatarsal stress fractures, metatarsalgia, sesamoid pain, and adjacent joint arthritis.

### Lateral Column Arthrodesis

Management strategies for lateral column arthritis are varied and controversial. Due to the relative increased mobility of the lateral column and function in accommodation on uneven surfaces, most agree that arthrodesis of the lateral column should be avoided at all costs. Komenda et al. argue that fusion may result in increased rates of metatarsal stress fractures [35], chronic lateral foot pain, and increased rates of nonunion. However, Raikin and Schon argue that lateral column arthrodesis may benefit patients with lateral column collapse, rocker bottom deformity, and severe pain [43].

In light of the controversy surrounding the importance of maintaining lateral column motion, two attractive motion-sparing procedures to treat lateral column arthritis have been described. Lateral TMT joint resection with peroneus tertius soft-tissue interposition and ceramic interpositional arthroplasty have both been used and reported. Available studies indicate that both procedures result in significantly decreased pain [44, 45], although each may have its own unique complications.

### Rehabilitation and Back-to-Sports

The patient is kept non-weight-bearing for 6 weeks. Radiographs are obtained at that point and if satisfactory union is occurring, the patient is allowed to bear weight in a cast. Once satisfactory union is demonstrated on radiographs and clinical exam, the patient is progressed to full weight-bearing. This typically occurs at 10–12 weeks.

### Prevention

A high index of suspicion is needed to accurately diagnose subtle Lisfranc joint injuries early to avoid the late sequelae of posttraumatic arthritis. Upon return to sports, stress across the midfoot fusion site can be limited with a stiff sole orthotic or shoe modification with a rocker bottom.

### Summary

1. The Lisfranc joint complex is the most commonly injured area of the midfoot, and posttraumatic arthritis is its most common complication.
2. Delayed or missed diagnosis of subtle Lisfranc injuries predisposes the foot to chronic instability, deformity, and pain from posttraumatic arthritis.
3. The 2nd TMT joint is the least mobile of the midfoot joints and most prone to development of symptomatic arthritis. The 4th and 5th TMT joints of the lateral column are the most mobile, allowing for balance and accommodation on uneven ground, and therefore fusion of these joints should be avoided.
4. Careful preoperative planning for TMT joint arthrodesis includes determination of the pathologic joints, presence of deformity, and presence of heel cord contracture.
5. Multiple fixation constructs have been described for TMT joint arthrodesis, but no gold standard exists.

### References

1. Czyzewski DI, Eakin MN, Lane MM, Jarrett M, Shulman RJ. Recurrent abdominal pain in primary and tertiary care: differences and similarities. *Child Health Care J Assoc Care Child Health*. 2007;36(2):137–53.
2. Canale ST, Kelly Jr FB. Fractures of the neck of the talus. Long-term evaluation of seventy-one cases. *J Bone Joint Surg Am*. 1978;60(2):143–56.
3. Bezes H, Massart P, Delvaux D, Fourquet JP, Tazi F. The operative treatment of intraarticular calcaneal fractures. Indications, technique, and results in 257 cases. *Clin Orthop Relat Res*. 1993(290):55–9.
4. Sanders R, Fortin P, DiPasquale T, Walling A. Operative treatment in 120 displaced intraarticular calcaneal fractures. Results using a prognostic computed tomography scan classification. *Clin Orthop Relat Res*. 1993;290:87–95.
5. Paley D, Hall H. Intra-articular fractures of the calcaneus. A critical analysis of results and prognostic factors. *J Bone Joint Surg Am*. 1993;75(3):342–54.
6. Peterson C, Hodler J. Evidence-based radiology (part 2): is there sufficient research to support the use of therapeutic injections into the peripheral joints? *Skeletal Radiol*. 2010;39(1):11–8.
7. Mei-Dan O, Carmont M, Laver L, Mann G, Maffulli N, Nyska M. Intra-articular injections of hyaluronic acid in osteoarthritis of the subtalar joint: a pilot study. *J Foot Ankle Surg Off Publ Am Coll Foot Ankle Surg*. 2013;52(2):172–6.
8. Flemister A. Hindfoot. Osteoarthritis and fusion. Orthopaedic knowledge update 4: foot and ankle. 15. Rosemont, Illinois: American Academy of Orthopaedic Surgeons; 2008. p. 195–200.
9. Mann RA, Baumgarten M. Subtalar fusion for isolated subtalar disorders. Preliminary report. *Clin Orthop Relat Res*. 1988;226:260–5.
10. Astion DJ, Deland JT, Otis JC, Kenneally S. Motion of the hindfoot after simulated arthrodesis. *J Bone Joint Surg Am*. 1997;79(2):241–6.
11. Muraro GM, Carvajal PF. Arthroscopic arthrodesis of subtalar joint. *Foot Ankle Clin*. 2011;16(1):83–90.

12. Scranton Jr PE. Comparison of open isolated subtalar arthrodesis with autogenous bone graft versus outpatient arthroscopic subtalar arthrodesis using injectable bone morphogenic protein-enhanced graft. *Foot Ankle Int.* 1999;20(3):162–5.
13. Garras DN, Santangelo JR, Wang DW, Easley ME. Subtalar distraction arthrodesis using interpositional frozen structural allograft. *Foot Ankle Int.* 2008;29(6):561–7.
14. DeCarbo WT, Berlet GC, Hyer CF, Smith WB. Single-screw fixation for subtalar joint fusion does not increase nonunion rate. *Foot Ankle Spec.* 2010;3(4):164–6.
15. Chuckpaiwong B, Easley ME, Glisson RR. Screw placement in subtalar arthrodesis: a biomechanical study. *Foot Ankle Int.* 2009;30(2):133–41.
16. Lee JY, Lee YS. Optimal double screw configuration for subtalar arthrodesis: a finite element analysis. *Knee Surg Sports Traumatol Arthrosc Off J ESSKA.* 2011;19(5):842–9.
17. Easley ME, Trnka HJ, Schon LC, Myerson MS. Isolated subtalar arthrodesis. *J Bone Joint Surg Am.* 2000;82(5):613–24.
18. Haskell A, Pfeiff C, Mann R. Subtalar joint arthrodesis using a single lag screw. *Foot Ankle Int.* 2004;25(11):774–7.
19. Tiemann A, David A, Jakob M, Muhr G. [Corrective arthrodesis in isolated post-traumatic malalignment of the subtalar joint]. *Chirurg Z Alle Geb Oper Med.* 1998;69(8):866–71.
20. Sammarco VJ. The talonavicular and calcaneocuboid joints: anatomy, biomechanics, and clinical management of the transverse tarsal joint. *Foot Ankle Clin.* 2004;9(1):127–45.
21. Main BJ, Jowett RL. Injuries of the midtarsal joint. *J Bone Joint Surg.* 1975;57(1):89–97.
22. DuVries HL. *Surgery of the foot.* St. Louis: Mosby; 1959. p. 300 p.
23. Thelen S, Rutt J, Wild M, Logters T, Windolf J, Koebke J. The influence of talonavicular versus double arthrodesis on load dependent motion of the midtarsal joint. *Arch Orthop Trauma Surg.* 2010;130(1):47–53.
24. Peicha G, Labovitz J, Seibert FJ, Grechenig W, Weiglein A, Preidler KW, et al. The anatomy of the joint as a risk factor for Lisfranc dislocation and fracture-dislocation. An anatomical and radiological case control study. *J Bone Joint Surg.* 2002;84(7):981–5.
25. Ouzounian TJ, Shereff MJ. In vitro determination of midfoot motion. *Foot Ankle.* 1989;10(3):140–6.
26. Myerson MS, Fisher RT, Burgess AR, Kenzora JE. Fracture dislocations of the tarsometatarsal joints: end results correlated with pathology and treatment. *Foot Ankle.* 1986;6(5):225–42.
27. Meyer SA, Callaghan JJ, Albright JP, Crowley ET, Powell JW. Midfoot sprains in collegiate football players. *Am J Sports Med.* 1994;22(3):392–401.
28. Goossens M, De Stoop N. Lisfranc's fracture-dislocations: etiology, radiology, and results of treatment. A review of 20 cases. *Clin Orthop Relat Res.* 1983(176):154–62.
29. Arntz CT, Veith RG, Hansen Jr ST. Fractures and fracture-dislocations of the tarsometatarsal joint. *J Bone Joint Surg Am.* 1988;70(2):173–81.
30. Patel A, Rao S, Nawoczenski D, Flemister AS, DiGiovanni B, Baumhauer JF. Midfoot arthritis. *J Am Acad Orthop Surg.* 2010;18(7):417–25.
31. Mann RA, Prieskorn D, Sobel M. Mid-tarsal and tarsometatarsal arthrodesis for primary degenerative osteoarthritis or osteoarthritis after trauma. *J Bone Joint Surg Am.* 1996;78(9):1376–85.
32. Rao S, Baumhauer JF, Tome J, Nawoczenski DA. Comparison of in vivo segmental foot motion during walking and step descent in patients with midfoot arthritis and matched asymptomatic control subjects. *J Biomech.* 2009;42(8):1054–60.
33. Hardcastle PH, Reschauer R, Kutscha-Lissberg E, Schoffmann W. Injuries to the tarsometatarsal joint. Incidence, classification and treatment. *J Bone Joint Surg.* 1982;64(3):349–56.
34. Chiodo CP, Myerson MS. Developments and advances in the diagnosis and treatment of injuries to the tarsometatarsal joint. *Orthop Clin North Am.* 2001;32(1):11–20.
35. Komenda GA, Myerson MS, Biddinger KR. Results of arthrodesis of the tarsometatarsal joints after traumatic injury. *J Bone Joint Surg Am.* 1996;78(11):1665–76.
36. Nunley JA, Vertullo CJ. Classification, investigation, and management of midfoot sprains: Lisfranc injuries in the athlete. *Am J Sports Med.* 2002;30(6):871–8.
37. Rao S, Baumhauer JF, Becica L, Nawoczenski DA. Shoe inserts alter plantar loading and function in patients with midfoot arthritis. *J Orthop Sports Phys Ther.* 2009;39(7):522–31.
38. Drakonaki EE, Kho JS, Sharp RJ, Ostlere SJ. Efficacy of ultrasound-guided steroid injections for pain management of midfoot joint degenerative disease. *Skeletal Radiol.* 2011;40(8):1001–6.
39. Suh JS, Amendola A, Lee KB, Wasserman L, Saltzman CL. Dorsal modified calcaneal plate for extensive midfoot arthrodesis. *Foot Ankle Int.* 2005;26(7):503–9.
40. Marks RM, Parks BG, Schon LC. Midfoot fusion technique for neuroarthropathic feet: biomechanical analysis and rationale. *Foot Ankle Int.* 1998;19(8):507–10.
41. Bibbo C, Anderson RB, Davis WH. Complications of midfoot and hindfoot arthrodesis. *Clin Orthop Relat Res.* 2001;391:45–58.
42. Sangeorzan BJ, Veith RG, Hansen Jr ST. Salvage of Lisfranc's tarsometatarsal joint by arthrodesis. *Foot Ankle.* 1990;10(4):193–200.
43. Raikin SM, Schon LC. Arthrodesis of the fourth and fifth tarsometatarsal joints of the midfoot. *Foot Ankle Int.* 2003;24(8):584–90.
44. Berlet GC, Hodges Davis W, Anderson RB. Tendon arthroplasty for basal fourth and fifth metatarsal arthritis. *Foot Ankle Int.* 2002;23(5):440–6.
45. Shawen SB, Anderson RB, Cohen BE, Hammit MD, Davis WH. Spherical ceramic interpositional arthroplasty for basal fourth and fifth metatarsal arthritis. *Foot Ankle Int.* 2007;28(8):896–901.

Tetsuro Yasui

**Abstract**

When an adolescent athlete presents with pain in the foot and ankle region, the diagnosis of tarsal coalition should be considered. Tarsal coalition is a congenital anomaly characterised by bony, cartilaginous or fibrous union of the bones in the hindfoot and midfoot. Most such coalitions are talocalcaneal or calcaneonavicular. Pain, peroneal spasm, rigid flatfoot, and a bony prominence inferior to the medial malleolus are typical signs and symptoms of such a coalition. The diagnosis can usually be made by plain X-ray. MRI, CT, and SPECT-CT are additional useful diagnostic modalities. In talocalcaneal coalition, the C sign and talar beaking are frequently visible in a lateral radiograph. Oblique radiographs of the foot are useful for detecting calcaneonavicular coalition. If conservative treatment such as reduced activity and cast immobilization is ineffective, excision of the coalition should be considered.

**Keywords**

Tarsal coalition • Adolescent athlete • Peroneal spasm • Flatfoot

**Etiology and Pathomechanism**

Tarsal coalition is a congenital anomaly characterised by bony, cartilaginous or fibrous union of the bones in the hindfoot and midfoot [1]. Symptomatic coalition is uncommon, the reported occurrence rate being 1–2%; however, asymptomatic coalition is reportedly not as rare [2, 3]. In their MRI-based study, Nalaboff et al. reported that 12% of the population have coalition [4]. In another study of 100 cadavers, Solomon et al. reported the incidence of tarsal coalition was 12.72% [5].

Due to the rigidity of the hindfoot and therefore biomechanically hyper-stress of the ankle joint, individuals with coalitions are thought to sustain ankle injuries more frequently than those without them. In support of this contention, Snyder et al. reviewed the X-ray films of 89 patients who had sustained ankle sprains during sports activity and

reported that 58 of these subjects (65%) had roentgenographic evidence of calcaneonavicular coalition [6].

The accepted likely cause of tarsal coalition is a failure of primitive mesenchyme to segment and produce a normal joint complex in the embryonic period [1]. An autosomal dominant pattern of inheritance has been reported [7].

Approximately 90% of symptomatic coalitions are talocalcaneal or calcaneonavicular, followed by the rare forms of talonavicular, calcaneocuboid, and naviculocuneiform [8, 9]. Coalitions usually become symptomatic between the ages of 10 and 15 years [10].

Although rare, stress fractures of the anterior process of the calcaneus in athletes with calcaneonavicular coalition have been reported [11, 12].

**Symptoms**

The major symptoms are rigidity and pain. The pain occurs at the coalition site, typically being slightly inferior to the tip of medial malleolus in talocalcaneal coalition and in the

T. Yasui, MD, PhD (✉)

Department of Orthopaedic Surgery, The University of Tokyo Hospital, 7-3-1 Hongo Bunkyo-ku, Tokyo 113-8655, Japan  
e-mail: [yasuit-ort@h.u-tokyo.ac.jp](mailto:yasuit-ort@h.u-tokyo.ac.jp)

anterior portion of the sinus tarsi in calcaneonavicular coalition. The pain is caused by restricted mobility of the affected bones and articulations [13].

Adolescent athletes sometimes present with vague pain around the hindfoot or frequent ankle sprains. When adolescent athletes present with hindfoot or midfoot pain, coalition should always be considered.

Rigid flatfoot with spasm of the peroneal tendons is a typical symptom and should suggest the diagnosis of tarsal coalition. Prominent peroneal tendons in the lateral aspect of lower leg are apparent when these tendons are in spasticity. In addition, when an examiner attempts to invert the hindfoot, the patient experiences pain extending upward along the peroneal tendons. Although the mechanisms of peroneal spasms in patients with coalition are unclear, such spasm sometimes leads to peroneal spastic flatfoot. Far less commonly, patients develop tibialis spastic varus foot rather than peroneal spastic flatfoot [14].

In subjects with talocalcaneal coalition, a bony prominence inferior to medial malleolus is detectable either by palpation or visual examination. This prominence is due to a talocalcaneal bar and sometimes causes tarsal tunnel syndrome [10].

## Diagnosics, Classification

After history taking and physical examination, a diagnosis of tarsal coalition is usually achieved by X-ray and advanced diagnostics as CT scan, MRI, or SPECT-CT.

In talocalcaneal coalition, a C-shaped line created by the medial outline of the talar dome and the sustentaculum tali (“C sign”) is visible in lateral ankle radiographs (Fig. 35.1) [15]. Anterior talar beaking, a secondary manifestation of lack of subtalar motion, is sometimes observable. Axial views of the calcaneus or anteroposterior views of the ankle joint sometimes show the site of talocalcaneal coalition (Fig. 35.2).

In calcaneonavicular coalition, lateral oblique views of the foot are useful for detecting the characteristic bar (Fig. 35.3). Although the calcaneus does not border the navicular in subjects with normal anatomy, in a typical calcaneonavicular coalition there is an irregular bony border between the anterior process of the calcaneus and the lateral side of the navicular. Lateral views show a long anterior process of the talus; this is sometimes described as the “anteater nose sign” (Fig. 35.4) [16].

CT scans reveal the location and extent of coalition more precisely than X-ray films (Figs. 35.5 and 35.6) [17, 18].

Because most coalitions are asymptomatic, clinicians should assess whether detected tarsal coalitions are the cause of the presenting pain. Tenderness at the coalition site, bone edema and/or reactive inflammatory changes on MRI scans and uptake on SPECT-CT are helpful in ascertaining this



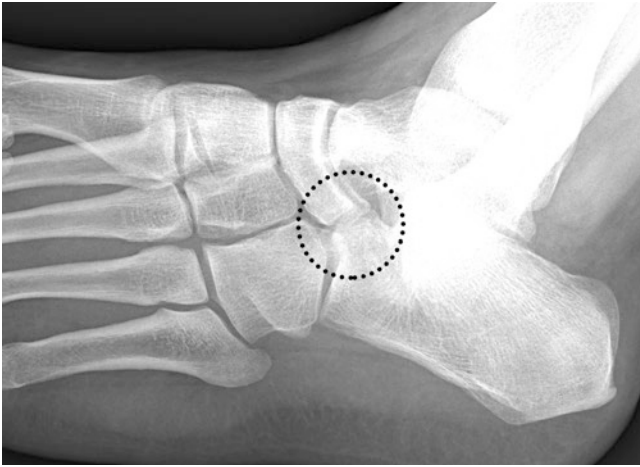
**Fig. 35.1** Lateral ankle radiograph of a patient with a talocalcaneal coalition. The *arrow heads* indicate the C sign



**Fig. 35.2** Anteroposterior radiograph of the ankle of a patient with a talocalcaneal coalition. The *arrowhead* indicates the coalition

(Fig. 35.7) [19]. Local anaesthetic injections at the coalition site can also help in assessing whether the coalition is symptomatic or not.





**Fig. 35.3** Oblique radiograph of the foot of a patient with a calcaneonavicular coalition. The *dotted circle* indicates the coalition



**Fig. 35.4** Lateral radiograph of the foot of a patient with a calcaneonavicular coalition. The *arrowheads* indicate the anteater nose sign

There is no well-accepted classification system; however, Kumar classified talocalcaneal coalition into the following three types based on CT findings: type 1, an osseous bridging of the middle facet joint; type 2, a cartilaginous coalition in which marked narrowing of the middle facet joint is visible; and type 3, a fibrous coalition in which only slight narrowing of the facet is apparent [20]. More recently, Rozansky et al. proposed more detailed classification system [21].

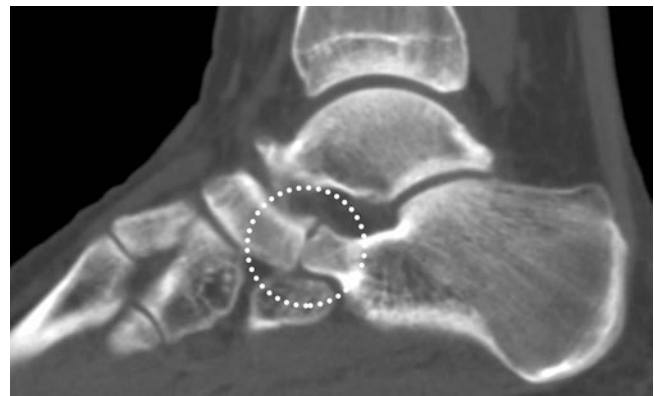
## Therapy

Conservative treatment comprises reduced activity or cast immobilization. A 6-week short-leg walking cast is sometimes very effective [22]. Appropriate insoles and lace-up ankle braces or both are useful for assisting athletes to return to sports activity.

Surgical treatment should be considered when the results of conservative treatment are not satisfactory. The two major surgical options are excision of the coalition and arthrodesis.

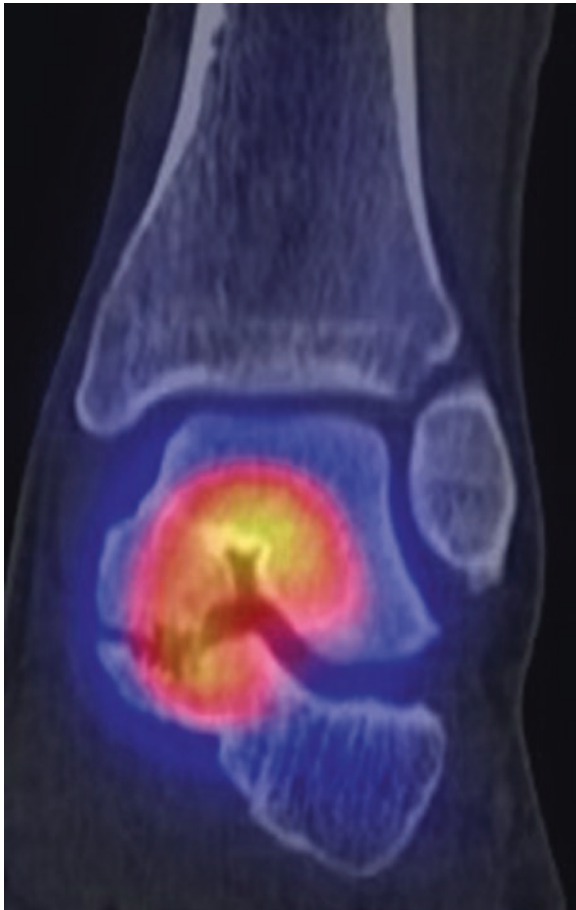


**Fig. 35.5** CT image of a patient with a talocalcaneal coalition. The *arrowhead* indicates the coalition



**Fig. 35.6** CT image of a patient with a calcaneonavicular coalition. The *dotted circle* indicates the coalition.

For talocalcaneal coalitions, strong indications for excision of the coalition are minimal or no degenerative change in the talocalcaneal joint and the coalition involving  $\leq 50\%$  of the posterior talocalcaneal joint (Fig. 35.8) [23, 24]. Wilde et al. reviewed 20 feet with talocalcaneal coalitions and reported that fair or poor results were observed in the feet in which preoperative CT had shown the area of relative coalition to be greater than 50%; all such feet had heel valgus of  $>16^\circ$  [24]. On the other hand, the presence of talar beaking, which is believed to be a secondary manifestation of impairment of subtalar mobility, does not contraindicate resection of a tarsal coalition [25].



**Fig. 35.7** SPECT-CT image of a patient with a talocalcaneal coalition

Surgeons should study the CT images carefully preoperatively to ensure that they excise the entire coalition from the medial side. Care should be taken not to damage the plantar nerves. When a coalition has been adequately excised, the talocalcaneal joint becomes mobile. Coalitions can recur if excision has been incomplete; conversely, excessive resection of bone can lead to instability and increased risk of fracture. Recurrence of coalition can be avoided by pasting bone wax and interposing fat tissue in the excision site or both. In cases of severe preoperative pes planovalgus et abductus an additional lateral calcaneal lengthening osteotomy or other osteotomy might be necessary to avoid increase of the flat-foot deformity and therefore pain after coalition resection.

When more than 50% of the posterior facet is involved in a coalition or definite degenerative change is observed, the recommended surgical procedure is subtalar or triple arthrodesis.

For calcaneonavicular coalition, the standard procedure is excision of the coalition and interposition of the extensor digitorum brevis muscle (Fig. 35.9). Surgeons should be aware that the coalition site is usually about 2 cm deep and that the depth is easily underestimated preoperatively. Incomplete excision of bones leads to recurrence of coalition.



**Fig. 35.8** CT image of a patient with a talocalcaneal coalition after excision of the coalition. Same patient as shown in Fig. 35.5



**Fig. 35.9** Oblique radiograph of a patient with a calcaneonavicular coalition after excision of the coalition. Same patient as shown in Fig. 35.3

In patients with osteoarthritic changes in adjacent joints, triple arthrodesis should be considered although it may by nature limit athletic performance.

When appropriate surgery is performed on carefully selected patients, the clinical results of excision of talocalcaneal or calcaneonavicular coalitions are favourable even in athletic subjects. Morgan et al. reported that seven of eight adolescent athletes with symptomatic tarsal coalitions were able to return

to competitive athletics after surgical resection [13]. O'Neil et al. reviewed 20 feet of athletes treated surgically and reported achieving excellent or good objective results in 19 of 20 feet and satisfactory subjective results in 17 of 20 [25].

There are no published recommendations for managing stress fractures around coalition sites. Pearce et al. reported treating a case of stress fracture of the anterior process of the calcaneus in a professional rugby player with calcaneonavicular coalition by screw fixation [11]. Nilsson et al. reported conservative treatment of another case of stress fracture of the anterior process caused by training for a marathon [12].

## Rehabilitation and Back-to-Sports

Postoperatively, walker or a split cast support for 2–4 weeks followed by physical therapy to maintain joint motion is recommended [10, 13, 25]. Once the swelling and pain have decreased, the patient is allowed to return to participating in sport. Use of arch supports/insoles is sometimes helpful in preventing increasing of flatfoot deformity postoperatively and facilitating return to sport.

## Prevention

Because tarsal coalition is a congenital anomaly, prevention is not possible.

## Evidence

No level I/II studies regarding tarsal coalitions have been published. However, there are several level III retrospective studies. Takakura et al. reported relevant clinical variables and outcomes of 67 feet of 42 patients with symptomatic talocalcaneal coalitions [10]. Khoshbin et al. reported the long-term functional outcomes after resection of tarsal coalitions in 32 cases [8]. Luhmann et al. reported the results of excision of talocalcaneal coalitions in 25 feet and discussed the indications for surgery [23]. As for athletes, O'Neill and Micheli retrospectively reviewed 20 feet of adolescent athletes and reported the objective and subjective outcomes [25] and Morgan et al. studied 12 adolescent athletes with tarsal coalitions [13].

## Summary

1. When an adolescent athlete presents with pain in the foot/ankle region, coalition should be considered as a possible diagnosis.

2. On physical examination, the typical signs of coalition are peroneal spasm, rigid flatfoot, and a bony prominence inferior to the medial malleolus.
3. Plain X-ray examination is important in screening for tarsal coalition. The C sign and talar beaking in a lateral radiograph are frequently observed in talocalcaneal coalition, whereas an oblique radiograph of the foot is useful for detecting calcaneonavicular coalition.
4. CT, MRI, and SPECT-CT scans are useful for accurate and precise assessment of symptomatic tarsal coalitions.
5. When surgical treatment is indicated, excision of the coalition is the first choice. Care must be taken to excise the necessary amount of bone.

## References

1. Harris BJ. Anomalous structures in the developing human foot. *Anat Rec.* 1955;121(2):399.
2. Harris RI, Beath T. Etiology of peroneal spastic flat foot. *J Bone Joint Surg.* 1948;30B(4):624–34.
3. Kulik Jr SA, Clanton TO. Tarsal coalition. *Foot Ankle Int.* 1996;17(5):286–96.
4. Nalaboff KM, Schweitzer ME. MRI of tarsal coalition: frequency, distribution, and innovative signs. *Bull NYU Hosp Jt Dis.* 2008;66(1):14–21.
5. Solomon LB, Ruhli FJ, Taylor J, Ferris L, Pope R, Henneberg M. A dissection and computer tomograph study of tarsal coalitions in 100 cadaver feet. *J Orthop Res Off Pub Orthop Res Soc.* 2003;21(2):352–8.
6. Snyder RB, Lipscomb AB, Johnston RK. The relationship of tarsal coalitions to ankle sprains in athletes. *Am J Sports Med.* 1981;9(5):313–7.
7. Leonard MA. The inheritance of tarsal coalition and its relationship to spastic flat foot. *J Bone Joint Surg.* 1974;56B(3):520–6.
8. Khoshbin A, Law PW, Caspi L, Wright JG. Long-term functional outcomes of resected tarsal coalitions. *Foot Ankle Int.* 2013;34(10):1370–5.
9. Stormont DM, Peterson HA. The relative incidence of tarsal coalition. *Clin Orthop Relat Res.* 1983;181:28–36.
10. Takakura Y, Sugimoto K, Tanaka Y, Tamai S. Symptomatic talocalcaneal coalition. Its clinical significance and treatment. *Clini Orthop Relat Res.* 1991;269:249–56.
11. Pearce CJ, Zaw H, Calder JD. Stress fracture of the anterior process of the calcaneus associated with a calcaneonavicular coalition: a case report. *Foot Ankle Int.* 2011;32(1):85–8.
12. Nilsson LJ, Coetzee JC. Stress fracture in the presence of a calcaneonavicular coalition: a case report. *Foot Ankle Int.* 2006;27(5):373–4.
13. Morgan Jr RC, Crawford AH. Surgical management of tarsal coalition in adolescent athletes. *Foot Ankle.* 1986;7(3):183–93.
14. Simmons EH. Tibialis spastic varus foot with tarsal coalition. *J Bone Joint Surg.* 1965;47:533–6.
15. Sakellariou A, Sallomi D, Janzen DL, Munk PL, Claridge RJ, Kiri VA. Talocalcaneal coalition. Diagnosis with the C-sign on lateral radiographs of the ankle. *J Bone Joint Surg.* 2000;82(4):574–8.
16. Oestreich AE, Mize WA, Crawford AH, Morgan Jr RC. The “ant-eater nose”: a direct sign of calcaneonavicular coalition on the lateral radiograph. *J Pediatr Orthop.* 1987;7(6):709–11.

17. Masciocchi C, D'Archivio C, Barile A, Fascetti E, Zobel BB, Gallucci M, et al. Talocalcaneal coalition: computed tomography and magnetic resonance imaging diagnosis. *Eur J Radiol.* 1992;15(1):22–5.
18. Wechsler RJ, Schweitzer ME, Deely DM, Horn BD, Pizzutillo PD. Tarsal coalition: depiction and characterization with CT and MR imaging. *Radiology.* 1994;193(2):447–52.
19. Scharf S. SPECT/CT imaging in general orthopedic practice. *Semin Nucl Med.* 2009;39(5):293–307.
20. Kumar SJ, Guille JT, Lee MS, Couto JC. Osseous and non-osseous coalition of the middle facet of the talocalcaneal joint. *J Bone Joint Surg Am.* 1992;74(4):529–35.
21. Rozansky A, Varley E, Moor M, Wenger DR, Mubarak SJ. A radiologic classification of talocalcaneal coalitions based on 3D reconstruction. *J Child Orthop.* 2010;4(2):129–35. Pubmed Central PMCID: 2832879.
22. Bohne WH. Tarsal coalition. *Curr Opin Pediatr.* 2001;13(1):29–35.
23. Luhmann SJ, Schoenecker PL. Symptomatic talocalcaneal coalition resection: indications and results. *J Pediatr Orthop.* 1998;18(6):748–54.
24. Wilde PH, Torode IP, Dickens DR, Cole WG. Resection for symptomatic talocalcaneal coalition. *J Bone Joint Surg.* 1994;76(5):797–801.
25. O'Neill DB, Micheli LJ. Tarsal coalition. A followup of adolescent athletes. *Am J Sports Med.* 1989;17(4):544–9.

Arno Frigg

## Abstract

Hallux valgus and hallux rigidus are the two most frequent pathologies around the MTP-1-joint. Hallux valgus is more common in the general population, and hallux rigidus is more common among athletes. Stiffness of the MTP-1-joint is one of the most dreaded complications after hallux surgery. In the case of recreational sports, hallux valgus and rigidus can be treated in the same way as in the general population. Professional athletes require a different treatment. Surgery should be delayed as long as possible because there is a high risk of potentially career-ending scarring of the joint. For hallux valgus in professional athletes, the aim is to convert a decompensated bunion into a compensated one. This can usually be achieved with a simple chevron osteotomy, which has a low risk of scarring and complications. More extensive surgery should be delayed until the end of the competitive career. In the case of hallux rigidus, a simple cheilectomie, possibly with an additional closing wedge osteotomy of the proximal phalanx and/or shorting of the distal first metatarsal, is sufficient. More extensive surgery, again, ought to be delayed until the end of the competitive career. To decrease the risk of scarring, minimal invasive surgical techniques should be used more frequently for treatment of hallux valgus and rigidus.

## Keywords

Hallux valgus • Hallux rigidus • OCD • MTP-1-joint • Sports • Treatment • Minimal invasive surgery

## Etiology and Pathomechanism

The first ray is the most important weight-bearing part of the forefoot. During the stance phase, the hallux bears 40–60% of the body weight, twice as much as the lesser toes [1]. During walking, forces on the hallux increase to 2–3 times the body weight, and during running to up to 8 times the body weight [1]. It is estimated that the foot of an average person absorbs 64 t of weight while walking one mile, and 110 t while running

one mile [2]. At toe off, the center of pressure is located under the hallux. Studies in runners showed that most of the plantar pressure is located in the distal-most 20–40% of the shoe, indicating that most time is spent on the forefoot [2]. Therefore athletes can injure the first ray either due to an acute accident or due to chronic overuse. Hallux valgus and rigidus often occur together in athletes and are therefore described together in this chapter. In the general population, hallux valgus is the most common pathology around the MTP-1-joint (meta-tarso-phalangeal), and hallux rigidus is the second most common. By contrast, in athletes hallux rigidus is the most common pathology [3]. Furthermore hallux rigidus is more painful and more disabling than hallux valgus [4].

A. Frigg  
Fusschirurgie Zürich, Bahnhofstrasse 56, Zürich 8001, Switzerland  
e-mail: [mail@arnofrigg.com](mailto:mail@arnofrigg.com)

## Hallux Valgus

### Etiology

The incidence of hallux valgus has two peaks: about half of the cases occur in patients who are younger than 20 years (juvenile hallux valgus), and the other half occurs in patients aged between 30 and 50 years (acquired hallux valgus) [5]. Metatarsus primus varus, hyperlaxity and flatfeet are frequent etiologies in juvenile and acquired hallux valgus [6]. The primary cause of the acquired hallux is the body mass pressing the foot on the ground over decades, and for this reason the acquired hallux is aggravated by obesity, age and inappropriate footwear such as high heels and narrow shoes. In sports, repetitive activities coupled with flatfeet may lead to progressive deformity [5]. Rare causes of traumatic hallux valgus with rupture of the medial collateral ligament of plantar plate have been reported [7].

Hallux valgus occurs with a frequency of about 2–4% in different ethnicities, but is symptomatic and more frequent in shoe-wearing societies [8]. Different ethnicities have a different anatomy of the foot and different incidence of hallux valgus [9]. The higher incidence of hallux valgus in women is caused by high heels, constricting foot wear and different soft tissue.

### Sports

There is no unanimity on the issue whether dance has an influence on the occurrence of hallux valgus. Some authors report that although dancers put an extreme stress on the MTP-1-joint, it is unlikely that dancing causes hallux valgus. Dancers are found not more prone to bunions than the rest of the population [10, 11], and hallux valgus was not more frequent in dancers than non-dancers [11]. By contrast, Van Dijk [12] found a greater number of cases of hallux valgus in dancers than in a control group. Dancing en-pointe and demi-pointe increases the forces around the MTP-1-joint, which may exacerbate an existing bunion [10, 11].

In rock climbers a high incidence of hallux valgus is described, and it is attributed to the widespread use of tight climbing shoes, which are on average two sizes smaller than the normal shoes [13].

### Pathomechanism

The MTP-1-joint is stabilized by the extensor hallucis longus and the extensor hallucis brevis, the flexor hallucis longus and the flexor hallucis brevis, the abductor and adductor hallucis, the medial and lateral capsule and collateral ligaments. Injury to any of these structures can alter the position of the hallux. The insertion of the adductor hallucis onto the lateral sesamoids and the plantar base of the proximal phalanx plays a main role in the development

of the deformity: The insertion of the adductor hallucis exerts not only a lateral force on these structures but also a rotational component [5]. As a consequence, with increasing hallux valgus deformity the great toe pronates (the nail turns towards the medial side), the sesamoids subluxate laterally, the medial aspect of the first metatarsal head becomes more prominent, and weight bearing shifts from the first metatarsal head to the second metatarsal head causing metatarsalgia. As the hallux moves laterally, it may occupy the space of the second toe leading to a hammertoe or cross-over deformity [2].

## Hallux Rigidus

### Etiology

Causes leading to hallux rigidus include the following [3, 14, 15]:

- Primary or idiopathic
  - a long narrow foot
  - a pronated foot
  - a long first metatarsal
  - metatarsus primus elevatus
- Secondary:
  - poor footwear
  - post-traumatic acute or chronic injury
  - obesity
  - age
  - neuromuscular disorders
  - arthritis (rheumatoid, psoriasis)
  - immobilization

### Sports

In beach soccer players OCD (osteochondral defects) are caused by hyperflexion or hyperextension [16]. Usually in kicking sports such as soccer, the dominant foot is affected with an acute fracture or repetitive stress [1]. Seventeen percent of soccer players were affected by hallux rigidus [17]. A higher occurrence of hallux rigidus was found in ballet dancers than in the control group [12].

### Pathomechanism

The normal ROM (range of motion) of the MTP-1-joint is flexion/extension 20–0–70° (to 30–0–80°). The amount of dorsiflexion needed in sports is greater (80°–100°) than in normal ambulation (65–75°) because of the prolonged propulsive phase, the longer stride length in running, and the increased ROM required for squatting or push-off activities [3, 14]. Relative forces press the base of the proximal phalanx against the dorsal portion of the metatarsal head, which causes articular damage and osseous proliferation.

## Symptoms

### Hallux Valgus

Hallux valgus is characterized by a medial prominence of the great toe and medial or varus deviation of the first metatarsal, accompanied by inflamed bursa and lateral or valgus deviation [8]. It may also be associated with pain under the sesamoids, metatarsal heads (metatarsalgia) and toe deformities (hammer toes, claw toes, cross-over toes). Hallux valgus may also be associated with sesamoiditis and stressfractures as a consequence of a flatfoot.

Since athletic activity puts a high load on the MTP-1-joint, overuse may lead to early degenerative changes. Therefore hallux valgus cannot be considered and treated alone but should be treated in conjunction with hallux rigidus.

In ballet it is not uncommon to see a clinically obvious bunion with an inflamed bursa but no pain [10]. In dancers there are two types of hallux valgus [10, 18]:

- Compensated bunion: slowly progressive bunions with a normal ROM and a congruous joint.
- Decompensated bunion: are rapidly progressive bunions. The dancer is affected by loss of ROM and progressive pain.

### Hallux Rigidus

Hallux rigidus is an osteoarthritic condition of the MTP-1-joint. It is characterized by a limited ROM, the formation of osteophytes, and associated synovitis. Some authors refer to a decrease in ROM as a hallux limitus and to a restricted dorsiflexion of  $<30^\circ$  as a hallux rigidus [3, 14]. At the same time, these authors do not distinguish between hallux limitus and hallux rigidus, but instead prefer a grading of hallux rigidus (please see section “Classification”). A functional hallux rigidus exhibits a normal ROM during the clinical examination in unloaded condition, but a limited ROM in response to load [14]. This is seen in any condition that functionally elevates the first ray, effectively blocking dorsiflexion, e.g. hypermobility of the first ray and metatarsus primus elevatus [14].

The main problem in athletic contexts is that most sports require a large ROM and a limitation causes disability. In running, for instance, a sprinter requires an extreme ROM, while the ROM needed in long or middle distance runners is less [8]. In ballet dancing  $80\text{--}90^\circ$  of dorsiflexion are required [10]. The IP-joint often compensates a restricted ROM of the MTP-1-joint with hyperextension. In particular in sports, this may force the nail of the hallux into the toe-box of the shoe, resulting in subungual hematoma and dystrophic nail changes [14].

## Diagnostics, Classification

For both hallux valgus and hallux rigidus radiographs, dorso-plantar and lateral, of the weight-bearing foot are needed. In case of a foot deformity, ankle radiographs and a hindfoot alignment view are helpful too. As a second step, MRI is required to evaluate cartilage, sesamoids and soft-tissues. In complicated cases a third step is recommended, namely the use of SPECT-CT to verify occult pathologies such as osteochondral defects, bone necrosis and CRPS.

### Hallux Valgus

The values of the most important anatomic angles characterizing a normal hallux are the following [5]:

- IMA = intermetatarsal angle  $<9^\circ$
- HVA = hallux valgus angle  $<20^\circ$
- DMAA = distal metatarsal articular angle  $<10^\circ$
- PPAA = proximal phalangeal articular angle, describing the inclination of the proximal articular surface to the axis of the proximal phalanx. The presence of a significant angulation defines a hallux interphalangeus.

Coughlin [5] differentiates between a congruous and a non-congruous MTP-1-joint. Congruity describes the orientation of the articular surfaces of the proximal phalanx and the metatarsal head:

- Congruent joint: The center of the metatarsal head corresponds to the center of the proximal phalanx. This is considered as a stable configuration and deformity does not progress with time.
- Incongruent joint: There is subluxation of the MTP-1-joint. The proximal phalanx deviates laterally in relation to the metatarsal head. With an incongruous joint there is significant risk of hallux valgus progression.

### Hallux Rigidus

On radiographs, the hallux rigidus is graded according to the degenerative changes in the MTP-1-joint [1, 5, 14]:

- Grade 0 = decreased ROM without articular changes
- Grade I = dorsal spur, minimal changes
- Grade II = osteophytes dorsal, lateral, medial,  $<25\%$  joint space narrowing, flattening of the head
- Grade III = destruction of articular cartilage,  $>25\%$  joint space narrowing, sesamoids enlarged, cystic changes
- Grade IV = presence or development of ankylosis

## Therapy: Conservative Treatment and Surgery

### Hallux Valgus

Stiffness of the MTP-1-joint is one of the most dreaded complications after hallux valgus surgery [19]. Therefore a conservative approach should be taken to a hallux valgus in athletes whenever possible. Many world record holders and Olympic gold medalists have clinically severely deformed feet, and yet they perform at a top level. The fact that one can be a world champion with a pathology shows that the clinical relevance of a deformity is only relative.

Stiffness is a greater impediment to athletic activity than pain. Since every surgical intervention runs the risk of resulting in stiffness, surgery should be considered only if pain and discomfort of the hallux impose serious limitations on athletic activity. In particular in the case of professional athletes, surgery should be delayed until the athlete either retires or the problems reach a level of severity that makes athletic performance impossible [5, 10, 11]. This is because foot and ankle surgeries that routinely show good results in the general population can prematurely end an athletic career [4].

### Conservative Treatment

Conservative measures include the use of pads and insoles both in sport shoes and everyday shoes. No form of brace or night splint has been found to be effective [10]. Furthermore, shoes with a wide toe box are recommended because they avoid friction and blistering. Shoes with a medial support and flat heel are preferable [4].

### Surgical Treatment

The goal of a treatment should be the functional return to sports. Most compensated bunions can be treated conservatively [2]. If surgery is unavoidable, a simple chevron osteotomy is sufficient. A complete correction of the deformity is not always possible. This is not a major problem, though, because all that is needed to restore functionality is the conversion of a decompensated bunion into a compensated bunion [2]. More extensive surgical procedures such as proximal osteotomies or Lapidus arthrodesis should be avoided because of the extensive nature of these procedures, the risk of joint stiffness, and the prolonged recovery time [2, 4, 18]. Hence, professional athletes should be given tentative treatments until they retire [6, 18]. By contrast, amateur athletes can given definitive treatments as long as they are aware that some functionality may be lost [18].

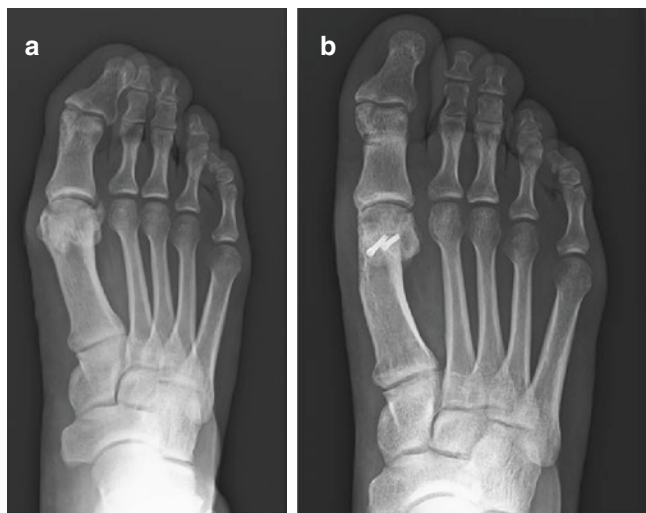
There are a large number of different hallux valgus surgeries. The following is an overview of the author's preferred methods:

1. Extraarticular hallux surgery (HVA  $<20^\circ$ ):
  - (a) McBride: Distal soft tissue alignment and resection of the medial eminence (bunionectomy, open or minimally invasive).
  - (b) Akin-osteotomy: Medial closing wedge osteotomy of the proximal phalanx (open or minimally invasive) (Fig. 36.1).
2. Intraarticular surgery:
  - (a) Chevron/short Scarf-osteotomy: The aim of the surgery is to restore normal anatomic angles. The IMA should not be overcorrected below  $4^\circ$ , otherwise the risk of hallux varus increases. The aim is to achieve an IMA of  $4\text{--}5^\circ$ . How much the head has to be shifted laterally is decided during the preoperative planning. If the shift is  $<50\%$ , the surgeons perform a chevron or short scarf osteotomy (2 cm L-shape, IMA usually  $<14^\circ$ ). This technique can be used in a way that is minimally invasive (Fig. 36.2)
  - (b) Long or classical Scarf-osteotomy: If more than  $50^\circ$  of shifting is needed a long "classical" scarf (until 1.5 cm distal to the TMT-1-joint) is performed as long as the IMA is not more than  $18\text{--}20^\circ$  [19]. The problem with scarf osteotomies is that with large IMA not only a lateral shift but also a lateral rotation is made, which creates a pathologic DMAA leading to an incomplete hallux valgus correction or recurrence in the postoperative course. Furthermore in the case of pathological DMAA, the scarf-osteotomy allows only minimal correction of the DMAA and is therefore not indicated [19]. In such cases proximal metatarsal osteotomies are preferred.



**Fig. 36.1** Ultra-Marathon runner before (a) and after percutaneous lateral release, cheilectomy and Akin-osteotomy (b)





**Fig. 36.2** (a) Professional Baseball player with hallux valgus and rigidus. (b) A shortening Chevron osteotomy and percutaneous Akin-osteotomy were performed. Prolonged rehabilitation over 6 months followed by a return to 100% professional competition

### 3. Proximal metatarsal osteotomies:

Proximal osteotomies are indicated at an IMA over  $16\text{--}18^\circ$ . Depending on the length of the first metatarsal in relation to the second metatarsal, an open or closing wedge osteotomy is performed. To correct all anatomic pathologies, we have a preference for the so-called triple osteotomy, which is a proximal osteotomy combined with a chevron osteotomy for the correction of the DMAA and an Akin-osteotomy. This triple osteotomy gives solid and reliable results also in amateur athletes. There are three types of proximal osteotomies:

- (a) Proximal open wedge osteotomy: In case of an index plus situation (second metatarsal longer than the first one), an open wedge osteotomy is recommended as it lengthens the first metatarsal. Furthermore it can stabilize unstable TMT-1-joints and is beneficial in the case of flatfeet (Fig. 36.3).
- (b) Proximal closing wedge osteotomy: In case of a long first metatarsal in relation to the second metatarsal (index minus), this kind of osteotomy is recommended because it shortens the first metatarsal bone (Fig. 36.4).
- (c) Crescentic osteotomy: This is a curved osteotomy characterized by a 1.5 cm distal to the TMT-1-joint described by Coughlin [5] with pure rotation of the metatarsal bone. It may be technically demanding and shortens the bone by the amount of the saw-blade (approximately 2 mm). We therefore prefer the open wedge osteotomy.

4. Lapidus-Arthrodesis: This is the fusion of the TMT-1-joint. It is indicated in TMT-1-hypermobility. The main problem with this procedure is the shortening of the first ray by about 4 mm leading to metatarsalgia and as result to more surgery, more scarring and more disability [18]. Therefore we try to avoid this surgery.

## Hallux Rigidus

### Conservative Treatment

Conservative treatments include a stiff sole, rocker bottom shoes, shoes with a wide toe box to accommodate the enlarged joint, pain medication and taping [14, 15].

### Surgical Treatment

Surgery is only recommended if all conservative measures have failed. The following is an overview of the author's preferred methods:

#### 1. Cheilectomy:

Resection of the dorsal osteophytes and removal of about 25–30% of the dorsal joint to allow dorsiflexion of  $110\text{--}120^\circ$  [4, 10]. The athlete has to be warned, however, that this treatment only lowers symptoms and does not restore normal functionality. The reason for this is that hallux rigidus is a degenerative disease (Fig. 36.5).

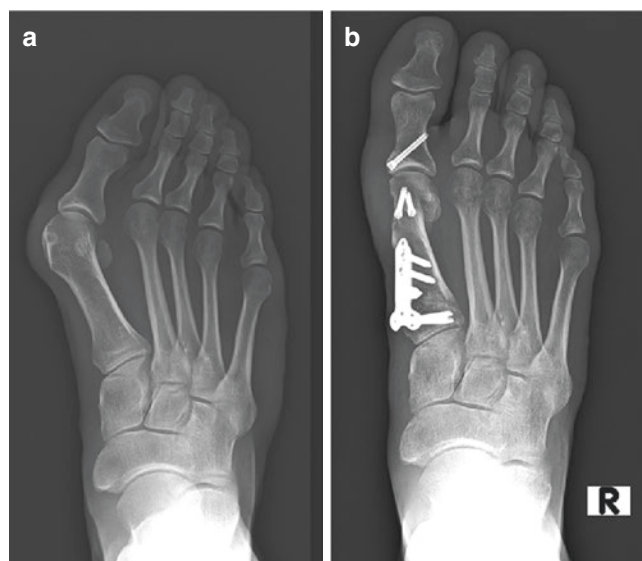
#### 2. Periarticular Osteotomies:

- Moberg osteotomy: Dorsiflexion osteotomy of the proximal phalanx to increase dorsiflexion.
- Waterman-Green Osteotomy: Removal of a dorsal wedge from the first metatarsal. The joint is preserved and decompressed. This technique requires an intact cartilage on the plantar and central aspect of the joint.
- Barouk-Osteotomy [20]: This is an oblique osteotomy similar to a Weil-osteotomy of the lesser metatarsals. The elevated first metatarsal head is lowered and the joint decompressed by shortening the first ray (Fig. 36.2).

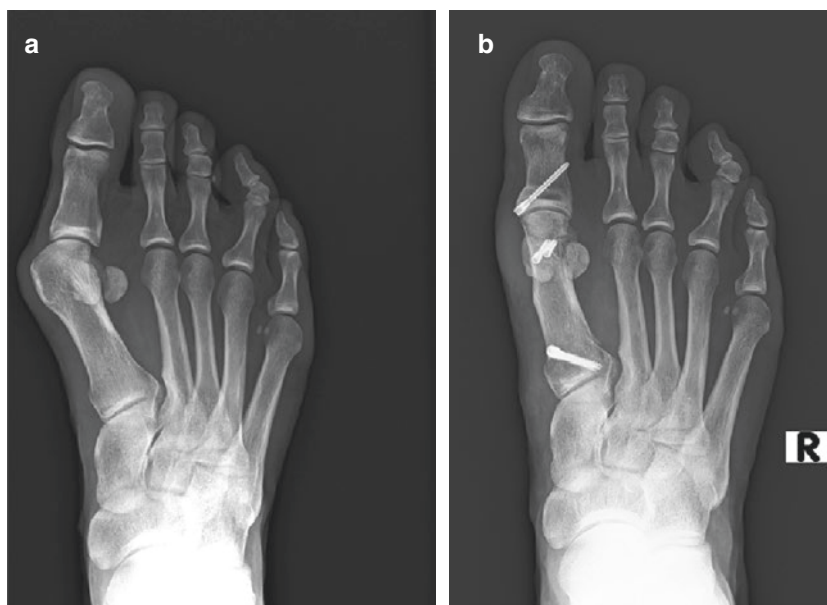
3. Osteochondral defects: OCD should be treated with debridement and drilling holes into the bleeding bone [16, 21]. For defects  $>50\text{ mm}^2$  or cystic lesions an osteochondral autograft transfer from the lateral edge of the trochlea of the knee should be performed [22].

4. Arthroscopic surgery of the MTP-1-joint is possible for dorsal impingement syndrome, OCD, and painful sesamoid bones [23]. However we prefer minimally invasive techniques for dorsal impingement syndromes.

**Fig. 36.3** Ballet student with symptomatic flatfoot, painful bunion, and index-plus variation. A triple osteotomy with proximal open wedge was performed and insoles for the flatfoot prescribed. Full return to dancing

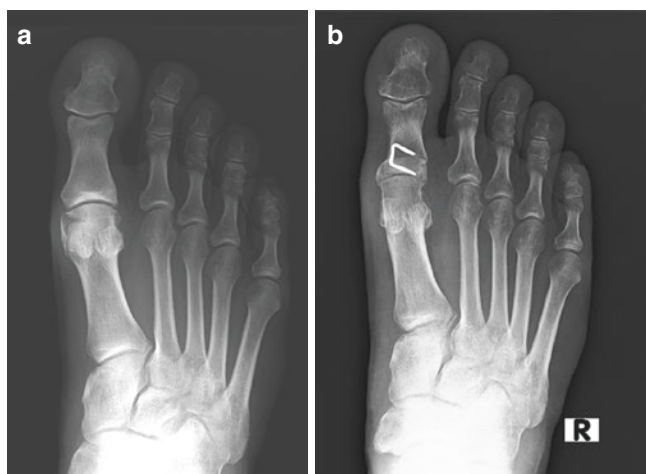


**Fig. 36.4** Recreational mountain-biker and squash player with index-minus variation. A triple osteotomy with proximal closing wedge was performed. Return to full amateur competitive level after surgery

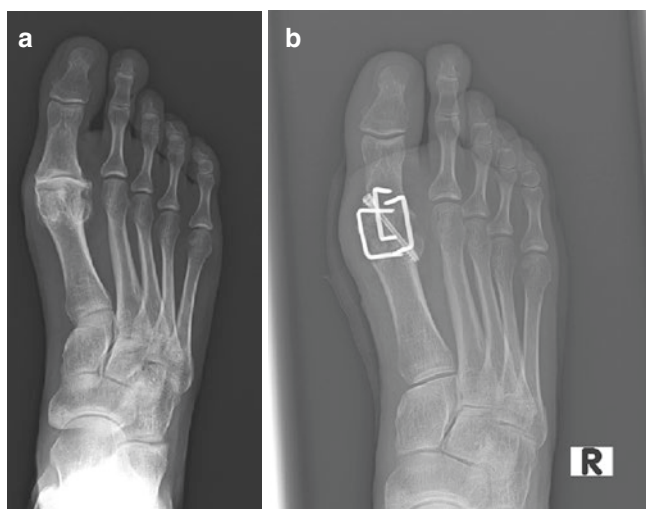


We advise strongly against the use of the following extensive techniques for professional athletes; however, they remain an option for recreational sports:

1. Arthrodesis: Procedure is limited to individuals who do not require motion at the MTP-1-joint. It is a good option for weekend athletes and recreational sports but not for competitive athletes [2] (Fig. 36.6).
2. Keller-Brandes Procedure: Resection arthroplasty with resection of a part of the proximal phalanx and metatarsal head and interposition with joint capsule or an allograft matrix. It should not be used in athletes because it disrupts the plantar plate and flexor mechanism and will result in decreased push off [2]. Furthermore 15% of cock-up deformity can occur [5].
3. Silicone hemiarthroplasty (Swanson) or total joint arthroplasty: Both are contraindicated in athletes because the decreased weight bearing capacities of the first ray may lead to transfer metatarsalgia. The increased load in sports may lead to early failure of the implant [8]. Silicone arthroplasty showed poor results as regards joint stiffness, soft tissue reaction due to bony resorption around the implant, and loosening of the prosthesis [2, 15]. Also total joint replacement has not been consistently successful because of joint stiffness, synovitis, implant failure and loosening [1, 2].



**Fig. 36.5** Police officer with hallux rigidus before and after cheilectomy and Moberg osteotomy. Equipped with insoles and stiff sole over the MTP-1-joint he made a full return to work and running sports



**Fig. 36.6** Recreational tennis player with hallux rigidus (a) before and (b) after MTP-1-arthrodesis with staples and one screw. Doing more sports after surgery than before

### Minimally Invasive Surgery

To date, it has not been possible to formulate clear recommendations for the use of MIS for hallux valgus or hallux rigidus surgery. However, preliminary results are encouraging [24]. One can distinguish between percutaneous and minimally invasive surgery:

1. Percutaneous surgery is performed through the smallest possible working incisions (usually 1–3 mm) without direct visualization of the target structures, using a mini-blade for soft-tissue incision and a rotatory burr for bony procedures under fluoroscopy.

2. MIS is performed through the smallest incision necessary to perform the surgery (usually 1–3 cm) using a traditional scalpel for soft-tissue incision and saw blades for bony procedures under direct visualization of the structures.

Minimally invasive procedures reduce the surgical trauma and operating time. This is advantageous due to potentially fewer complications, especially stiffness, and quicker recovery [19]. The Reverdin-Isham procedure [25] can be performed up to an IMA of 15° and a HVA of 40°. It is not yet clear, however, whether the procedure leads to reduced stiffness. A 17% loss of ROM was observed which is comparable to the standard open procedures [19]. A potential explanation of this fact is that the working area was insufficiently cleaned and irrigated of bony debris and fragments [24]. The clinical results obtained with percutaneous procedures for the correction of mild to moderate hallux valgus are currently comparable to, but not better than, results obtained in most series of open surgery [24].

### Rehabilitation and Back-to-Sports

The average time to return to low-level athletic activity (15–20 min of running or practice) after a first metatarsal osteotomies is 9 weeks; after a bunionectomies for hallux valgus 6 weeks; and after cheilectomies for hallux rigidus between 7 weeks and 6 months [11, 26]. Athletes must be informed that even if the surgery is successful, they may not regain the required ROM for sports [11]. The average time to return to recreational sports such as swimming, golf, and jogging is 16 weeks [22]. The return to training can be advanced, for instance, by wearing cast boots. Return to competition can require months because surgical results continue to improve 6–12 months postoperatively. Dancers often continue dancing during recovery, which often prolongs, and at times complicates, treatment [4]. No dancing should be allowed for at least 3 months after surgery [10].

After MTP-1-joint arthroplasty with the Toefit-plus joint replacement 91% of patients were able to resume a recreational activity after 18 months. Low-impact sports (such as walking, hiking, swimming, dancing, golf, and cycling) were recommended because of the risk of loosening and the early failure associated with more intense physical activity [27].

### Prevention

Hallux valgus can partially be prevented by wearing shoes with a wide toe box and by choosing them large enough to avoid compression of the forefoot. In the case of flatfeet the use of orthopedic insoles can avert the onset of hallux valgus.

Sport itself is controversially discussed as cause of an increased deformity progression. However, the only way to avoid a hallux valgus due to sports is to avoid sports itself, which is impractical. Hallux rigidus is also triggered by acute or repetitive trauma to the MTP-1-joint during sports. Stiff insoles and a wide toe box can prevent a hallux rigidus from becoming symptomatic.

Patients who are diagnosed with symptomatic hallux valgus and rigidus at a young age should be advised not to pursue a career as professional athletes. This is because these conditions are incurable and have the potential to bring an athletic career to a premature end at any time [6].

## Evidence

The following is a description of highest evidence with an indication of the Level and Grade of evidence:

Bauer 2009 Level IV [19]  
 Danilidiis 2008 Level IV [27]  
 Mullier 1999 Level IV [3]  
 Kim 2014 Level III [22]  
 Maffulli 2011 Level III [24]

## Summary: 5 Take-Home Lessons

First, hallux valgus and hallux rigidus are the two most frequent pathologies of the MTP-1-joint. Hallux valgus is more common in the general population, while hallux rigidus is more common among athletes. Athletes often suffer from both conditions simultaneously.

Second, in case of professional athletes, surgery should be avoided for as long as possible. Only if the condition gets unbearable an operation should be carried out. This is because of possible scarring of the joint and which has the potential to bring an athletic career to an untimely end.

Third, as regards hallux valgus, the aim is to convert a decompensated bunion into a compensated one. This can usually be achieved with a simple chevron osteotomy which has a low risk of scarring and complications.

Fourth, as regards hallux rigidus, a simple cheilectomie is usually sufficient, potentially accompanied by a closing wedge osteotomy of the proximal phalanx or a shortening of the distal first metatarsal. More extensive surgery ought to be delayed until the end of the career as a professional athlete.

Fifth, if symptomatic hallux valgus or rigidus develop at an young age, a professional career should be discouraged.

## References

1. Nihal A, Trepman E, Nag D. First ray disorders in athletes. *Sports Med Arthrosc.* 2009;17(3):160–6.
2. Hockenbury RT. Forefoot problems in athletes. *Med Sci Sports Exerc.* 1999;31(7 Suppl):S448–58.
3. Mulier T, Steenwerckx A, Thienpont E, Sioen W, Hoore KD, Peeraer L, Dereymaeker G. Results after cheilectomy in athletes with hallux rigidus. *Foot Ankle Int.* 1999;20(4):232–7.
4. Brown TD, Micheli LJ. Foot and ankle injuries in dance. *Am J Orthop (Belle Mead NJ).* 2004;33(6):303–9.
5. Coughlin MJ, Saltzman CL. *Surgery of the foot and ankle.* 8th ed. Mosby Elsevier; 2006. Philadelphia, USA
6. Howse J. Disorders of the great toe in dancers. *Clin Sports Med.* 1983;2(3):499–505.
7. Fabeck LG, Zekhnini C, Farrokh D, Descamps PY, Delincé PE. Traumatic hallux valgus following rupture of the medial collateral ligament of the first metatarsophalangeal joint: a case report. *J Foot Ankle Surg.* 2002;41(2):125–8.
8. DeLee J, Drez D, Miller M. *Orthopaedic sports medicine.* 2nd ed. Saunders; 2003. Philadelphia, USA
9. Gurney JK, Kersting UG, Rosenbaum D. Dynamic foot function and morphology in elite rugby league athletes of different ethnicity. *Appl Ergon.* 2009;40(3):554–9.
10. Kennedy JG, Collumbier JA. Bunions in dancers. *Clin Sports Med.* 2008;27(2):321–8.
11. Kadel NJ. Foot and ankle injuries in dance. *Phys Med Rehabil Clin N Am.* 2006;17(4):813–26.
12. van Dijk CN, Lim LS, Poortman A, Strübbe EH, Marti RK. Degenerative joint disease in female ballet dancers. *Am J Sports Med.* 1995;23(3):295–300.
13. Schöffl V, Küpper T. Feet injuries in rock climbers. *World J Orthop.* 2013;4(4):218–28.
14. Lichniak JE. Hallux limitus in the athlete. *Clin Podiatr Med Surg.* 1997;14(3):407–26.
15. Hawkins BJ, Haddad Jr RJ. Hallux rigidus. *Clin Sports Med.* 1988;7(1):37–49.
16. Altman A, Nery C, Sanhudo A, Pinzur MS. Osteochondral injury of the hallux in beach soccer players. *Foot Ankle Int.* 2008;29(9):919–21.
17. Steinacker T, Steuer M, Höltke V. Injuries and overload-damages at players of the German lady-soccer-national-team. *Sportverletz Sportschaden.* 2005;19(1):33–6.
18. Baxter DE. Treatment of bunion deformity in the athlete. *Orthop Clin North Am.* 1994;25(1):33–9.
19. Bauer T, de Lavigne C, Biau D, De Prado M, Isham S, Laffenétre O. Percutaneous hallux valgus surgery: a prospective multicenter study of 189 cases. *Orthop Clin North Am.* 2009;40(4):505–14.
20. Louis-Samuel B. *Forefoot reconstruction.* 2nd ed. Springer; 2005. Parris, France
21. Street CC, Shereff MJ. Traumatic osteochondral defect of the first metatarsal head: a case report. *Am J Orthop (Belle Mead NJ).* 1999;28(10):584–6.
22. Kim YS, Park EH, Lee HJ, Koh YG, Lee JW. Clinical comparison of the osteochondral autograft transfer system and subchondral drilling in osteochondral defects of the first metatarsal head. *Am J Sports Med.* 2012;40(8):1824–33.
23. van Dijk CN, Veenstra KM, Nuesch BC. Arthroscopic surgery of the metatarsophalangeal first joint. *Arthroscopy.* 1998;14(8):851–5.
24. Maffulli N, Longo UG, Marinozzi A, Denaro V. Hallux valgus: effectiveness and safety of minimally invasive surgery. A systematic review. *Br Med Bull.* 2011;97:149–67.
25. De Prado M, Ripoll PL, Golano P. *Minimally invasive foot surgery.* About Your Health Publisher; 2009. Barcelona, Spain
26. Saxena A. Return to athletic activity after foot and ankle surgery: a preliminary report on select procedures. *J Foot Ankle Surg.* 2000;39(2):114–9.
27. Danilidis K, Martinelli N, Marinozzi A, Denaro V, Gosheger G, Pejman Z, Buchhorn T. Recreational sport activity after total replacement of the first metatarsophalangeal joint: a prospective study. *Int Orthop.* 2010;34(7):973–9.

Andrew R. Hsu and Robert B. Anderson

## Abstract

Turf toe is a hyperextension injury to the hallux metatarsophalangeal (MTP) joint that commonly occurs during athletics when an axial load is delivered to the heel with the ankle in plantarflexion and the hallux in dorsiflexion or extension. Hyperextension at the hallux MTP joint can lead to tearing and/or attenuation of the plantar capsuloligamentous structures, which in turn may lead to joint instability, pain, and decreased function. The incidence of turf toe has risen over the past four decades as awareness of the injury has grown along with advances in surgical management and rehabilitation. A thorough history and clinical exam focusing on hallux MTP stability and flexion strength is critical to ensure a timely diagnosis. Proximal migration of the sesamoids should be carefully examined on standard radiographs, dynamic stress views, and MRI. Management of turf toe injuries depends on the grade of the injury, with surgical intervention recommended in cases of complete disruption of the plantar structures with persistent joint instability and pain despite non-operative treatments. Direct repair of the plantar capsuloligamentous complex can be successfully achieved using a two-incision approach (medial and plantar). Post-operative rehabilitation is critical to final outcome and return to athletics is permitted 3–4 months after surgery depending on sport and position played.

## Keywords

Plantar plate • Turf toe • Instability • Sesamoid complex • Hallux • Reconstruction • Rehabilitation

## Introduction

Injuries to the sesamoid complex and capsule of the first metatarsophalangeal (MTP) joint, commonly known as turf toe, were first described in 1976 among American football players [1]. Turf toe typically occurs as a result of a hyperextension force to the hallux MTP joint that injures the plantar plate structures. These injuries can be debilitating to athletes

as the first ray is pivotal during jumping, running, and cutting maneuvers [2]. Over the past four decades, these injuries have received increasing attention and research as awareness and identification have risen along with the prevalence of synthetic playing surfaces and lighter, more flexible shoes among athletes [3–9]. With correct and timely diagnosis and treatment combined with sport-specific rehabilitation, athletes can recover from turf toe injuries and return to play at their pre-injury level of competition. The purpose of this review is to discuss the anatomy, etiology, work-up, treatment, and rehabilitation of turf toe injuries with a focus on evidence from the literature to guide clinical decision-making.

A.R. Hsu, MD (✉) • R.B. Anderson, MD  
OrthoCarolina Foot & Ankle Institute,  
2001 Vail Avenue, Suite 200B, Charlotte, NC 28207, USA  
e-mail: [andyhsu1@gmail.com](mailto:andyhsu1@gmail.com); [Robert.Anderson@orthocarolina.com](mailto:Robert.Anderson@orthocarolina.com)

## Anatomy

The hallux MTP joint is stabilized by osseous and soft-tissue structures such as the short flexor complex, ligaments, and capsule that limit the amount of plantarflexion and dorsiflexion to prevent injury (Fig. 37.1). The primary osseous stabilizer is the articulation between the metatarsal and proximal phalanx, but there is little inherent bony stability as the proximal phalanx has a shallow cavity in which the hallux metatarsal head articulates. Dynamic tendon restraints include the flexor hallucis brevis (FHB) that contains the tibial and fibular sesamoids, abductor hallucis (medially), and adductor hallucis (laterally). The FHB tendon inserts on the proximal phalanx and is in confluence with the plantar plate. The sesamoids contained within the FHB function to directly increase the lever arm of the FHB and indirectly increase the lever arm of the flexor hallucis longus (FHL) to mechanically increase flexion and push-off strength at the MTP joint [10].

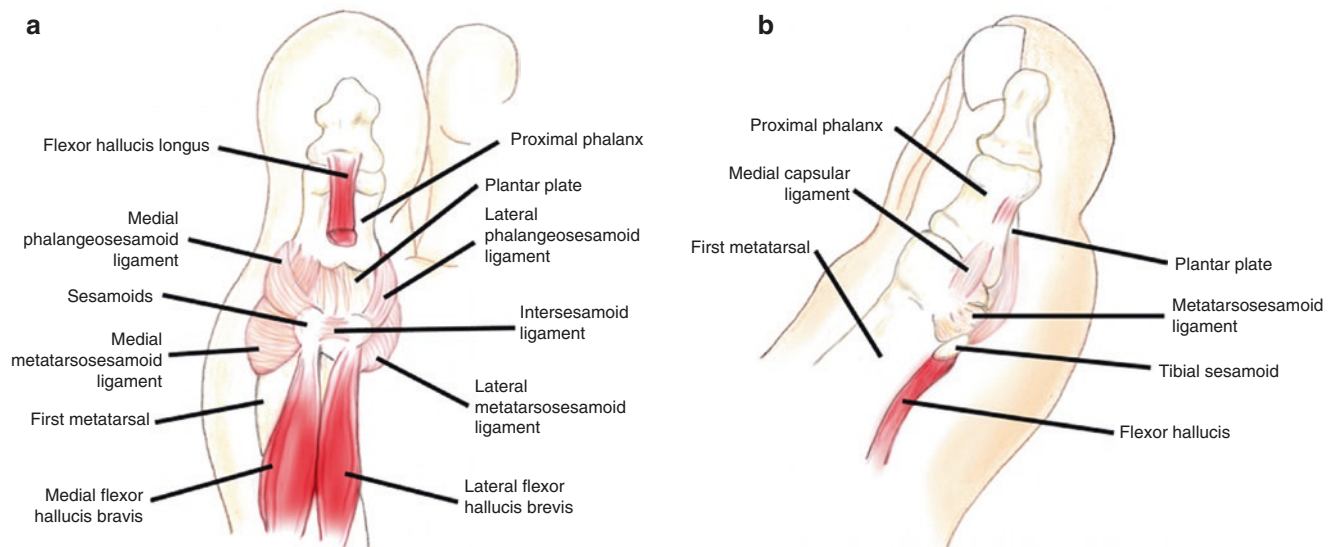
Ligamentous support comes from the intermetatarsal ligament and fan-shaped medial and lateral collateral ligaments that provide valgus and varus stability, respectively. The plantar plate is composed of the thickened joint capsule that travels from the weaker metatarsal head attachment to the stronger proximal phalanx attachment. The plantar plate also attaches to the transverse head of the abductor hallucis, flexor tendon sheath, and deep transverse intermetatarsal ligament. During normal gait mechanics, the capsule ligamentous complex of the hallux MTP joint supports 40–60%

of body weight [11]. This force can increase to 2–3 times body weight during athletic activity and reach as high as eight times body weight during high-impact activities [11, 12].

## Etiology

Turf toe injuries are more prevalent in contact athletic sports played on rigid surfaces such as American football. The most common mechanism of injury is hyperextension of the hallux MTP joint as an axial load is applied to the heel with the ankle in plantarflexion and the hallux in dorsiflexion or extension [13]. The forefoot is engaged in the field surface, and this combination of force and joint positioning in hyperextension leads to attenuation or tearing of the plantar capsule ligamentous complex, most often off of the proximal phalanx. A spectrum of injuries can result, ranging from a sprain of the plantar plate to complete disruption of the plantar structures with joint dislocation. In a cadaveric study of first MTP joint sprains, Frimenko et al. found that there was a 50% risk of plantar plate injury at 78° of hallux dorsiflexion from anatomical zero [6].

Osteochondral lesions (OCDs) of the MTP joint and subchondral edema of the metatarsal head may occur concurrently as the proximal phalanx impacts or shears across the metatarsal head articular surface [14]. Additional injuries may include sesamoid fractures and stress fractures of the proximal phalanx. A traumatic hallux valgus deformity can result when a more medial (valgus) force is applied to the



**Fig. 37.1** (a) Plantar and (b) lateral views of hallux metatarsophalangeal (MTP) joint anatomy including the bony, capsular, ligamentous, and short flexor complex restraints (a and b reproduced with

permission from *Medscape Drugs & Diseases* (<http://emedicine.medscape.com/>), 2014, available at: <http://emedicine.medscape.com/article/1236962-overview>)

hallux MTP joint during hyperextension. In this scenario, more force is directed across the medial and plantar-medial ligamentous structures and the tibial sesamoid complex, thus causing contracture of the lateral sesamoid complex and adductor hallucis [15, 16].

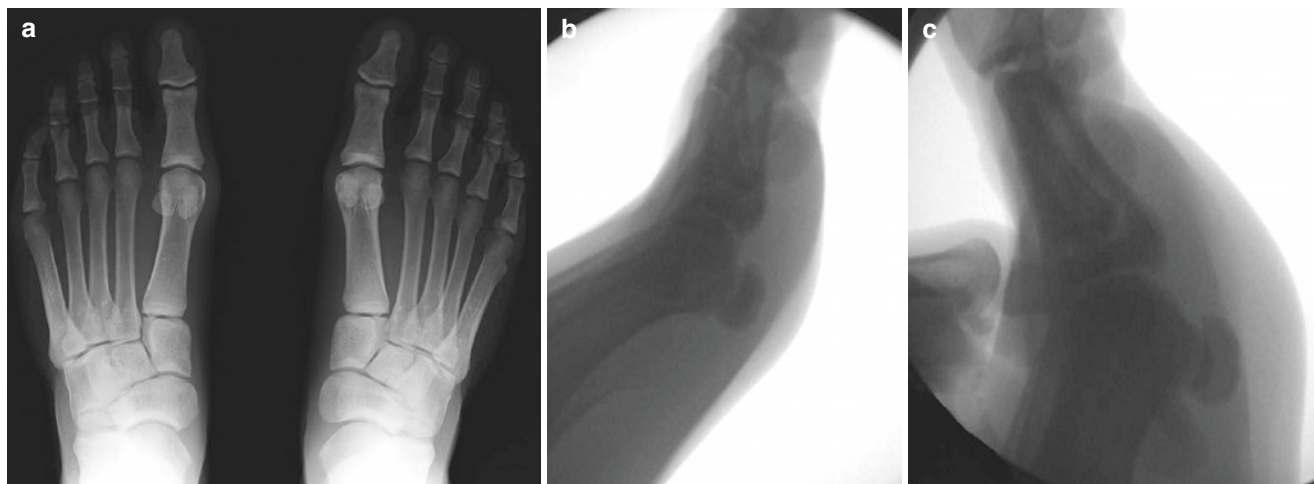
## Work-Up

Common symptoms after turf toe injury include pain, stiffness, and swelling. However, physical exam findings can be variable with underlying pathology difficult to isolate given the numerous adjacent anatomic structures. A high index of suspicion should be held in any patient with persistent MTP joint pain and swelling after an acute injury. Defining characteristics include a decreased ability to push-off at the hallux MTP joint, increased hallux dorsiflexion compared with the contralateral unaffected side, and reduce agility in cutting and pivoting maneuvers. On physical exam, patients will often have plantar swelling and ecchymosis at the hallux MTP joint with pain during active and passive range of motion. Varus and valgus stress should be applied to evaluate the collateral ligaments and active flexion and extension can be used to assess the extensor and flexor tendons and plantar plate. Localizing pain proximal to the sesamoids suggests a strain of the FHB musculocutaneous junction rather than an unstable turf toe injury [17]. In unstable turf toe, pain is typically localized distal to the sesamoids and exacerbated with hallux hyperextension with a vertical (dorsal-plantar) Lachman test showing increased laxity compared with the contralateral side. Evaluation of gait mechanics will often

show a shortened time spend after heel rise to reduce the amount of hallux dorsiflexion.

Initial imaging should include standard weight-bearing anteroposterior (AP), oblique, and lateral radiographs of the foot to evaluate the sesamoid-to-joint distance, proximal migration of the sesamoids, and presence of sesamoid fracture (Fig. 37.2). Additional views can be obtained to further delineate the extent of injury including sesamoid axial views and forced dorsiflexion views under live fluoroscopy. A small fleck of bone pulled off of the proximal phalanx or distal sesamoid may indicate a capsular avulsion or disruption [17]. In cases of subtle xray findings it can be helpful to obtain standing radiographs of the contralateral limb to compare side-to-side sesamoid-to-joint distances.

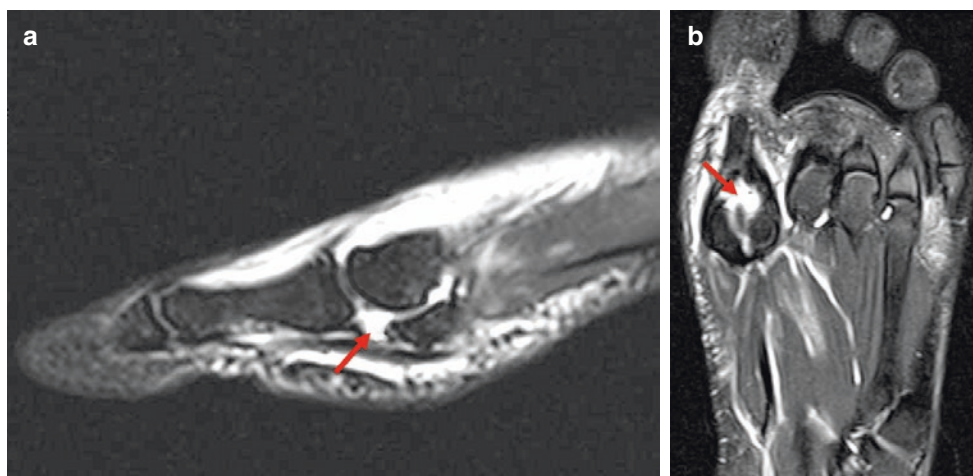
Patients with Grade III injury will have proximal migration of one or both sesamoids. The sesamoids will not track distally with hallux dorsiflexion and remain beneath the metatarsal head during all radiographs. This finding can be highlighted with a stress dorsiflexion lateral radiograph and/or comparison to the contralateral uninjured side. Fluoroscopic imaging has also been found to be very helpful as a diagnostic tool, and educational to the patient as well. Magnetic resonance imaging (MRI) can be obtained in cases of persistent pain, swelling, and decreased hallux push-off in the setting of normal appearing radiographs [18]. T2-weighted MRI cuts will often show disruption of the capsule ligamentous complex distal to the sesamoids and can also help rule out stress fractures of the proximal phalanx (Fig. 37.3). MRI can also help delineate any concurrent pathology affecting the FHL tendon and articular surfaces.



**Fig. 37.2** (a) Standing anteroposterior (AP) radiograph of bilateral feet showing proximal sesamoid retraction on the injured left foot compared with normal sesamoid position on the right foot. Lateral dynamic fluoroscopic image of sesamoids under the metatarsal head at (b) rest

and (c) after forced hallux MTP dorsiflexion showing lack of distal tracking of the sesamoids associated with rupture of the capsule ligamentous complex

**Fig. 37.3** (a) T2-weighted sagittal short-tau inversion recovery (STIR) and (b) axial fat-saturated MRI cuts of the hallux showing edema and disruption of the capsule ligamentous complex distal to the medial sesamoid (shown by red arrows)



## Classification

Diagnosis of turf toe injury is often correlated with a hyperextension mechanism of injury to the hallux MTP joint and the type of athletic shoe and surface played on at time of injury. Grade I injury is a sprain or attenuation of the plantar capsule ligamentous complex of the hallux MTP joint [17]. Patients typically have localized swelling and minimal ecchymosis on exam. Treatment is symptomatic and patients are typically able to return to play as tolerated. Grade II injury is a partial rupture of the plantar soft tissues of the MTP joint with moderate swelling and restricted motion due to pain [17]. Patients typically require 2–3 weeks to recover after a period of immobilization in a controlled ankle motion (CAM) boot as needed. Patients have also require taping of the hallux MTP joint upon return to athletic activity. Grade III injury is a complete rupture of the plantar plate structures of the hallux MTP joint with significant swelling and ecchymosis along with hallux flexion weakness and frank instability of the joint. Treatment consists of long-term (10–16 weeks depending on sport and position played) immobilization in a CAM boot or short-leg cast versus surgical reconstruction [17].

## Non-operative Treatment

Non-operative treatment for all grades of turf toe injury begins with rest, ice, compression, elevation (RICE), and non-steroidal anti-inflammatories (NSAIDs) to reduce pain and swelling. Immobilization is achieved with a combination of taping, toe spica extension, stiff-sole shoes, and/or CAM boot. Conservative treatment is indicated in the majority of Grade I–III injuries with early symptomatic care with icing

and rest initiated first. Taping of the hallux MTP joint in plantarflexion can protect the joint from hyperextension while opposing the injured plantar soft tissue structures. However, taping is not indicated in the acute setting as it can worsen swelling and compromise local circulation to the hallux. Stiff-sole shoes or CAM boots help limit hallux MTP motion with more severe injuries warranting a short-leg cast for 2–6 weeks. Once swelling and pain had decreased, progressive, controlled motion with physical therapy is started with gradual incremental progression back to athletic activity.

For Grade I injuries, patients are allowed to return to athletic competition as tolerated with minimal loss of playing time. Taping is performed routinely during the recovery process to limit motion and provide compression in combination with a stiff-sole shoe with a turf toe plate insert or custom orthotic with a Morton's extension to limit hallux motion [17]. Passive plantarflexion of the injured hallux can begin 3–5 days after injury to decrease sesamoid adhesions while protecting injured tissues [19]. Patients must be followed closely as deformity can develop during rehabilitation as impact levels are increased. Grade II injuries typically result in a loss of 2–3 weeks playing time owing to partial rupture of the capsule ligamentous complex. Similar to Grade I injuries, focus is on early gentle range of motion exercises with hallux protection with immobilization. Activities are progressed as tolerated with routine taping and footwear modifications upon return to full activity. Grade III injuries are complete ruptures of the plantar plate structures of the hallux MTP joint requiring long-term immobilization for up to 10–16 weeks [17]. The hallux MTP joint should have at least 50–60° of painless passive dorsiflexion before high-impact activities are initiated [19]. Patients may require up to 6–8 months of rehabilitation before returning to athletics without symptoms.



## Surgical Treatment

Surgical repair of turf toe is reserved for Grade III injuries that fail conservative treatment or cases that involve one or a combination of the following: (1)- large capsular avulsion with unstable MTP joint, (2)- diastasis of bipartite sesamoid, (3)- diastasis of sesamoid fracture, (4)- retraction of sesamoid, (5)- traumatic hallux valgus deformity, (6)- vertical instability (positive Lachman test), (7)- loose body in MTP joint, or (8)- chondral injury in MTP joint [20]. Elite athletes may often undergo initial treatment with surgical repair in the presence of severe Grade III injuries in an effort to decrease rehabilitation times and return to play faster. The goal of surgery is to restore the normal anatomy of the hallux MTP joint along with stability during full range of motion.

A variety of techniques have been described to repair the plantar capsule ligamentous complex including a medial approach with J-shaped incision extending across the plantar crease of the hallux MTP joint, medial approach alone, and combined medial and plantar approach [5, 21, 22]. It is our current preference to use a combined medial and plantar approach through two separate incisions. We believe this technique helps decrease wound-healing complications, in particular skin edge necrosis, reduce iatrogenic injury to the plantar cutaneous nerves, and improve medial and lateral exposure of the plantar plate.

Patients are placed in a supine position using general or regional anesthesia with an ankle tourniquet in place to control bleeding. Under anesthesia, the cock-up deformity of the hallux MTP joint is tested by dorsiflexing the joint determine the clinical degree of joint instability and lack of plantar restraint. The degree of proximal sesamoid migration can be examined on a lateral view of the hallux MTP joint under dynamic fluoroscopy. A medial approach is made using a longitudinal incision just plantar to midline extending from the metatarsal neck to the flexion crease at the base of the hallux (Fig. 37.4). The skin and subcutaneous incision is carried down to the capsule and then the capsule is carefully taken off of bone in the interval plantar to the metatarsal head and dorsal to the medial sesamoid. The capsulotomy extends from the metatarsal neck to the mid-phalanx to provide complete visualization of the sesamoid complex. Using dissection scissors, the sesamoid complex and capsule are identified along with the plantar plate tear. Attenuated tissue, a partial tear, or complete tear distal to the sesamoids warrants an open reconstruction. Care must be taken to dissect and identify the plantar medial cutaneous nerve along the inferior aspect of the incision.

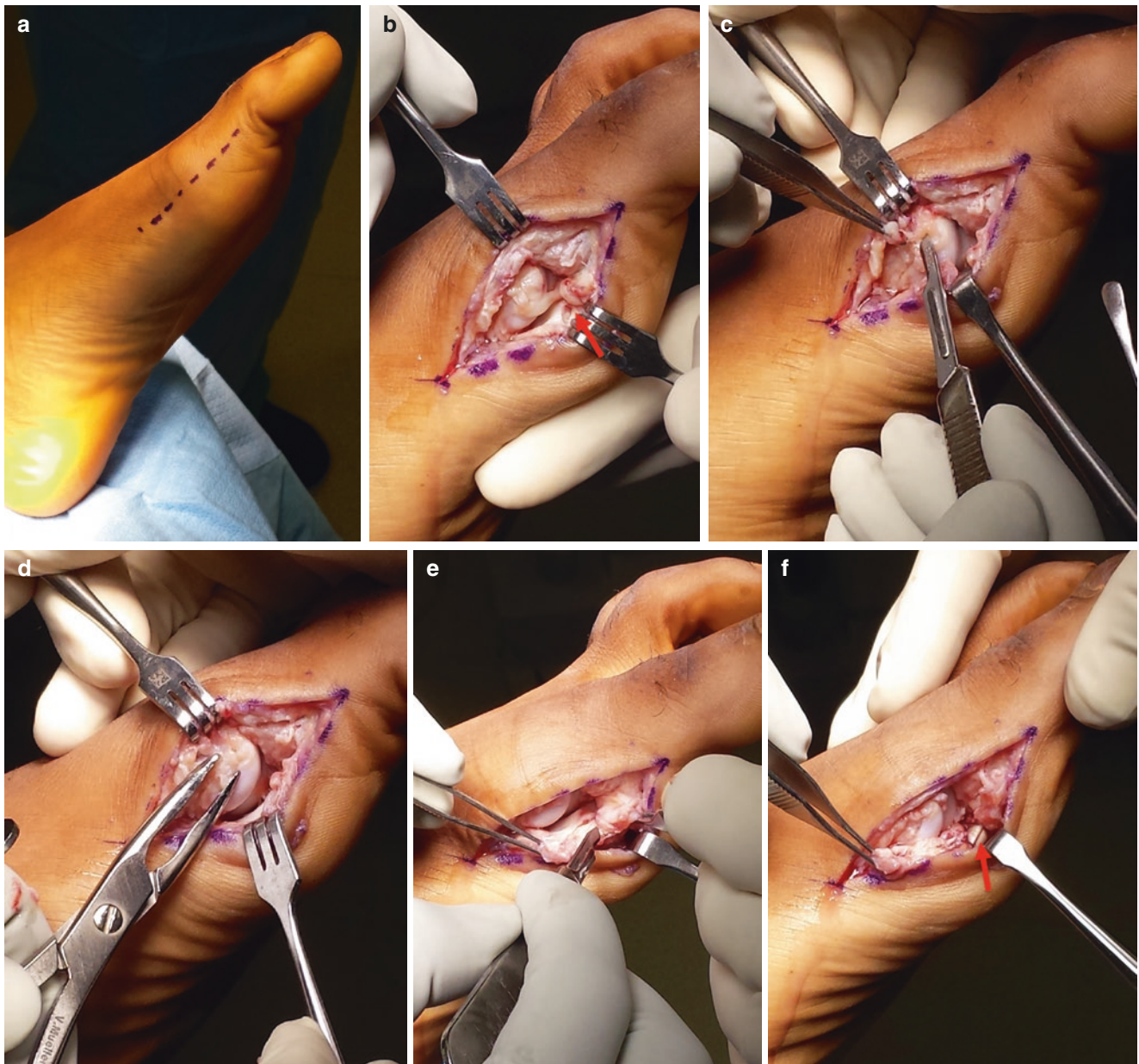
The collateral ligaments are often torn away from the metatarsal head during the initial dissection and the plantar plate tear is distal to the sesamoid complex. With proper dissection the plantar plate torn ends should be visualized along

with the FHL tendon distally. Redundant capsule is excised and there is typically a stump of plantar plate tissue remaining on the base of the proximal phalanx for subsequent repair. A small rongeur is used to remove a small amount of bone from the medial aspect of the metatarsal head to create a bleeding bony surface for ligament-to-bone healing. A freer can be placed into the lateral aspect of the sesamoid complex to see where it extends along the plantar aspect of foot as this will be where the plantar incision is centered.

If a traumatic hallux valgus is present with primarily medial soft tissue injury, a percutaneous adductor tenotomy is required to balance the hallux MTP joint along with a medial eminence resection as needed to obtain a capsulodesis [17]. The metatarsal head is inspected for the presence of OCD lesions and the FHL is examined for any tears. If one of the sesamoids is fractured or fragmented, one pole of the sesamoid should be preserved as a smaller distal pole is amenable to excision. A small drill hole in the larger proximal pole may permit soft tissue repair to the sesamoid bone. If complete sesamoidectomy is necessary, the abductor hallucis tendon should be transferred into the soft tissue defect of the excised sesamoid to allow the abductor to function as a plantar restraint [17]. In this scenario we would also recommend that an adductor tenotomy be performed.

A plantar incision is then made along the lateral border of hallux MTP-sesamoid complex just lateral to the weight-bearing surface of the hallux to decrease wound-healing complications (Fig. 37.5). A knife is used for skin incision followed by dissection scissors to spread down to fascia and dissect out the plantar lateral cutaneous nerve and its associated branches. It is important to have direct access to the plantar aspect and sesamoid complex while retracting the nerves laterally under a self-retaining retractor to prevent injury. If sufficient healthy tissue exists to perform a primary repair, three 2-0 non-absorbable sutures (Fiberwire, Arthrex Inc., Naples, FL) are placed in a figure-of-eight fashion through the plantar capsule with sutures starting lateral to medial to reduce injury to the nerve. Small needles are needed due to the small size of the operative field along with loop magnification for surgeon visualization of key neurovascular structures. Each pair of sutures are tagged and clamped separately for later identification. A 2-0 Vicryl suture is used to approximate the deep edges of the plantar plate.

A series of two to three 2-0 non-absorbable sutures in a figure-of-eight fashion are placed in the medial aspect of the sesamoid complex through the previously used medial approach (Fig. 37.6). Sutures are placed in both distal and proximal aspects of the plantar plate. Each pair of sutures are tagged and clamped separately for later identification. Sutures are left out of the MTP joint as they can potentially cause an inflammatory joint reaction and post-operative

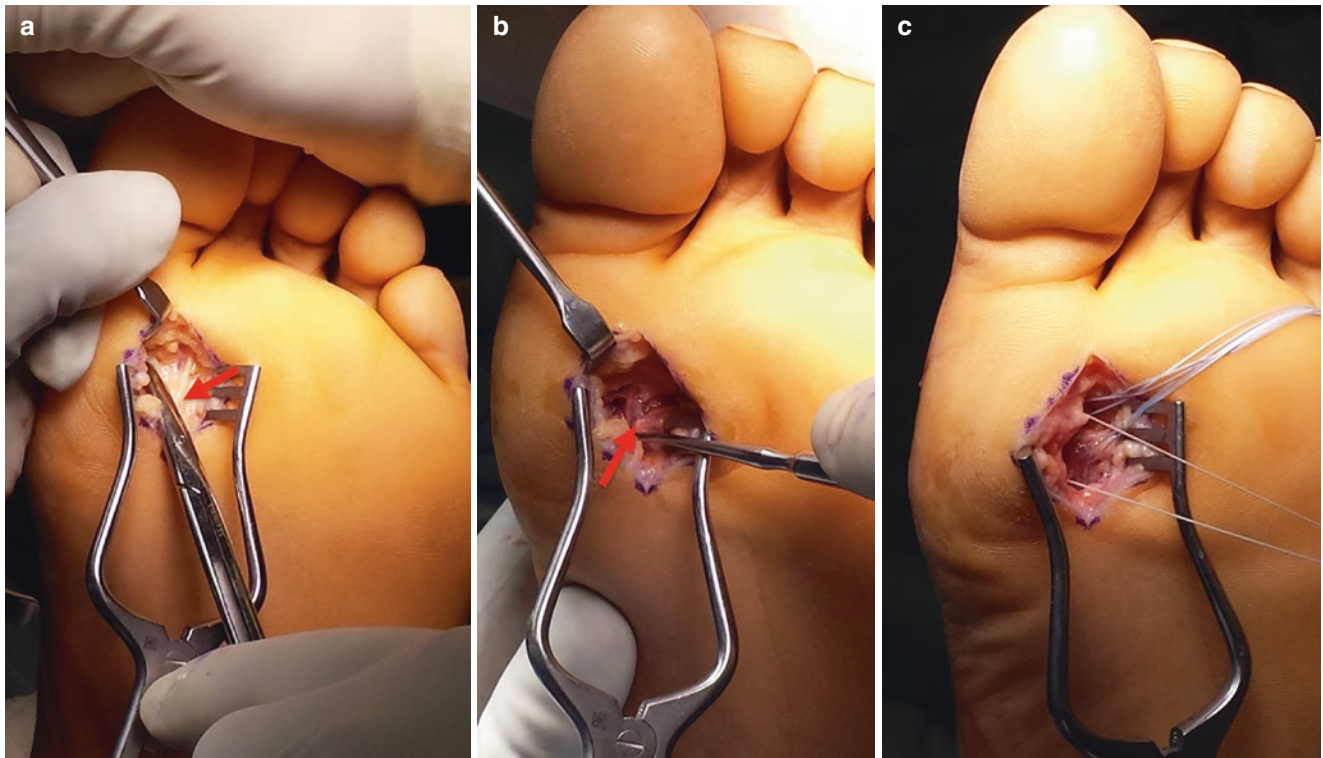


**Fig. 37.4** (a) During turf toe open surgical reconstruction, a medial incision is first made using a longitudinal incision just plantar to midline extending from the metatarsal neck to the flexion crease at the base of the hallux. (b) A capsulotomy is performed extending from the metatarsal neck to the mid-phalanx to provide complete visualization of the sesamoid complex (*red arrow* indicates plantar plate rupture distal to

medial sesamoid). (c) Remaining collateral ligaments are dissected off the metatarsal head and (d) a rongeur is used to remove a small amount of bone from the medial aspect of the metatarsal head to create a bleeding bony surface for ligament-to-bone healing. (e) Remaining plantar plate tissue is taken off of the proximal phalanx with (f) resultant visualization of the FHL tendon (*red arrow*)

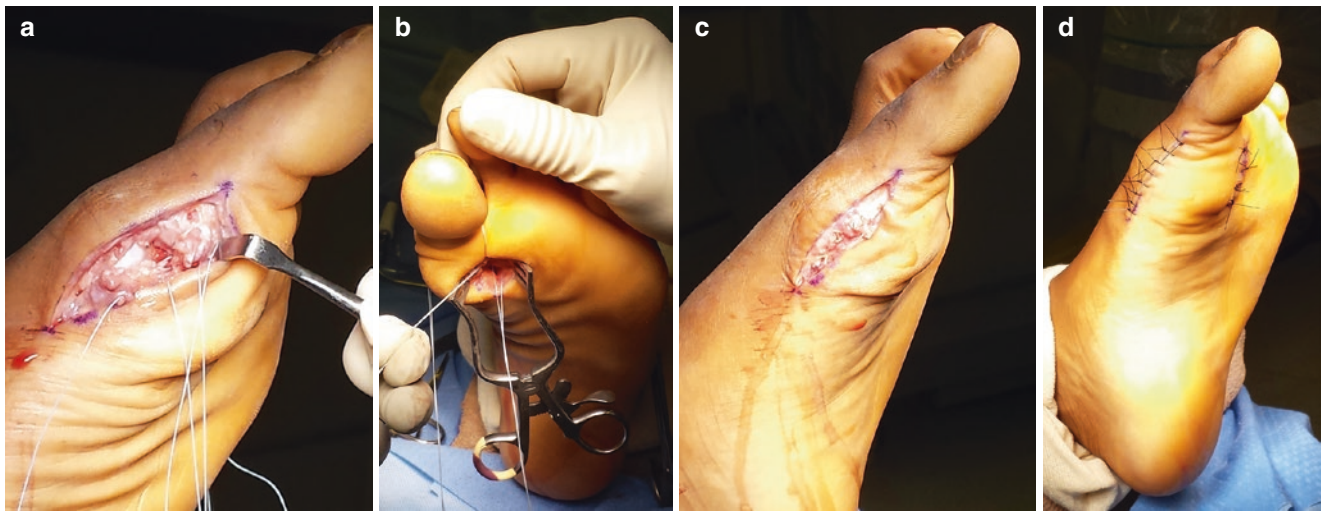
synovitis. Sutures are placed inferiorly away from the nerve with the goal of achieving a primary repair to the base of the proximal phalanx. If the tear cannot be primarily repaired due to inadequate healthy tissue a plantar plate advancement can be performed directly onto the base of the proximal phalanx using drill holes or suture anchors. If suture anchors are used they should be placed plantarly and confirmed fluoroscopically in order to avoid any supination deformity as the plantar complex is advanced distally.

The medial capsule is approximated and advanced using 2-0 Vicryl suture and this same suture can be used to repair any disruptions in the abductor hallucis tendon. After completion of suture placement medially, attention is then turned back to the plantar wound. The tagged pairs of sutures plantarly are sequentially tied with the hallux plantarflexed 10–15° followed by tying of the tagged pairs of sutures medially. 2-0 Vicryl sutures can be added to reinforce the medial repair as needed. After final repair the



**Fig. 37.5** (a) A plantar incision is then made along the lateral border of hallux MTP-sesamoid complex just lateral to the weight-bearing surface of the hallux and the branches of the plantar lateral cutaneous nerve are carefully dissected (*red arrow*). (b) The nerve is gently retracted laterally to get down to the plantar aspect of the sesamoid

complex (*red arrow*). (c) Three 2-0 non-absorbable sutures (*blue*) are placed in a figure-of-eight fashion through the plantar capsule with sutures starting lateral to medial to reduce injury to the nerve. A 2-0 Vicryl suture (*white*) is used to approximate the deep edges of the plantar plate

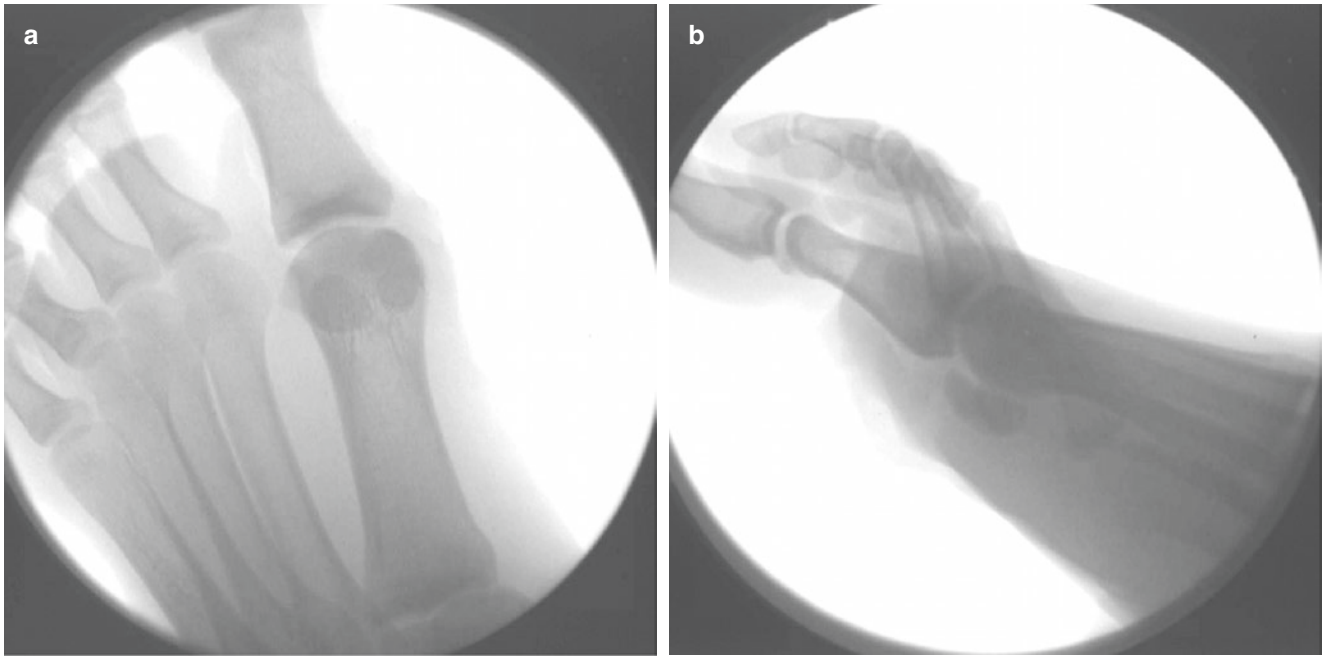


**Fig. 37.6** (a) A series of two to three 2-0 non-absorbable sutures in a figure-of-eight fashion are placed in the medial aspect of the sesamoid complex through the previously used medial approach. Sutures are placed in both distal and proximal aspects of the plantar plate. (b) After completion of suture placement medially, attention is then turned back

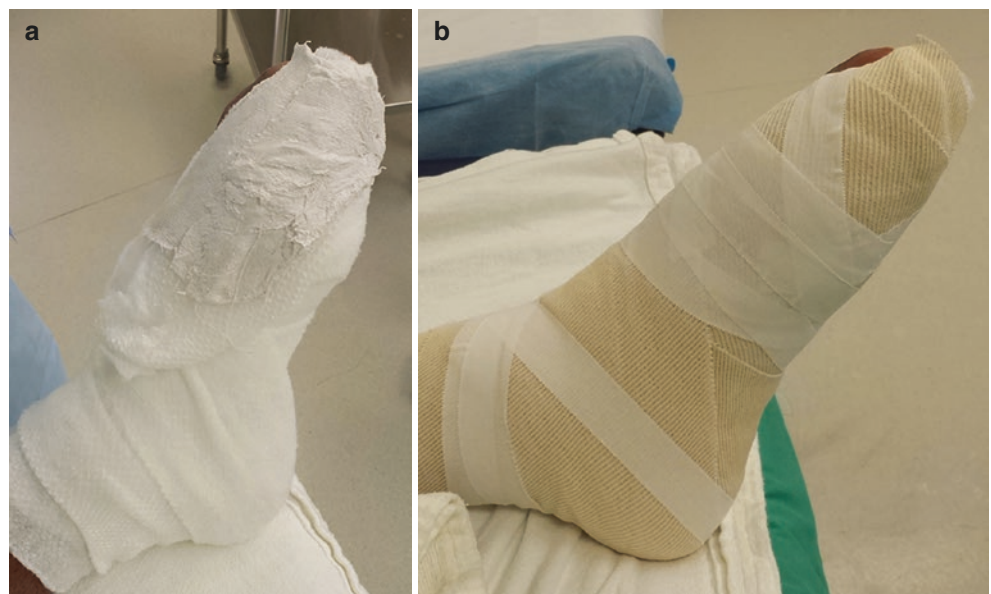
to the plantar wound and sutures are sequentially tied with the hallux plantarflexed 10–15° followed by tying of the tagged pairs of sutures medially. (c) After final repair the hallux should be aligned straight and slightly plantarflexed with maintenance of alignment (d) after complete skin closure

hallux should be aligned straight and slightly plantarflexed. The soft-tissue fixation can be tested by gently shucking the toe and dorsiflexing it to test tension. Intraoperative radiographs should demonstrate the anatomic location of the sesamoids relative to the MTP joint more distal than before surgery (Fig. 37.7). Gentle dynamic fluoroscopy can be used to assess joint stability throughout a range of motion and to visualize movement of the sesamoids with hallux dorsiflexion.

During closure, the plantar medial and lateral cutaneous nerves are checked to make certain they are intact and not under tension. 3-0 nylon sutures are used to close medial and plantar skin incisions followed by local anesthetic injection (10 cc combination of 5 cc 1% lidocaine and 5 cc 0.5% bupivacaine) into both wounds. Incision are dressed with a petrolatum dressing, gauze, and a 3-piece plaster hallux spica splint to immobilize the hallux MTP joint in 5–10° plantarflexion (Fig. 37.8). The spica splint is overwrapped in



**Fig. 37.7** (a) Intraoperative AP and (b) lateral radiographs after surgical repair should demonstrate anatomic reduction of the sesamoids relative to the MTP joint and location more distal than before surgery



**Fig. 37.8** (a) Incision are dressed with a petrolatum dressing, gauze, and a 3-piece plaster hallux spica splint to immobilize the hallux MTP joint in 5–10° plantarflexion. (b) The spica splint is overwrapped in cast padding and covered with a soft bandage and reinforced with silk tape

cast padding and covered with a soft bandage and reinforced with silk tape.

Complications of surgery include iatrogenic injury to the plantar medial and/or lateral cutaneous nerves during dissection and wound-healing complications. Superficial and deep infections are rare complications that can be decreased by strict non-weight bearing precautions for 4 weeks after surgery. Late sequela of turf toe injury includes decreased athletic performance, degenerative arthritis, decreased range of motion, and hallux valgus deformity [20, 23].

---

## Rehabilitation

After surgery, there is a delicate balance between incision protection and early range of motion to reduce arthrofibrosis between the capsule and the MTP sesamoid complex that must be directed by the surgeon working together with physical therapist and/or trainer. An important aspect of recovery is setting appropriate and realistic player expectations regarding return to play. Patients are non-weight bearing in a CAM boot for 4 weeks with the hallux MTP joint in plantarflexion. We typically begin passive range of motion exercises by 5–7 days after surgery depending on patient compliance and wound healing status. Passive plantar flexion is initiated as soon as the incision is completely healed with transition to active range of motion by 4 weeks. Pool therapy can at this time with gentle weight bearing and gait exercises. Patients are moved into an accommodative shoe with orthotic or turf toe plate at 8 weeks with a return to running activity at 12 weeks. Return to contact sports is permitted at 3–4 months after surgery and contact athletes may require up to 6–12 months for full recovery. Prevention of recurrent injury focuses on taping and shoe-wear modifications as needed to reduce hallux MTP dorsiflexion.

---

## Evidence

There is limited evidence in the literature regarding outcomes following turf toe management with studies consisting of retrospective case series (Level IV). Coker et al. reported 18 injuries of the first MTP joint complex that resulted in 92 missed practices and 7 missed games during the course of 3 football seasons [24]. Nine athletes had turf toe injuries resulting in long-term joint pain and stiffness with athletic activity. Clanton et al. reported on a series of 20 athletes with turf toe injuries and found that 50% of patients had persistent hallux MTP pain and stiffness at 5 year follow-up [25]. In a survey of 80 active National Football League (NFL) players, Rodeo et al. reported that 36 players (45%) indicated that they had suffered a turf toe injury

during their professional career with the majority of injuries (83%) occurring on artificial turf [3].

Anderson reported on the outcomes of 19 elite athletes with Grade III turf toe injuries and found that 9 required surgical repair and all but 2 returned to full athletic activity with restoration of joint stability without surgical complications [21]. Brophy et al. investigated the relationship between turf toe and plantar foot pressures and found that turf toe was associated with significantly decreased MTP motion (7.8° less motion with turf toe) and increased hallux pressures (121 kPa higher in turf toe) in a group of 44 National Football League (NFL) players [4]. In a cadaveric model of plantar plate injury, Waldrop et al. found that an increase of 3 mm in the distance from the sesamoids to the proximal phalanx was significant and predictive of a severe plantar plate injury to at least three of the four ligaments of the plantar plate complex [8].

Recently, an analysis of the NCAA's Injury Surveillance System (ISS) for five football seasons (2004–2009) showed the overall incidence of turf toe injuries in football players to be 0.062 per 1000 athlete-exposures [7]. Athletes were nearly 14 times more likely to sustain a turf toe injury during games compared with practice with an average of 10.1 days of athletics lost to injury. Fewer than 2% of injuries required operative intervention according to the authors.

---

## Summary

1. The evaluation and treatment of injuries to the first MTP capsule ligamentous complex (turf toe) are improving as these injuries are becoming better recognized and diagnosed.
2. The most common mechanism of injury is hyperextension of the hallux MTP joint as an axial load is applied to the heel with the foot in an fixed equinus position.
3. Grade I injury is a sprain or attenuation of the plantar capsule ligamentous complex of the hallux MTP joint, Grade II injury is a partial rupture of the plantar soft tissues, and Grade III injury is a complete rupture of the plantar plate structures with significant swelling, hallux flexion weakness, and frank joint instability.
4. Non-operative treatment for all grades of turf toe injury begins with rest, ice, compression, elevation (RICE), and non-steroidal anti-inflammatories (NSAIDs) to reduce pain and swelling.
5. Surgical repair of turf toe is reserved for Grade III injuries that fail conservative treatment or cases that involve one or a combination of the following: large capsular avulsion with unstable MTP joint, diastasis of bipartite sesamoid, diastasis of sesamoid fracture, retraction of sesamoid, traumatic hallux valgus deformity, vertical instability (positive Lachman test), loose body in MTP joint, or chondral injury in MTP joint.

6. With correct and timely diagnosis and treatment combined with sport-specific rehabilitation, athletes can recover from turf toe injuries and return to play at their pre-injury level of competition.

No part of this study has been submitted or duplicated elsewhere prior. This study has been read and approved by all authors and each author believes that the manuscript is valid and represents honest work. No research or industry funds were received in relation to the research or publication of this study.

**Relevant Disclosures Outside of the Current Study** Dr. Hsu has no disclosures; Dr. Anderson receives royalties from Arthrex, Inc., DJ Orthopaedics, and Wright Medical Technology, Inc. and is a paid consultant for Amniox, Wright Medical Technology, Inc., and Arthrex, Inc.

## References

- Bowers Jr KD, Martin RB. Turf-toe: a shoe-surface related football injury. *Med Sci Sports*. 1976;8(2):81–3.
- Frimenko RE, Lievers W, Coughlin MJ, Anderson RB, Crandall JR, Kent RW. Etiology and biomechanics of first metatarsophalangeal joint sprains (turf toe) in athletes. *Crit Rev Biomed Eng*. 2012;40(1):43–61.
- Rodeo SA, O'Brien S, Warren RF, Barnes R, Wickiewicz TL, Dillingham MF. Turf-toe: an analysis of metatarsophalangeal joint sprains in professional football players. *Am J Sports Med*. 1990;18(3):280–5.
- Brophy RH, Gamradt SC, Ellis SJ, Barnes RP, Rodeo SA, Warren RF, et al. Effect of turf toe on foot contact pressures in professional American football players. *Foot Ankle Int*. 2009;30(5):405–9.
- Doty JF, Coughlin MJ. Turf toe repair: a technical note. *Foot Ankle Spec*. 2013;6(6):452–6.
- Frimenko RE, Lievers WB, Riley PO, Park JS, Hogan MV, Crandall JR, et al. Development of an injury risk function for first metatarsophalangeal joint sprains. *Med Sci Sports Exerc*. 2013;45(11):2144–50.
- George E, Harris AH, Dragoo JL, Hunt KJ. Incidence and risk factors for turf toe injuries in intercollegiate football: data from the national collegiate athletic association injury surveillance system. *Foot Ankle Int*. 2014;35(2):108–15.
- Waldrop 3rd NE, Zirker CA, Wijdicks CA, Laprade RF, Clanton TO. Radiographic evaluation of plantar plate injury: an in vitro biomechanical study. *Foot Ankle Int*. 2013;34(3):403–8.
- Meyers MC, Barnhill BS. Incidence, causes, and severity of high school football injuries on FieldTurf versus natural grass: a 5-year prospective study. *Am J Sports Med*. 2004;32(7):1626–38.
- Aper RL, Saltzman CL, Brown TD. The effect of hallux sesamoid excision on the flexor hallucis longus moment arm. *Clin Orthop Relat Res*. 1996;325:209–17.
- Stokes IA, Hutton WC, Stott JR, Lowe LW. Forces under the hallux valgus foot before and after surgery. *Clin Orthop Relat Res*. 1979;142:64–72.
- Stokes IA, Hutton WC, Stott JR. Forces acting on the metatarsals during normal walking. *J Anat*. 1979;129(Pt 3):579–90.
- Mullen JE, O'Malley MJ. Sprains – residual instability of subtalar, Lisfranc joints, and turf toe. *Clin Sports Med*. 2004;23(1):97–121.
- Nihal A, Trepman E, Nag D. First ray disorders in athletes. *Sports Med Arthrosc*. 2009;17(3):160–6.
- Douglas DP, Davidson DM, Robinson JE, Bedi DG. Rupture of the medial collateral ligament of the first metatarsophalangeal joint in a professional soccer player. *J Foot Ankle Surg*. 1997;36(5):388–90.
- Fabeck LG, Zekhnini C, Farrokh D, Descamps PY, Delincé PE. Traumatic hallux valgus following rupture of the medial collateral ligament of the first metatarsophalangeal joint: a case report. *J Foot Ankle Surg*. 2002;41(2):125–8.
- Anderson RB, Shawen SB. Great-toe disorders. In: Porter DA, Schon LC, editors. *Baxter's the foot and ankle in sport*. Philadelphia: Elsevier Health Sciences; 2007. p. 411–33.
- Crain JM, Phanco JP, Stidham K. MR imaging of turf toe. *Magn Reson Imaging Clin N Am*. 2008;16(1):93–103.
- McCormick JJ, Anderson RB. Rehabilitation following turf toe injury and plantar plate repair. *Clin Sports Med*. 2010;29(2):313–23.
- McCormick JJ, Anderson RB. The great toe: failed turf toe, chronic turf toe, and complicated sesamoid injuries. *Foot Ankle Clin*. 2009;14(2):135–50.
- Anderson RB. Turf toe injuries of the hallux metatarsophalangeal joint. *Tech Foot Ankle Surg*. 2002;1(2):102–11.
- McCormick JJ, Anderson RB. Turf toe: anatomy, diagnosis, and treatment. *Sports Health*. 2010;2(6):487–94.
- Coughlin MJ, Kemp TJ, Hirose CB. Turf toe: soft tissue and osteo-cartilaginous injury to the first metatarsophalangeal joint. *Phys Sportsmed*. 2010;38(1):91–100.
- Coker TP, Arnold JA, Weber DL. Traumatic lesions of the metatarsophalangeal joint of the great toe in athletes. *Am J Sports Med*. 1978;6(6):326–34.
- Clanton TO, Butler JE, Eggert A. Injuries to the metatarsophalangeal joints in athletes. *Foot Ankle*. 1986;7(3):162–76.

Erik C. Nilssen and William K. Whiteside

**Abstract**

Metatarsalphalangeal (MTP) joint disorders involving the 2nd through 5th toes are not the most common injury of the ankle and foot in athletes but can be problematic if not recognized. A good history and physical examination along with weight bearing radiographs of the affected foot can identify most of these disorders. Generalized synovitis of the forefoot can occur with overuse and can result in plantar plate instability. Plantar plate injuries can be difficult to treat but surgical options do exist and continue to evolve. Freiberg disease can be likened to avascular necrosis of the hip, and should be considered specifically in the athlete with pain localized over the second MTP joint. Bunionettes typically respond well to conservative treatment in the athlete with over the counter pads and callus care; however, surgical options are well described based on the type with good outcomes.

**Keywords**

Metatarsals • Metatarsalgia • Plantar plate • Freiberg disease • Bunionettes

**Introduction**

In any sport, repetitive activities often lead to excessive stress on joints, bones and soft tissues alike in the forefoot of the athlete. In the metatarsals (MTs), the most recognizable condition that athletes are susceptible to are stress fractures but other entities includes plantar plate disruption with instability, synovitis, Freiberg disease, and bunionettes. All together, metatarsalgia is not that common in the healthy, young athletic population and when suspected one should think of Freiberg disease or metatarsalphalangeal (MTP) joint instability [1–4].

**Etiology and Pathomechanism**

Occasionally acute traumatic events can lead to disorders of the metatarsals 2–5, but they are more commonly from chronic overuse. The second ray is most commonly involved due to its position next to the hallux and that it is typically the longest metatarsal. This can lead to pressure transfer and overload in the lesser MTs or to increase in local pressure beneath the MT head respectively. The repeat stresses on this joint can lead to synovitis which can cause attenuation of the plantar plate and associated collateral ligaments. This results in MTP joint instability and deformity in the form of a cross over toe or frank dislocation over time [1, 2, 5].

Freiberg disease is a specific condition of the lesser metatarsal head most commonly involving the second metatarsal (Fig. 38.1). This disease usually is seen in adolescent females. The exact etiology is unknown but osteonecrosis of the subchondral bone leads to joint collapse and degenerative changes similar to avascular necrosis in the femoral head. It is thought that increased pressure on the metatarsal head possibly results in vascular disruption [6].

E.C. Nilssen, MD • W.K. Whiteside, MD (✉)  
Andrews Research and Education Institute,  
1040 Gulf Breeze Pky, Ste 210, Gulf Breeze, FL 32561, USA  
e-mail: [nilssenmd@yahoo.com](mailto:nilssenmd@yahoo.com); [will.whiteside@yahoo.com](mailto:will.whiteside@yahoo.com)



**Fig. 38.1** Freiberg disease in the second MT head of a 14 year old cross country runner. Figure from personal collection of author

A bunionette is similar to hallux valgus but involves the fifth metatarsal instead. Medial based pressure on the fifth metatarsal or hallux valgus indirectly can lead to this condition. Anatomically, an enlarged lateral condyle of the fifth metatarsal or a widened 4–5 intermetatarsal angle can result in symptomatic protrusion of the joint [7].

## Symptoms

Patients describe symptoms ranging from pain to a sense of fullness in the MTP joints. Typically the symptoms are exacerbated by the sport and activities that cause extreme motions in the forefoot such as sprinting or the demi-pointe position in dancers. They also may notice deformity of the toes in the form of swelling, or a change in position of the toe over time as in cross over toes due to instability. Callosities may occur dorsally, plantarly, or laterally as in bunionettes. The clinician must remember that Freiberg disease can be symptomatic for as long as 6 months before it appears on x-ray and should be considered in the young athlete with forefoot pain.

## Diagnostics, Classification

A thorough physical exam of the forefoot is essential to evaluate the metatarsals and metatarsal joints specifically. Inspection should be done in the weight bearing as well as seated position with a focus on the position of each of the joints of the toes and noting any deformity of the hallux is

essential to a diagnosis. Several disorders in the MTP joints are the result of hallux valgus or rigidus which can lead to overload of the lesser toes especially the second toe. In plantar plate disruption, initially the toes deviates medially most commonly and with time dorsally, and will cross over the great toe with time. Palpation is performed next focusing on the location of pain whether it is over the joint rather than the webspaces which would more likely be a neuroma. In athletes, it is often difficult to localize the pain during the initial examination since the pain often occurs only during the sport. It is helpful to have the athlete go through the motions of the sport prior to physical exam to identify the exact location of their symptoms. During palpation, check range of motion and for callosities both dorsal and plantar which can be seen with overload; inspect for bony prominences over the lateral condyles and specifically over the lateral fifth MTP joint as in bunionettes. If after an acute injury, usually after extreme dorsiflexion positions of the forefoot, ecchymosis can be seen with plantar plate disruption. Synovitis is often accompanied by swelling of the MTP joints, mild erythema may be seen, and limited range of motion which can be evident in Freiberg disease as well. Nontraumatic synovitis may deserve a rheumatologic work up on a case by case basis. The tightness of the calf with the Silfverskiold test should be evaluated since this can lead to excessive pressure on the forefoot.

Thompson and Hamilton described the anterior drawer test which is a provocative maneuver that can be used to examine the stability of the MTP joint [8]. The examiner holds the head of the metatarsal between their index finger and thumb, and with the other hand grasps the base of the proximal phalanx and applies dorsally directed pressure checking the translation during this maneuver in comparison to the other toes as well as the reproduction of the patient's painful symptoms. Doty and Coughlin described a clinical staging of MTP joint instability where Stage 0 has no malalignment; Stage 1 with mild malalignment, widened web space and medial deviation; Stage 2 with moderate malalignment, deformity in any plane, hyperextension; Stage 3 with severe malalignment, dorsal deformity; Stage 4 with severe malalignment, dislocation, and a fixed hammertoe [2]. On physical exam one will notice joint pain, and swelling in Stage 0 through 2, and decreasing toe purchase as demonstrated by the paper pull-out test where a strip of paper is positioned under the affected toe and the examiner asks the patient to resist pulling the paper from under the toe [2, 9, 10].

Imaging in the form of three weight bearing radiographs of the foot should be evaluated for forefoot disorders. A bunionette may be diagnosed radiographically (Fig. 38.2) as an enlarged fifth metatarsal head (Type I), as a bowing of the metatarsal (Type II), or as a widened 4–5 intermetatarsal angle of more than 8° (Type III) [7]. Radiographs can also be evaluated for widening, narrowing, degenera-



tion, or deviation of the MTP joints indicating synovitis, dislocation or subluxation, Freiberg disease, or collateral ligament/plantar plate injury respectively. An appreciation of the cascade of the metatarsals can also be determined on the standing AP of the foot looking at the lengths of the metatarsals and for any splaying of the rays.

Freiberg disease can be classified radiographically into 4 types: Type I where there is localized osteonecrosis of the metatarsal head without articular cartilage defect; Type II where there is collapse of the head with osteophytes development over the dorsal aspect of the MT head; Type III demonstrates head collapse and articular cartilage destruction; Type IV involves multiple heads and a congenital epiphyseal disorder should be considered [6].

MRI can be helpful to confirm Freiberg disease or platar plate disruption [6, 11]. We believe that a 3 Tesla magnet is essential when looking at the plantar plate due to the intricate anatomy of the MTP joint.



**Fig. 38.2** Type 1 bilateral bunionette deformities. Figure from personal collection of author

## Therapy

The goals of nonsurgical treatment of metatarsal disorders include reduction of inflammation and pressure on the metatarsals. This can be in the form of shoe wear selection of softer, flexible shoe uppers or modification of current shoes with stretching the toebox or the addition of a custom made orthotics with metatarsal pads, or bars. Taping of the affected toe to the neighboring toes can be utilized or single toe sling type taping to control instability (Fig. 38.3). A Budin splint can also be used for instability. Bunionettes may respond to an over the counter callus pad applied to the irritated area or by self paring of calluses over the hypertrophied area. Physical therapy with a focus on triceps surae stretching to lengthen muscles and decrease pressure on the forefoot can improve symptoms as well. Cross training with lower impact activities and a decrease in total mileage may be required. Short periods of rest in a cast with non weight bearing, or a walking boot can also be used depending on the severity at the time of presentation. Nonsteroidal anti-inflammatory drugs in oral, topical, or patch form can be used for symptomatic treatment. Corticosteroid injections can be used for synovitis in particular but the clinician must keep in mind the risk of further attenuation of the capsular structures [10, 12]. If the athlete fails to respond to these modalities, then surgical options can be considered.

The surgical treatment of MTP instability is based on clinical assessment of the deformity. Mild deviation of the toe can be treated with capsular reefing with synovectomy. If the MTP joint is subluxed, capsular reefing and a flexor tendon transfer is often required. The flexor to extensor transfer or Girdlestone-Taylor procedure is described in the lesser toe section. If the deformity is completely dislocated or fixed, a Weil osteotomy is often required. For each of these procedures, a dorsal longitudinal incision is made over the MTP joint, typically the EDL is Z-lengthening during this procedure and the EDB is transected. A dorsal capsulotomy is performed. If there is medial deviation of the toe, the medial capsule and collaterals are released completely and the



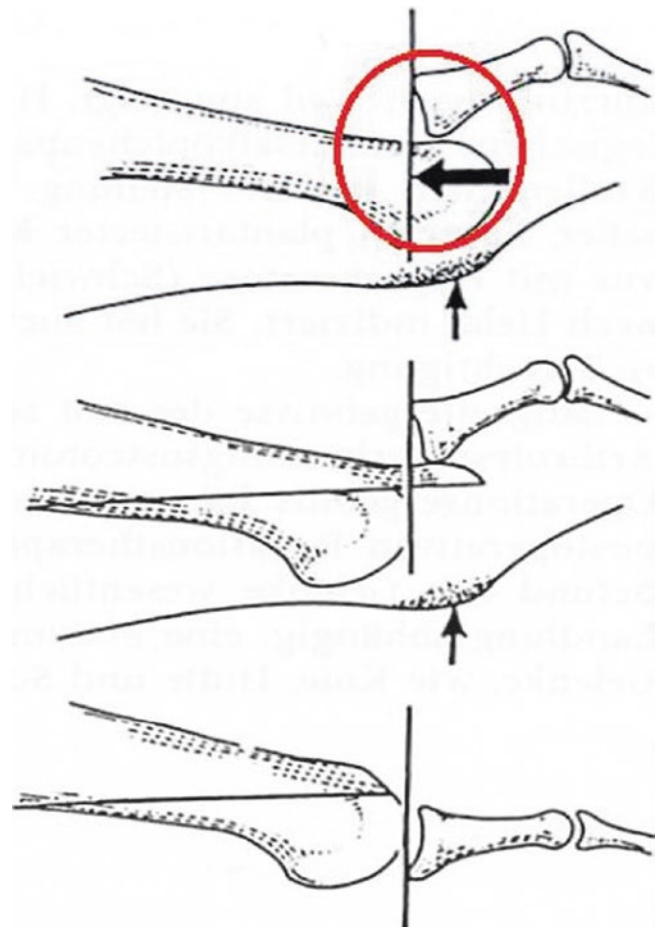
**Fig. 38.3** (a) Sling type taping for single toe MTP joint instability-A. dorsal view, (b) plantar view. Figure from personal collection of author

lateral capsule is reefed. Numerous techniques have been described for cross over toe due to plantar plate and collateral ligament disruption with no clear consensus. One of the most common techniques if there is cross over component of the toe, due to plantar plate and collateral insufficiency hyperextension, or dorsal-plantar instability is an extensor digitorum longus (EDL) to extensor digitorum brevis (EDB) transfer for varus/valgus toe deformity as a result of plantar plate instability. This involves a similar approach as described above with capsulotomy and release of the tight collateral and plantar plate. A drill hole is then created transversely from medial to lateral in the proximal phalanx in a medial deviated toe and the distal end of the EDL is passed through this drill hole and sutured to the EDB while the toe is held in a reduced position with a K-wire across the MTP joint. This procedure creates a checkrein on the lateral aspect of the MTP joint with the EDL tendon [3]. Patients are placed in a soft dressing and allowed heel weight bearing. The K-wire is removed at 6 weeks and taping of the toe in a corrected position can be continued on an as needed basis.

Several implant companies have recently developed plantar plate repair systems which involve anatomic unilateral or bilateral collateral and plantar plate repair through tensioning and advancement of the soft tissue structures with suture through bone tunnels in the proximal phalanx. Exposure of the MTP joint as described above and elevating the MTP head out of the wound with either Hohmanns or a McGlamry elevator for improved exposure of the plantar plate is necessary for these procedures. Joint distraction devices are used for adequate exposure. Often a Weil osteotomy is performed to allow improved exposure of the plantar plate as well. The specific technique guides can be referenced for further information on these surgical techniques.

A Weil osteotomy (Fig. 38.4) is a shortening osteotomy that can be used for an irreducible joint after soft tissue procedures or to reduce the local pressure that resulted in the condition with the goal of maintaining the normal cascade of the rays with the second ray near the same level as the first MT. If this is not performed a soft tissue procedure will often fail [13]. A longitudinal incision is made typically in the webspace if more than one metatarsal is involved. An incision directly over the MTP joint can also be used with single MTP joint involvement. A capsulotomy is performed and plantarflexion of the toe allows exposure of the metatarsal head. A hand held oscillating saw is used to create an osteotomy parallel to the weight bearing surface of the foot aiming for the heel. The osteotomy begins at the dorsal aspect of the MT articular surface. The capital fragment is then displaced proximally and fixed with screws. The overhanging bone is then trimmed with a rongeur. The capsule is then reefed as necessary and closed. If a Weil osteotomy is done, we recommend 6 weeks of heel weight bearing.

Synovitis typically responds to conservative measures. However, in the nonresponsive patient the affected MTP



**Fig. 38.4** Weil osteotomy (With kind permission of Springer Science + Business Media: Arnold [13])

joint is explored through a dorsal incision with capsulotomy of the affected MTP joint with complete synovectomy. The collateral ligaments may need to be released, and EDB and EDL lengthening may be needed for reduction of a subluxed or dislocated joint. If the involved metatarsal is excessively long and plantar callus under the MTP head is present, a Weil osteotomy is performed for pressure relief as described above. Post operatively the patient is placed in a soft dressing in a walking boot for heel weight bearing for a minimum of 2 weeks. Range of motion exercises are started at this point if only a soft tissue procedure was performed.

Freiberg disease can be treated with MTP joint synovectomy and core decompression if minimal metatarsal head deformity is present. If cartilage loss is present, a dorsiflexion metatarsal neck osteotomy can be done to rotate native cartilage into the articulation and temporarily held with K-wires. If a significant portion of the metatarsal head is involved, metatarsal head resection as in a DuVries arthroplasty can be considered along with capsular or flexor/extensor tendon interposition. The risks with these procedures include permanent instability and transfer lesions to the other toes especially in the competitive athlete. If a synovectomy alone

was performed a soft dressing is applied in a walking boot to allow for heel weight bearing for a minimum of 2 weeks until the soft tissues heal. If bony work was done, a 6 week minimum of immobilization is required allowing heel weight bearing in a walking boot.

Bunionettes are typically treated based on their radiographic stage. Type I bunionettes are treated with lateral partial condylectomy to produce a smooth lateral border taking care to excise the inflamed bursa as well in the soft tissues. Type II bunionettes are treated with a distal chevron osteotomy typically as in hallux valgus surgery. This is done through a lateral approach with resection of the lateral eminence as in Type I deformities. A distal based chevron osteotomy is performed, and can be fixed with K-wires or a single screw, and the capsule is tightly closed. The limits of this procedure are due to the small nature of the fifth metatarsal and risk of avascular necrosis of the metatarsal head. Type III bunionettes are treated with a metatarsal shaft osteotomy. This osteotomy needs to be a long oblique cut that allows solid bony contact. The osteotomy should not be performed in the proximal diaphysis due to the tenuous blood supply of the proximal fifth metatarsal. As with hallux valgus surgery, there is a risk of recurrence, incomplete pain relief, and painful scar. Post operatively for bunionette surgery, the patients are placed in a dorsal and plantar plaster sandwich for 6 weeks in which they can bear weight on their heel. At the 6 week mark, the K-wire is removed for Type II and III bunionettes.

The primary goal is pain relief in these surgeries, and cosmesis of limited concern. Recurrence and stiffness do occur and are the primary complications. It is important to remember that associated hallux valgus or rigidus if not addressed can lead to continued second toe malalignment. Transfer metatarsalgia to the other MTP joints can also occur if the second ray is excessively shortened or during bunionette correction if the metatarsal head is dorsally translated. Nonunion and malunion is also of concern with bony procedures in the forefoot but are usually rare.

---

## Rehabilitation and Back-to-Sport

Rehabilitation is typically instituted at the 2 week mark for soft tissue surgery as in synovitis and Freiberg infarction that only involves a synovectomy. This involves patient self manipulation of the involved toes after surgical wound healing. If a plantar plate repair is undertaken then 6 weeks of immobilization is required. This is similar for bunionette correction and Weil osteotomies where therapy is initiated after the K-wire is removed at the 6 week mark. Radiographic healing should be confirmed if osteotomies have been performed as in bunionette surgery and Freiberg disease. Athletic activity is then increased in a graduated manner as swelling and pain diminish. It is important to remember, that

surgical treatment in such a small area as the lesser metatarsals take time to heal and some element of stiffness and swelling can remain for a long period of time sometimes up to a year. This can be concerning to athletes; and preoperative counseling is necessary so expectations of the surgeon and athlete align. In general, it takes an athlete 3–6 months to return to sport without restrictions.

---

## Prevention

The goal should be to avoid problems by education of the athlete on good footwear and proper training and conditioning. Highly flexible shoes with narrow toe boxes should not be worn to prevent 2–5 MT conditions and injuries.

---

## Evidence

As a whole, there is limited if any Level I evidence with Grade A recommendations on treating disorders of MTs 2–5. Most of the evidence discussed below are Level IV studies and Level II at best with a limited number of patients and studies regarding athletes in general.

When diagnosing plantar plate instability Sung et al. found that MRI is an accurate and valid test in evaluated plantar plate disruption with sensitivity of 95 % and specificity of 100 % (Level II) and a Grade A recommendation can be made for this modality based on this study [11]. Hofstaetter et al. found that Weil osteotomies are a safe and effective treatment method for subluxed or dislocated MTP joints with >80 % good to excellent results at 1 and 7 year follow up (Level IV) with a Grade B recommendation [14]. Studies where a Weil osteotomy was done specifically for plantar plate instability are limited. Nery et al. described a prospective study on direct plantar plate repair with a Weil osteotomy on 28 patients, 55 MTP joints, with at least 12 month follow up found that there was improvement in pain, MTP joint hyperextension, and instability in 68 % of patients [15]. This was a level II study and can be given a Grade A recommendation. Barca et al. studied EDL transfer to treat 30 crossover toes with average 2 year follow up had 83 % good to excellent result with only one recurrence (Level IV); Grade B recommendation [16].

Bunionettes are treated based on their severity. Kitaoka and Holiday reported results in 16 patients who underwent lateral condylar resection [17]. 23 % of patients had recurrent or persistent lateral pain (Level IV). Boyer and DeOrto achieved correction of the 4–5 IM angle from an average of 9.1° to an average of 1.4°, using the chevron osteotomy and securing it with a bioabsorbable pin (Level IV) [18]. Coughlin reported on 30 patients treated for Type II and III bunionettes with oblique metatarsal osteotomies. The 4–5 IM angle improved from an average of 16° to 0.5°. All osteotomies

healed by 8 weeks (Level IV) [19]. Cooper recently reported on a subcapital oblique osteotomy of the fifth metatarsal for Type I bunionette deformity and found it to provide reliable clinical results for correction of painful type I bunionette deformity with 88% patients having no limitation in activity (Level IV) [20]. A Grade B recommendation exists for bunionette surgery based on the Level of evidence in these studies.

Carmont et al. did an extensive review of the literature on Freiberg disease in 2009 and graded the level of evidence for the various treatment modalities [6]. Freiberg initially used core decompression alone for treatment of the disease (Level IV evidence). This was later modified to include debridement of avascular bone and K-wire fixation by Helal et al. [21]. Sproul et al. found good results in athletes were debridement and synovectomy was performed where all patients had improvement of symptoms (Level IV evidence) [22]. Core decompression has a Grade I recommendation and Grade B recommendation for open dorsal approach with debridement.

When there is later stage disease, there is a Grade B recommendation based on Level IV studies of wedge osteotomies to rotate normal cartilage into the articulation. 15 patients in one study were all able to return to sporting activities that they were unable to do prior to surgery Kinnard [23]. Interpositional arthroplasty is reserved for the arthritic joint and has been described by Thompson and Hamilton with good pain relief but limited range of motion. The evidence for this is Level IV with a Grade B recommendation [8, 24].

## Summary

- Aside from stress fractures, MTP joint instability due to plantar plate injury, Freiberg disease, synovitis, and bunionettes should be considered in athletes with forefoot pain. A good history and physical examination can usually identify these disorders.
- Plantar plate instability can be treated with taping and rest in the acute stage however surgical options are evolving with good short term results.
- Freiberg disease is an uncommon condition but should be considered specifically in the female athlete with forefoot pain localized over the second MTP joint without radiographic evidence of pathology.
- Synovitis can lead to disability and pain in the athlete resulting in attenuation of surrounding tissue causing plantar plate tears. If acute, it usually resolves with conservative measures.
- Bunionettes typically respond to conservative treatment in the athlete with off the shelf pads and callus care, however surgical options exist with good outcomes.

## References

1. Coughlin M, Saltzman C. *Surgery of the foot and ankle*, 8th ed. Elsevier Health Sciences; Mosby, Philadelphia, PA. 2007.
2. Doty JF, Coughlin MJ. Metatarsophalangeal joint instability of the lesser toes and plantar plate deficiency. *J Am Acad Orthop Surg*. 2014;22(4):235–45.
3. Kitaoka H. *The foot and ankle*. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2013.
4. Porter D, Schon L. *Baxter's the foot and ankle in sport*. 2nd ed. Philadelphia: Mosby, Inc.; 2008.
5. Mizel MS, Yodkowski ML. Disorders of the lesser metatarsophalangeal joints. *J Am Acad Orthop Surg*. 1995;3:166–73.
6. Carmont MR, Rees RJ, Blundell CM. Current concepts review: Freiberg's disease. *Foot Ankle Int*. 2009;30(2):167–76.
7. Cohen BE, Nicholson CW. Bunionette deformity. *J Am Acad Orthop Surg*. 2007;15(5):300–7.
8. Thompson FM, Hamilton WG. Problems of the second metatarsophalangeal joint. *Orthopedics*. 1987;10(1):83–9.
9. Bouché RT, Heit EJ. Combined plantar plate and hammertoe repair with flexor digitorum longus tendon transfer for chronic, severe sagittal plane instability of the lesser metatarsophalangeal joints: preliminary observations. *J Foot Ankle Surg*. 2008;47(2):125–37.
10. Coughlin MJ, Baumfeld DS, Nery C. Second MTP joint instability: grading of the deformity and description of surgical repair of capsular insufficiency. *Phys Sportsmed*. 2011;39(3):132–41.
11. Sung W, Weil Jr L, Weil Sr LS, Rolfes RJ. Diagnosis of plantar plate injury by magnetic resonance imaging with reference to intraoperative findings. *J Foot Ankle Surg*. 2012;51(5):570–4.
12. Coughlin MJ. Subluxation and dislocation of the second metatarsophalangeal joint. *Orthop Clin North Am*. 1989;20(4):535–51.
13. Arnold H. Operation von Schneiderballen, Hammer- und Krallenzechen. *Orthopadie Rheuma*. 2014;17(2):16–8.
14. Hofstaetter SG, Hofstaetter JG, Petroutsas JA, Gruber F, Ritschl P, Trnka HJ. The Weil osteotomy: a seven-year follow-up. *J Bone Joint Surg Br*. 2005;87(11):1507–11.
15. Nery C, Coughlin MJ, Baumfeld D, Mann TS. Lesser metatarsophalangeal joint instability: prospective evaluation and repair of plantar plate and capsular insufficiency. *Foot Ankle Int*. 2012;33(4):301–11.
16. Barca F, Acciaro AL. Surgical correction of crossover deformity of the second toe: a technique for tenodesis. *Foot Ankle Int*. 2004;25(9):620–4.
17. Kitaoka HB, Holiday Jr AD. Lateral condylar resection for bunionette. *Clin Orthop Relat Res*. 1992;278:183–92.
18. Boyer ML, Deorio JK. Bunionette deformity correction with distal chevron osteotomy and single absorbable pin fixation. *Foot Ankle Int*. 2003;24(11):834–7.
19. Coughlin MJ. Treatment of bunionette deformity with longitudinal diaphyseal osteotomy with distal soft tissue repair. *Foot Ankle*. 1991;11:195–203.
20. Cooper MT, Coughlin MJ. Subcapital oblique osteotomy for correction of bunionette deformity: medium-term results. *Foot Ankle Int*. 2013;34(10):1376–80.
21. Helal B, Gibb P. Freiberg's disease: a suggested pattern of management. *Foot Ankle*. 1987;8(2):94–102.
22. Sproul J, Klaaren H, Mannarino F. Surgical treatment of Freiberg's disease in athletes. *Am J Sports Med*. 1993;21(3):381–4.
23. Kinnard P, Lirette R. Freiberg's disease and dorsiflexion osteotomy. *J Bone Joint Surg Br*. 1991;73(5):864–5.
24. DiGiovanni CW, et al. Osteonecrosis in the foot. *J Am Acad Orthop Surg*. 2007;15:208–27.

Johnny Lau and David Santone

**Abstract**

This chapter will focus on athletic injuries specific to the forefoot. While these injuries are rare, there has been a noted increase in their incidence. The majority of these injuries are minor, however, if not identified in a timely fashion and managed appropriately, they can result in chronic pain, deformity and eventual loss of function and inability to partake in sport. Forefoot sports injuries include a vast number of injuries. This chapter will address injuries involving turf toe, stress fractures of the second metatarsal, traumatic hallux valgus and sesamoid disorders.

**Keywords**

Sports Injuries • Forefoot • Turf toe • Stress fracture • Hallux valgus • Sesamoid disorder

**Traumatic Hallux Valgus****Etiology and Pathomechanism**

Traumatic hallux valgus is a rare condition that has been attributed to various pathologies including acute MCL rupture, lisfranc ligament injury, first metatarsal fracture, medial plantar nerve entrapment and displaced lesser metatarsal neck fractures resulting in disruption of the transverse ligamentous system [1–5]. Injury to these structures commonly results in a gradual hallux valgus deformity. While traumatic hallux valgus usually results from an injury to either the 1st metatarsal, the 1st MTP joint or the 1st MT-cuneiform joint, recent evidence suggests that an injury to the transverse “tie bar” system can also result in traumatic hallux valgus [5].

J. Lau (✉)

Department of Orthopedic Surgery, University Health Network,  
Toronto Western Hospital, 399 Bathurst St, 438, 1 East Wing,  
Toronto, ON M5T 2S8, Canada  
e-mail: [johnny.lau@utoronto.ca](mailto:johnny.lau@utoronto.ca); [drjohnnylau@sympatico.ca](mailto:drjohnnylau@sympatico.ca)

D. Santone

Department of Orthopedic Surgery, Markham Stouffville Hospital,  
381 Church Street, PO Box 1800, Markham, ON L3P 7P3, Canada  
e-mail: [dave.santone@gmail.com](mailto:dave.santone@gmail.com); [dsantone@msh.on.ca](mailto:dsantone@msh.on.ca)

The transverse tie bar system is comprised of the MTP plantar plates and the deep transverse metatarsal ligaments. An intact tie bar prevents splaying of the forefoot. Disruption of the tie bar will allow neighboring metatarsals to splay. Traumatic hallux valgus has also been described as a variant of turf toe [6]. A valgus directed force causes injury to the medial and plantar medial complex of the 1st MTP.

**Symptoms**

The initial presentation is that of erythema, pain and tenderness localized to the medial aspect of the 1st MTP joint. Joint range of motion is limited. Gross deformity or joint instability is usually absent.

**Diagnostics and Classification**

Physical examination and a high index of suspicion of medial sided injury is required to identify this injury. Standing bilateral AP, lateral and oblique foot xrays can identify mild deformity or fractures that result in traumatic hallux valgus. There is no classification for traumatic hallux valgus.

## Therapy

There has been little written on the management of traumatic HV given its rarity. Initial therapy consists of rest, ice and elevation of the affected foot with anti-inflammatory medications. Patients who fail non operative measures and continue to complain of pain or instability during sport without radiographic explanation for hallux valgus development, should be worked up further with advanced imaging such as MRI. Based on the case reports thus far, management should consist of reduction of any associated fractures and repair of torn collateral ligaments. In the case of a medial collateral ligament tear, a direct repair through a standard medial incision has been performed [7]. Protection of the dorsal hallucal nerve is essential during the approach. Post operative management involves weight bearing as tolerated in a post op shoe or other hard soled shoe, followed by early passive range of motion exercises of the toe.

## Rehabilitation and Back to Sports

In non-operatively treated patients, gradual return to sport, over 3–6 weeks, is done after a short period of immobilization and physiotherapy. In operatively treated patients with MCL pathology, progressive increased weight bearing and a gradual return to sport over 8–10 weeks is the goal [7].

## Prevention

Prevention is best achieved with appropriate footwear and avoidance of foot positions that place the medial structures at risk.

## Evidence

Due to the rarity of this injury, there is limited evidence to support operative management of traumatic hallux valgus.

---

## Turf toe

### Etiology and Pathomechanism

The term turf toe is the eponym given to a sprain or tear of the capsuloligamentous structures of the 1st MTP joint [8]. Turf toe is a common athletic injury especially in sports such as soccer, football, tennis and basketball. It results from an axial load applied to the heel with the foot in fixed equinus and the 1st MTP joint in dorsiflexion. As a result of this injury, the plantar ligaments and capsule can be torn from the

metatarsal neck, as the toe is hyperdorsiflexed, and pulled distally. Associated osteochondral injuries on either the articular surface of the base of the proximal phalanx or the metatarsal head, are not uncommon. Dorsal dislocation of the toe may also result.

## Symptoms

Tenderness, ecchymosis and swelling around the medial and plantar-medial aspect of the 1st MTP joint is typically observed.

## Diagnostics, Classification

History and physical exam of the toe is critical for diagnosis. Exam should begin with inspection of the 1st MTPJ to assess for swelling, ecchymosis and deformity. Palpation of dorsal and plantar structures should then be undertaken. Stability of the joint should be assessed with a varus-valgus stress test as well as a dorso-plantar drawer test. Range of motion of the joint should be assessed. Decreased resistance to dorsiflexion is an indicator of injury to the plantar structures. Assessment of great toe flexion and extension strength can help determine involvement of respective tendons. AP and lateral foot xrays with a sesamoid axial view are obtained. While xrays are typically normal, they can sometimes identify avulsion fractures. Forced dorsiflexion lateral radiographs may be helpful, along with xrays of the contralateral, uninjured foot [9]. MRI is now more commonly obtained for evaluation of the extent of turf toe injury.

A clinical grading system has been published by Anderson [6]. This system divides turf toe injuries into 3 grades and gives recommendations for length of loss of playing time and surgical vs. non surgical management. Grade 1 injuries are sprains, grade 2 injuries are partial tears and grade 3 injuries are complete tears. Non-operative treatment is indicated for grades 1 and 2, while in grade 3 injuries, surgical management may be considered.

## Therapy: Conservative Treatment and Surgery (Approach, Technique, Risks, After Treatment)

All grades are initially treated with rest, ice, compression, elevation and anti inflammatory medication to help reduce swelling and pain. Taping and shoe wear modification can aid return to sport and may require use for up to 6 months post-injury [8]. Management is guided by grade (see above).

While surgery for turf toe is rare, there are limited indications. These include large capsular avulsion with unstable joint, diastasis of bipartite sesamoid, displaced sesamoid

fracture, retraction of sesamoids, traumatic hallux valgus deformity, vertical MTPJ instability, loose bodies, chondral injury and failed conservative management [8].

Surgery entails either a medially based J incision or more recently, a 2 incision technique with a medial based and plantar lateral based incisions according to Hunt et al. [8]. Once the plantar plate defect is identified, non absorbable sutures are used in interrupted fashion to repair the defect. The MTPJ is placed in about 15° of plantarflexion while the sutures are tied.

### Rehabilitation and Back-to-Sports

Post-op rehab involves a short period of immobilization (5–7 days) followed by early passive ROM to prevent arthrofibrosis. The patient remains NWB with a protective boot. Protected WB is commenced 4 weeks post-op and the boot is discontinued 8 weeks post-op. Return to contact activity occurs at 3–4 months and complete recovery may take up to 6 months.

### Prevention

As turf toe is typically caused by increased surface friction associated with artificial turf and increased motion associated with lighter more flexible shoes, avoiding these contributing factors are the best way to prevent this injury [10]. Alternatively, continuing with these high risk sports but altering shoe design to increase forefoot stiffness is another method to prevent this type of injury.

### Evidence

There is limited data on surgical or nonsurgical success of turf toe injury. Anderson has previously reported on his series of 19 collegiate or professional athletes with disabling turf toe injury. 9 underwent surgical repair. All but 2 returned to sports.

---

## Metatarsal Stress Fracture

### Etiology and Pathomechanism

Metatarsal stress fractures are relatively common in athletes. It has been suggested that metatarsal stress fractures make up 10% of all sports overuse injuries [11]. The substantial and repetitive loads that the metatarsals are exposed to during sport, places them at risk for fracture. The 2nd and 3rd metatarsals are the longest and most rigid and thus are the most

prone to fracture, however, the 4th and 5th metatarsals are also susceptible to fracture. Well known risk factors for stress fractures include limb length inequality, cavus foot, excessive running and menstrual irregularities [8].

### Symptoms

Pain and swelling on the dorsum of the foot in the region of the fracture with inability to continue with sports is the most common presentation. The examiner will be able to elicit tenderness to palpation directly over the fracture site.

### Diagnostics

Xrays are the mainstay for diagnosis of metatarsal stress fracture, especially in the chronic state, as metatarsal stress fractures are known to produce a significant periosteal reaction [12]. Technetium 99 three phase bone scans can be used for early detection of stress fractures as they are typically positive as early as 1 week after injury [8]. MRI is typically the test of choice as it can also detect early stress fractures with high accuracy [13].

### Therapy: Conservative Treatment and Surgery (Approach, Technique, Risks, Aftertreatment)

Initial management of stress fractures includes temporary avoidance of the offending sport, rest, ice and immobilization. The vast majority of stress fractures can be treated non operatively. These fractures typically heal within 6–8 weeks. Any delay in healing may require additional therapeutic modalities such as bone stimulation or prolonged immobilization.

While surgery is rare, it may be required for displaced fractures or fracture non-unions. In either case, a small longitudinal incision can be made centered over the fracture site with a dorsal plate applied to the metatarsal, with or without the requirement for bone grafting.

### Rehabilitation and Back-to-Sports

Return to sport is gradual and is guided by radiographic and clinical exam, as well as pain.

### Prevention

Prevention mainly requires minimization of high intensity sports involvement in addition to ensuring the use of proper

footwear. Optimizing nutritional health and other conditions such as irregular menstruation will aid in preventing these fractures.

## Sesamoid Disorders

### Etiology and Pathomechanism

Although small in structure, injury to the hallucal sesamoids may result in considerable pain, discomfort and inability to partake in sporting activities. Due to its larger size, the tibial sesamoid occupies a larger portion of the plantar aspect of the distal 1st metatarsal and thus absorbs more weight bearing forces during activities. This results in more frequent injuries to the tibial, relative to the fibular, sesamoid [14]. Most injuries to the sesamoids involve overuse injuries such as stress fracture (40%), sesamoiditis (30%), acute fracture (10%), osteochondritis (10%), osteoarthritis (5%) and bursitis (5%) [14]. The etiology depends on the type of sesamoid disorder. Etiologies include crush injury, hyperextension injury to the MTP, chondromalacia, impingement or repetitive overloading of the medial forefoot [8]. Patients with a subtle forefoot driven cavovarus deformity have mild forefoot pronation with plantarflexion of the first ray (peroneal overdrive). This places increased pressure under the first metatarsal head and can result in irritation of the soft tissues surrounding the sesamoids. The forefoot pronation deformity can be subtle and should be looked for on physical examination. Subtle contractures of the Achilles tendon should also be ruled out, particularly of the gastrocnemius (Silfverskiold test).

### Symptoms

Patients with sesamoid pathology present with generalized pain around the great toe with weight bearing (specifically in the terminal part of the stance phase of gait) and hyperextension of the toe.

### Diagnostics

Physical exam will demonstrate swelling and pain with palpation directly over the sesamoids, along with decreased plantarflexion strength of the great toe. Loss of active and passive dorsiflexion may also be present [10]. The AP foot xray together with a medial and lateral oblique foot xray to assess the tibial and fibular sesamoids respectively, as well as an axial sesamoid xray, will all be useful in evaluating for sesamoid pathology [14]. Bone scans have been utilized in cases of suspected sesamoid pathology however it is difficult for the scan to differentiate between sesamoid pathology and

intraarticular MTP pathology. CT scans and MRI can identify conditions such as fracture, nonunion, osteomyelitis and post traumatic arthritis much better.

### Therapy: Conservative Treatment and Surgery (Approach, Technique, Risks, Aftertreatment)

Treatment is dependent on the type of sesamoid pathology present. Fractures, osteochondrosis and osteonecrosis can be initially treated with orthoses, activity modification, footwear modification or casting, with the goal of avoiding dorsiflexion of the toe [10]. Shoe modification may include a removable walking boot or a shoe modification with a full length steel shank and anterior rocker bottom, with an orthotic insert recessed in the sesamoid region [10]. Surgical treatment for sesamoid pathology is reserved for those who have exhausted conservative management and continue to have intractable pain. While surgical intervention is generally avoided due to potential complications including weakness, residual pain or neuritic pain, either partial or complete resection of one or both sesamoids may be considered. Complete resection of both sesamoids is generally avoided. If surgical intervention is required, the tibial sesamoid is usually approached through a small plantar medial incision, protecting the medial branch of the plantar digital nerve. The toe is flexed and the intersesamoid ligament is identified and incised and the sesamoid extracted [14]. The fibular sesamoid is approached through a dorsal incision in the 1st webspace. The deep peroneal nerve is identified and protected and the interval between the adductor hallucis and the joint capsule is developed [14]. The tendon of the adductor hallucis is separated from the lateral sesamoid and the intersesamoid ligament is identified and incised and the sesamoid extracted.

### Rehabilitation and Back-to-Sports

Post operatively the foot is immobilized in a splint for up to 6 weeks. After 6 weeks the patient can be advanced to a hard soled shoe with possibly a turf toe plate or mortons extension.

## References

1. Fabeck LG, Zekhnini C, Farrokh D, Deschamps PY, Delince PE. Traumatic hallux valgus following rupture of the medial collateral ligament of the first metatarsophalangeal joint: a case report. *J Foot Ankle Surg.* 2002;41(2):125–8.
2. Bohay DR, Johnson KD, Mannoli A. The traumatic bunion. *Foot Ankle Int.* 1996;17:383–7.
3. Ganel A, Israeli A, Horoszowski H. Post-traumatic development of hallux valgus. *Orthop Rev.* 1987;16:667–70.
4. Johal S, Sawalha S, Pasapula C. Post-traumatic acute hallux valgus: a case report. *Foot (Edinb).* 2010;20:87–9.



5. Lui TH. Acute traumatic hallux valgus: a case report. *Foot (Edinb)*. 2013;23:104–6.
6. Anderson RB. Turf toe injuries of the hallux metatarsophalangeal joint. *Tech Foot Ankle Surg*. 2002;1(2):102–11.
7. Douglas DP, Davidsaon DM, Robinson JE, Bedi DG. Rupture of the medial collateral ligament of the first metatarsophalangeal joint in a professional soccer player. *J Foot Ankle Surg*. 1997;36(5):388–90.
8. Hunt KJ, McCormick J, Anderson RB. Management of forefoot injuries in the athlete. *Oper Tech Sports Med*. 2010;18:34–45.
9. Rodeo SA, O'Brien S, Warren RF, et al. Turf toe: an analysis of metatarsophalangeal joint sprains in professional football players. *Am J Sports Med*. 1990;18:280–5.
10. Nihal A, Trepman E, Nag D. First ray disorders in athletes. *Sports Med Arthrosc Rev*. 2009;17:160–6.
11. Korpelainen R, Orava S, Karpakka J, Siira P, Hulkko A. Risk factors for recurrent stress fractures in athletes. *Am J Sports Med*. 2001;29(3):304–10.
12. Brockwell J, Yeung Y, Griffith J. Stress fractures of the foot and ankle. *Sports Med Arthrosc Rev*. 2009;17:149–59.
13. Bergman AG, Fredericson M. MR imaging of stress reactions, muscle injuries and other overuse injuries in runners. *Magn Reson Imaging Clin N Am*. 1999;7:151–74.
14. Richardson EG. Hallucal sesamoid pain: causes and surgical treatment. *J Am Acad Orthop Surg*. 1999;7:270–8.

Tim Schneider and Elango Selvarajah

## Abstract

Tarsal tunnel syndrome is an entrapment neuropathy of the posterior tibial nerve or its terminal branches as they pass into the foot through a fibro osseous tarsal tunnel below and behind the medial malleolus.

It presents with pain and paraesthesia, or numbness in the distribution of the posterior tibial nerve. The diagnosis generally requires a positive Tinel's sign and positive electrophysiological studies.

In the athletic individual TTS tends to be associated with running, jumping or impact sports. It has been associated with tarsal coalitions, accessory muscles and planovalgus foot deformity.

Treatment includes conservative measures such as ice, anti inflammatory medication, orthotics and modified activity. Surgical decompression needs to include the entire distal tarsal tunnel. The timing of surgery is controversial, but there is a strong body of evidence to suggest that earlier decompression avoids permanent nerve damage and fibrosis.

## Keywords

Tarsal Tunnel Syndrome • Tibial Nerve • Heel Pain • Plantar Nerve • Flexor Retinaculum • Traumatic Neuropathy

## Introduction

Tarsal tunnel syndrome (TTS) is an entrapment neuropathy of the posterior tibial nerve or its terminal branches below or posterior to the medial malleolus as they pass into the foot through a fibro osseous tarsal tunnel.

It was first described by Keck [1] and Lam [2] independently in 1962.

It presents with pain in the tarsal tunnel and paraesthesia, or numbness in the distribution of the posterior tibial nerve

and its branches. The diagnosis generally requires a positive Tinel's sign and may be supported by positive electrophysiological studies

Literature pertaining to TTS in the sports setting is sparse. In the athletic individual TTS tends to be associated with running, jumping or impact sports. There is a high incidence of associated pathologies such as tarsal coalitions, hindfoot valgus, or space occupying lesions.

LEVEL IV EVIDENCE

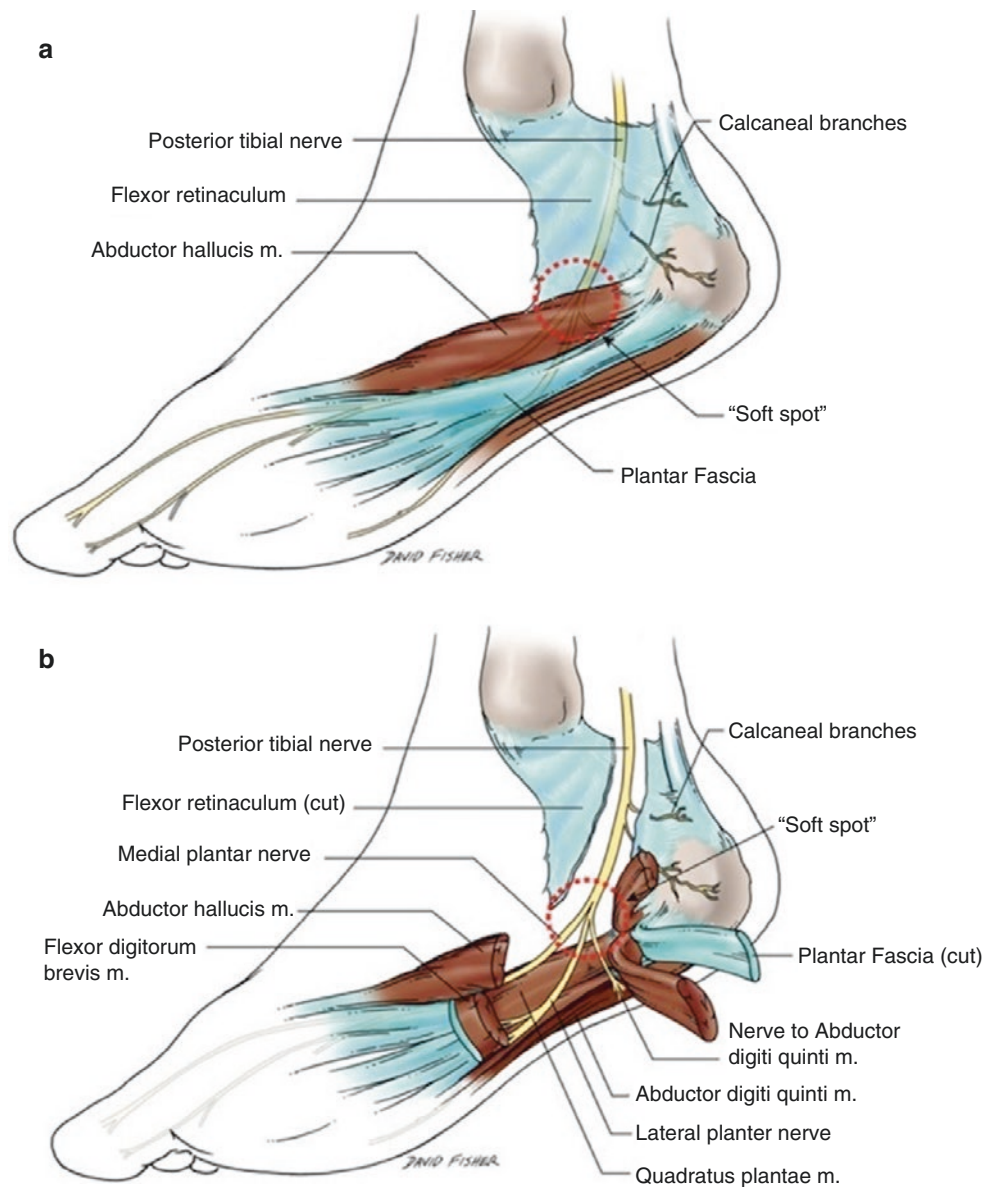
## Anatomy

The tarsal tunnel is a fibro osseous tunnel behind and the beneath the medial malleolus. It allows passage of the postero-medial tendons, and neurovascular structures including the posterior tibial nerve and its branches. It has a proximal portion deep to the flexor retinaculum and a

T. Schneider (✉)  
Melbourne Orthopaedic Group,  
33 The Avenue, Windsor, VIC 3181, Australia  
e-mail: [taschneider@mog.com.au](mailto:taschneider@mog.com.au)

E. Selvarajah  
Foot and Ankle Department, Melbourne Orthopaedic Group,  
33 The Avenue, Windsor, VIC 3181, Australia  
e-mail: [elango\\_selvarajah@yahoo.com](mailto:elango_selvarajah@yahoo.com)

**Fig. 40.1** Artists depiction of the tarsal tunnel and its contents (reprinted with permission courtesy of John S Gould). Posterior Tibial Nerve passes deep to the flexor retinaculum and Abductor Hallucis muscle belly (a) To access and decompress the entire nerve including the important distal components it is necessary to cut and reflect the Abductor Hallucis muscle belly (b)



distal portion which is divided variably by fibrous septa which can create points of compression or entrapment [3, 4] (Fig. 40.1).

#### LEVEL IV EVIDENCE

The posterior tibial nerve enters the tarsal tunnel as one of the more posterior structures. In 95% of cases it divides into its terminal branches (Lateral Plantar Nerve and Medial Plantar Nerve) near lower margin of the flexor retinaculum. In a small number of cases it bifurcates above the flexor retinaculum [4].

Distally the medial plantar nerve passes forward beneath abductor hallucis while the lateral plantar nerve passes through abductor hallucis and deep to quadratus plantae [3].

The calcaneal branch of the posterior tibial nerve originates at or above flexor retinaculum and courses down with

the main nerve before penetrating the flexor retinaculum to supply the medial side of the heel [4].

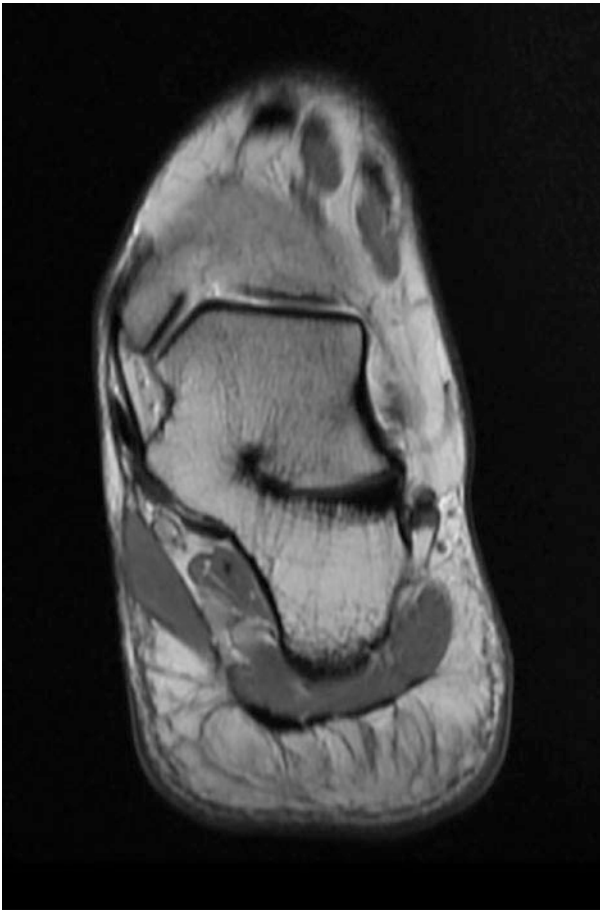
The nerve to abductor digiti minimi is the first branch from the lateral plantar nerve and courses laterally distal to the calcaneal tuberosity. It is prone to compression under the calcaneum in runners [5].

#### LEVEL IV EVIDENCE

### Aetiology

The cause of tarsal tunnel syndrome can be broadly classified as:

1. Anatomical changes to the fibro osseous tunnel.
2. Space occupying lesions
3. Traumatic lesions



**Fig. 40.2** MRI image of a tarsal coalition of the subtalar joint in a patient with tarsal tunnel syndrome including a positive Tinel sign

Changes to the fibro osseous tunnel include osteophytes or coalition at the medial subtalar joint (Fig. 40.2) causing impingement. Thickening of the plantar fascia or hypertrophy of the abductor hallucis can be seen in the athletic individual [6]. Variations in the fibrous septa dividing the distal compartments in the tarsal tunnel can also contribute to entrapment [3].

Dynamic alterations to the foot posture such as hindfoot inversion or eversion can alter the cross sectional area of the tunnel. Forefoot pronation in runners has been associated with injury to the tibial nerve through excessive stretching [7]. These dynamic causes are thought to either stretch the medial planar nerve against the navicular or compress the posterior tibial nerve within the tarsal tunnel itself [3].

Space occupying lesions can include varicosities, ganglia, neuromas and lymphomas [7] (Fig. 40.2). Tenosynovitis of the Tibialis Posterior or long flexor tendons may also have this effect [7–9].

#### LEVEL IV EVIDENCE

Traumatic lesions are the most common cause of TTS [6, 9] This group can overlap with the other two groups and include tarsal fractures, (such as calcaneal fractures

and the *Cedell* fracture of the medial tubercle of the talus) [10], traumatic tenosynovitis, and post traumatic epineural scarring following haemorrhage [6, 9]. Rask [11] and Jackson [12] described dynamic deformities whereby the medial plantar nerve is stretched against the navicular in a valgus hindfoot. Other traumatic lesions include plantar fascial tears with an associated inflammatory mass and crush injuries to the Nerve to Adductor Digiti Minimi against the calcaneum [5].

#### LEVEL IV EVIDENCE

Prolonged compression of a peripheral nerve results in loss of endoneurial microvessel function and oedema [13]. If this persists for more than 6 months, in primate models, it can result in perineural fibrosis and demyelination [13]. The double crush syndrome was described by Upton and McComas in 1973 [14]. This describes compression of the nerve over an extended length creating characteristic myelin and axonal degeneration. It is one of the causes of failure of decompression surgery [13] and warrants extended decompression of the entire tarsal tunnel.

#### LEVEL IV EVIDENCE

## Symptoms and Presentation

Patients describe dysaesthesia, or anaesthesia on the plantar aspect of the foot. There is often associated burning pain radiating distally or proximally into the calf. There is often burning sensation at night and particularly after activity. This was described by Gould and others as “after burn” [15]. Symptoms are usually relieved by rest and made worse with activity.

Clinical signs include objective evidence of decreased sensation on the plantar aspect of the foot, intrinsic muscle weakness, (which is much more difficult to assess) and a positive Tinel’s sign. The Tinel’s sign is elicited by percussion along the course of the posterior tibial nerve or its branches. It is seen as an important component of the clinical diagnosis [6, 9, 13, 16, 17]. Symptoms may be provoked by combined dorsiflexion and eversion [18]. Clinically there maybe a feeling of fullness along the course of the tarsal tunnel associated with a space occupying lesion such as tenosynovitis, ganglia or engorged veins [6].

#### LEVEL IV EVIDENCE

In athletes, tarsal tunnel syndrome is most commonly seen with repetitive impact activities such as running or jumping sports [6, 8, 17] In these individuals there is also a high incidence of predisposing anatomical factors, which cause alteration to the tarsal tunnel itself [8].

Kinoshita [8] reported on a series of 18 athletes reporting symptoms of tarsal tunnel syndrome who underwent surgery. Seven patients had tarsal coalitions, 3 had accessory muscles and four patients had planovalgus feet.



**Fig. 40.3** MRI image of a ganglion of the flexor tendon sheath with the tarsal tunnel below the sustentaculum tali

#### LEVEL IV EVIDENCE

Rask [11] described “joggers foot” which was attributed to compression of the medial plantar nerve under the medial navicular in joggers. It is a recurrent impact injury and is worsened by hindfoot valgus which brings the medial plantar nerve under the navicular in the distal medial tarsal tunnel. This creates a point of compression.

Labib and Gould [19] described a heel pain triad being a combination of plantar fasciitis, posterior tibial dysfunction and tarsal tunnel syndrome. Clearly these three entities will overlap in a certain number of patients so the diagnosis and subsequent treatment rationale is often confused.

Sammarco [9] pointed out the role of post traumatic scarring of the posterior tibial nerve and its branches in the tarsal tunnel. This may be a contributing factor to TTS in athletes involved in repetitive impact activity.

Baxter and Pfeffer [5] described release of the first branch of the lateral plantar nerve in a group of patients, half of whom related their pain to sporting activity. This was most commonly long distance running but also included tennis, volleyball, basketball and aerobics [5].

The differential diagnosis for heel pain in the athlete should include fat pad insufficiency, plantar fasciitis, nerve entrapment including tarsal tunnel syndrome or first branch of the lateral plantar nerve (Fig. 40.4) and stress fractures of the calcaneum [8, 17, 19, 20].

#### LEVEL V EVIDENCE



**Fig. 40.4** MRI image of a disruption of the plantar fascia creating a thickening which was impinging on the nerve to abductor digiti minimi

## Diagnosis

Routine weight bearing radiology may reveal evidence of tarsal coalition, an osteophyte in the tarsal tunnel or a planovalgus posture which could contribute to compression of the nerve on weight bearing [6, 13]. CT scanning will add further detail to the plain radiological imaging, however they are generally not weight bearing.

Ultrasonography can demonstrate tenosynovitis, venous structures or ganglia within the tarsal tunnel [13, 21].

MRI scanning is the modality of choice for defining most anatomical details concerning the tarsal tunnel [6, 13]. It can identify bony and soft tissue anomalies, including tendon, muscular, and vascular anomalies, as well as some neural detail [6, 9, 13, 21].

Electrophysiological investigations can demonstrate slow sensory conduction or reduced amplitude of motor potentials [9]. Conduction times are characteristically increased with nerve compression causing focal demyelination [22]. False negative rates of up to 50% have been reported although it is felt that a positive result strongly supports the diagnosis of Tarsal Tunnel Syndrome [13, 23].

#### LEVEL IV EVIDENCE

## Treatment

Assessment of Tarsal Tunnel Syndrome in the athletic individual should include the role of repetitive impact such as in distance runners, muscle hypertrophy (such as flexor hallucis longus or abductor hallucis), entrapment or injury to the first branch of the lateral plantar nerve and the role of a planovalgus foot.

Initial conservative treatment includes rest, ice, non-steroidal anti-inflammatories and orthotics to alter the pattern of heel strike and reduce tension in the nerve in a valgus hindfoot [6, 17] (Fig. 40.5). Gould describes a specific total contact orthotic with a posteromedial channel to unload the distal tarsal tunnel [15]. Several others recommend a calf stretching program to alleviate the contribution from the plantar fascia [15, 24, 25]

The timing of surgery is controversial, but there is a strong body of evidence to suggest that earlier decompression avoids permanent nerve damage and fibrosis [9, 13, 17, 26]. Surgical decompression performed within 12 months from the onset of symptoms are thought to achieve more favourable outcomes [9].

Surgical decompression should include release of the proximal and distal components of the tarsal tunnel. Reports of surgical release of only the proximal component under the flexor retinaculum had 50% or more unsatisfactory outcomes [23, 27], more extensive release of both the proximal and distal components of the tarsal tunnel report more than 80% satisfactory results [19, 20]. Gould reported an 82% satisfactory rate when the exploration was combined with a plantar fascia release [28].

### LEVEL IV EVIDENCE

Surgery needs to include removal of any space occupying lesion. Care needs to be taken to identify the calcaneal branches as they penetrate the flexor retinaculum, both plantar nerves, and in particular the first branch of the lateral plantar nerve to abductor digiti minimi. Haemostasis is important to prevent subsequent scarring within the tarsal tunnel, which is seen as a cause of recurrent symptoms [20, 24, 29].

Tarsal tunnel surgery fails because of inaccurate initial diagnosis, inadequate release of all components of the tarsal tunnel, inadequate haemostasis and subsequent scarring or traction neuritis, double crush syndrome, persistent hypersensitivity of the nerve, damage to the nerve or its branches at the time of surgery and intrinsic damage to the nerve either as a result of delayed release or some other primary nerve pathology such as diabetic neuropathy [6, 13, 24, 29].

### LEVEL IV EVIDENCE

## Rehabilitation and Back-to-Sports

Post operative management should involve early immobilisation and compression during the initial wound healing phase and then mobilisation to prevent tethering of the tibial nerve and its branches.



**Fig. 40.5** Orthotics are an initial management strategy to correct hind-foot valgus and relieve the pressure under the tarsal tunnel with a custom cut away channel

Early series reported prolonged post operative immobilization. Results were less than optimal and more recently authors have advocated an initial period of rest for wound healing followed by mobilization and retraining as soon as the wound is stable [6, 15, 20]. Active and passive mobilization with full weight bearing can begin at or around 2 weeks [6], with increasing intensity over the next 4 to 6 weeks. As well as limiting functional loss from inactivity it will minimize the risk of perineural scarring. As there are no concerns for structural stability, full impact loading can begin as soon as the soft tissue envelope will tolerate it.

### LEVEL V EVIDENCE

On returning to training and sport particular attention needs to be paid to proprioceptive function and any subtle loss of foot balance as the result of ongoing neurological deficit. This is relevant to the running, jumping, and turning athlete where subtle loss of motor and sensory input will compromise the athlete's performance in the rehabilitation phase. Specific attention to these issues may help the athlete make an earlier return to optimal performance. Optimal post operative function occurs by around 9 months [9] although this can be quite variable.

### LEVEL IV EVIDENCE

## Summary

1. Tarsal tunnel syndrome is an entrapment neuropathy of the posterior tibial nerve and its branches as it passes behind the medial malleolus. It presents with neuritic heel pain which often lasts after the cessation of activity (“after burn”)
2. Beware of false negative electrophysiological studies, however positive studies strongly support a diagnosis of tarsal Tunnel Syndrome
3. Trauma is the most common aetiology, however, one needs to consider space occupying lesions and changes to the conformation of the tarsal tunnel. It often occurs in association with plantar fasciitis and Tibialis Posterior dysfunction
4. In the athletic patient, there is a high association with repetitive impact activities such as running and jumping
5. In the athletic patient there is a high association with other lesions such as a planovalgus foot deformity, tarsal coalition or a space occupying lesions.
6. Early conservative measures such as anti inflammatory medication, calf stretching, insoles or gait retraining may reduce symptoms. Surgery should be considered after 3–6 months of failed non operative treatment to reduce the formation of perineural scarring and traumatic neuropathy

## References

1. Lam SJS. A tarsal-tunnel syndrome. *Lancet*. 1962;2:1354–5.
2. Keck C. The tarsal-tunnel syndrome. *J Bone Joint Surg*. 1962;44A:180–2.
3. Singh G, Kumar VP. Neuroanatomical basis for the tarsal tunnel syndrome. *Foot Ankle Int*. 2012;33:513–8.
4. Kelikian AS, editor. *Sarrafian’s anatomy of the foot and ankle: descriptive, topographical, functional*. 3rd ed. Philadelphia: Lippincott Williams Wilkins; 2011. p. 134–48, p 403–417.
5. Baxter DE, Pfeiffer GB. Treatment of chronic heel pain by surgical release of the first plantar nerve. *Clin Orthop*. 1992;279:229–36.
6. Lau JTC, Daniels TR. Tarsal tunnel syndrome: a review of the literature. *Foot Ankle Int*. 1999;20(3):201–9.
7. Daniels TR, Lau JTC, Hearn T. The effect of foot position and load on tibial nerve tension. *Foot Ankle Int*. 1998;19:73–8.
8. Kinoshita M, Okuda R, Yasuda T, Abe M. Tarsal tunnel syndrome in athletes. *Am J Sports Med*. 2006;34(8):1307–12.
9. Sammarco GJ, Chang L. Outcome of surgical treatment of tarsal tunnel syndrome. *Foot Ankle Int*. 2003;24(2):125–31.
10. Cedell CA. Rupture of the posterior talotibial ligament with the avulsion of a bone fragment from the talus. *Acta Orthop Scand*. 1974;45:454–61.
11. Rask M. Medial plantar neuropraxia (joggers foot). *Clin Orthop*. 1978;134:193–5.
12. Jackson DL, Haglund BL. Tarsal tunnel syndrome in runners. *Sports Med*. 1992;13:146–9.
13. Ahmad M, Tsang K, Mackenney PJ, Adedapo AO. Tarsal tunnel syndrome : a literature review. *Foot Ankle Surg*. 2012;18:149–52.
14. Upton AR, McComas AJ. The double crush syndrome in nerve entrapment syndromes. *Lancet*. 1973;2:359–61.
15. Gould JS. Tarsal tunnel syndrome. *Foot Ankle Clin N Am*. 2011;16:275–86.
16. Dellon AL. The four medial ankle tunnels: a critical review of perceptions of tarsal tunnel syndrome and neuropathy. *Neurosurg Clin N Am*. 2008;19:629–48.
17. Lorei MP, Hershman EB. Peripheral nerve injuries in athletes: treatment and prevention. *Sports Med*. 1993;16(2):130–47.
18. Kinoshita M, Okuda R, Morikawa J. The dorsiflexion eversion test for diagnosis of tarsal tunnel syndrome. *J Bone Joint Surg*. 2001;83-A(12):1835–9.
19. Labib SA, Gould JS, Rodriguez-del-Rio FA, Lyman S. Heel pain triad (HPT): the combination of plantar fasciitis, posterior tibial tendon dysfunction and tarsal tunnel syndrome. *Foot Ankle Int*. 2002;23:212–20.
20. Dellon AL. Nerve disorders plantar heel pain, A. Lower extremity nerve injuries in athletes’. In: Porter DA, Schon LC, editors. *Baxter’s the foot and ankle in sport*. 2nd ed. Philadelphia: Mosby; 2008. p. 205–24.
21. Lopez-Ben R. Imaging of nerve entrapment in the foot and ankle. *Foot Ankle Clin N Am*. 2011;16:213–24.
22. Lehman RM. A review of neurophysiological testing. *Neurosurg Focus*. 2004;16:1–16.
23. Sung KS, Park SJ. Short-term operative outcome of tarsal tunnel syndrome due to benign space-occupying lesions. *Foot Ankle Int*. 2009;30:741–5.
24. Gould JS. The failed tarsal tunnel release. *Foot Ankle Clin N Am*. 2011;16:287–93.
25. Davies MS, Weiss GA, Saxby TS. Plantar fasciitis: how successful is surgical intervention. *Foot Ankle Int*. 1999;20:803–7.
26. Takakura Y, Kitada C, Sugimoto K, Tanaka Y, Tamai S. Tarsal tunnel syndrome: causes and results of operative treatment. *J Bone Joint Surg*. 1991;73B:125–8.
27. Pfeiffer WH, Cracchiolo A. Clinical results after tarsal tunnel decompression. *J Bone Joint Surg*. 1994;76A:1222–30.
28. DiGiovanni BF, Abuzzahab FS, Gould JS. Plantar fasciitis release with proximal and distal tarsal tunnel release: surgical approach to chronic disabling plantar fasciitis with associated nerve pain. *Tech Foot Ankle Surg*. 2003;2:254–61.
29. Gould JS. Recurrent tarsal tunnel syndrome. *Foot Ankle Clin*. 2014;19(3):451–67.

Christopher E. Gross and James A. Nunley II

**Abstract**

Nerve entrapments are an under-appreciated and under-diagnosed cause of many recalcitrant aches and pains in athletes. These entrapments are difficult to diagnose due to the dynamic nature of symptoms. The goal of this chapter is to discuss the etiology, treatment strategies, rehabilitation, and prevention of six common foot and ankle nerve entrapments experienced by athletes—Morton's neuroma, tarsal tunnel syndrome, anterior tarsal tunnel syndrome, and entrapment of the superficial peroneal nerve, Baxter's nerve, and jogger's nerve.

**Keywords**

Nerve compression • Neuritis • Jogger's foot • Baxter's • Morton's • Tarsal tunnel

Nerve entrapments are an under-appreciated and under-diagnosed cause of many recalcitrant aches and pains in athletes. Often, these entrapments are difficult to diagnose due to the dynamic nature of symptoms: only certain training activities or positioning of the feet during maneuvers may elicit symptoms.

An accurate diagnosis begins with taking a thorough history. The athlete should be asked when he began to experience symptoms. Rarely patients can point to specific injury, such as an inversion ankle sprain, that led to the beginnings of symptoms. Commonly, patients note a gradual pain that was occasionally nagging and now occurs more frequently with specific activities. This pain can evolve into pain that does not improve with rest or a change in shoes, intensity of training, or physical therapy. Some common neurogenic symptoms that cause suspicion for a nerve entrapment include: pain (burning, stabbing, radiating), paresthesias, and weakness. The specific anatomic distribution of these symptoms should be explored. The athlete's past medical history, especially known rheumatologic, endocrine, and neurologic issues should be queried. One may need to rule out exertional com-

partment syndrome which often presents in patients as a dull, achy pain that is made worse with exertion, most often running. It is important to note the location of the pain while exercising as this will help direct more focused questioning/examining as well as point to which muscle groups are possibly affected. The athlete's training schedule is also reviewed.

The physical examination should be directed by the surgeon's knowledge of foot and ankle anatomy. Following a routine physical examination of the foot, palpation and percussion of the nerve of interest is attempted to elicit symptoms. The clinician may try to simulate nerve constriction by holding external compression. At times, the radiating nerve symptoms may be mimicked.

Conservative treatment is the mainstay for the majority of neurologic compression injuries. Athletes are asked to remove all extrinsic factors that could be causing potential sources of constriction: tight shoes or shoe laces, taping, and straps. Next, the training itself should be modified such as playing/training surface and muscle stretching and strengthening. Rest, a regimented course of non-steroidal anti-inflammatory drugs, massage, acupuncture, transcutaneous electrical nerve stimulation, ice, heat, and local anesthetic/corticosteroid injections may prove successful.

The goal of this chapter is to discuss the etiology, treatment strategies, rehabilitation, and prevention of six com-

C.E. Gross, MD (✉) • J.A. Nunley II, MD  
Department of Orthopaedic Surgery, Duke University Medical  
Center, 4709 Creekstone Dr., Durham, NC 27703, UK  
e-mail: [cgross144@gmail.com](mailto:cgross144@gmail.com); [james.nunley@duke.edu](mailto:james.nunley@duke.edu)



mon foot and ankle nerve entrapments experienced by athletes—Morton’s neuroma, tarsal tunnel syndrome, anterior tarsal tunnel syndrome, and entrapment of the superficial peroneal nerve, Baxter’s nerve, and jogger’s nerve.

## Morton’s Neuroma

### Etiology and Pathomechanism

Morton’s neuroma is a paroxysmal neuralgia that presents as a sharp, burning pain in the toe webspace. It most commonly affects the 3rd interdigital nerve in the third webspace between the third and fourth toes (80–85%) and next commonly, the 2nd common digital nerve in the second webspace (10–15%) [1]. It is not a neuroma in the formal definition of the word, but rather a perineural fibrosis. Runners and dancers are often most commonly affected [2]. In dancing, the forefoot is compressed in the following maneuvers: *en pointe*, *relevé*, and *tendu* [3].

The exact etiology and pathogenesis of Morton’s neuroma is still a point of contention. Morton suggested that enlargement or a neuroma of the digital branches of the lateral plantar nerve was the source of pain [4]. Neuritis may also occur as contraction of the flexor digitorum brevis causes the nerve to shear against the intermetatarsal ligament, which in turn, causes inflammation [5]. Another theory states that the intermetatarsalphalangeal bursa balloons out and causes traction on the digital nerve adjacent to the digital nerve, which leads to an ischemic effect on the nerve [6]. Current theories believe that Morton’s neuroma is a consequence of the combination of repetitive mechanical and ischemic trauma, entrapment, and tethering [7].

The epidemiology is not clearly defined as the incidence and prevalence are not known [8]. Women aged 45–50 are most commonly affected. Though men are affected, women account for the overwhelming majority [9]. Both feet are equally affected, though bilateral complaints are rare. It is also uncommon to find two neuromas in the same foot.

### Diagnostics, Classification

The differential diagnosis must be carefully crafted to exclude more serious disorders of the forefoot. The differential diagnosis for Morton’s neuroma includes: intermetatarsal bursitis, metatarsophalangeal joint instability/capsulitis, metatarsalgia, lumbar radiculopathy, tarsal tunnel syndrome, stress fracture, Freiberg’s infraction, infection, tumors, painful callosities associated with toe deformities, peripheral neuropathy [1, 7].

Imaging is necessary to rule out the differential diagnosis listed above. Often, an x-ray may show a faint radiopaque mass and lateral toe deviation [7]. An MRI may show inflammation

surrounding the neuroma as seen in T2 weighted images. An ultrasound may show a non-compressible hypoechoic interdigital mass with or without a bursal effusion.

While early studies of imaging showed that the clinical utility of these modalities is questionable [10], most studies did not evaluate a Morton’s neuroma with today’s more sensitive MRI magnets and ultrasounds. Owens and colleagues [11] showed that an MRI may have a sensitivity of 97% in patients with symptomatic neuromas, but showed neuromas in 25% of patients without symptoms. Sharp et al evaluated confirmed cases of Morton’s neuroma that were evaluated with an MRI, ultrasound and physical examination. MRI and ultrasound’s accuracy was dependent on the size of the lesions. Physical exam proved to be the most sensitive and specific modality [12].

### Symptoms

A patient with a Morton’s neuroma often complains of a burning, sharp pain that is located between the third and fourth toes. This pain is often plantar at the metatarsal heads and radiates distally on either side of the toe. It can often radiate from the forefoot up the leg proximally. The pain is exacerbated with wearing tight or constricting shoes and frequently alleviated while walking barefoot. A patient may describe characteristically and temporally different pain. The first, intense pain lasts for about 5–10 min and is precipitated by direct compression of the neuroma. This pain then becomes a dull ache for the next 2–3 h [13]. One feature of a Morton’s neuroma is that a patient may need to rest after walking, remove the shoe and massage the area of discomfort for moderate relief [14]. This may occur several times throughout the day.

On palpation, the usual location of pain is at the interspace between the metatarsal heads. Therefore, one must examine the metatarsophalangeal joints for synovitis or instability (metatarsophalangeal drawer test). Interdigital skin sensation is often decreased. A useful test for Morton’s neuroma is the “lateral squeeze test”. This test is performed with the index and thumb on the dorsal and plantar aspect of the painful intermetatarsal space. The forefoot is then compressed with the opposite hand by squeezing together the metatarsal heads. The test is positive if a painful or palpable click is felt. This “Mulder’s click” is likely due to subluxation of the neuroma between the metatarsal heads [15].

### Therapy: Conservative Treatment and Surgery

When a diagnosis of Morton’s neuroma is made, some surgeons believe that conservative management is never warranted since the pain will persist and worsen as the neuroma enlarges [13]. While there is no agreed-upon treatment

algorithm, most believe conservative treatments should be tried from 3 months to a year [1, 7].

Firstly, a patient should wear wide, comfortable shoes with large toe boxes [1]. Custom orthotics may limit foot subtalar pronation and limit the mobility of the first ray and reduce forefoot abduction [16].

Another treatment modality is an intermetatarsal corticosteroid and local anesthetic injection. It is imperative that the needle traverses the intermetatarsal ligament in order to enter the bursa and infiltrate the neurovascular bundle. While some studies show that 47% of people may experience relief with injections [17], others fail to show any long-term relief [18]. Recently studies have looked at injection of either phenol or alcohol. An electrode-guided injection of phenol proved to be effective in 80.3% of cases [19]. Ultrasound-guided injection of alcohol gave 84% of patients total relief with partial relief to 94% [20].

The evidence for conservative therapy is relatively weak [8]. If a patient fails these measures, surgery is warranted. Many surgical methods have been described, however, the most common technique includes a dorsal incision with interdigital nerve excision after intermetatarsal ligament division.

A dorsal approach allows for immediate weight bearing and suture removal after 2 weeks

## Rehabilitation and Back-to-Sports

A patient usually transitions to a normal shoe by 3–4 weeks, with return to sports in 4–6 weeks. The most common complication results from wound healing keloid formation or superficial infection [7]. In one series, 1.1% of patients had a wound infection while 2.2% had keloid formation over the scar [7]. Recurrent neuroma formation is another complication. This usually results from inadequate proximal resection of the common digital nerve. This neuroma stump is then trapped by the metatarsal heads, compressed, and causes recurrent pain.

Despite the above complications, patients describe high satisfaction rates from 80 to 96% [17, 21].

## Prevention

There are no formalized studies that evaluate strategies to avoid interdigital neuromas in the athletic population.

---

## Tarsal Tunnel Syndrome

### Etiology and Pathomechanism

The tarsal tunnel is a fibrous tunnel made of the medial malleolus, the medial wall of the calcaneus, the

flexor retinaculum (lacinate ligament), the posterior talus, and the abductor hallucis. The contents of the tarsal tunnel include the tibial nerve, posterior tibial artery, the flexor digitorum longus (FDL), the flexor hallucis longus (FHL), and the posterior tibialis tendon. At this tunnel, the tibial nerve divides into four main branches (medial and lateral plantar and the medial and inferior calcaneal nerves) to supply the majority of the plantar sensation and intrinsic musculature.

The nerve can be compressed by both intrinsic and extrinsic factors which include: ganglion cysts, varicose veins, tenosynovitis, lipomas, tendinopathy, osteophytes, tight laces, scarring, and edema. Dancers may have a hypertrophied FHL muscle belly and may experience symptoms when they are in relevé [3]. In a review of a series of 18 athletes treated for tarsal tunnel syndrome [22], activities that triggered symptoms sprinting, jumping, and performing ashi barai in judo or karate. These activities may overload the ankle joint. They noticed that certain physical factors could increase the risk for tarsal tunnel syndrome such as: pes planus, accessory muscles, talocalcaneal coalition, and accessory muscles.

## Symptoms

Due to the complexity of the tibial nerve branches, presentation of tarsal tunnel syndrome is highly variable. It may include numbness, tingling, and aching in the specific branches affected. The syndrome may be exacerbated in those who need to stand for longer periods of time (baseball outfielders). Often athletes do not notice a loss of strength or wasting of the intrinsic muscles unless it is severe. In advanced cases, the surgeon may be able to appreciate muscle atrophy. Percussion or manual compression of the tarsal tunnel (and the distal tibial nerve branches) may be positive. Using a monofilament test, sensory changes in the distribution of the tibial nerve branches may be elicited.

## Diagnostics, Classification

Radiographs, an MRI, or ultrasound may be helpful in locating any osseous fragments or space-occupying lesions in the tarsal tunnel [23]. An electrodiagnostic test is another diagnostic modality, however a negative result does not exclude the problem. A nerve conduction study (NCV) can see prolongation of conduction velocity of any of the number of branches of the tibial nerve (most often the medial or lateral plantar nerves). An electromyography (EMG) should focus on the intrinsic muscles: abductor hallucis, interossei, and abductor digiti quinti. An ultrasound-guided local anesthetic and corticosteroid injection can be both diagnostic and therapeutic.

## Therapy: Conservative Treatment and Surgery

In patients with space-occupying lesions of the tarsal tunnel, conservative treatment is initiated as outlined above. More often than not, those with mechanical symptoms have already tried a period of conservative therapy before reaching the clinic setting either directed intuitively or by a trainer. Therapy may focus on stretching the plantar fascia and the gastrocnemius soleus complex. An medial arch support may invert the foot, offloading the medial column and improving symptoms [3].

Surgery consists of an extensive release of the flexor retinaculum, removal of any mechanical lesions, a partial release of the abductor hallucis fascia, and partial release of the plantar fascia. A cast is applied with the suture removal after 2 weeks.

## Rehabilitation and Back-to-Sports

A patient usually transitions to full weight bearing at 2–4 weeks with a return to sports in 12 weeks. In the previously-mentioned study of 18 athletes, 67% returned to their sport at the same functional level at final follow-up. In a population that includes both athletes and non-athletes, 82% experience complete recovery [24] with a symptomatic plateau at an average of 19.6 months (Grade IV evidence: range, 6–30 months) [23].

## Prevention

There are no formalized studies that evaluate strategies to avoid tarsal tunnel syndrome in the athletic population.

---

## Deep Peroneal Nerve (Anterior Tarsal Tunnel)

### Etiology and Pathomechanism

Approximately 5 cm proximal to the ankle joint, the deep peroneal nerve lies between the extensor digitorum communis (EDC) and the extensor hallucis longus (EHL) tendons. It then passes underneath the superior extensor retinaculum where 1 cm proximal to the joint, the nerve divides into medial and lateral branches. The lateral branch innervates the extensor digitorum brevis (EDB) and lateral tarsal structures. The purely sensory medial branch passes under the Y-shaped inferior extensor retinaculum (near the talonavicular joint) where compression occurs. Compression may be due to ribbons from dancing shoes impinging on the talonavicular joint and straps from stationary bicycles. Those who perform sit-ups with the dorsal foot against a bar and runners

who place a key under the tongue of their shoes may also experience discomfort [25].

## Symptoms

Athletes typically complain of vague pain on the dorsum of the foot and may sometimes localize pain and paresthesias to the 1st dorsal webspace. Plantarflexion, places the nerve on stretch, and may exacerbate symptoms. Typically, exertion exacerbates the pain and rest alleviates the pain. More so than any other nerve entrapment syndromes, deep peroneal nerve compression is associated with night pain [26].

Some athletes relate a history of multiple ankle sprains. During the injury, the deep peroneal nerve is stretched as the ankle plantarflexes and supinates [2].

## Diagnostics, Classification

Diagnosis is again made clinically. The entire course of the deep peroneal nerve should be palpated and percussed. Symptoms may often be reproduced when the ankle is forcibly plantarflexed and inverted. Subtle difference in the strength and bulk of the EDB may be ascertained with resistance of both of the great toes in dorsiflexion. Palpation of the dorsal talonavicular joint may reproduce symptoms.

In deep peroneal nerve compression, radiographs of the ankle and foot should be obtained. This type of imaging is important as dorsal osteophytes at the talonavicular joint may cause compression. MRI is important if one is concerned about a space-occupying lesion. Electrodiagnostic studies focus on the EDB (lesion would be proximal to inferior retinaculum) and conduction velocities. One study demonstrated that there is reduced EDB motor recruitment in 38% asymptomatic individuals and abnormal conduction velocities in 76% of asymptomatic people [27].

## Therapy: Conservative Treatment and Surgery

The primary goal is to try to reduce any extrinsic device that is causing external compression on the dorsal foot, such as a tight ski boot. Non-restrictive shoes can often reduce symptoms.

Surgery is considered when conservative therapy fails. Localization of the entrapment dictates the surgical approach and the extent of the dissection. However, it may be necessary to release the superior extensor retinaculum all the way distal to the 1st web space.

## Rehabilitation and Back-to-Sports

Rehabilitation and return to sports is dependent on how extensive the decompression was. If they decompression was a simply dorsal osteophyte removal, activities can be restarted as soon as the wound heals.

### Prevention

Athletes should try to limit any devices, straps, or activities that constrict the dorsal foot

---

## Medial Plantar Nerve

### Etiology and Pathomechanism

Entrapment of the medial plantar nerve (jogger's foot) commonly affects runners with a male predominance without a specific age distribution [28]. Entrapment occurs at the Master Knot of Henry. Though the mechanism of compression is not entirely clear, Kopell and Thompson postulated that the entrapment is related to hallux rigidus. An overactive tibialis anterior leads to denervation of the medial plantar nerve which in turn causes increased stress in the 1st metatarsophalangeal joint [29]. Schon and Baxter proposed that patients trying to reduce motion of their 1st MTP joint, may have spasming of the abductor hallucis and flexor brevis which, in turn, caused compression of the medial plantar nerve [2].

### Symptoms

Athletes complain of pain that radiates from the medial arch to the medial toes. It can also shoot proximally. Pain is often elicited by running on flat surface. Orthotics often exacerbate the symptoms, especially if the place the heel into valgus or pronate the forefoot [2]. At times, it may also mimic tibial sesmoiditis (medial hallucal nerve).

### Diagnostics, Classification

Diagnosis is based on a thorough physical examination. Patients will often have tenderness at the medial plantar arch or just proximal to the tibial sesamoid. Pain and numbness may be present after the patient jogs. Pain may be elicited with percussion of the nerve or by everting the hindfoot or having the patient walk on the tiptoes.

## Therapy: Conservative Treatment and Surgery

Conservative therapy includes the above-mentioned modalities. The offending orthotic should be removed from the patient's shoe. Sometimes a lamb's wool pad within a ballet slipper may also reduce symptoms [3].

If conservative treatment fails to reduce the symptoms to a manageable level, a decompression surgery from a medial approach should be performed. There are no published studies regarding surgical outcomes.

## Rehabilitation and Back-to-Sports

Rehabilitation and back-to-sports is dependent on the type of sport. Depending of the amount of release, running is allowed anytime from 3 to 6 months.

### Prevention

There are no studies that evaluate strategies to avoid medial plantar nerve compression.

---

## First Branch of the Lateral Plantar Nerve (Baxter's Nerve)

### Etiology and Pathomechanism

Entrapment of the lateral plantar nerve is often mistakenly referred to as jogger's foot and should not be confused with medial plantar nerve entrapment. Baxter's nerve compression is a rare, but significant cause of heel pain. It often occurs in runners. Entrapment occurs as the nerve passes between the deep fascia of the abductor hallucis muscle (which is often hypertrophied) and the caudal margin of the medial head of the quadratus plantae muscle.

### Symptoms

Patients often have non-specific heel pain that eventually localizes to the medial heel. It is worse with running and may prevent full athletic effort. However, some patients notice that the pain actually improves during running as the pain transitions to paresthesias. Symptoms are often confused with plantar fasciitis as the pain is often at the origin of the plantar fascia. However, whereas plantar fasciitis pain will go away after a few minutes of activity, entrapment of Baxter's nerve can persist throughout the day.

## Diagnosics, Classification

Baxter's nerve compression is often a diagnosis of exclusion. Pain can be replicated with direct palpation of the medial heel. The pain can also be made worse with ankle dorsiflexion. Other sources of heel pain (plantar fasciitis, stress fracture of the calcaneus) must be excluded.

## Therapy: Conservative Treatment and Surgery (Approach, Technique, Risks, Aftertreatment)

Often patients are treated for with heel cups, injections, and plantar fascia and gastrocnemius-soleus stretching. In a study of 69 surgically treated heels, 91 % had used cushioned heel cups, 83 % had taken NSAIDS, and 86 % used received a corticosteroid injection [30]. The average duration of conservative treatment was 14 months.

Classically, surgical treatment uses a long medial incision from the medial malleolus to the medioplantar heel. The tibial nerve is carefully dissected out until the medial and lateral branching can be seen. The superficial and deep fascia of the abductor hallucis muscle is released along with the quadratus plantae and FDB muscle. It is possible to endoscopically release the abductor hallucis as well.

In Baxter's study of 69 heels surgically treated [30] (Level IV study) for compression, 89 % had good to excellent results with 83 % experiencing a complete relief of their symptoms. Close to 50 % of these patients first noticed their pain during athletic activities. In a study of 18 patients, 50 % had complete resolution of their symptoms, while the other 50 % experienced minor pain after extended activities [31].

## Rehabilitation and Back-to-Sports

For the first couple weeks, the leg is elevated as often as possible. After the wound is healed, weight bearing is progressed according to the pain level. Soft insoles are used for 12 weeks. The patient may not run or jump for 12–18 weeks.

## Prevention

There are no studies that evaluate strategies to avoid Baxter's nerve entrapment in the athletic population.

---

## Superficial Peroneal Nerve

### Etiology and Pathomechanism

The superficial peroneal nerve is a branch of the peroneal nerve that innervates the peroneal brevis and longus in the

anterolateral compartment of the leg. It becomes superficial nerve approximately 10 cm proximal to the ankle joint. 4 cm distal, the nerve divides into the intermediate and medial dorsal cutaneous nerve. The intermediate dorsal cutaneous nerve supplies dorsal sensation to the 4th toe and portions of the 3rd and 5th toe and lateral ankle. The medial dorsal cutaneous nerve provides sensation to the dorsal surfaces of the 2nd and 3rd toes and medial ankle and hallux.

Trauma to the nerve is the most common reason for entrapment [25]. Activities that place traction on the nerve include kneeling and squatting for long periods of time. Athletes with chronic ankle instability or lateral ligament instability may also stretch the nerve. Dancers with hypertrophied peroneals may compress the nerve as it goes from deep to superficial [3]. Fascial defects of the lateral compartment can also be a site of compression.

## Symptoms

Patients complain of pain at the junction of the distal 1/3 and medial 1/3 tibia. Pain starts anteriorly and can radiate to the dorsum of the foot. Entrapment distal to the innervation of the peroneals has no motor functional loss. 66 % of patients will not have any sensory loss [32].

## Diagnosics, Classification

Styf and Moberg described three maneuvers to provoke superficial peroneal nerve symptoms [33]. The examiner should hold pressure over the potential site of entrapment while the patient dorsiflexes and everts the foot against resistance. The examiner can then passively plantarflex and invert the ankle. With the ankle in inversion, the course of the superficial peroneal nerve course should be percussed.

In the fourth toe flexion sign [34], when the fourth toe is passively plantarflexed, the skin overlying the superficial peroneal nerve is tented. In patients who had a positive sign (26/30), injection of anesthetic in this area resulted in numbness in the distribution intermediate dorsal cutaneous nerve in all cases. In this study, this sign had a 100% specificity and 85 % specificity.

## Therapy: Conservative Treatment and Surgery

Superficial peroneal nerve compression related to ankle stability should have physical therapy that concentrates on improving proprioception and lateral ankle stability and strengthening. Lateral heel wedges may offload a varus ankle [25].

Surgery is performed when conservative efforts fail. Before the patient is given anesthesia, the site of nerve compression must be identified. The nerve is released by local

fasciotomy where it emerges through the deep fascia. The lateral compartment should be released if the compression is related to chronic exertional compartment syndrome.

In a study of a complete decompression of the fascial tunnel from which the superficial peroneal nerve emerges superficially, 80% of legs were completely asymptomatic (Level IV evidence) [33]. In a study of twelve patients treated with a local fasciotomy, all twelve had a resolution of their symptoms [35].

## Rehabilitation and Back-to-Sports

The athlete does not have to do any specific rehabilitation beyond local wound control. When the wound is healed, the patient can slowly begin to resume all activities.

## Prevention

There are no studies that evaluate strategies to avoid superficial peroneal nerve entrapment.

## Summary

- Nerve entrapment syndromes are under-diagnosed and should be on the differential for any chronic foot and ankle pain in the athlete.
- A careful history and physical examination are the two most important diagnostic tools in the surgeon's armamentarium.
- Conservative management has variable results in nerve compression. Most importantly, all possible external sources of compression must be removed.
- Surgery can provide excellent relief and a return to sports in most patients. Localized decompression is warranted in most nerve compression; tarsal tunnel release merits an extensive release to decompress all possible sources of compression.

## References

1. Singh AIJ, Chiodo C. The surgical treatment of Morton's neuroma. *Curr Orthop*. 2005;19:379–84.
2. Schon LC, Baxter DE. Neuropathies of the foot and ankle in athletes. *Clin Sports Med*. 1990;9:489–509.
3. Kennedy JG, Baxter DE. Nerve disorders in dancers. *Clin Sports Med*. 2008;27:329–34.
4. Morton TG. The classic. A peculiar and painful affection of the fourth metatarso-phalangeal articulation. Thomas G. Morton, M.D. *Clin Orthop Relat Res*. 1979;4–9.
5. Betts L. Morton's metatarsalgia. *Med J Aust*. 1940;1:514–5.
6. Nissen KI. Plantar digital neuritis; Morton's metatarsalgia. *J Bone Joint Surg*. 1948;30B:84–94.
7. Wu KK. Morton's interdigital neuroma: a clinical review of its etiology, treatment, and results. *J Foot Ankle Surg Off Publ Am Coll Foot Ankle Surg*. 1996;35:112–9; discussion 87–8.
8. Thomson CE, Gibson JN, Martin D. Interventions for the treatment of Morton's neuroma. *Cochrane Database Syst Rev*. 2004;(3):CD003118.
9. Thompson CCR, Wood A, Rendall G. *Disorders of the adult foot*. 6th ed. Edinburgh: Churchill Livingstone; 2001.
10. Resch S, Stenstrom A, Jonsson A, Jonsson K. The diagnostic efficacy of magnetic resonance imaging and ultrasonography in Morton's neuroma: a radiological-surgical correlation. *Foot Ankle Int/Am Orthop Foot Ankle Soc/Swiss Foot Ankle Soc*. 1994;15:88–92.
11. Owens R, Gougoulias N, Guthrie H, Sakellariou A. Morton's neuroma: clinical testing and imaging in 76 feet, compared to a control group. *Foot Ankle Surg Off J Eur Soc Foot Ankle Surg*. 2011;17:197–200.
12. Sharp RJ, Wade CM, Hennessy MS, Saxby TS. The role of MRI and ultrasound imaging in Morton's neuroma and the effect of size of lesion on symptoms. *J Bone Joint Surg*. 2003;85:999–1005.
13. Grace D. The lesser rays. In: Helal BRD, Cracchiolo A, Myerson M, editors. *Surgery of disorders of the foot and ankle*. London: Lippincott-Raven; 1996. p. 348–51.
14. Lutter L. *Atlas of adult foot and ankle surgery*. St. Louis: Mosby; 1997.
15. Mulder JD. The causative mechanism in morton's metatarsalgia. *J Bone Joint Surg*. 1951;33-B:94–5.
16. Kilmartin TE, Wallace WA. Effect of pronation and supination orthosis on Morton's neuroma and lower extremity function. *Foot Ankle Int/Am Orthop Foot Ankle Soc/Swiss Foot Ankle Soc*. 1994;15:256–62.
17. Bennett GL, Graham CE, Mauldin DM. Morton's interdigital neuroma: a comprehensive treatment protocol. *Foot Ankle Int/Am Orthop Foot Ankle Soc/Swiss Foot Ankle Soc*. 1995;16:760–3.
18. Rasmussen MR, Kitaoka HB, Patzer GL. Nonoperative treatment of plantar interdigital neuroma with a single corticosteroid injection. *Clin Orthop Relat Res*. 1996:188–93.
19. Magnan B, Marangon A, Frigo A, Bartolozzi P. Local phenol injection in the treatment of interdigital neuritis of the foot (Morton's neuroma). *La Chirurgia degli organi di movimento*. 2005;90:371–7.
20. Hughes RJ, Ali K, Jones H, Kendall S, Connell DA. Treatment of Morton's neuroma with alcohol injection under sonographic guidance: follow-up of 101 cases. *AJR Am J Roentgenol*. 2007;188:1535–9.
21. Mann RA, Reynolds JC. Interdigital neuroma – a critical clinical analysis. *Foot Ankle*. 1983;3:238–43.
22. Kinoshita M, Okuda R, Yasuda T, Abe M. Tarsal tunnel syndrome in athletes. *Am J Sports Med*. 2006;34:1307–12.
23. Gould JS. Tarsal tunnel syndrome. *Foot Ankle Clin*. 2011;16:275–86.
24. DiGiovanni BFAF, Gould JS. Plantar fascia release with proximal and distal tarsal tunnel release: surgical approach to chronic disability plantar fasciitis with associated nerve pain. *Tech Foot Ankle Surg*. 2003;2:254–61.
25. Flanigan RM, DiGiovanni BF. Peripheral nerve entrapments of the lower leg, ankle, and foot. *Foot Ankle Clin*. 2011;16:255–74.
26. Borges LF, Hallett M, Selkoe DJ, Welch K. The anterior tarsal tunnel syndrome. Report of two cases. *J Neurosurg*. 1981;54:89–92.
27. Rosselle N, Stevens A. Unexpected incidence of neurogenic atrophy of the extensor digitorum brevis muscle in young normal adults. In: Desmedt JE, editor. *New developments in electromyography and clinical neurophysiology*. Basel: Karger; 1973.
28. Murphy PC, Baxter DE. Nerve entrapment of the foot and ankle in runners. *Clin Sports Med*. 1985;4:753–63.
29. Koppell HPTW. *Peripheral entrapment neuropathies*. Malabar: Krieger Publishing; 1976.

30. Baxter DE, Pfeffer GB. Treatment of chronic heel pain by surgical release of the first branch of the lateral plantar nerve. *Clin Orthop Relat Res.* 1992;229–36.
31. Goecker RM, Banks AS. Analysis of release of the first branch of the lateral plantar nerve. *J Am Podiatr Med Assoc.* 2000;90:281–6.
32. Baxter DE. Functional nerve disorders in the athlete's foot, ankle, and leg. *Instr Course Lect.* 1993;42:185–94.
33. Styf J, Morberg P. The superficial peroneal tunnel syndrome. Results of treatment by decompression. *J Bone Joint Surg.* 1997;79:801–3.
34. Stephens MM, Kelly PM. Fourth toe flexion sign: a new clinical sign for identification of the superficial peroneal nerve. *Foot Ankle Int/Am Orthop Foot Ankle Soc/Swiss Foot Ankle Soc.* 2000;21:860–3.
35. Malavolta M, Malavolta L. Surgery for superficial peroneal nerve entrapment syndrome. *Oper Orthop Traumatol.* 2007;19:502–10.

Hamza M. Alrabai, Yousef Alrashidi, Victor Valderrabano,  
and Marino Delmi

## Abstract

Morton's neuroma is a common cause of metatarsalgia which characterized by enlargement of interdigital nerve possibly due to nerve entrapment. Morton's neuroma is usually common among middle age population with female gender predominance. Diagnosis of Morton's neuroma is essentially clinical. Imaging modalities may help to delineate and localize the nerve lesion. Prone position foot MRI is associated with higher visibility of Morton's neuroma. Identification of Morton's neuroma, as a cause of metatarsalgia, tends to be missed by clinicians. Hence, high index of suspicion should be invested for this purpose. Proper footwear awareness should be emphasized among athletes. The cross-training shoes are discouraged for long distance running. Local steroid injection may provide temporary relief. Ultrasound-guided injection can improve the localization of injection. Neurectomy of the thickened nerve is considered the standard surgical treatment to date. Majority of surgeons prefer dorsal approach for first-time excision of Morton's neuroma despite of its limited exposure reserving the plantar approach to the revision cases. Plantar surgical approach provides direct anatomical access and allows wide exploration of the area of neuroma. Recurrent neuroma is a major concern after neurectomy. Deep burial of nerve stump within the intermetatarsal soft tissues might minimize the risk of recurrent neuroma formation.

## Keywords

Interdigital neuroma • Intermetatarsal neuroma • Metatarsalgia • Morton's neuroma • Neuralgia

H.M. Alrabai, MD (✉)  
College of Medicine, King Saud University,  
7805, Riyadh 11472, Riyadh, Saudi Arabia  
e-mail: [hamzarabai@gmail.com](mailto:hamzarabai@gmail.com)

Y. Alrashidi  
Orthopedic Surgery Department, Taibah University,  
College of Medicine,, Almadinah Almunawwarah, Saudi Arabia

Orthopaedic Department, SWISS ORTHO CENTER,  
Schmerzklinik Basel, Swiss Medical Network, Basel, Switzerland  
e-mail: [yalrashidi@gmail.com](mailto:yalrashidi@gmail.com)

V. Valderrabano, MD, PhD  
Orthopaedic Department, SWISS ORTHO CENTER,  
Schmerzklinik Basel, Swiss Medical Network, Basel, Switzerland  
e-mail: [vvalderrabano@gsmn.ch](mailto:vvalderrabano@gsmn.ch)

M. Delmi  
Clinic Grangettes, Geneva, Switzerland  
e-mail: [marino.delmi@grangettes.ch](mailto:marino.delmi@grangettes.ch)

## Introduction

Neuroma is a thickening of the nerve tissue due to irritation or compression. Morton's (interdigital) neuroma is typically located in the 3rd or 2nd intermetatarsal space [1]. It acquired its eponym after Thomas George Morton who reported this condition in 1876 [2]. In fact, Civinini and Durlacher preceded Morton in describing such a condition in 1835 and 1845, respectively [3, 4]. Later on, many synonyms were suggested to describe Morton's neuroma such as: Morton neuralgia, Morton metatarsalgia, interdigital neuroma and intermetatarsal neuroma. Until recently, no agreement exists on a standard medical terminology to describe this condition [5].



## Incidence

Currently, there is no accurate estimate of the incidence or prevalence of Morton's neuroma due to lack of solid epidemiological studies on this condition [6]. Morton's neuroma is the commonest nerve lesion involving the forefoot. It was found to be higher at middle age and women. Studies have not proved any side predilection. The usual location of the neuroma is the third intermetatarsal space followed by second intermetatarsal space. Bilateral feet involvement may occur and occasionally more than one lesion could be detected in the same foot [1].

## Aetiology and Pathomechanism

The exact cause of development of Morton's neuroma is not yet well known. However, it seems that neuroma formation is associated with conditions in which the peripheral nerve is subject to irritation or repetitive trauma. Some authors emphasized the role of transverse metatarsal ligament in the pathogenesis of the disease as a compressing structure [1]. Ischemic hypothesis was advocated by Giakoumis et al. depending on a histological evidence of digital arteries luminal occlusion [7]. Poor-fitting footwear particularly high-heeled shoes with tight toe box could lead to Morton's neuroma. Certain foot deformities make the foot more susceptible to develop Morton's neuroma such as bunion, flatfoot, and hammertoes.

Morton's neuroma is associated with some types of sports requiring forefoot excessive stress such as: running, dancing and racquet (Table 42.1). Repetitive hyperdorsiflexion of metatarsophalangeal joint may pull the interdigital nerve against the transverse metatarsal ligament leading to nerve irritation [8]. Metatarsophalangeal joints are subject to high degrees of dorsiflexion during the toe-off phase of running gait cycle and in certain ballet dance maneuvers like demi-pointe, relevé and grand plié [9]. Certain types of firm footwear may increase the forefoot workload such as cross-training and racket shoes and thus should be avoided especially during the long distance running [10].

## Symptoms and Signs

Patients with Morton's neuroma mainly present with neuropathic forefoot pain in the form of tingling, burning and numbness, and often in association with metatarsalgia.

Occasionally, some patients report a subjective feeling of something like a firm particle inside the ball of the foot, elevated ridge in the shoe or sock fold. Tight shoes and weight bearing aggravate symptoms of neuroma, while removal of compressive shoes, and performing a foot massage help to relieve the symptoms.

## Classification

Up to our knowledge, no classification system of clinical value is available for Morton's neuroma.

## Diagnostics

Diagnosis of Morton's neuroma is established mainly on clinical basis [1]. Obtaining a thorough medical history is vital. Physical examination helps the physician to elicit signs of the disease. Meticulous palpation of metatarsal head and metatarsophalangeal joint may give a clue about possible metatarsophalangeal synovitis or Freiberg's disease. Web space tenderness is usually present. Mulder sign can be elicited by compressing the metatarsals against each other while the examiner's contralateral thumb perceives a click on the plantar surface of the target web space. Sensory deficit can be found in some cases. Positive Tinel's sign at the tarsal tunnel indicates that the nerve is entrapped on higher level [10].

Weight-bearing plain radiographs are essential to exclude other causes of metatarsalgia such as metatarsal stress fracture and Freiberg's disease [11]. Magnetic Resonance Imaging (MRI) and Ultrasonography (US) are useful adjuncts to demonstrate the suggestive signs of Morton's neuroma. However, presence of nerve lesion without remarkable neuroma formation is possible [1]. On the other hand, interdigital nerve enlargement can be detected in asymptomatic population [12]. These factors limit the value of imaging diagnostic tools and in turn stress on clinical diagnostic role of Morton's neuroma. Further, a recent meta-analysis has shown no remarkable difference between MRI and US in terms of diagnostic ability for Morton's neuroma [13].

MRI showed high sensitivity (87%) and specificity (100%) for Morton's neuroma detection [14]. Prone position MRI with foot in plantar flexion significantly enhances the visibility of Morton's neuroma. Morton's neuroma can be visualized on T1-weighted MRI images as an isointense

**Table 42.1** Sports associated with a higher risk of Morton's neuroma

Sport type	Specific risky maneuvers
Running	Excessive MTPJ hyperdorsiflexion in toe-off stage during running gait cycle Long distance running with cross-training shoes.
Dancing	Ballet (relevé, grand plié, demi-pointe)

Abbreviations: MTPJ Metatarsophalangeal joint

well-defined mass relative to muscle tissue and hypointense area on T2-weighted MRI images relative to fat [15].

Histologic examination of the excised neuromas is performed routinely in some centers to confirm the diagnosis [16]. The main histologic findings are prominent fibrosis, edema, nerve demyelination, hyalinization and partial obliteration of blood vessels and degenerative changes [7]. Strikingly, presence of these histologic changes was demonstrated histologically in interdigital nerves of asymptomatic individuals [17].

In opinion of authors, persistent forefoot pain after Weil osteotomy for metatarsalgia should alert the surgeon about the possibility of missed Morton's neuroma as the primary etiology of pain. The foot specialist should pay attention to other causes of metatarsalgia, which may mimic presentation of Morton's neuroma such as; metatarsophalangeal synovitis, plantar plate disruption, ganglion, stress fracture, rheumatoid nodule and metatarsal head avascular necrosis [16, 18, 19]. In few occasions, forefoot neoplastic conditions could be confused with Morton's neuroma [20].

## Therapy

Early detection of the disease at its initial stages is likely to yield a satisfactory outcome with conservative remedies. It is imperative to counsel patients with Morton's neuroma to modify their lifestyle in terms of substituting poor-fitting footwear with accommodative wide toe box shoes and avoiding activities with a potential harm to the diseased nerve. Athletes should avoid cross-training shoes [10]. Padding can help supporting the metatarsal arch and keeping the metatarsal bones apart from each other to relieve the relatively crushed nerve in between. Similarly, the use of a metatarsal bar is a good option as it works by shifting the body weight force away from the forefoot and offloading the area of interest.

Steroid injections became a reasonable option after exhaustion of the conservative means and before considering surgical intervention. Some researchers advocate the use of ultrasound (US) as an adjunctive tool to ensure proper placement of injection. Adding to the cost of US, obstruction of the dorsal field of injection by the US transducer remains an

unfavorable issue for this technique [21, 22]. Combining anesthetic agent with steroid showed a better outcome as compared to steroid alone for 3 months in a randomized trial published by Thomson and co-researchers. Their recommended dose per injection was 40 mg of methylprednisolone (1 mL) and 2% lignocaine (1 mL) [23].

Radiofrequency Thermoneurolysis Therapy (RTT) of Morton's neuroma is an alternative minimally invasive treatment modality with a relatively promising outcome. The desired location of ablation is identified clinically before intervention [24]. Ultrasound-assisted radiofrequency was attempted to increase the accuracy of probe aiming [25]. RTT efficacy is still a matter of controversy.

Surgery is indicated when the non-operative treatment did not provide relief. It is worth to mention that the current pool of literature related to Morton's neuroma has not provided yet an evidence-based answer concerning whether the operative treatment outweighs the conservative treatment measures, in terms of a better outcome [6]. Excision is still the standard surgical intervention for Morton's neuromas, which were refractory to nonoperative treatment. The ideal surgical approach by which the excision can be conducted is a matter of debate. A comparison between dorsal and plantar approaches for excision of Morton's neuroma is summarized in Table 42.2. Division of the intermetatarsal transverse ligament can improve intraoperative isolation of the neuroma, which is usually hidden underneath. Furthermore, ensuring that plantar branches of the intermetatarsal nerves are adequately excised, by performing the neurectomy level at approximately 3 cm proximal to the intermetatarsal transverse ligament, may minimize the chance of recurrence. Plantar-directed nerve branches tend to adhere to adjacent structures, if not resected, and consequently prevent back-retraction of stump away from the weight-bearing area (Fig. 42.1) [26]. On the other hand, plantar approach is likely to end up with a painful surgical scar and subsequent walking discomfort. Nery et al. suggested that the use of transverse planter incision, if made distally in a relatively low-weight bearing area, is associated with lower rate of surgical scar-related complications [27].

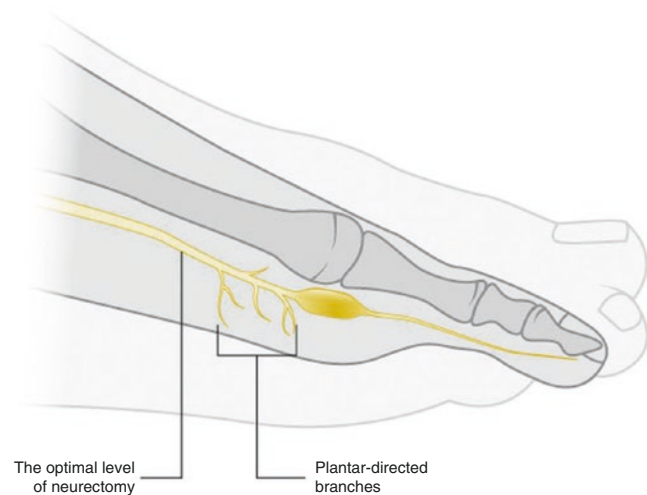
In athletes, neuroma resection through plantar incision is the authors' preferred approach. Plantar approach allows

**Table 42.2** Comparison between dorsal and plantar approaches for excision of Morton's neuroma

	Dorsal approach	Planter approach
Exposure	Limited	Adequate
Wound healing	No significant problems	Occasional painful scarring
Transverse metatarsal ligament	Division is often needed to access proximal part of neuroma	It is possible to leave it intact
Weight bearing	Early	Delayed
Recurrence	Higher	Less
Patients' satisfaction	No difference	No difference

complete resection of neuroma without need to section the transverse metatarsal ligament. The plantar skin is incised longitudinally over the affected interspace. Care to be taken to keep the incision away from the high-pressure areas under the metatarsal heads. Soft tissue meticulous dissection is carried out to explore the neuroma. Generous resection of the thickened nerve is conducted. After neuroma excision, the proximal nerve end should be concealed within the intermetatarsal soft tissues to minimize stump neuroma formation.

Some foot surgeons advocate endoscopic nerve decompression by selective division of the transverse intermetatarsal ligament without neurectomy. Believing that Morton's neuroma is merely an entrapment nerve syndrome rather than a true neuroma makes the rational basis of isolated decompression treatment approach [28].



**Fig. 42.1** The neurectomy level should be made around 3 cm proximal to neuroma lesion to untie the nerve stump from the plantarly-directed branches which tend to prevent stump back retraction leading to subsequent neuroma formation

In recurrent neuromas, the plantar approach should be considered as it offers a direct access through which the neuroma stump can be easily identified and resected. The presence of remarkable scarring around the neuroma stump renders the dorsal approach quite demanding nevertheless; Mann and Reynolds recommend dorsal approach for redo neuroma resection [29].

## Rehabilitation and Back-to-Sports

Post-operative immediate weight bearing is possible if the neuroma was excised through dorsal approach. Full weight bearing mobilization is allowed once the plantar surgical wound healing is ensured.

## Prevention

Avoidance of footwear with high heels, tapered narrow toe box and cross-training firm outsole plays a remarkable role of preventing Morton's neuroma development.

## Evidence

Table 42.3 shows levels of the best available evidence on treatment of Morton's neuroma.

## Summary

1. The most important differential diagnosis of Morton's neuroma is metatarsophalangeal joint pain.
2. Magnetic resonance imaging (MRI) for Morton's neuroma is preferred to be done in prone position with foot in plantarflexion.

**Table 42.3** Illustration of selected best evidence available on therapy of Morton's neuroma

Authors	Journal, year	Study topic	Number of patients	Evidence level
Rungprai et al. [30]	<i>FAI</i> , 2015	Simple neurectomy versus neurectomy with intramuscular implantation for interdigital neuroma: a comparative study	118	III
Villas et al. [31]	<i>FAI</i> , 2008	Neurectomy versus neurolysis for Morton's neuroma	69	III
Nery et al. [27]	<i>JBJS Am</i> , 2012	Plantar approach for excision of a Morton neuroma: a long-term follow-up study	227	IV
Barret et al. [28]	<i>Open J Orthop</i> , 2012	Endoscopic Decompression of Intermetatarsal Nerve (EDIN) for the treatment of mortons entrapment multicenter retrospective review	193	IV
Kasperek et al. [1]	<i>Int Orthop</i> , 2013	Surgical treatment of Morton's neuroma: clinical results after open excision	111	IV

3. Morton's neuroma is found in common with sports requiring forefoot excessive stress and repetitive hyperdorsiflexion of metatarsophalangeal joints.
4. Certain footwear with relatively rigid outsole such as cross-training and racket shoes may increase the risk of development of neuroma.
5. Although there is no clear evidence to support superiority of a surgical approach over the other in treatment of Morton's neuroma, the authors prefer a plantar incision in athletes for neuroma excision as it is not necessary to divide the intermetatarsal ligament.

## References

1. Kasperek M, Schneider W. Surgical treatment of Morton's neuroma: clinical results after open excision. *Int Orthop*. 2013; 37(9):1857–61.
2. Mostofi S, editor. *Who's who in orthopedics*. London: Springer; 2005.
3. Durlacher L, editor. *Treatise on corns, bunions, the diseases of nails and the general management of the feet*. London: Simpkin-Marshall; 1835.
4. Pasero G, Marson P. Filippo Civinini (1805–1844) and the discovery of plantar neuroma. *Reumatismo*. 2006;58(4):319–22.
5. Larson EE, Barrett SL, Battiston B, Maloney Jr CT, Dellon AL. Accurate nomenclature for forefoot nerve entrapment: a historical perspective. *J Am Podiatr Med Assoc*. 2005;95(3): 298–306.
6. Thomson CE, Gibson JN, Martin D. Interventions for the treatment of Morton's neuroma. *Cochrane Database Syst Rev*. 2004(3): CD003118.
7. Giakoumis M, Ryan JD, Jani J. Histologic evaluation of intermetatarsal Morton's neuroma. *J Am Podiatr Med Assoc*. 2013; 103(3):218–22.
8. Ferkel E, Davis WH, Ellington JK. Entrapment neuropathies of the foot and ankle. *Clin Sports Med*. 2015;34(4):791–801.
9. Raikin SM, Schon LC. Nerve entrapment in the foot and ankle of an athlete. *Sports Med Arthroscopy Rev*. 2000;8(4):387–94.
10. Hockenbury RT. Forefoot problems in athletes. *Med Sci Sports Exerc*. 1999;31(7 Suppl):S448–58.
11. Pastides P, El-Sallakh S, Charalambides C. Morton's neuroma: a clinical versus radiological diagnosis. *Foot Ankle Surg Off J Eur Soc Foot Ankle Surg*. 2012;18(1):22–4.
12. Symeonidis PD, Iselin LD, Simmons N, Fowler S, Dracopoulos G, Stavrou P. Prevalence of interdigital nerve enlargements in an asymptomatic population. *Foot Ankle Int*. 2012;33(7):543–7.
13. Bignotti B, Signori A, Sormani MP, Molfetta L, Martinoli C, Tagliafico A. Ultrasound versus magnetic resonance imaging for Morton neuroma: systematic review and meta-analysis. *Eur Radiol*. 2015;25(8):2254–62.
14. Zanetti M, Ledermann T, Zollinger H, Hodler J. Efficacy of MR imaging in patients suspected of having Morton's neuroma. *AJR Am J Roentgenol*. 1997;168(2):529–32.
15. Weishaupt D, Treiber K, Kundert HP, Zollinger H, Vienne P, Hodler J, et al. Morton neuroma: MR imaging in prone, supine, and upright weight-bearing body positions. *Radiology*. 2003;226(3):849–56.
16. O'Connor KM, Johnson JE, McCormick JJ, Klein SE. Correlation of clinical, operative, and histopathologic diagnosis of interdigital neuroma and the cost of routine diagnosis. *Foot Ankle Int*. 2015;37(1):70–4.
17. Bourke G, Owen J, Mchet D. Histological comparison of the third interdigital nerve in patients with Morton's metatarsalgia and control patients. *Aust N Z J Surg*. 1994;64(6):421–4.
18. Umans H, Srinivasan R, Elsinger E, Wilde GE. MRI of lesser metatarsophalangeal joint plantar plate tears and associated adjacent interspace lesions. *Skeletal Radiol*. 2014;43(10):1361–8.
19. Miller SD. Technique tip: forefoot pain: diagnosing metatarsophalangeal joint synovitis from interdigital neuroma. *Foot Ankle Int*. 2001;22(11):914–5.
20. Prieskorn DW, Irwin RB, Hankin R. Clear cell sarcoma presenting as an interdigital neuroma. *Orthop Rev*. 1992;21(8):963–70.
21. Smith RW. Steroid injection for Morton neuroma – data-based justification. *J Bone Joint Surg Am*. 2013;95(9):e641–2.
22. Schon L. An injection of corticosteroid plus anesthetic was more effective than anesthetic alone for Morton neuroma. *J Bone Joint Surg Am*. 2014;96(4):334.
23. Thomson CE, Beggs I, Martin DJ, McMillan D, Edwards RT, Russell D, et al. Methylprednisolone injections for the treatment of Morton neuroma: a patient-blinded randomized trial. *J Bone Joint Surg Am*. 2013;95(9):790–8, s1.
24. Moore JL, Rosen R, Cohen J, Rosen B. Radiofrequency thermoneurolysis for the treatment of Morton's neuroma. *J Foot Ankle Surg Off Pub Am Coll Foot Ankle Surg*. 2012;51(1):20–2.
25. Deniz S, Purtuloglu T, Tekindur S, Cansiz KH, Yetim M, Kilickaya O, et al. Ultrasound-guided pulsed radio frequency treatment in Morton's neuroma. *J Am Podiatr Med Assoc*. 2015;105(4):302–6.
26. Amis JA, Siverhus SW, Liwnicz BH. An anatomic basis for recurrence after Morton's neuroma excision. *Foot Ankle*. 1992; 13(3):153–6.
27. Nery C, Raduan F, Del Buono A, Asaumi ID, Maffulli N. Plantar approach for excision of a Morton neuroma: a long-term follow-up study. *J Bone Joint Surg Am*. 2012;94(7):654–8.
28. Barrett SL, Rabat E, Buitrago M, Rascon VP, Applegate PD. Endoscopic Decompression of Intermetatarsal Nerve (EDIN) for the treatment of Morton's entrapment—multicenter retrospective review. *Open J Orthop*. 2012;2:19–24.
29. Mann RA, Reynolds JC. Interdigital neuroma – a critical clinical analysis. *Foot Ankle*. 1983;3(4):238–43.
30. Rungprai C, Cychosz CC, Phruetthiphath O, Femino JE, Amendola A, Phisitkul P. Simple neurectomy versus neurectomy with intramuscular implantation for interdigital neuroma: a comparative study. *Foot Ankle Int*. 2015;36(12):1412–24.
31. Villas C, Florez B, Alfonso M. Neurectomy versus neurolysis for Morton's neuroma. *Foot Ankle Int*. 2008;29(6):578–80.

Rik Osinga, Andreas Gohritz, Martin D. Haug,  
and Dirk J. Schaefer

## Abstract

Successful soft tissue reconstruction of the foot and ankle is challenging and often needs an interdisciplinary approach, especially when composite defects are present. This chapter provides a coherent concept to treat cases of severe skin and soft tissue damage of the foot and ankle. A functional and anatomical subdivision of the foot and ankle region is proposed depending on its pressure distribution, soft tissue quality, zones of shear and the structural anatomy. A treatment algorithm, highlighted by two clinical cases, is presented considering common and well established local, regional and micro-surgical reconstructive options to restore maximum sensibility, motor function and aesthetics.

## Keywords

Foot • Ankle • Soft tissue defect • Functional and anatomical subdivision • Reconstruction • Flap • Treatment algorithm

## Introduction

The ankle and foot region are highly specialized regions of the lower extremity. To bear body weight in an upright position and to efficiently allow locomotion, evolution has led to a functional structure that is only covered by a thin layer of soft tissue according to the principle ‘form follows function’. The sole of the foot plays a key role and provides sensory protection and proprioception and its special anatomy allows it to withstand high pressure loads and shearing forces due to a unique plantar fibro-fatty pad which serves as a hydraulic pressure system.

## Etiology and Pathomechanism

The most common etiologic factors for soft tissue defects of the foot and ankle include:

1. Direct trauma
2. Postoperative dehiscence
3. Compartment syndrome
4. Tumor (e.g. sarcoma)
5. Infection (incl. osteomyelitis)
6. Radiation
7. Vascular insufficiency (arterial/venous)
8. Diabetes

## Diagnostics, Classification

Patients present themselves with various degrees of soft tissue damage. Although it may be a point of discussion what qualifies to be called a ‘severe case’, it usually involves more than one subunit of the foot and ankle and can involve exposed fractures, tendons and neurovascular structures.

Preoperative patient evaluation and wound analysis includes above all:

- ABCDE principles: Complete trauma evaluation is of paramount importance

R. Osinga (✉) • A. Gohritz • M.D. Haug • D.J. Schaefer  
Department of Plastic, Reconstructive, Aesthetic and Hand  
Surgery, University Hospital of Basel,  
Spitalstrasse 21, Basel CH-4031, Switzerland  
e-mail: rik.osinga@usb.ch; andreas.gohritz@usb.ch;  
martin.haug@usb.ch; dirk.schaefer@usb.ch

- Vascular examination (e.g. palpable pulses, Doppler examination)
- Neurological examination
- Assessment of size, depth, and involvement of vital structures of the wound
- Radiographic examination (X-ray, CT, MRI)
- Comorbidities, general and nutrition status
- Personal history

---

## Principles of Reconstruction

Treatment depends on size, depth and localization of the wound and the exposure of vital structures such as nerves/vessels, bone and tendons. In the foot and ankle region, restoration of plantar proprioception, sensibility, bony stability and freely moving joints is of paramount importance for early mobilization and weight-bearing and thus for rehabilitation and social reintegration of the patient. In particular, the integrity of skin over a sufficient layer of soft tissue allows unimpaired shoe wearing in the absence of ulcer or pain.

---

## General Principles

1. Surgical debridement of all devitalized tissue to obtain healthy wound bed
2. Restoration of vascularity, bony stability, structure and joint function
3. Obliteration of dead space
4. Durable coverage of vital structures (e.g. bone, tendon, nerves and vessels)
5. Minimal donor site morbidity
6. Adequate aesthetic result

## Timing of Reconstruction

If not contraindicated by life-threatening injuries or comorbidities, soft tissue replacement requires an orderly progression of interventions. Godina in 1986 [1] pronounced that any open lower extremity wound should be closed as immediately as possible, although this is logistically not always possible. It is nowadays believed that closure in the acute wound phase (first week) is needed, especially if bone, tendon, nerves and vessels are exposed.

## Surgical Debridement

Surgical debridement is imperative and reduces the risk of infection. Therefore, all devitalized tissue must be removed, even if it were of structural importance until only viable

tissue remains. Furthermore, tissue samples for microbiological analysis and histology to identify possible osteomyelitis should be taken.

## Fracture and Vessel Management

Fractures must be reduced by an orthopedic or podiatric surgeon and vascular insufficiencies must be restored not only to sustain foot viability, but also to preserve the possibility of local foot flaps and potential vascular recipient sites for a free flap.

## Wound Conditioning

Negative-pressure wound therapy reduces the frequency of dressing changes, minimizes patient discomfort and prevents wound desiccation. It enhances the formation of granulation tissue and facilitates wound contracture, therefore it may sometimes result in spontaneous healing of a small defect or allow the use of an autologous skin graft only. However, the risk of an unstable scar and an insensible scarred area, where contracture can impair joint function and cause nerve compression, remains. For the vast majority of cases therefore, it is nothing more than a bridge to the definitive method to restore soft tissue and skin integrity. Then, the newly formed granulation tissue would have to be removed before coverage with a flap.

---

## Subunit Reconstruction

The foot and ankle region is divided into anatomical subunits:

1. Ankle (including Achilles tendon)
2. Dorsum of the foot
3. Sole of the foot

## Single Subunit Reconstruction

If only one subunit of the foot or ankle region has been damaged, soft tissue integrity is restored by using the reconstructive algorithm consulting the full armamentarium of reconstructive options from skin graft over pedicled flaps to free flaps [2].

## Multiple Subunit Reconstruction

If more than one subunit is involved, a pedicled or free flap is considered to be the gold standard. A free flap provides the

possibility to reconstruct large soft tissue defects, allows adaptation of the thickness of the flap, can avoid skin grafting and is greatly independent in placement of the flap because of little constraint of the vascular pedicle.

### Principles of Subunit Reconstruction

Early descriptions of the subunit principle divided the foot and ankle into various zones according to the need of specific tissue requirements [3]. For example, the highly specialized glabrous skin of the planter surface of the foot is difficult to replace with a durable yet thin flap. The dorsum of the foot on the other hand must also be thin, but the final aesthetic appearance is of much greater importance. This also counts for the ankle, but any restriction of motion is a functional concern.

The subunit principle has been updated [4, 5] so that any flap selected must meet the functional and aesthetic demands of the zone given, with bulk or contour that does not impede the use of shoe wear and proper amputation.

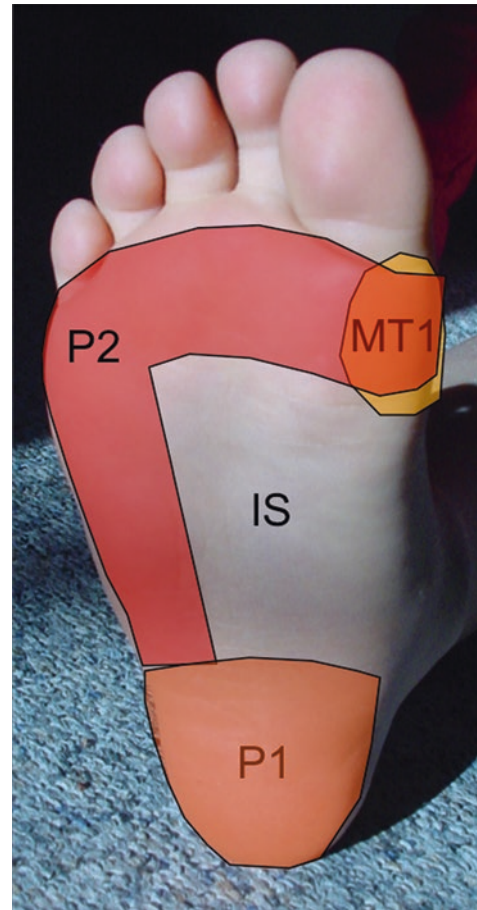
### Pressure Zones of the Foot

Literature distinguishes between direct and indirect pressure zones of the foot and ankle [4]. Direct pressure is applied on the sole of the foot, where four zones are distinguished (Fig. 43.1): P1: Plantar 1=heel, P2: Plantar 2=lateral border of the foot and metatarsal II-V, MT1: Metatarsal I, IS: InStep=arch of the foot. Indirect pressure is applied on the dorsum (D), on the malleoli (M) and on the calcaneal region (C) (Fig. 43.2). Between the direct and indirect pressure zone is a transition zone, where high shear forces are applied and scars should be minimized.

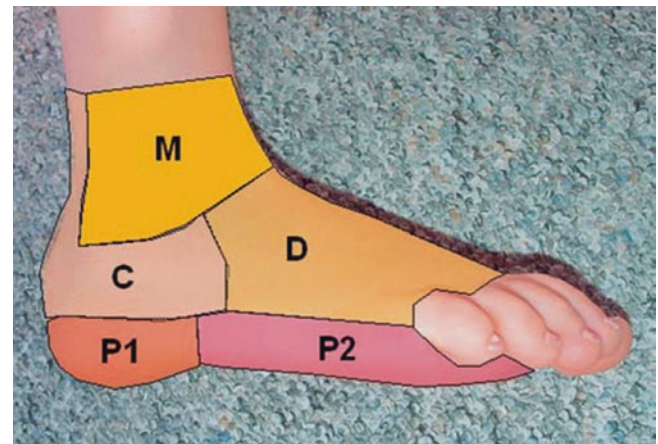
Several donor sites for free flaps provide enough tissue to cover a single subunit, but may not be able to provide enough volume and surface for the typical mangled foot and ankle deformity which by definition involves two or more subunits simultaneously. Therefore, local foot flaps [6, 7] and distally based neurocutaneous flaps from the more proximal lower leg [8] are inadequate in the mangled foot and ankle. Used often in the last century as an alternative to free flaps, a cross-leg flap could be an option despite long-term limb immobilization and need for a staged procedure in very special indications [9].

### Reconstructive Options and Evaluation

A variety of local, regional and free flaps have been described as reconstructive options. Table 43.1 gives an overview of the most clinically relevant fascial, fascio-cutaneous and



**Fig. 43.1** Zones of direct pressure. P1 Plantar 1=heel, P2 Plantar 2=lateral border of the foot and metatarsal II-V, MT1 Metatarsal I, IS InStep=arch of the foot [4] (Used with permission of © Georg Thieme Verlag KG)



**Fig. 43.2** Zones of indirect pressure. D dorsum, M malleolar region, C calcaneal region, P1 Plantar 1=heel, P2 Plantar 2=lateral border of the foot and metatarsal II-V [4] (Used with permission of © Georg Thieme Verlag KG)

muscle flaps, the type of flap perfusion is not considered. To select the right flap best matching the patient's needs, the

surgeon has to take into account both morphology and type of involved tissues (muscle, tendon, fascia, skin) of the defect as well as the functional and anatomical features of the according subunit.

The evaluation of the quality of the reconstructive option is dependent on the morphological structure and type of tissue (muscle, fascia, skin) and must consider the functional and anatomical properties of the zones described in Figs. 43.1 and 43.2. Tissue volume, donor site morbidity, microsurgical possibility of resensitisation, the ability to withstand shear and pressure and the thickness of the transplanted flap must be considered (Table 43.2).

## Treatment Algorithm

The evaluation of the pressure zones proposed and the reconstructive options discussed allow the surgeon to select a flap for a defined defect (Table 43.3).

## Regional Propeller Flaps

These recently devised local island fascio-cutaneous flaps are based on a single dissected perforator [10]. They are designed like a propeller with two blades of unequal length with the perforator forming the pivot point so that when the blades are switched, the long arm fills the defect. The ability of this flap to rotate any angle up to 180° makes it extremely versatile for reconstructing defects of the distal lower extremity where

it was originally conceived. They are useful around the ankle joint, including the medial and lateral malleolus, which are common sites of poor healing following compound fractures and internal fixation (Figs. 43.3, 43.4, 43.5, and 43.6).

## Special Considerations Regarding the Reconstruction of Specific Subunits

### Ankle Reconstruction

Ankle defects require not only wound coverage, but also regional restoration of form, function and aesthetics (Figs. 43.3, 43.4, 43.5, and 43.6).

### Achilles Tendon Reconstruction

Achilles tendon defects often occur in infected wounds and may require tendon debridement and biopsies, before composite reconstruction can be initiated (Figs. 43.7, 43.8, and 43.9).

### Ankle Reconstruction – Composite Defects

Soft tissue defects are frequently complicated by additional segmental bone defects, mainly of traumatic origin directly or due to pseudarthrosis or osteomyelitis.

**Table 43.1** Reconstructive options of the foot and ankle

	Local	Regional	Free flap
Fascio-cutaneous	Perforator flaps Plantar instep flap Supramalleolar flap	Sural flap Propeller flap Plantar instep flap	Lateral arm flap Scapula/parascapular flap Anterolateral thigh flap Plantar instep flap
Muscular	Abductor hallucis flap Abductor digiti minimi flap	Peroneus brevis flap	Gracilis flap Latissimus dorsi flap
Fascial	Adipo-fascial flap	Adipo-fascial flap Dorsal pedis flap	Serratus anterior flap Temporal artery fascia flap

**Table 43.2** Evaluation of quality of reconstructive options

Type of flap	Defect – size	Sensibility	Pressure	Shear	Thickness
Local flaps	—	+	+++	+++	+++
Plantar instep flap (pedicled)	++	+++	+++	+++	+++
Plantar instep flap (free)	++	+/-	+++	+++	+++
Sural flap	++	–	+	++	++
Free fascio-cutaneous flap	+++	+++	+	—	+/-
Free muscle flap	+++	–	++	++	+++

Used with permission of © Georg Thieme Verlag KG

+++ very good, ++ good, + fair, +/- indifferent, – not so good, – – poor, – – – very poor



**Table 43.3** Algorithm for flap selection for foot and ankle reconstruction

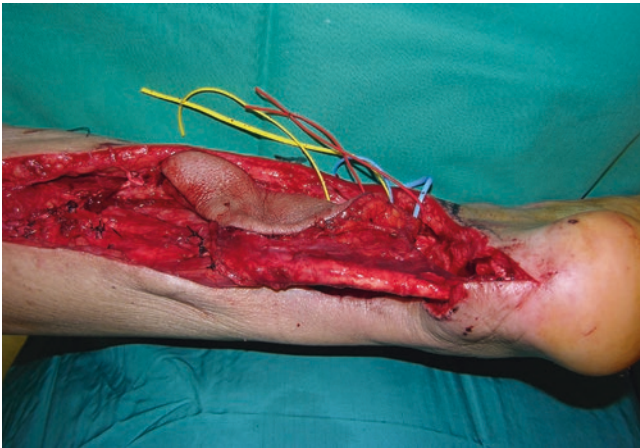
Foot subunit	Defect size	Flaps
P1	Small	Local rotation or transposition flap, plantar rotation flap
	Large	Pedicled/free plantar instep flap, gracilis flap, latissimus dorsi flap+SSG
P2	Small	Local flap, plantar rotation flap
	Large	Free plantar instep flap, gracilis flap, latissimus dorsi flap+SSG
C	Small	Supramalleolar flap, sural artery flap, perforator flap
	Large	Free gracilis flap
	Achilles Tendon	Sural artery flap, lateral arm flap (tendo-osteo-fascio-cutaneous, distal part of the triceps tendon)
M	Small	Supramalleolar flap, perforator propeller flap
	Large	Sural artery flap, lateral arm flap, anterior lateral thigh flap
D	Small	Local/rotation flap
	Large	Lateral arm flap, serratus anterior flap, temporal artery fascia flap, groin flap

SSG split skin graft

**Fig. 43.3** Supramalleolar lateral ankle defect. Ventral incision to raise a propeller flap based on a perforator of the fibular artery. The foreceps are next to the perforator, indicating the pivot point**Fig. 43.5** Propeller flap turned 180° to completely cover the defect**Fig. 43.4** Completely mobilized propeller flap**Fig. 43.6** Propeller flap at the end of the operation



**Fig. 43.7** Chronic infected wound with exposed, sutured Achilles tendon



**Fig. 43.8** Wound after debridement and with extended lateral arm flap including fascio-tendinous component taken from the distal triceps tendon to reconstruct the Achilles tendon after microvascular anastomosis



**Fig. 43.9** Flap after wound closure

## Osteo-cutaneous Composite Flaps

Various composite (osteo-cutaneous) donor sites have proven useful [11]:

- Fibula flap
- Iliac crest flap
- Serratus anterior and rib
- Scapula/Metatarsal bone/Radius
- Lateral arm flap with bony component
- Medial femur condyle

Alternative techniques are

- Fascio-cutaneous flaps combined with bone grafts
- Ilizarov technique (ring fixateur for callus distraction of the lower leg, but not for the ankle and foot)

## Composite Ankle Reconstruction

Osteo-myo-cutaneous chimeric flaps are composed from multiple components harvested from the same donor site, e.g. scapula bone, serratus anterior muscle and the thoracodorsal artery perforator (TAP) flap.

## Reconstruction of the Dorsum of the Foot

A close relation exists between the anatomy and function in this region regarding thin skin coverage of the dorsum of the foot, the extensor tendons and various sensory nerve branches. Composite defects mostly occur due to traumatic origin or due to postoperative deformities and have to be evaluated individually.

## Sole Reconstruction

Reconstructions in this region should primarily be sensible and withstand pressure and shear stress through good cushioning properties. Secondly, they should restore form (contour, no bulking), function (tendon, bone, joint), sensibility and aesthetics (skin colour, no patchwork) [12–17].

Various concepts have been devised regarding coverage of the sole of the foot :

### 1. Muscle Flaps with Split Skin Graft (SSG)

These methods, e.g. the latissimus dorsi muscle flap and SSG provide a good cushioning, but no sensibility.

## 2. Fascio-cutaneous Flaps

Fascio-cutaneous muscle flaps often provide sensibility, but have proven suboptimal regarding the shearing stress forces specific to the sole of the foot.

## 3. Local Plantar Instep Pedicled Flaps

The plantar instep flap can be used as

- Instep Island Flap
- Distally pedicled Instep Flap
- Cross-foot Instep flap

A pedicled instep island flap is useful to cover the heel or metatarsal region.

Distally pedicled instep flaps are well-suited for forefoot defects (cross-foot instep flap). If a pedicled flap is impossible, microsurgical transplantation may be used.

compared to multiple stage procedures. Flap choice depends on an algorithmic approach based on a subunit adapted reconstruction system.

3. Although almost all soft tissue defects may be covered today using modern sophisticated techniques (including microsurgery), each patient must still be treated individually taking into account his physical, social, psychological and also economical needs.
4. The ultimate goal is to maximize restoration of the patient's lifestyle with the best possible ability to ambulate with absence of pain and ulcerations, unimpaired shoe wearing and full weight bearing and, if possible, to perform sports independently again.

---

## Rehabilitation and Back-to-Sports

Postoperative flap monitoring is essential as almost all anastomotic failures occur within the first 3 days. Rehabilitation starts by dangling and walking protocols depending on the overall the extent of injury and involvement of other body systems. Edema management requires limb elevation for an indeterminate period. Shoewear should avoid constriction at the recipient site and pressure on the flap, especially if sensation is impaired. Usually, long-term secondary procedures require flap re-elevation, e.g. if bone grafting is needed. Care must be taken while raising the flap not to compromise its perfusion. Often, flap contours need readjustment, especially to reduce bulk, which can be avoided by using a muscle flap atrophying over time. Instability of the flap, especially due to intrinsic mobility or wobbling, is most commonly seen over the heel and a concern both for muscle and perforator flaps that may also need correction.

---

## Evidence

The advice given in this book chapter is based on case series and therefore Evidence Level IV according to the Oxford Centre for Evidence-based Medicine.

---

## Summary

Five most important points in one to two sentences each.

1. The decision to salvage the mangled foot and ankle requires thorough preoperative planning, infection control by surgical debridement and stable osteosynthesis.
2. Early single-stage reconstruction with soft tissue or composite flaps are feasible and provide many distinct advantages

---

## References

1. Godina M. Early microsurgical reconstruction of complex trauma of the extremities. *Plast Reconstr Surg.* 1986;78:285–92.
2. Ullman Y, Fodor L, Ramon Y, et al. The revised reconstructive ladder and its applications for high-energy injuries to the extremities. *Ann Plast Surg.* 2006;56:401–5.
3. Hidalgo DA, Shaw WW. Reconstruction of foot injuries. *Clin Plast Surg.* 1986;13:663–80.
4. Haug MD, Valderrabano V, Rieger UM, Pierer G, Schaefer DJ. Anatomically and biomechanically based treatment algorithm for foot and ankle soft tissue reconstruction. *Handchir Mikrochir Plast Chir.* 2008;40:377–85.
5. Medina ND, Kovach III SJ, Levin LS. An evidence-based approach to lower extremity acute trauma. *Plast Reconstr Surg.* 2011;127:926–34.
6. Hallock GG. Local fasciocutaneous flap skin coverage for the dorsal foot and ankle. *Foot Ankle.* 1991;11:275–81.
7. Hallock GG. Distally based flaps for skin coverage of the foot and ankle. *Foot Ankle Int.* 1996;17:343–8.
8. Nakajima H, Imanishi N, Fukuzumi S, et al. Accompanying arteries of the cutaneous veins and cutaneous nerves in the extremities: anatomical study and a concept of the venoadipofascial and/or neuroadipofascial pedicled fasciocutaneous flap. *Plast Reconstr Surg.* 1998;102:779–91.
9. Lu L, Liu A, Zhu L, et al. Cross-leg flaps: our preferred alternative to free flaps in the treatment of complex traumatic lower extremity wounds. *J Am Coll Surg.* 2013;217:461–71.
10. Teo LC. The propeller flap concept. *Clin Plast Surg.* 2010;37:615–26.
11. Yazar S, Lin CH, Wei FC. One-stage reconstruction of composite bone and soft-tissue defects. *Plast Reconstr Surg.* 2004;114:1457–66.
12. Milanov NO, Adamyan RT. Functional results of microsurgical reconstruction of plantar defects. *Ann Plast Surg.* 1994;32:52.
13. Hong JP, et al. Sole reconstruction using AL perforator flaps. *Plast Reconstr Surg.* 2007;119:186.
14. Santanelli F, Tenna S, Pace A, et al. Free flap reconstruction of the sole of the foot with or without sensory nerve coaptation. *Plast Reconstr Surg.* 2002;109:2314–22.
15. Scheufler O, Kalbermatten DF, Pierer G. Instep free flap for plantar soft tissue reconstruction: indications and options. *Microsurgery.* 2007;27(3):174–80.
16. Peek A, Giessler G. Free composite heel reconstruction. *Ann Plast Surg.* 2006;56(6):628–34.
17. Durham JW, et al. Outcome after free flap reconstruction of the heel. *Foot Ankle Int.* 1994;15:250.

---

## Part III

### Sports Specific Injuries

Christian Plaass, Christoph Becher,  
and Hauke Horstmann

#### Abstract

Training in health clubs is in many countries rated as the most performed sports activity. The general risk of injuries is low during aerobic and fitness sports and most injuries are due to overuse. Especially the Achilles tendon and the plantar fascia are at risk to become inflamed. Shin splints and stress fractures can also develop. Most overuse injuries can be prevented or treated by adequate regeneration periods, shoe-wear and training modifications.

Changed training methods and the introduction of unstable surfaces in the training can increase the risk of acute ankle sprains, which also occur during aerobics especially when using steps and in exhausted athletes. The treatment of these acute injuries follows the general principles for these injuries.

#### Keywords

Aerobics • Achilles tendinitis • Elliptic trainer • Step aerobics • Crossfit • Unstable surface

## Injuries

Nearly three-quarters of all injuries in aerobics and fitness sports are chronic injuries [1]. The tendinous structures are especially at risk from overuse.

### Achilles Tendinitis

Painful swellings around the Achilles tendon are common in these sports. Mid-portion and insertional Achilles tendinitis should be differentiated in patients.

C. Plaass, Dr. Med (✉) • C. Becher, PD, Dr. Med  
Orthopedic Clinic, Hannover Medical School,  
Anna-von-Borries Strasse 1-7, 30625 Hannover, Germany  
e-mail: [Christian@Plaass.de](mailto:Christian@Plaass.de); [Christoph.Becher@ddh-gruppe.de](mailto:Christoph.Becher@ddh-gruppe.de)

H. Horstmann, MD  
Orthopedic Clinic, Hannover Medical School,  
Anna-von-Borries Strasse 1-7, 30625 Hannover, Germany

Institute of Sports Medicine, Hannover Medical School,  
Carl-Neuberg Strasse 1, 30625 Hannover, Germany  
e-mail: [Horstmann.Hauke@mh-hannover.de](mailto:Horstmann.Hauke@mh-hannover.de)

## Shin Splints and Stress Fracture

Shin splints are a common cause of pain. Patients experience pain on the distal tibia, especially after long training pauses, technique change or too rapid increase in training intensity.

Another overuse injury described in aerobics is stress fracture. These happen most often in the lesser metatarsal bones and begin with either slowly developing pain over days or a sharp onset of pain. Swelling in the foot and lower leg is also common. If not treated properly this can cause ongoing pain.

### Ankle Sprain

Ankle sprains can occur during aerobics or training on unstable surfaces as is proposed in some newer fitness plans. These are also more likely and can be more severe when training with additional weights or during exhaustion.

## Plantar Fasciitis and Heel Spurs

The plantar fascia can become inflamed, resulting in pain on the underside of the foot. This can be severe and hinder the patient from walking properly.

## Forefoot Injuries (Metatarsalgia, Sesamoiditis and Lesser Toe Deformities)

Forefoot complications are another problem frequently reported by patients participating in aerobics and fitness sports. Patients report a sharp or dumb pain under the metatarsal heads, mostly under the second and third. Pain under the sesamoids can be due to an inflammation or even fracture of the bones. In jumping sports such as aerobics deformities of the lesser toes can develop, with pain due to the development of horns dorsally on the proximal interphalangeal joints or on the fingertips.

## Etiology and Pathomechanism

### Achilles Tendinitis

Achilles tendinitis is often caused by chronic overload of the tendon or typically by technique errors during exercise. One common technique error in step aerobics is poor foot placement on the bench. When the foot is not fully positioned on the bench, the heel can drop below the top of the step and the tendon gets repeatedly stretched causing an Achilles tendinitis [2]. Further causes of Achilles tendinitis are the shortening of the calf muscles and tightness of the large muscles in the back of the leg as well as incorrect footwear.

In fitness sports the use of elliptical trainers – often suggested for general foot and ankle rehabilitation training – can lead to overload problems of the Achilles tendon. Elliptical trainers simulate a movement similar to Nordic skiing. This allows a whole body, low impact workout with controlled movement. Nevertheless in our experience, due to the repeatedly forced dorsal extension of the ankle and push off, this can lead to irritation of the tendons. As the machines are of a standardized size this tends to affect smaller exercisers as they are forced into greater dorsal extension at the end of the movement (Fig. 44.1).

### Stress Fractures and Shin Splints

Stress fractures and shin splints are caused by repeated loads on the foot or ankle that exceed the strength and repair potential of the bones. They occur mostly when the progression of training activity is too rapid, the training plan is changed or with inadequate equipment. Despite conflicting results poor



**Fig. 44.1** On elliptical trainers the feet are forced in repeated loaded dorsi-flexion of the ankle, which can lead to irritations of the Achilles tendon insertion

shoe selection and non-resilient surfaces are generally thought to contribute to the development of these overuse injuries. One technique error that can increase the risk of stress fractures during step aerobics is bouncing or hopping up and down off the bench instead of stepping. Another issue is the step height; an increase in bench height will increase the load on the leg and foot and thus the risk of the development of stress fractures [2].

### Ankle Sprain

Ankle sprains in aerobics and fitness are often a result of fatigue or insufficient technique. Towards the end of aerobic classes athletes can tire, consequently hitting the step awkwardly and slipping causing a distortion. In fitness sports the increasing use of destabilizing equipment such as a BOSU® leads to increasing numbers of sprains. The addition of weights can further increase the distorting force making the injury more severe (Fig. 44.2).

## Plantar Fasciitis and Heel Spurs

Plantar fasciitis and heel spurs can be caused by frequent stress on the plantar aspect. Forefoot and rearfoot instability, with excessive pronation, or tightness of the calf muscles may result in plantar fasciitis.

## Forefoot Injuries (Metatarsalgia, Sesamoiditis and Lesser Toe Deformities)

Often the repetitive high loads on the foot, especially the forefoot, can cause metatarsalgia or sesamoiditis. In aerobics



**Fig. 44.2** Ankle sprains can occur during aerobic and fitness sports. Athletes are at a higher risk when for example hitting the step during aerobics wrong (a) when getting exhausted or using unstable surfaces, like a BOSU® (b)

the bench height and the position where the foot lands influence the impact on the foot during training. Using a higher step increases the forces due to higher impact and larger steps. Furthermore, when the foot lands further away from the step, it requires a lot of flexibility in the toe joints, which can overload the plantar structures. If the participant's foot has structural deformities of the metatarsals or lesser toes the likelihood of developing metatarsalgia or sesamoiditis is increased. Inadequate shoe wear increases the risk of developing symptoms.

## Epidemiology

The number of members of health clubs is increasing worldwide, for example in the United States health club memberships have increased from 41.3 million in 2005 to 58.5 million in 2013 [3]. With 61 % participation rate, training at health clubs is rated as the most performed sports activity [4]. The average usage is approx. 90 visits/year [3, 5]. Twenty-four million people participate in aerobic dancing in the United States.

The mean rate of injuries is 7.8/1000 training hours in recreational fitness activities, leading to a physician visit rate of 9.5 % of all injured patients [6]. Specifically, injury rate is 3.1/1000 h for CrossFit® [7] and 1.8 to 2.7/1000 h for aerobics [8]. Despite the general low incidence the high number of participants makes these injuries relevant. Up to a third of the injuries concern the ankle or the foot, respectively [9, 10]. The ankle region suffers from acute injury in approx. 60 % of injuries [6].

In aerobic instructors 73 % of injuries of the lower extremity are chronic and mostly tendinopathies (up to 21 % of all injuries) [1], in which Achilles tendinopathy makes up to 18.3 % [11]. Shin splints are the most often occurring injury in aerobics, causing approx. 40 % of injuries [12].

Fatigue is attributed as the primary cause of injury, with secondary factors being the type of floor, footwear or step used [9, 11].

In recent years new training techniques such as instability resistance training and CrossFit® have gained increasing popularity and may change injury patterns and rates [7].

Patients with a history of injuries were at least twice more likely to sustain similar injuries, compared to uninjured participants [9].

---

## Therapy (On the Field, Conservative and Surgical)

### Achilles Tendinitis

The primary therapy of Achilles tendinitis is conservative. Initial therapy consists of the classical RICE concept of Rest, Cooling, Compression and Elevation. In addition to RICE, local application of anti-inflammatory medication as well as drugs such as NSAIDs and arnica can be applied. After the acute phase physiotherapy should be considered. In most patients cooling, friction therapy, ultrasound and eccentric training are the most important techniques, but therapy has to be individualized, which includes all forms of osteopathy, e.g. including the Fascial Distortion Model. The training technique should be evaluated with the trainer and physiotherapist and modification of external factors (shoe wear, training floor) should be considered. In chronic cases, additional shock wave therapy, oral low-dose aspirin or sclerotherapy may be applied. Surgery is reserved for severe and therapy-resistant patients.

### Stress Fractures and Shin Splints

Treatment for a stress fracture of the foot includes activity modification and use of a stiff-soled shoe for approximately 2–4 weeks after injury. Depending on the location of the fracture, immobilization in a cast or use of crutches may be needed. Vitamin D and calcium supplementation should be considered. Fractures in the fifth metatarsal, talus and navicular bone heal very slowly or may not heal at all due to poor blood supply. Longer periods of treatment or surgical intervention may be necessary in order to heal these types of fractures.

Shin splint pain is treated with cooling immediately after the workout to reduce inflammation. Anti-inflammatory drugs accelerate the healing process. Proper stretching before the workout, together with a strong set of foot muscles, prevent the onset of shin splints (see chapter 17 and 19).

### Ankle Sprain

Depending on the clinical finding diagnostic radiology or even MRI should be considered to exclude accompanying

lesions. Although initial treatment is conservative in general, surgery may be appropriate for high-level athletes.

After a period of rest, mobilizing and stabilizing therapies should be performed by a physiotherapist. The Fascial Distortion Model may be used. The application of local NSAIDs or other anti-inflammatory medications promote recovery. In patients with suspected ligamentous lesions an orthosis is recommended for 6 weeks. Surgery is indicated when concomitant lesions are found, in high-performance athletes or with persistent instability or pain after sufficient conservative treatment.

### Plantar Fasciitis and Heel Spurs

Initial therapy with the physiotherapist should include stretching of the flexing structures, from the gastrocnemius complex to the toes, and friction therapy. Ultrasound and electrophoresis can be added. Shock wave therapy shows good results in otherwise therapy-refractory patients. Shoe wear modifications including individual-adapted soft-bedding in the shoes should be used (see Chap. 21).

### Forefoot Injuries (Metatarsalgia, Sesamoiditis and Lesser Toe Deformities)

Potential risk factors during training should be identified. Recognizing actions that place high stress on the forefoot and applying training modifications to avoid these are most efficient. Furthermore, as shortening of the calf muscles are a risk factor for these injuries, adequate stretching should always be emphasized for these patients. The use of anti-inflammatory drugs should be considered. Shoe wear modifications with individualized soft-beddings for the forefoot and retrocapital support are most important.

---

## Rehabilitation and Back-to-Sports

### Achilles Tendinitis

After the acute phase of the injury has passed a controlled rehabilitation and back-to-sports schedule should be used. High impact exercises and those with dorsal extension of the ankle should be avoided initially.

Proper footwear for the specific type of exercise should be worn. Resumption of an exercise program should be slow and gradual, with an emphasis on proper and sufficient stretching and limbering warm-ups.



## Stress Fractures and Shin Splints

Monitoring of the healing progress of stress fractures is best done clinically. Radiologic and MRI findings often do not correlate well with clinical symptoms. It is most important to select the appropriate footwear and start out slowly when returning to an exercise program. Increments of no greater than 10% of the training intensity and volume per week are recommended when restarting training.

## Ankle Sprain

An early return to fitness sports on resilient floor surfaces is often possible using stabilizing orthoses, as most exercises are performed under controlled conditions. Elliptical trainers and bikes without cleats can be used. Training on unstable surfaces and aerobics can be started after achieving sufficient proprioception and should be done in close cooperation with the treating physiotherapist. Initially an ankle orthosis should be worn.

## Plantar Fasciitis and Heel Spurs

When returning to sports the athlete should start with low impact exercise, for example biking or elliptical training. Weight lifting sports, especially using the upper extremities, can usually be continued during treatment for a plantar fasciitis.

## Forefoot Injuries (Metatarsalgia, Sesamoiditis and Lesser Toe Injuries)

The primary focus of the rehabilitation protocol should be stretching exercises for the calf muscles. On returning to sport adequate shoe beddings should be utilized to allow pain-free mobility. Exercises to strengthen foot muscles should be done regularly.

---

## Prevention

### Achilles Tendinitis

In sports with frequent side-to-side movements, such as aerobics, participants who do not wear special stabilizing shoes have been shown to be at increased risk of injury [8]. Newer studies do not support this, but possibly because athletic shoes have been designed for some time to reduce excessive movements of the rear foot [11].

Correct technique during exercise should be checked. In step aerobics the height of the step is especially important; lower steps decrease the load on the joints without significantly reducing the efficiency of the exercise.

Lifting of the heel during push off on elliptical trainers might help to reduce the risk of injury recurrence. Warm up and down, including stretching exercises, should be performed conscientiously.

## Stress Fractures and Shin Splints

The best way to avoid a stress fracture of the foot when starting an exercise program is to start slowly and follow a sensible high quality program. Increasing the intensity by 10% per week has been shown to be an appropriate goal.

## Ankle Sprain

Correct stabilizing shoe wear, especially in sports with many sideways movements like aerobics, are essential to lower the rate of sprains. In fitness sports unstable resistance training uses lighter weights than conventional training, therefore the athletes are tempted to increase the weights more quickly. Exercises to enhance the ability of proprioception should be done on a regular basis.

## Plantar Fasciitis and Heel Spurs

Shoes with proper support in the arch often prevent plantar fasciitis.

Both plantar fasciitis and heel spurs can be avoided by a proper warm-up that includes stretching the band of tissue on the underside of the foot. In patients with even mild hindfoot deformities custom made insoles may help to decrease shear stresses on the Achilles tendon.

## Forefoot Injuries (Metatarsalgia, Sesamoiditis and Lesser Toe Injuries)

Proper shoe wear and sufficient warm-up can reduce the risk of forefoot injuries. In aerobics bouncing instead of stepping on the bench should be avoided and the bench should be positioned at the right height. Excessive dorsiflexion of the toes can occur during elliptical training or weight-bearing lunging but should be avoided.

## Evidence

All cited publications are level three to four studies.

---

## Summary

1. Proper shoe wear is important to minimize the risk of acute and chronic injuries during aerobics and fitness sports.
2. Small foot deformities can lead to ongoing foot problems in sporting activities and insoles may be necessary.
3. Correct technique helps to reduce overload injuries. Factors to be considered include appropriate bench height in step aerobics, gradual progression of weights in functional training (e.g. Crossfit) and lifting of heels on elliptical trainers.

---

## References

1. Rothenberger LA, Chang JI, Cable TA. Prevalence and types of injuries in aerobic dancers. *Am J Sports Med.* 1988;16(4):403–7.
2. Furman A. American Academy of Podiatric Sports Medicine [Internet]. [www.aapsm.org](http://www.aapsm.org). 2014 [cited 2014 Sep 13]. pp. 1–2. Available from: <http://www.aapsm.org/ct1198.html>.
3. U.S. health club membership exceeds 50 million, up 10.8%; industry revenue up 4% as new members fuel growth [Internet]. [www.physicalactivitycouncil.com](http://www.physicalactivitycouncil.com). 2014 [cited 2014 Sep 10]. pp. 1–3. Available from: [http://www.physicalactivitycouncil.com/PDFs/2013\\_PAC\\_Overview\\_Report\\_Final.pdf](http://www.physicalactivitycouncil.com/PDFs/2013_PAC_Overview_Report_Final.pdf).
4. Schiller D. 2013 participation report [Internet]. [www.physicalactivitycouncil.com](http://www.physicalactivitycouncil.com). 2014 [cited 2014 Sep 10]. pp. 1–15. Available from: [http://www.physicalactivitycouncil.com/PDFs/2013\\_PAC\\_Overview\\_Report\\_Final.pdf](http://www.physicalactivitycouncil.com/PDFs/2013_PAC_Overview_Report_Final.pdf).
5. Gym membership statistics [Internet]. [www.physicalactivitycouncil.com](http://www.physicalactivitycouncil.com). 2014 [cited 2014 Sep 10]. pp. 1–4. Available from: [http://www.physicalactivitycouncil.com/PDFs/2013\\_PAC\\_Overview\\_Report\\_Final.pdf](http://www.physicalactivitycouncil.com/PDFs/2013_PAC_Overview_Report_Final.pdf).
6. Requa RK, DeAvilla LN, Garrick JG. Injuries in recreational adult fitness activities. *Am J Sports Med.* 1993;21(3):461–7.
7. Hak PT, Hodzovic E, Hickey B. The nature and prevalence of injury during CrossFit training. *J Strength Cond Res.* 2013 Nov 22. [Epub ahead of print].
8. Potter H. Lower limb injuries in aerobics participants in Western Australia: an incidence study. *Aust J Physiother.* 1996;42(2):111–9.
9. Garrick JG, Gillien DM, Whiteside P. The epidemiology of aerobic dance injuries. *Am J Sports Med.* 1986;14(1):67–72.
10. du Toit V, Smith R. Survey of the effects of aerobic dance on the lower extremity in aerobic instructors. *J Am Podiatr Med Assoc.* 2001;91(10):528–32.
11. Malliou P, Rokka S, Beneka A, Gioftsidou A, Mavromoustakos S, Godolias G. Analysis of the chronic lower limb injuries occurrence in step aerobic instructors in relation to their working step class profile: a three year longitudinal prospective study. *J Back Musculoskelet Rehabil.* 2014;27(3):361–70.
12. Hayes GW. Injuries arising from aerobic fitness classes. *Can Fam Physician Coll Fam Physician Can.* 1985;31:1517.

Mark E. Magill and Robert B. Anderson

**Abstract**

It is an understatement to say that American football is a high impact sport. Injuries are common; often the result of high-energy trauma and can be quite significant. Given the cleat-surface interactions, a multitude of foot and ankle injuries can occur and it is not within the scope of this chapter to cover all of these injuries and the intricacies of their management. However, a few common injuries will be discussed with a focus on the subtleties to the treatment of American football players who have sustained these injuries. Epidemiologic studies are available, unfortunately, level 1 and level 2 studies are for the most part not available regarding football related foot and ankle studies. Additionally, significant work has been done by the league in conjunction with the senior author to improve cleat-surface interactions and this has not been published.

**Keywords**

American football • Football • Turf toe • Jones fracture • Lisfranc • Achilles tendon rupture • Syndesmotic ankle sprain • High ankle sprain

No part of this study has been submitted or duplicated elsewhere prior. This study has been read and approved by all authors and each author believes that the manuscript is valid and represents honest work. No research or industry funds were received in relation to the research or publication of this study.

Relevant disclosures outside of the current study: Dr. Magill has no disclosures; Dr. Anderson receives royalties from Arthrex, Inc., DJ Orthopaedics, and Wright Medical Technology, Inc. and is a paid consultant for Amniox, Wright Medical Technology, Inc., and Arthrex, Inc.

M.E. Magill, MD (✉)  
Department of Orthopedic Surgery, Charlotte Medical Center,  
Charlotte, NC, USA  
e-mail: [magillme@gmail.com](mailto:magillme@gmail.com)

R.B. Anderson, MD  
OrthoCarolina Foot & Ankle Institute, 2001 Vail Avenue, Suite  
200B, Charlotte, NC 28207, USA  
e-mail: [Robert.Anderson@orthocarolina.com](mailto:Robert.Anderson@orthocarolina.com)

**Turf Toe****Etiology and Pathomechanism**

Turf toe is defined as a disruption of the plantar capsule – sesamoid complex. In a study of professional football players, 85% of turf toe injuries were secondary to forced hyperextension of the great toe [1]. Additionally, shoe wear is an important determinant of great toe mobility and should be examined during initial evaluation as a possible etiology and ultimately, treatment of turf toe.

**Epidemiology**

Older population studies of individual university football teams and professional athletes showed an incidence of 4–6 cases per year [1–3]. In the study of 80 football players alluded to above, 45% of players surveyed reported a turf toe at some point in their career. Players with a history of turf toe had an increased age and an increased duration of professional football playing

years. In a study of collegiate athletes, players were 14 times more likely to be injured during games as opposed to practice [4]. In this study less than 2% required surgery.

### Therapy (On the Field, Conservative, Surgical)

Initial management begins with maintaining the great toe in plantar flexion. A plantar flexion great toe brace or plantar flexion taping of the great toe can be helpful. Weight bearing x-rays of both feet should be obtained for comparison, specifically sesamoid station and hallux malalignment. Fluoroscopy or radiographs with forced dorsiflexion lateral view should be considered and compared to the uninjured side. Attention is focused on whether the sesamoids move distally with dorsiflexion (Fig. 45.1). MRI is useful in the diagnostic process and also assesses for intra-articular injury or bone contusion. Patients with stable injuries can typically be treated non-operatively and are placed in either a short or tall boot or a toe spica cast with the toe in plantar flexion. Surgical intervention is typically reserved for patients who fail conservative management with loss of push off strength, instability, or progressive clawing.

### Rehabilitation and Back-to-Sports

Patients with a “classic” but stable turf toe injury typically lose 9 days while those that involve a sesamoid fracture lose an average 42 days [4].

### Prevention

In the most recent epidemiologic study, football players were 85% more likely to sustain a turf toe on third generation artificial fields compared to grass fields [4]. Shoe wear modifications with stiffer footplates may be able to help prevent the forced dorsiflexion commonly resulting in turf toe injuries, however, there are no studies to substantiate this claim.

---

## Jones Fracture

### Etiology and Pathomechanism

Fractures at the junction of the metaphysis and diaphysis of the fifth metatarsal are designated “Jones” fractures. There are both biologic and biomechanical reasons for the propensity for fracture and nonunion at this location. Players with a cavus foot are at risk for lateral column overload and fracture. The lateral portion of the foot outside the supportive foot bed of the cleat is also a risk factor for fracture. The blood supply in this region is considered a watershed region and thus also has a propensity for delayed union and nonunion [5].

### Epidemiology

The NFL surveillance system from 1996 to 2001 noted a 17.8% incidence of all foot fractures [6]. The incidence of proximal fifth metatarsal fractures at the NFL combines between 2004 and 2009 was 3.42% [7]. Nearly 10% of players with a fifth metatarsal fracture will have bilateral fifth metatarsal fractures.

### Therapy (On the Field, Conservative, Surgical)

Initially, patients can be placed in a tall walking boot and weightbear as symptoms allow. X-rays of the foot should be obtained and are usually sufficient for diagnosis. MRI and CT can assist in diagnosing incomplete or subtle varieties. Seventy-six percent of Jones fractures at the NFL combines between 2004 and 2009 were treated operatively [7] (Fig. 45.2). Conservative management is an option, however, it typically requires 6–8 weeks of non-weightbearing in a cast or boot and the average time to union is still 21 weeks [8], with an inherent risk of refracture.

### Rehabilitation and Back-to-Sports

In a study of collegiate athletes, operative management was shown to be a reliable way to return athletes to play in an expeditious manner, on average 8.5 weeks from surgery [9] Axial screw fixation, typically performed percutaneously, assists with rehabilitation and minimizes refracture risk.

### Prevention

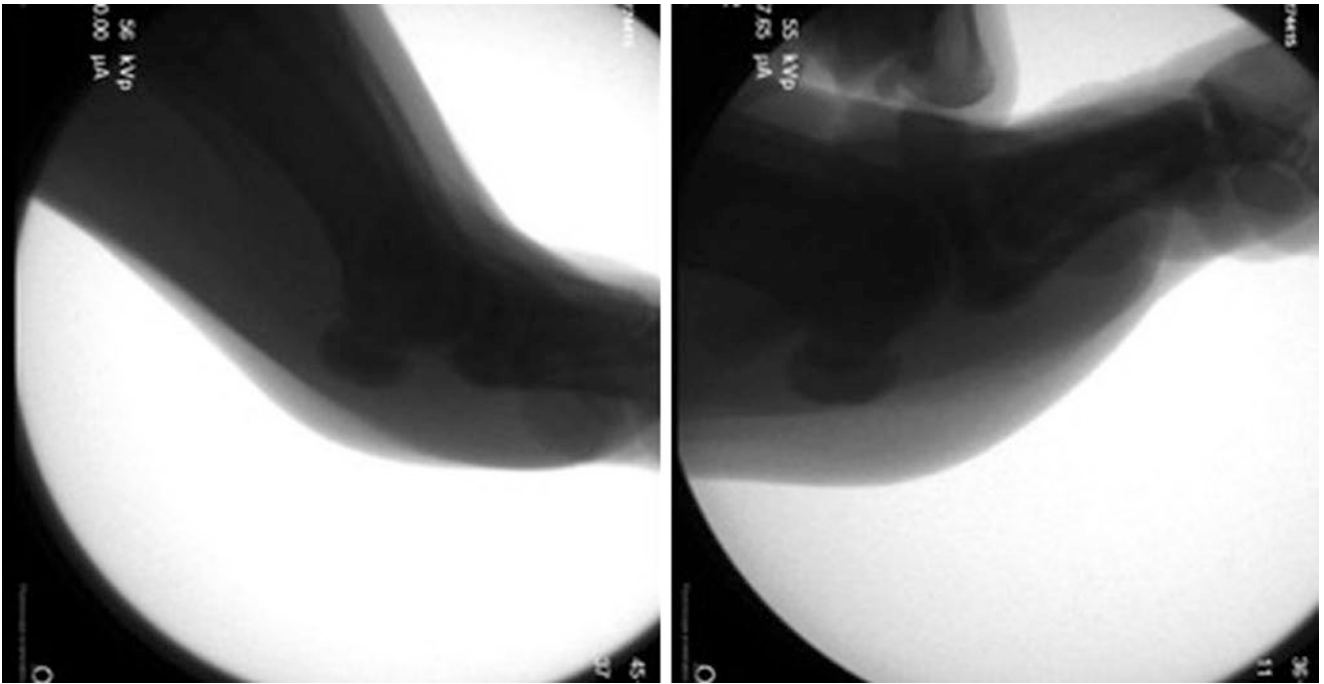
There are no studies to date to look at prevention of Jones fractures. However, an orthotic device with a lateral heel wedge and lateral forefoot post may help patients with a flexible cavus foot to lessen lateral column overload. Additionally, players with a wide foot should be educated on appropriate shoe wear so the foot is not outside the foot bed of the shoe during weightbearing.

---

## Lisfranc

### Etiology and Pathomechanism

Lisfranc injuries and midfoot sprains are typically the result of high-energy mechanisms. Historically, these were seen when the forefoot was fixed to the stirrups of a saddle as a rider fell from a horse. In the general population, these are most commonly the result of motor vehicle collisions. In



**Fig. 45.1** (a) Lateral and (b) forced dorsiflexion lateral fluoroscopic images of the hallux shows that the sesamoids do not move with great toe dorsiflexion in an NFL player



**Fig. 45.2** (a) Lateral and (b) AP imaging of the foot after Jones screw fixation in a collegiate football player

American football players, these injuries are typically purely ligamentous and result from direct compression of the mid-foot (37%), twisting injury with the forefoot cleats fixed (50%), or direct axial impact on the heel by an opposing player on a fixed foot [10]. There are many variations in injury patterns because of these varied mechanisms. Of interest, it is the non-contact twisting mechanism that appears to be increasing in the sport. This mechanism often results in a proximal extension of the ligamentous injury to involve the intercuneiform and naviculo-cunieform joints.

## Epidemiology

In a study of a single collegiate football team, an average of 5 midfoot injuries occurred per year over a 5-year period [10]. In this study, 30% occurred in offensive linemen. NFL surveillance data from 2004 to 2012 showed an average 11 injuries per year with 86% occurring during games. In a study of NFL combine athletes found that 3.4% of the 320 participants reported a Lisfranc sprain [11].

## Therapy (On the Field, Conservative, Surgical)

Appropriate treatment depends on early accurate diagnosis. A high index of suspicion should be maintained for these injuries. Variations in injury patterns should be expected. It is important to look for associated fractures of the cuboid as well as MP head fractures and MP dislocations. Appropriate diagnosis and management often relies on fluoroscopic stress examination with pronation and supination views as well as three views of the foot with the patient standing (Fig. 45.3). Stable injuries, those without significant displacement or diastasis on stress examination, can typically be treated non-operatively. Unstable Lisfranc injuries in athletes are typically managed with open reduction and internal fixation to stabilize all injured joints (Fig. 45.4). Fusion should be avoided if at all possible. In a review of a consecutive series of 25 NFL athletes, 21 were treated operatively at our institution [12]. Three of the 4 treated non-operatively had stable second TMT – medial cuneiform diastasis. Twelve of the 21 treated operatively were proximal variants with navicular – medial cuneiform instability. Thirteen of 21 (62%) underwent removal of hardware on average 5 months from their index surgery.

## Rehabilitation and Back-to-Sports

In the series above of 25 NFL Lisfranc injuries, 84% were able to return to play [12]. Both column injuries are associated with worse outcomes and lower return to play. Non-



**Fig. 45.3** An NFL athlete getting tackled resulting in a Lisfranc injury and MCL knee injury

operatively treated Lisfranc injuries returned to play from 12 to 30 weeks post injury while operatively treated Lisfranc injuries returned 28–48 weeks post-operatively.

## Prevention

There are no studies to date showing methods of prevention for midfoot and Lisfranc injuries in football players. Cleat configuration may alter the ability of the cleat to disengage the turf or grass during forefoot rotation, however this will not alter direct impact or axial load type injuries.

## Achilles Tendon Rupture

### Etiology and Pathomechanism

Achilles tendon ruptures can occur through multiple mechanisms including sudden plantar flexion of the ankle with an extended knee, sudden dorsiflexion of a plantar flexed foot, which typically occurs with landing after jumping, or direct blow to a contracted tendon. Ruptures typically occur at the hypovascular region approximately 2–6 cm proximal to the insertion resulting in an average gap of 2.7 cm [13].

### Epidemiology

A study of NFL players between 1997 and 2002 showed 31 Achilles tendon ruptures during this period amounting to approximately 5 Achilles tendon ruptures per year with an overall incidence of .93% [14]. The average age of the



**Fig. 45.4** (a) AP and (b) AP stress views showing diastasis and instability of the Lisfranc joint in an NFL player

players was 29, and the average time in the league was 6 years. The injuries were more likely to occur during a game (65%) as opposed to preseason (35.4%).

### Therapy (On the Field, Conservative, Surgical)

Initial management hinges on making the correct diagnosis early. Patients should be immobilized in a splint (or boot with multiple heel wedges) and made non-weightbearing. Athletes are managed operatively to assist with rehabilitation, accelerate recovery, improve plantar flexion strength and resting tension of the tendon, and to decrease the incidence of re-rupture (Fig. 45.5). However, operative management has risks including wound complications, infection and possible nerve injury. These risks can be decreased with newer minimally invasive techniques (Fig. 45.6). In a retrospective study of nine NFL athletes who underwent minimally open Achilles repair, there were no postoperative wound complications, nerve injuries or tendon re-ruptures [15].

### Rehabilitation and Back-to-Sports

In the study of NFL athletes with 31 Achilles ruptures, players took on average between 9 and 12 months to return to

play and 32.3% of players never returned to play [14]. The more recent study of nine NFL players managed with a minimally invasive technique found a quicker return to play with an average return to play of 8.9 months and one athlete returning in 5.4 months [15].

### Prevention

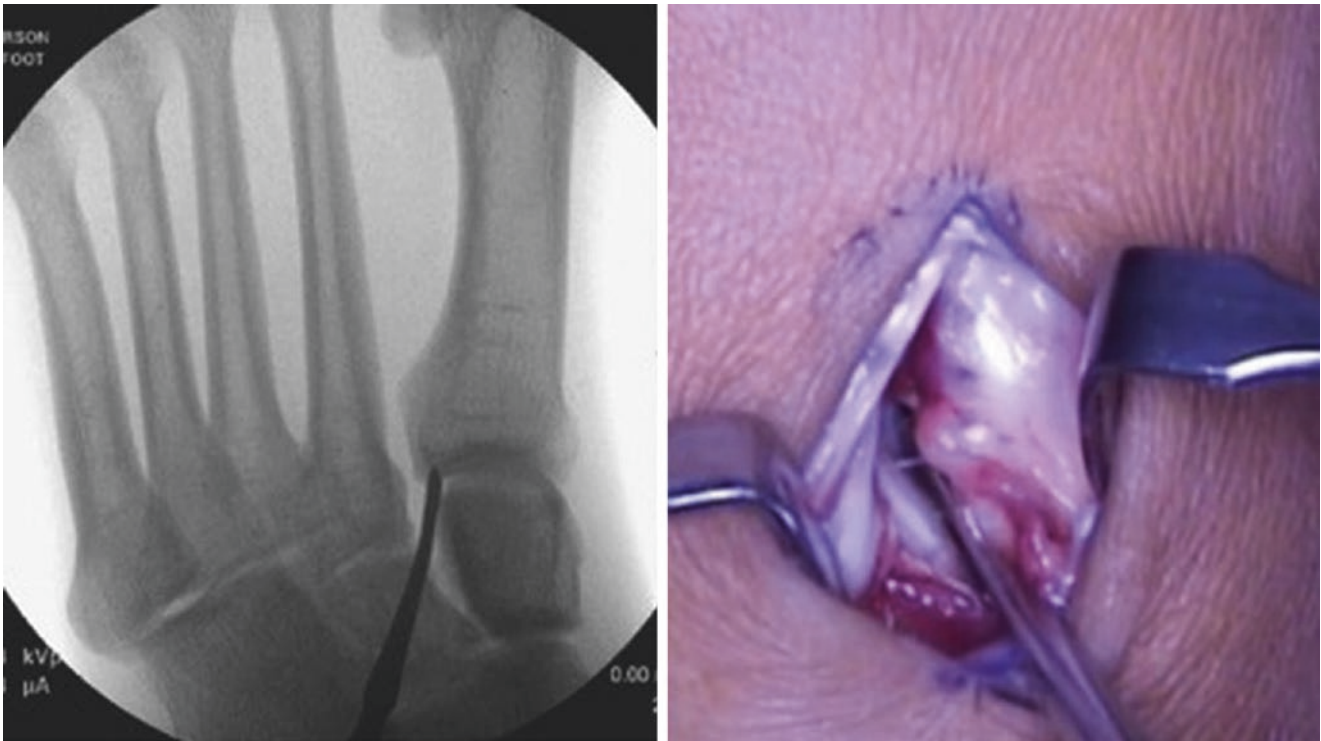
There are currently no high quality studies to date looking at prevention of Achilles tendon ruptures in athletes.

---

## Syndesmotic Ankle Sprains

### Etiology and Pathomechanism

Syndesmotic ankle injuries are defined by injuries to any component of the syndesmosis including anterior inferior tibiofibular ligament, interosseous ligament, inferior transverse ligament, posterior inferior tibiofibular ligament or interosseous membrane. Injury mechanism includes forced external rotation, and often dorsiflexion. 75.2% occur with contact with another player, usually a direct force to the lateral leg that can result in an associated medial collateral ligament strain of the knee [16].



**Fig. 45.5** (a) Fluoroscopic and (b) operative pictures showing the corresponding intraoperative findings from the same NFL football player in Fig. 45.3



**Fig. 45.6** Operative incision after minimally invasive percutaneous Achilles tendon repair in an NFL athlete

Standard treatment includes a thorough physical exam, radiographs and an MRI (Fig. 45.8). Patients treated non-operatively are placed in a cast with weightbearing as symptoms allow. They transition to a tall walking boot for 2–4 weeks based on the severity of injury. Patients can then progress utilizing an ankle strengthening and proprioceptive rehabilitation program. Operative fixation should be considered for any diastasis on ankle x-rays or documented instability based on weightbearing ankle x-rays, fluoroscopic stress exams or arthroscopic exam. Less than 1% of incomplete syndesmotomies require surgery [16]. However, if the injury is complete, 30% require surgery.

## Epidemiology

Ankle sprains are one of the most common football related injuries with syndesmotomies accounting for approximately 10–24.6% [16, 17] (Fig. 45.7). The incidence of ankle sprains is 22% higher on Field Turf than on grass [18]. In a study of the Vikings football team over a 6 year period, they averaged 2.5 high ankle sprains per year [19].

## Therapy (On the Field, Conservative, Surgical)

Initial management of syndesmotomies is typically more aggressive than for standard ankle sprains.

## Rehabilitation and Back-to-Sports

Syndesmotomies are typically predictive of long-term ankle dysfunction and are associated with a more prolonged recovery compared to lateral ligamentous ankle sprains [17, 20]. Typically players lose an average 16 days for partial syndesmotomies and a significantly greater 31 days for complete injuries [16]. In our experience, both non-operatively and operatively high ankle sprains have a longer post op course. Non-operatively treated sprains typically return by 8 weeks while operatively treated injuries returned an average of 4 months post-op.





**Fig. 45.7** A professional football player being tackled resulting in a complex high ankle sprain



**Fig. 45.8** Non-weightbearing mortise ankle x-rays of a patient with a syndesmotic ankle sprain showing medial clear space widening and loss of tib-fib overlap

## Prevention

In one study of ankle sprains in high school football players, overweight players were 19 times more likely to sustain a noncontact ankle sprain [21]. A frequently cited study of collegiate volleyball players, showed that a proprioceptive based ankle training program resulted in significantly fewer ankle sprains compared to a normal training program [22]. In a level 1 study, the prophylactic use of a lace up ankle brace by high school football players reduced the incidence of all ankle injuries (fractures, medial, lateral and syndesmotic sprains) in players with a prior ankle injury by 70% and in players with no history of injury by 57% [23]. The incidence of high ankle sprains is significantly higher on third generation artificial surfaces compared to grass [16].

## References

1. Rodeo SA, et al. Turf-toe: an analysis of metatarsophalangeal joint sprains in professional football players. *Am J Sports Med.* 1990;18(3):280–5.
2. Clanton TO, Butler JE, Eggert A. Injuries to the metatarsophalangeal joints in athletes. *Foot Ankle.* 1986;7(3):162–76.
3. Coker TP, Arnold JA, Weber DL. Traumatic lesions of the metatarsophalangeal joint of the great toe in athletes. *Am J Sports Med.* 1978;6(6):326–34.
4. George E, et al. Incidence and risk factors for turf toe injuries in intercollegiate football: data from the national collegiate athletic association injury surveillance system. *Foot Ankle Int.* 2014;35(2):108–15.
5. Shereff MJ, et al. Vascular anatomy of the fifth metatarsal. *Foot Ankle.* 1991;11(6):350–3.
6. Low K, et al. Jones fractures in the elite football player. *J Surg Orthop Adv.* 2004;13(3):156–60.

7. Carreira DS, Sandilands SM. Radiographic factors and effect of fifth metatarsal Jones and diaphyseal stress fractures on participation in the NFL. *Foot Ankle Int.* 2013;34(4):518–22.
8. Clapper MF, O'Brien TJ, Lyons PM. Fractures of the fifth metatarsal. Analysis of a fracture registry. *Clin Orthop Relat Res.* 1995;315:238–41.
9. Mindrebo N, et al. Outpatient percutaneous screw fixation of the acute Jones fracture. *Am J Sports Med.* 1993;21(5):720–3.
10. Meyer SA, et al. Midfoot sprains in collegiate football players. *Am J Sports Med.* 1994;22(3):392–401.
11. Kaplan LD, et al. Incidence and variance of foot and ankle injuries in elite college football players. *Am J Orthop (Belle Mead NJ).* 2011;40(1):40–4.
12. McCullough K, Corn C, Anderson R. Lisfranc injury in the elite football athlete. 2014.
13. Cetti R, Henriksen LO, Jacobsen KS. A new treatment of ruptured Achilles tendons. A prospective randomized study. *Clin Orthop Relat Res.* 1994;308:155–65.
14. Parekh SG, Brimmo O, Drager RW, Behbahani A, Pedowitz DI, Reddy S, Sennett BJ, Wapner KL. Epidemiology and outcomes of Achilles tendon ruptures in the National Football League. *Univ Pa Orthop J.* 2007;19.
15. McCullough KA, Anderson RB. Mini-open repair of Achilles rupture in the National Football League. *J Surg Orthop Adv.* 2014;23(4):179–83.
16. Hunt KJ, et al. Epidemiology of syndesmosis injuries in intercollegiate football: incidence and risk factors from National Collegiate Athletic Association injury surveillance system data from 2004–2005 to 2008–2009. *Clin J Sport Med.* 2013;23(4):278–82.
17. Gerber JP, et al. Persistent disability associated with ankle sprains: a prospective examination of an athletic population. *Foot Ankle Int.* 1998;19(10):653–60.
18. Hershman EB, et al. An analysis of specific lower extremity injury rates on grass and FieldTurf playing surfaces in National Football League Games: 2000–2009 seasons. *Am J Sports Med.* 2012;40(10):2200–5.
19. Boytim MJ, Fischer DA, Neumann L. Syndesmotic ankle sprains. *Am J Sports Med.* 1991;19(3):294–8.
20. Hopkinson WJ, et al. Syndesmosis sprains of the ankle. *Foot Ankle.* 1990;10(6):325–30.
21. Tyler TF, et al. Risk factors for noncontact ankle sprains in high school football players: the role of previous ankle sprains and body mass index. *Am J Sports Med.* 2006;34(3):471–5.
22. Verhagen E, et al. The effect of a proprioceptive balance board training program for the prevention of ankle sprains: a prospective controlled trial. *Am J Sports Med.* 2004;32(6):1385–93.
23. McGuine TA, et al. The effect of lace-up ankle braces on injury rates in high school football players. *Am J Sports Med.* 2012;40(1):49–57.

Cristian Ortiz, Emilio Wagner, and Gonzalo Fernandez

**Abstract**

Track and field sports involve mainly running, throwing and jumping activities. Injuries will occur mainly due to traumatic or overuse cause. The greater proportion of injuries occurs during training (more than 60%), 20% occur during competition. Inadequate remodelling of bone structures and a failed healing response in tendons after repetitive load, may lead to injury. The ankle region is most frequently injured in jumps. The foot is damaged most frequently in sprints. Achilles tendon ruptures, Achilles tendinopathy and navicular stress fracture are discussed in detail, relative to their diagnosis and treatment. Briefly stated, Achilles tendinopathy should be first treated conservatively, as initial navicular stress fractures. Our treatment recommendation for achilles rupture is surgical using a minimally invasive approach. In order to obtain a safe return to sports, different tests have to be performed including range of motion test, balance and proprioception tests such as the star excursion balance test, agility tests and strength tests such as the vertical jump test. In general since track and field does not include contact a more predictable return to sports is generally expected.

**Keywords**

Track • Field • Foot and ankle • Overuse • Tendinopathy • Stress fracture • Injury prevention

**Athletic Injuries**

Athletics generally refers to track and field activities. It is a sport which combines various athletic activities based on the skills of running, jumping, and throwing. The running events include sprints, middle and long distance events. Jumping includes hurdling (running and jumping over an obstacle at speed), steeplechase, long jump, triple jump, high jump and pole vault. Throwing can consider javelin throw, hammer throw and discus throw. There are also “combined events”, such as heptathlon and decathlon, in which athletes compete in a number of the above events. Most track and field events

are individual sports, although some are relay races. In this chapter we will discuss mainly injuries related to sprint activities and jumping activities, as long distance running will be covered in a different chapter, and throwing mainly affects the upper extremity.

**Etiology and Pathomechanism**

Most of the injuries observed in athletics are related to overuse. In running, the overuse of musculoskeletal structures and the excessive traction of the involved muscles leads to inflammation. These combined with inadequate remodelling of bone structures from repetitive load and impact may lead to injury [1]. In tendons the concept of failed healing response is used to explain where they start to accumulate damaged areas which do not possess the capacity to heal spontaneously

C. Ortiz, MD • E. Wagner, MD (✉) • G. Fernandez, MD  
Department of Orthopedics and Traumatology, Clinica Alemana,  
Av. Vitacura 5951, Universidad del Desarrollo, Santiago, Chile  
e-mail: [caortizm@gmail.com](mailto:caortizm@gmail.com); [emiliowagner@gmail.com](mailto:emiliowagner@gmail.com);  
[gfernandez@alemana.cl](mailto:gfernandez@alemana.cl)

[2]. Jumping disciplines comprise four phases, run-up, take-off, flight and landing phases. Injuries in these activities tend to be associated with jump-landing movements. A stiffer jump-landing technique is a risk factor in the development of overuse injuries and acute injuries, as it will have a lesser shock absorbing component. This may be caused by less active motion in the lower extremity joints and by an increased valgus position of the knee during the jump-landing maneuver [3]. Besides overuse pathology, traumatic injuries can also be found, where the most common include acute achilles tendon ruptures, ankle sprains, syndesmotic ankle sprains, metatarsal fractures and midfoot sprains. Most of the tendon ruptures occur from a rapid eccentric load applied to a stiff foot due to muscle contraction during a fast pushoff or a sudden jump [4]. Ankle sprains will occur with inversion of a plantar flexed ankle as when landing on irregular surfaces. This same mechanism may be involved in the production of fifth metatarsal base fractures. Syndesmotic ankle sprains may happen when an external rotation torque is applied to the foot. Relative to pole vaulting (Fig. 46.1), some specific biomechanical studies have been focused on improving performance and decreasing the risk of injuries like the one by Schade, who concluded that although the timing of the pole plant influences the interactions between the vaulter, the pole, and the ground, it does not affect the athlete's performance. Although a late pole plant decreases the loss of energy by the vaulter during the take-off, this is counterbalanced by a decrease in the energy stored in the pole at take-off [5].

## Epidemiology

In a systematic review of overuse injuries in foot and ankle Sobahni analyzed 84 studies on 23 sports and concluded that study design and reporting methods were heterogeneous.



**Fig. 46.1** Picture example of pole vault. Timing is important to achieve good performance in this discipline

Most studies suffered from a weak methodology and poor reporting. The most common weaknesses were lack of a clear case definition, describing assessment procedures and reporting sample characteristics. Due to methodological heterogeneity of studies, inter-sports and intra-sports comparisons and meta-analysis were not possible. Methodology of most studies on incidence and prevalence of ankle and foot overuse injuries is insufficient [6].

In 2012 Jacobsson reported a retrospective review of 321 Swedish track and field athletes. The body region showing the highest injury prevalence was the knee and lower leg with 15.0% prevalence in 1 year, followed by the Achilles tendon, ankle, and foot and toe with 11.7% prevalence [7]. In a recent report by Feddermann a total of 1470 injuries were reported, equivalent to 81% injuries per 1000 registrations of which 36% were expected to result in absence from sports. The incidence of time-loss injuries was significantly higher in competition (29%) than in training (5.8%), and in outdoor (46%) than in indoor (23%) or youth/junior championships (13%). While most in-competition time-loss injuries were reported during short distance events (32.5%), combined events had the highest incidence of in-competition time-loss injuries (106%). The most frequent diagnosis was thigh strain (28%), followed by lower leg strain and ankle sprain. Injury location varied between different discipline categories: in long distances the lower leg, in marathon the foot and in throws the upper extremity were mainly affected [8].

Older athletes injure more than younger; athletes involved in lower levels of competition injure more than athletes involved in higher levels. Sprints and hurdling injure more frequently than middle distance runners or jumps. The ankle region is most frequently injured in jumps (30% of injuries in jumps happen in the ankle area), followed



**Fig. 46.2** Picture example of triple jump, in final push off. Patellar tendon injuries can be seen due to the high energy release when jumping, as ankle ligament injuries if the foot is too supinated in the push off phase

by middle distance runners. The foot is damaged most frequently in sprints (22%), followed by long distance runners and jumps [9]. In middle and long distance running, the main injuries observed are medial tibial stress syndrome (incidence 13.6–20.0%; prevalence of 9.5%), Achilles tendinopathy (incidence 9.1–10.9%; prevalence 6.2–9.5%) and plantar fasciitis (incidence 4.5–10.0%; prevalence 5.2–17.5%) [1]. In jumping, the most frequent injuries seen are muscle injuries (isquicrural, trunk), tendon injuries where the patellar tendon is more frequently compromised than the achilles tendon, insertional tendinopathies, ligament injuries due to a supinated foot in take off, and stress fractures, mainly anterior tibial edge and navicular bone [10] (Fig. 46.2). Data from the London Summer Olympic Games 2012 showed that only 35% of the injuries were expected to prevent the athlete from participating during competition or training. Relative to athletics, 17.7% of the athletes suffered some sort of injury, of which 39% led to time loss equal or greater than 1 day, and 16% led to time loss greater than 7 days [11]. In this same set of athletes, within athletics, the four most commonly reported injury mechanisms were overuse (34%), non-contact trauma (28%), contact with another athlete and with a stationary object. The distribution of injuries during competition and training was similar.

---

### Therapy (On the Field, Conservative, Surgical)

We will consider the most frequent and time consuming injuries described, i.e. achilles tendon ruptures, Achilles tendinopathy and navicular stress fractures. All the other foot and an ankle injuries that are common to every sports that includes running, have been described in detail in the other chapters of this book. On the field care will always vary depending on the pathology, i.e. if the injury was sustained acutely, and it consists in an acute fracture or tendon rupture, local ice, immobilization, weight bearing avoidance and transfer from the field will be paramount. Stress injuries will probably not need an urgent transfer or care, as it will present with signs and symptoms previously present in the athlete, and therefore, he/she will seek medical attention prior to the competition. Achilles tendinopathy will respond to rest, cryotherapy, and eventually analgesics. It is essential to start when possible, with an eccentric exercise program, which can improve patient's symptoms in up to 60% of the athletes. It has been suggested a synergistic effect between eccentric training and shock wave therapy [12], and this is currently our approach. When dealing with Achilles tendon ruptures, pain and physical impairment are intense, and therefore, urgent care for pain control and immobilization is indicated. We recommend immobilization with a removable

boot trying to keep the leg elevated and holding the ankle in equinus which will decrease pain. Some debate has been present over the last 2 years regarding the ideal treatment for Achilles tendon ruptures, since Soroceanu presented similar results comparing surgical versus non surgical treatment of an acute Achilles tendon rupture [13]. The main debate relates to the return to sports and physical activity probability, and only four articles present in this meta analysis dealt with it, and therefore was not conclusive. Most of the current recommendations support operative intervention due to the improved possibility of returning to the same level of sports, but minimizing complications trying to be minimally invasive [14]. Our treatment recommendation for achilles rupture is surgical in the athletic active population, using a minimally invasive approach, as it has shown excellent results with scarce complications and fast rehabilitation [15]. Relative to navicular stress fractures, sprinters and middle distance runners will present more frequently with these injury. They will present with a vague, not localized foot pain over the dorsum of the foot. Most of the time radiological exams result negative and magnetic resonance imaging has become the exam of choice [16]. Navicular stress fractures are considered high risk fractures due to their natural history, and thus require immediate treatment. Torg [17] published on 2010 his study where conservative treatment was recommended for every navicular stress fracture, as no statistical significant difference was found between treatment, independent on the type of fracture. Saxena published and commented on their results stating that navicular stress fractures with just a dorsal breach of the dorsal cortex can be treated conservatively (Type 1) and that types 2 and 3 (fracture extending into the navicular body or complete fracture) treatment should be surgical. This last approach has been also supported lately [16] and it is our current recommendation.

---

### Rehabilitation and Back-to-Sports

Rehabilitation is paramount for our athletes, and the current approach should coordinate the physician efforts to achieve healing, and the coach effort to coordinate return to sports. For stress fractures, full return to sports should be allowed when bone healing has been certified, which should be done with CT scans. Relative to the Achilles tendon, specific scores can be used to evaluate how recovered a certain athlete is, such as the VISA-A score [18]. After certifying healing, health and disability can be assessed in terms of function. Actually, the World Health Organization recommends using tests related to the particular activities involved in competitive play. These tests should assess pain, instability, kinematics and symmetry. General recommended tests include range of motion test, balance and proprioception tests such as the

star excursion balance test, agility tests and strength tests such as the vertical jump test [19]. In general since track and field does not include contact a more predictable return to sports is generally expected.

## Prevention

A key element in success is to correctly identify the overuse element that generated the injury in a specific athlete. After this is done, and healing is achieved, it should be well noted to the patient, family and coach in order to understand the origin of the problem and avoid in the future. It has been suggested in the literature that the variety in sport and exercise may limit the risk of overuse injuries. In addition, participation in a variety of sports may help prevent the development of neuromuscular deficits which are apparently the primary determinants of both acute and chronic injury risk [20]. In our group, the key element in decreasing injury recurrency has been a team work comprising the orthopedic surgeon subspecialist, the sports physician, physical therapist and team coach.

Although many studies have intended to prevent ankle injuries in athletes using proprioceptive training, there is still no definitive evidence to prove it [21].

Shoes seem to be important to prevent injuries and this has been suggested by several studies like the one by Debisao in which he showed that independently of the type of shoe, men and women loaded the foot differently during a jump landing. The bladed cleat shoe increased forefoot loading, which may increase the risk for forefoot injury. He recommends that the type of shoe should be considered when choosing footwear for athletes returning to activity [22].

The surface where the athlete practices or competes is also important. Taylor suggests that elite athletes may sustain more injuries, even when playing on the newer artificial surfaces [23].

Ankle stabilizers may help to reduce incidence of ankle sprain in athletes but although they do not seem to affect jumping or balance, they may compromise performance where agility is needed [24].

## Evidence

Achilles tendon rupture, Level 1, Level 3 and 4 articles.

Grade of evidence B.

Achilles Tendinopathy, Level 2 and 4 articles. Grade of Evidence B.

Navicular Stress fractures, Level 1 and 4 articles. Grade of Evidence B.

## Summary

Track and field sports involve mainly running, throwing and jumping activities. Injuries will occur mainly due to traumatic or overuse cause. Very few studies are specifically focused in foot and ankle injuries in athletics.

The greater proportion of injuries occurs during training (more than 60%), 20% occur during competition. Older athletes injure more than younger and lower levels of competition injure more than athletes involved in higher levels.

Sprints and hurdling injure more frequently than middle distance runners or jumps. The ankle region is most frequently injured in jumps, the foot is damaged most frequently in sprints.

Achilles tendon ruptures should be treated surgically. Achilles tendinopathy should be first treated conservatively, as initial navicular stress fractures.

The same intrinsic and extrinsic factors that have to be considered for foot and ankle overuse injuries in every sport that include running are also true for track and field athletes.

A complete interrelation between the treating physician, physical therapist, coach, patient and family is needed to achieve a complete rehabilitation and reliable prevention or recurrence.

## References

1. Lopes A, Hespanhol L, Yeung S, Pena L. What are the main running-related musculoskeletal injuries? *Syst Rev Sports Med.* 2012;42(10):891–905.
2. Rees JD, Maffulli N, Cook J. Management of tendinopathy. *Am J Sports Med.* 2009;37(9):1855–67.
3. Aerts I, Cumps E, Verhagen E, Verschuere J, Meeusen R. A systematic review of different jump-landing variables in relation to injuries. *J Sports Med Phys Fitness.* 2013;53(5):509–19.
4. Title C, Katchis S. Traumatic foot and ankle injuries in the athlete. *Orthop Clin North Am.* 2002;33:587–98.
5. Schade F, Arampatzis A. Influence of pole plant time on the performance of a special jump and plant exercise in the pole vault. *J Biomech.* 2012;45(9):1625–31.
6. Sobhani S, Dekker R, Postema K, Dijkstra P. Epidemiology of ankle and foot overuse injuries in sports: a systematic review. *Scand J Med Sci Sports.* 2013;23(6):669–86.
7. Jacobsson J, Timpka T, Kowalski J, Nilsson S, Ekberg J, Renström P. Prevalence of musculoskeletal injuries in Swedish elite track and field athletes. *Am J Sports Med.* 2012;40(1):163–9.
8. Feddermann-Demont N, Junge A, Edouard P, Branco P, Alonso JM. Injuries in 13 international Athletics championships between 2007–2012. *Br J Sports Med.* 2014;48(7):513–22.
9. D'Souza D. Track and field athletics injuries – a one-year survey. *Br J Sports Med.* 1994;28(3):197–202.
10. Engelhardt M, Dorr A. *Sports orthopedics*, Chap.4. Elsevier. Munchen, Germany 2011;p. 425–9.
11. Engebretsen L, et al. Sports injuries and illnesses during the London Summer Olympic Games. *Sports Med.* 2013;47:407–14.

12. Longo U, Ronga M, Maffulli N. Achilles tendinopathy. *Sports Med Arthrosc.* 2009;17:112–26.
13. Soroceanu A, Sidhwa F, Aarabi S, Kaufman A, Glazebrook M. Surgical versus non-surgical treatment of acute Achilles tendon rupture. A meta analysis of randomized trials. *J Bone Joint Surg Am.* 2012;94:2136–43.
14. Longo U, Petrillo S, Maffulli N, Denaro V. Acute Achilles tendon rupture in athletes. *Foot Ankle Clin.* 2013;18:319–38.
15. Keller A, Ortiz C, Wagner E, Wagner P, Mococain P. Mini-open tenorrhaphy of acute Achilles tendon ruptures: medium-term follow-up of 100 cases. *Am J Sports Med.* 2014;42(3):731–6.
16. McCormick F, Nwachukwu B, Provencher M. Stress fractures in runners. *Clin Sports Med.* 2012;31:291–306.
17. Torg J, Moyer J, Gaughan J, Boden B. Management of tarsal navicular stress fractures: conservative versus surgical treatment: a meta-analysis. *Am J Sports Med.* 2010;38:1048–53.
18. Robinson J, Cook J, Purdam C, Visentini P, Ross J, Maffulli N, Taunton J, Khan K. The VISA-A questionnaire: a valid and reliable index of the clinical severity of Achilles tendinopathy. *Br J Sports Med.* 2001;35(5):335–41.
19. Clanton T, Matheny L, Jarvis H, Jeronimus A. Return to play in athletes following ankle injuries. *Sports Health.* 2012;4(6):471–4.
20. Paterno M, Taylor-Haas J, Myer G, Hewett T. Prevention of overuse sports injuries in the young athlete. *Orthop Clin North Am.* 2013;44:553–64.
21. Ben Moussa Zouita A, Majdoub O, Ferchichi H, Grandy K, Dziri C, Ben Salah FZ. The effect of 8-weeks proprioceptive exercise program in postural sway and isokinetic strength of ankle sprains of Tunisian athletes. *Ann Phys Rehabil Med.* 2013;56(9–10):634–43.
22. DeBiasio J, Russell M, Butler R, Nunley J, Queen R. Changes in plantar loading based on shoe type and sex during a jump-landing task. *J Athl Train.* 2013;48(5):601–9.
23. Taylor S, Fabricant P, Khair M, Haleem A, Drakos M. A review of synthetic playing surfaces, the shoe-surface interface, and lower extremity injuries in athletes. *Phys Sportsmed.* 2012;40(4):66–72.
24. Ambegaonkar J, Redmond C, Winter C, Cortes N, Ambegaonkar S, Thompson B, Guyer S. Ankle stabilizers affect agility but not vertical jump or dynamic balance performance. *Foot Ankle Spec.* 2011;4(6):354–60.

Christopher E. Gross, Jeannie Huh, and James A. Nunley II

**Abstract**

Basketball-related injuries are the most common type of sports-related injury in patients under 25-years-old and second in those aged 25–40. According to the National Electronic Injury Surveillance System, basketball accounts for over 28 % of total injuries sport-related injuries that were treated in an emergency room. In a study of all injuries in the National Basketball League, the ankle was by far the most common joint injured. Some of the most common injuries include: ankle sprains and lateral instability, fractures of the base of the fifth metatarsal, Achilles tendon ruptures, osteochondral lesions of the talus, and anterior ankle impingement. Prevention strategies, appropriate operative intervention, and functional rehabilitation are all important in the management of the injured basketball player for safe and timely return to play.

**Keywords**

Basketball • Ankle sprain • Lateral ankle instability • Jones fractures • Achilles tendon ruptures • Osteochondral lesions of talus

**Introduction**

Basketball subjects the foot and ankle to acute and chronic strain. The sport consists of rapid acceleration and deceleration, cutting, jumping, and pivoting. Basketball-related injuries are the most common type of sports-related injury in patients under the age of 25 and second in those aged 25–40 [1]. In 2012, the National Electronic Injury Surveillance System (NEISS) recorded more than 1.9 million individuals who had a sports-related injury treated in an emergency department. Of those, basketball represented over 28 % of total injuries with 570,000 suffering injuries. Of those, the most common injury was related to the foot and ankle. In a study of all injuries in the National Basketball League between 1989 and 2007, the ankle was by far the most common joint

injured with 1850 injuries (14.7 % of all injuries) [2]. There were 1123 game-related ankle injuries (17.9 %) with an incidence of 3.4 injuries per 1000 athlete exposures. The foot contributed to 962 more injuries (7.6 % of all injuries).

**Injuries**

The ankle and foot are injured in many unique manners in basketball. While an ankle sprain is the most common injury in basketball, the following represent the spectrum of injuries:

- Ankle sprain
- Acute and chronic ankle instability
- Achilles tendon ruptures
- Osteochondral lesions of the talus
- Anterior ankle impingement
- Jones fracture
- Over-use injuries
  - Plantar fasciitis

C.E. Gross, MD (✉) • J. Huh, MD • J.A. Nunley II, MD  
Department of Orthopaedic Surgery, Duke University Medical  
Center, 4709 Creekstone Drive, 2nd Floor,  
Durham, NC 27703, USA  
e-mail: [cgross144@gmail.com](mailto:cgross144@gmail.com);  
[handyh@gmail.com](mailto:handyh@gmail.com); [james.nunley@duke.edu](mailto:james.nunley@duke.edu)



- Medial-sided stress fractures (navicular, medial malleolus)
- Calcaneal apophysitis
- Achilles tendinitis
- Intedigital neuromas
- Calcaneal beak fractures
- Navicular avulsions
- Tears of the superior peroneal retinaculum
- Os trigonum fractures

Due to the limited breath and scope of this specific chapter, we are going to concentrate on five of the most common foot and ankle injuries in basketball: ankle sprains and lateral instability, fractures of the base of the fifth metatarsal, Achilles tendon ruptures, osteochondral lesions of the talus, and anterior ankle impingement.

## Lateral Ankle Sprain and Instability

### Etiology and Pathomechanism

Lateral ankle sprains generally result from an inversion force on the plantar-flexed ankle. When the ankle is in plantar-flexion, the osseous stability of the ankle is reduced and the soft tissues are more susceptible to injury. The anterior talofibular ligament (ATFL), which is tight in plantar-flexion, is the most commonly injured portion of the lateral ligament complex [3], followed by the calcaneofibular ligament (CFL), and less frequently, the posterior talofibular ligament (PTFL). In basketball, the classic injury scenario occurs when a player inverts his ankle on another player's foot while landing after a jump [4]. Many ankle sprains are noncontact injuries, and may occur while taking an awkward step during cutting or turning maneuvers. In these scenarios, as the athlete attempts to work around a crowd of players, this focus of attention is shifted away from the playing surface, exposing the lower extremity to injury.

Lateral ankle sprains are frequently classified into three grades based on the severity of injury to the involved ligament. A Grade I sprain indicates a stretched lateral ligament. Clinically, the athlete will have pain and swelling, but will be able to walk without crutches. A Grade II injury indicates a partial tear of the lateral ligament complex and presents with swelling and ecchymosis, with an ability to walk a few steps unassisted. A Grade III injury indicates a complete tear and presents with swelling, ecchymosis, sensation of instability, and inability to walk [5].

Residual disability after ankle sprains is present in 32–76% of patients [6, 7]. After conservative or surgical treatment, patients may continue to have symptoms, including persistent synovitis or tendinitis, ankle stiffness, swelling, muscle weakness, and recurrent instability.

### Epidemiology

Ankle sprains are the most frequent orthopaedic injury sustained in basketball players [8–10]. In a series of professional basketball players reported by Deitch and colleagues [11], lateral ankle sprains were the most common injury diagnosis (13.7% of all injuries) in both the National Basketball Association (NBA) and the Women's National Basketball Association (WNBA). In a 17 year prospective study spanning the NBA seasons from 1988 to 2005, Drakos et al. [2] found that ankle sprains accounted for 8.8% of all games missed, making them the second most common reason for games missed by a player, following patellofemoral syndrome (Level II). The incidence of ankle sprain (3.2 per 1000 athlete exposures) was more than twice as common as any other injury. In a 2-year prospective study of female Greek professional basketball players, most ankle sprains occurred within the 3-point line and occurred during games rather than practice. Centers, followed by guards and forwards, had the highest rate of injury [12].

Though the incidence of ankle sprains is higher in athletes who have had a prior ankle sprain [13–15], there is conflicting evidence in the literature as to other demographic risk factors for ankle sprains. Among collegiate basketball players, Hosea et al. [16] found a higher rate of Grade I ankle sprains in women than in men, but for higher grade sprains the incidence was similar between groups. However, the study on professional basketball players previously mentioned above found no difference in incidence of ankle sprains between genders [11]. A study of high school athletes found that males with a higher body mass index (BMI) had a higher incidence of ankle sprains, however, this was not found to be so in a separate study among professional basketball players [2]. Similarly, in a study of 10,393 recreational basketball players, McKay et al. found that the risk of ankle injury was not related to gender, age, height or weight [15].

### Therapy (On the Field, Conservative, Surgical)

Functional, nonoperative management is the preferred method of treatment for all acute lateral ankle sprains, regardless of grade (Grade B). On the court, an athlete suspected of this injury is initially treated with rest, ice, compression, and elevation. A thorough physical exam should take place, keeping in mind several subtle injuries can mimic an ankle sprain, including: an anterior process of the calcaneus fracture, a lateral talar process fracture, a proximal fifth metatarsal fracture, peroneal tendon disruption, or a subtalar dislocation. If the athlete can bear weight and demonstrate the ability to jump, run, and cut on the injured extremity, they

may return to play. However, if the athlete is unable to do so, he or she should be kept out of the rest of the game.

A brief period of immobilization followed by functional rehabilitation allows the athlete to return to play within a week for Grade I sprains, 4–6 weeks for Grade II sprains, and 6–10 weeks for Grade III sprains, without the need for further treatment. During the recovery period, a combination of elastic wrap with an Air-Stirrup brace (Aircast, Inc, Summit, NJ) has been shown to be significantly faster at returning patients with Grade I or II ankle sprains to pre-injury function compared to either alone or a walking cast for 10 days (Level II, Grade B). Athletes with Grade III ankle sprains showed no difference in their return time to normal function between bracing and casting for 10 days [17] (Level II).

Primary repair of Grade III injuries may not be superior to nonsurgical management [18]. Even distal fibula avulsion fractures treated non-operatively have been shown to heal satisfactorily without late instability, when compared with purely ligamentous injuries [19].

If after the appropriate amount of rest and rehabilitation the athlete has persistent dysfunction, then further evaluation should be performed, which usually includes MRI evaluation. Findings may include a concomitant injury not previously appreciated, such as an osteochondral lesion of the talus or peroneal tendon pathology, which would warrant further management [20].

## Rehabilitation and Back-to-Sports

Upon return to play following an acute ankle sprain, use of an external ankle brace and/or taping provides the athlete with direct mechanical support and enhances proprioception through skin pressure. Combining external support with a dedicated functional rehabilitation program, focused on strengthening of the peroneal muscles and proprioceptive training, results in both successfully preventing and treating instability in more than 90% of cases [21] (Level II, Grade B). Such a program is especially effective for functional instability in the patient with chronic symptoms, where there are no objective measurements of ligament instability, but pain or sensation of instability exists.

Mechanical instability is clinically apparent when an athlete complains of recurrent “giving way” events and has associated measurable laxity on physical exam. Mechanical and functional instability can coexist. If after a dedicated rehabilitation and bracing trial fail and the athlete remains symptomatic, surgical intervention with anatomic lateral ligament repair is indicated. This is achieved by anatomic shortening of the ATFL (and CFL as indicated), reinforced with the inferior extensor retinaculum (Brostrom procedure with Gould modification). This method has shown excellent

results for the treatment of chronic lateral ankle instability with 26 years follow-up [22] (Level IV).

## Prevention

Given the frequency of lateral ankle sprains and the disability that results, including time away from play and increased risk of re-injury, much attention has been placed on the prevention of these injuries. A Cochrane review of 14 randomized trials with 8279 total subjects was performed to determine the effectiveness of various prevention strategies [23] (Level I). The only intervention that showed a significant reduction in ankle sprains in patients with a history of prior ankle sprains was the use of an external ankle support device (ie. semi-rigid orthosis, air-cast brace) [24, 25]. The Cochrane reviewers noted a possible, but unproven benefit using the same devices in those without a history of prior ankle sprain.

Although taping is frequently used among basketball players upon return to play after acute injury, it remains inconclusive as to whether this modality effectively prevents recurrent ankle sprains [23]. Taping has been shown to lose up to 50% of its mechanical effectiveness after approximately 20 min of exercise. It is also more difficult to adjust tape during a game, requires a skilled trainer or therapist to apply, and can be more time-consuming and more expensive than some external ankle support devices [26] (Level V).

Calf stretching programs [27] and the use of cushioned insoles were not found to effectively reduce the risk of ankle sprains in the Cochrane review. A separate study, in fact, found that the presence of air cells in the heel of the shoe was associated with a higher rate of injury [15] (Level IV). In terms of high-top shoes, a prospective study of intramural basketball players, found no difference in the rate of ankle injury [28] (Level I).

As noted previously, the most important risk factor for an ankle sprain is a previous ankle sprain. One study found a fivefold increase in ankle injuries in previously injured basketball players compared to those with no previous history of ankle injury [15]. This predilection is thought to be due to reduced proprioceptive function as well as reduced mechanical stability. One of the most predictable ways to prevent recurrent or chronic instability is to fully rehabilitate the initial injury by focusing on optimizing both proprioception and peroneal strength [29]. This has been shown to be effectively done through the use of a balance board or wobble board (also called ankle disk training) [30].

Prehabilitation programs that emphasize overall fitness, balance, and prevention strategies, such as landing with a wide-based stance may also provide players protection from future ankle sprains [31] (Level II). Such programs are currently used by most NBA teams [32].

## Jones Fractures

### Etiology and Pathomechanism

Sir Robert Jones was the first to bring attention to proximal fractures of the fifth metatarsal in 1902 when he described his own injury which he sustained indirectly while dancing [33]. Today, proximal fractures of the fifth metatarsal are generally divided into three anatomic zones: Zone 1 – tuberosity; Zone 2 – metaphyseal-diaphyseal junction; Zone 3 – proximal diaphysis. Each of these fracture types commonly occur in basketball players and are associated with a specific mechanism of injury.

Zone 1 fractures are tuberosity avulsion fractures that result from abrupt hindfoot inversion. An injury of this type may be sustained in basketball when a rebounding player lands on the foot of another player. As the hindfoot forcibly inverts, contraction of the peroneus brevis muscle [34] or tension on the lateral band of the plantar fascia results in an avulsion of the tuberosity.

The classic Jones fracture occurs in Zone 2 and is an acute fracture at the metaphyseal-diaphyseal junction, without distal extension beyond the four to five intermetatarsal articulation. The mechanism of injury is believed to be a large adduction force applied to the forefoot with simultaneous ankle plantar-flexion, such as when one missteps on the lateral border of the foot [35, 36]. This type of fracture has been recognized for its high rate of delayed union, nonunion, and refracture due to its location in a relatively avascular watershed zone with diminished osseous blood supply and resultant poor healing potential [37].

Zone 3 fractures consist of proximal diaphyseal stress fractures that result from repetitive overuse or overload applied under the metatarsal head. It has been noted that in basketball players, stress fractures of the proximal fifth metatarsal occur during games and practices in which the shuttle run and cutting tasks are frequently performed [38]. These repetitive injuries may be acute or chronic. Conditions that place increased stress or load to the lateral column of the foot, such as a varus hindfoot [39], can predispose the athlete to fifth metatarsal fracture. In a similar manner, there is evidence to suggest that generic use of non-customized off-the-shelf foot orthoses with medial arch support may increase fifth metatarsal loading and therefore risk of fracture [40].

The distinction between Jones (Zone II) and proximal diaphyseal stress (Zone III) fractures is often difficult because of their close anatomic proximity. Recent data also indicate similar healing characteristics and therefore treatment indications for both acute Jones and proximal diaphyseal stress fractures, suggesting that both zones should be regarded simply as “Jones” fractures [41].

### Epidemiology

The fifth metatarsal is the most commonly-fractured metatarsal in adults, and has been shown to be involved in up to 70% of all metatarsal fractures [42]. These fractures are common among running and jumping athletes. In particular, basketball players have an increased prevalence of proximal fifth metatarsal fractures compared to other athletic populations [43, 44].

### Therapy (On the Field, Conservative, Surgical)

Due to unacceptably high nonunion rates, refracture rates, and delayed return to activities with nonoperative treatment of Zone 2 and Zone 3 fractures, primary fixation is the accepted standard of care for the elite athlete [45–48]. The difficulty in managing proximal fifth metatarsal fractures conservatively in basketball players was first described in 1979 by Zelko and colleagues [49]. In their series of 21 patients, 9 fractures and 8 refractures occurred during basketball. They found that, compared to other athletes, basketball players were most disabled by this type of fracture with a greater tendency for reinjury, and were most successfully treated by early bone grafting. A later prospective, nonrandomized study of basketball players by Fernandez and colleagues [43] concluded that operative treatment was more reliable than nonoperative management, with faster return to sport (Level I). In nine stress fractures treated with compression screw fixation, all healed within 8–14 weeks. In four of eight acute fractures treated nonoperatively, half went onto nonunion after 12 weeks and required internal fixation (Level IV).

Surgery typically consists of internal fixation with an intramedullary screw with or without bone graft. Multiple biomechanical studies have been performed in an attempt to determine the optimal screw size. In general, for the skeletally mature athlete, the screw should be  $\geq 4.5$  mm, as smaller diameter screws have been associated with delayed union and nonunion [50] (Level IV). Some authors suggest using the largest screw that the canal can accommodate, particularly in the heavier athlete [51] (Level V). The surgeon should be cognizant, however, that too-large a screw can cause cortical compromise, including fracture and increased risk of stress shielding across the fracture site. Whether to use cannulated or solid screws and stainless steel or titanium has also been studied without conclusive results. Solid screws have been shown to be less likely to break, but it is unclear if this difference is clinically significant [52]. Biomechanical studies have suggested that stainless steel is more resistant to bending forces than titanium. However, a study by DeVries et al. [53] comparing cannulated stainless steel and titanium screws of multiple sizes showed

comparable rates of healing among all groups, demonstrating the absence of a clinical advantage in using one screw type over another. Regardless of screw size and type, it is important that the fixation is rigid and there is compression across the fracture site.

Before proceeding with surgery, the athlete should be made aware of the risks of surgery, including sural nerve injury, hardware irritation with possible need for subsequent removal, nonunion, delayed union, and refracture.

## Rehabilitation and Back-to-Sports

Postoperative rehabilitation protocols following intramedullary fixation of proximal fifth metatarsal fractures vary. In general, weight bearing is restricted for 2–4 weeks and the patient is kept protected in a cast or boot. Weight bearing is progressively advanced as tenderness subsides. The patient is transitioned to a molded orthotic device in a stiff soled shoe to decrease stress at the fracture site. Upon return to sport, functional bracing, shoe modification, or orthoses should be considered.

Given the tenuous nature of these fractures and risk for refracture, early return to play should be permitted with caution. At our institution, both clinical and radiographic evidence of healing should be present. Routine CT scanning of elite athletes to confirm union is recommended.

In a systematic review of 26 studies on intramedullary fixation of proximal fifth metatarsal fractures, the authors found that return to sports ranged from 4 to 18 weeks. In elite basketball players, return was less than 3 months in most series with low complication rates [54].

## Prevention

Non-customized off-the-shelf foot orthoses with medial arch support may increase maximum pressures and forces on the base of the fifth metatarsal during landing from a lay-up, and on the head and base of the fifth metatarsal during the stance phase of the shuttle run in basketball players [40]. Avoiding these orthotics in this population may prevent this particular injury pattern. Additionally, correction of any hindfoot varus or forefoot supination with custom orthotics to offload the lateral column may prevent injury [39]. The goal is to transfer weight and stress to the medial side of the midfoot and off the fifth metatarsal.

Refracture following intramedullary fixation of proximal fifth metatarsal fractures is a not uncommon complication in athletes. Recognized risk factors for refracture include inadequate screw size [49], premature return to sports activity [50], and varus hindfoot alignment with resultant lateral column overload [39] (Level IV). As discussed above,

prevention of refracture is enhanced by using an adequate screw size greater than 4.5 mm, confirming complete fracture union before return to sports via CT scan, and adding a custom rigid orthotic insert with a lateral hindfoot wedge extended to a lateral forefoot post in the athlete with any suggestion of varus alignment.

Also, because of the risk for recurrent fracture in high-performance athletes, it is recommended that the screw be retained after healing, as long as the player continues to compete [55].

## Achilles Tendon Ruptures

### Etiology and Pathomechanism

Injuries to the Achilles tendon occur during an eccentric contraction of the gastrocnemius-soleus complex when the ankle suddenly dorsiflexes while the foot is plantarflexed. Other times, the rupture occurs with pushing off with the knee in extension while the patient is weight-bearing.

Training errors account for a large percentage of injuries to the Achilles tendon in sports [56, 57]. Inappropriate footwear, increases in a training regimen, alterations in training routine, and changes in playing surface may also contribute to injuries in the athlete. Common risk factors associated with Achilles tendon ruptures – fluoroquinolone usage, corticosteroids, inflammatory arthritis, Haglund’s deformity – do not generally have any relevance in those playing basketball. Prodromal symptoms of pain in the Achilles tendon and stiffness of the ankle are commonly seen in the elite athlete [58].

### Epidemiology

In the United States, the most common sport involved with an acute Achilles rupture is basketball; this sport accounts for 48 % of all injuries [59]. The rising incidence of Achilles tendon ruptures over the past two decades is partially due to an older population participating in sports and recreational activities [60].

### Therapy (On the Field, Conservative, Surgical)

Diagnosing an acute Achilles tendon rupture is largely a clinical diagnosis. A thorough history and physical examination can lead to diagnosis in a majority of patients. An archetypal patient is a 30–45 year old weekend warrior who reports pain and inability to walk after playing basketball. This intense pain gradually diminishes after a few hours, and the patient is left with a functional deficit of weakness of plantarflexion and a gait disturbance often with an inability to bear weight.

The American Academy of Orthopaedic Surgeons (AAOS) Clinical Practice Guidelines [30] recommend that the diagnosis of Achilles tendon rupture be made with two or more of the following physical examination findings: positive Thompson test, decreased plantarflexion strength, palpable defect of the tendon, and increased passive ankle dorsiflexion (Level I). When the patient is supine, one can examine for bruising around the posterior ankle, strength of plantar flexion, and a palpable gap in the tendon. The patient may also attempt a single limb heel-raise on the affected leg. The patient is then turned prone with the knees at a 90° flexion angle. Firstly, the resting tension of the affected leg is observed. The calf of the affected leg is then compressed. A positive Thompson test is when compressing the calf does not result in plantar flexion of the foot.

Despite an increasing understanding of these injuries and rapid rise in its incidence, there is no clear consensus on non-operative versus surgical treatment. Options for treatment include cast immobilization, early functional rehabilitation with functional bracing, open repair, and percutaneous repair. AAOS Clinical Practice Guidelines state that both nonsurgical and surgical treatments are valid options for patients with acute Achilles tendon ruptures [61].

At the senior author's institution, the non-operative protocol, which is rarely utilized, includes placing the patient plantarflexed in a CAM boot weight-bearing as tolerated with three wedges. Starting at 3 weeks from injury, the patient is to remove one wedge per week. At 6 weeks from injury, the patient is placed into a CAM boot at neutral and formal physical therapy is started. At 10 weeks, the patient is back in normal athletic shoes (Level V).

There are three options for operative treatment for Achilles tendon ruptures: open, mini-open, or percutaneous. The risks of surgery include: superficial wound infection, sural nerve dysfunction, wound healing issues, deep infection, re-rupture, and deep vein thrombosis.

The most recent meta-analysis discussing operative versus non-operative treatment by Soroceanu et al. and colleagues [62] reviewed ten studies (Level II Evidence, Grade B). Each study reported the re-rupture rate with an absolute risk difference of 5.5% in favor of surgery. In a stratified analysis, there was no significant difference in the re-rupture rate if functional rehabilitation with early range of motion was used in the non-operative group. However, when the functional rehabilitation studies are removed, there was a significant absolute risk reduction by 8.8% in the surgery group. Comparing the complication rate between non-operative and operative management, there was a complication other than re-rupture for 1 in 7 patients treated surgically or a 3.9 times risk for a complication. Patients with surgical treatment returned 19.2 days earlier to work.

## Rehabilitation and Back-to-Sports

An Achilles tendon rupture is a burden to both the professional and recreational athlete. For the professional athlete, this injury may prevent a return to the sport at a competitive level. Post-injury changes of morphology and kinematics are also noted in the non-athletic population. At an average of 10.8 years from injury, Horstmann et al. [63] noted that there were smaller values of the injured side's calf circumference, ankle range of motion, and heel height during heel-raise tests and calf circumference as compared to the uninjured leg. This report underscores the observation that following repair of the ruptured Achilles tendon, muscle atrophy is a long-term consideration [36] (Level IV).

## Prevention

There are no formal studies which present evidence for preventing Achilles tendon ruptures. If one were to experience the prodromal symptoms of tendinopathy or symptoms which portend a rupture, then stretching and strengthening exercises should be performed.

---

## Osteochondral Lesions of the Talus (OLTs)

### Etiology and Pathomechanism

The pathophysiology of OLTs is as elusive as is its treatment. Literature does not have a definitive answer; rather, hypotheses range from repeated microtrauma, microvascular insufficiency, ankle instability, ankle trauma, and genetics [64]. Lateral lesions are associated with trauma close to 100% of the time, whereas medial lesions are trauma-related between 64% and 82% of the time [65, 66].

### Epidemiology

The true incidence of OLTs is unknown, however in a 10-year review of military personnel, there was a 27 OLTs per 100,000 person years with spikes of OLTs seen during periods of combat.

In a study of 283 ankle fractures who underwent an open reduction internal fixation and an arthroscopy, 73% of patients had a chondral lesion [67]. Most lesions occur in the centromedial (54.5%) and centrolateral (31.2%) aspect of the talus [68]. Another OLT morphologic study demonstrated that medial lesions were larger in depth and surface area compared to lateral OLTs [69] (Level IV, Grade C).

## Therapy (On the Field, Conservative, Surgical)

Diagnosis of OLTs are often delayed given the non-descript and vague symptoms that are present early in the disease. Patients present with an aching pain with weight-bearing and during practice or games. Often, these lesions are not associated with typical mechanical symptoms (locking, catching, clicking). A history of ankle trauma is often elicited.

Initial imaging should include three weight-bearing views of the ankle. If an OLT is not seen, but highly suspected, an ankle MRI should be obtained to look for cartilage irregularities and subchondral edema. CT scans are useful in surgical planning to determine the extent of subchondral bony involvement. If an OLT is discovered on enhanced imaging, one must determine if the lesion is the source of the pain. Therefore, an intra-articular anesthetic injection is administered to see if the pain is relieved (Level V).

Conservative therapy includes therapeutic stretching, range of motion exercises, sport-specific training, proprioceptive training, NSAIDs, and corticosteroids. Some surgeons will also attempt immobilization in efforts to reduce the synovitis of the anterior joint capsule (Expert opinion).

While exploring the numerous methods of treating OLTs (it is not within the scope) of this chapter to discuss all the procedures that can be performed: arthroscopic debridement and microfracture, anterograde/retrograde drilling, bone grafting, lesion reduction and internal fixation, structural allograft, osteochondral allo/autograft, OAT, ACL, and juvenile cartilage (see Chaps. 16 and 31).

## Rehabilitation and Back-to-Sports

Rehabilitation varies widely based on the extent of the procedure performed. If any sort of drilling, microfracture or reconstructive procedure is performed, weight bearing is restricted to allow fibrocartilage to form [70]. For lesions smaller than 1.5 cm<sup>2</sup>, weight bearing is resumed after 2 weeks. For lesions larger than 1.5 cm<sup>2</sup>, weight bearing is restricted for at least 4–6 weeks. Range of motion is allowed once the arthroscopy portals are healed.

Again, return to sport is variable depending on the extent of the primary procedure. Often, for smaller lesions, a player can return to drills by 3 months and practice/games by 4 months.

## Prevention

There is no literature to suggest that OLTs can be prevented, given that researchers do not understand how they form.

## Anterior Ankle Impingement

### Etiology and Pathomechanism

Ankle impingement can be a common source of chronic ankle pain in an athlete subjected to repeated ankle sprains and microtrauma. Specifically in basketball, high-impact load experienced during repetitive jumping and dorsiflexion likely contributes to the development of the condition. Recurrent microtrauma to the anterior structures of the ankle and ankle capsule traction will cause fibrocartilage and deep hypertrophic cartilage to undergo enchondral ossification [71]. With repeated dorsiflexion, there may be abutment of adjacent osteophytes limiting motion and producing pain caused by entrapment of soft tissues. It is the soft tissue impingement that leads to significant pain. Osteophyte formation may also be the result of chronic ankle instability as well. A lateral ankle sprain may be the instigating factor as the sprain causes increasing laxity of the lateral ankle structures which lead to inflammation and synovitis of the ankle capsule. As the synovial tissue hypertrophies, the soft tissue structures are impinged and osteophytes form in reaction to this stress.

The distal fascicle of the anterior inferior tibiofibular ligament (Bassett's ligament) can also become hypertrophied to due repetitive stress. This structure may produce anterolateral impingement. Symptoms of impingement are made evident when there is ankle laxity as this ligament comes into contact with the lateral dome of the talus [72]. Without the support of the anterior talofibular ligament, the talus translates slightly anterior during ankle motion, causing impingement on Bassett's ligament. Inversion ankle injuries are suspected as the main cause for the initiation of impingement [73].

### Epidemiology

There are no statistics in the literature that provide an incidence of impingement in basketball players. All reports of impingement in basketball are purely subjective and anecdotal. Basketball players with ankle inversion sprains and repetitive microtrauma frequently develop anterior impingement. After practice or games, these patients can present with ankle swelling, anterior ankle pain, and decreased ankle range of motion. On physical examination, the player will have swelling about the ankle joint. They may have some tenderness to palpation anterolaterally or anteromedially (at the shoulders of the joint line). In the clinic setting, one may replicate symptoms by forcefully dorsiflexing the ankle.

Radiographically, the ankle joint often shows no lines of osteoarthritis. In early impingement, the anterior distal tibia may lack the normal smoothness of the periosteal border. In later stages of impingement, the distal tibia has an osteophyte projecting anteriorly. This bony ridge does not involve the articular surface. On the talar side, osteophytes project superiorly and toward the ankle joint.

Since anteromedial osteophytes are often undetected due to superimposition of other talar structures [74], van Dijk recommends an oblique anteromedial impingement view to improve the sensitivity and specificity of the imaging. In this view, the foot is plantarflexed, the leg externally rotated 30°, and the beam is directed in 45° craniocaudal direction from a standard lateral x-ray view.

### Therapy (On the Field, Conservative, Surgical)

Following a diagnosis of impingement, conservative therapy is tried for at least 3–6 months. Conservative treatment includes therapeutic stretching, range of motion exercises, sport-specific training, proprioceptive training, NSAIDs, and corticosteroids. Some surgeons will also attempt immobilization in efforts to reduce the synovitis of the anterior joint capsule. Functional rehabilitation is generally favored over casting [72].

After conservative treatment has failed, arthroscopic removal of either Bassett's ligament [74] or the impinging osteophytes is recommended [71]. Most basketball players will opt for longer periods of conservative treatment as long as the impingement does not significantly affect the season. Scranton and McDermott compared open and arthroscopic resection of the impinging osteophytes. Patients treated arthroscopically recovered in roughly half the time, and had a month quicker return to sport. Those with larger osteophytes or evidence of tibiotalar arthritis had poorer functional outcomes.

In a study [75] (Grade IV Evidence) evaluating 80 patients at an average follow-up of 105 months demonstrated that patients with spurs localized to the tibia did better at follow-up than did those with minimal to no arthritic changes on X-ray. Negative prognostic factors included: age, history of ankle trauma, and a cavus foot. Overall, these patients did have a significant improvement in their AOFAS scores at final follow-up after arthroscopic treatment.

### Rehabilitation and Back-to-Sports

Rehabilitation is often limited to concomitant procedures that are performed with osteophyte removal. For isolated arthroscopic debridement, a brief period of immobilization in a splint allows wound and skin healing. After the wound is healed, we begin aggressive range of motion and weight bearing. Physical therapy may be necessary for some

patients. Patients are usually back to basketball within a few weeks post-bone spur removal.

### Prevention

There are no formal studies which look at preventing ankle impingement. Since microtrauma and lateral ankle instability contribute to anterior impingement, please refer to the section about ankle instability.

### Evidence

Level of evidence for this paper ranges from I-IV. Grade of evidence, depending on the topic, is Grade B (Fair).

### Conclusions

Injuries to the foot and ankle are frequent in both competitive and recreational basketball, and are an important source of significant time lost from play. Because of the rapid acceleration and deceleration, cutting, jumping, and pivoting maneuvers involved in basketball, the foot and ankle are particularly susceptible to both acute and overuse injuries. Prevention strategies, appropriate operative intervention when indicated, and dedicated functional rehabilitation are all important in the management of the injured basketball player for safe and timely return to play.

### Summary

1. The ankle is the most injured joint in basketball (Grade A).
2. Most ankle sprains are noncontact injuries, and may occur while taking an awkward step during cutting or turning maneuvers (Grade A).
3. The most important risk factor for an ankle sprain is a previous ankle sprain. One of the most predictable ways to prevent recurrent or chronic instability is to fully rehabilitate the initial injury by focusing on optimizing both proprioception and peroneal strength (Grade B).
4. Confirm union of a Jones fracture with a CT scan before returning an elite athlete to play (Expert opinion).
5. Basketball accounts for 48% of all Achilles ruptures (Grade A).

### References

1. Commission USCPS. NEISS data highlights 2012. Washington, DC; 2012.
2. Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the national basketball association: a 17-year overview. *Sports Health*. 2010;2:284–90.

3. Brostrom L. Sprained ankles. I. Anatomic lesions in recent sprains. *Acta Chir Scand*. 1964;128:483–95.
4. Powell JW, Barber-Foss KD. Sex-related injury patterns among selected high school sports. *Am J Sports Med*. 2000;28:385–91.
5. The team physician. A statement of the Committee on the Medical Aspects of Sports of the American Medical Association, September 1967. *J Sch Health*. 1967;37:497–501.
6. Gerber JP, Williams GN, Scoville CR, Arciero RA, Taylor DC. Persistent disability associated with ankle sprains: a prospective examination of an athletic population. *Foot Ankle Int*. 1998;19:653–60.
7. Bosien WR, Staples OS, Russell SW. Residual disability following acute ankle sprains. *J Bone Joint Surg Am*. 1955;37-A:1237–43.
8. Dick R, Hertel J, Agel J, Grossman J, Marshall SW. Descriptive epidemiology of collegiate men's basketball injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 through 2003–2004. *J Athl Train*. 2007;42:194–201.
9. Starkey C. Injuries and illnesses in the national basketball association: a 10-year perspective. *J Athl Train*. 2000;35:161–7.
10. Hootman JM, Dick R, Agel J. Epidemiology of collegiate injuries for 15 sports: summary and recommendations for injury prevention initiatives. *J Athl Train*. 2007;42:311–9.
11. Deitch JR, Starkey C, Walters SL, Moseley JB. Injury risk in professional basketball players: a comparison of Women's National Basketball Association and National Basketball Association athletes. *Am J Sports Med*. 2006;34:1077–83.
12. Kofotolis N, Kellis E. Ankle sprain injuries: a 2-year prospective cohort study in female Greek professional basketball players. *J Athl Train*. 2007;42:388–94.
13. Milgrom C, Shlamkovitch N, Finestone A, et al. Risk factors for lateral ankle sprain: a prospective study among military recruits. *Foot Ankle*. 1991;12:26–30.
14. McHugh MP, Tyler TF, Tetro DT, Mullaney MJ, Nicholas SJ. Risk factors for noncontact ankle sprains in high school athletes: the role of hip strength and balance ability. *Am J Sports Med*. 2006;34:464–70.
15. McKay GD, Goldie PA, Payne WR, Oakes BW. Ankle injuries in basketball: injury rate and risk factors. *Br J Sports Med*. 2001;35:103–8.
16. Hosea TM, Carey CC, Harrer MF. The gender issue: epidemiology of ankle injuries in athletes who participate in basketball. *Clin Orthop Relat Res*. 2000;372:45–9.
17. Beynon BD, Renstrom PA, Haugh L, Uh BS, Barker H. A prospective, randomized clinical investigation of the treatment of first-time ankle sprains. *Am J Sports Med*. 2006;34:1401–12.
18. Pihlajamaki H, Hietaniemi K, Paavola M, Visuri T, Mattila VM. Surgical versus functional treatment for acute ruptures of the lateral ligament complex of the ankle in young men: a randomized controlled trial. *J Bone Joint Surg Am*. 2010;92:2367–74.
19. Haraguchi N, Toga H, Shiba N, Kato F. Avulsion fracture of the lateral ankle ligament complex in severe inversion injury: incidence and clinical outcome. *Am J Sports Med*. 2007;35:1144–52.
20. Renstrom PA. Persistently painful sprained ankle. *J Am Acad Orthop Surg*. 1994;2:270–80.
21. Reed MFJ, Donley B, Giza E. Athletic ankle injuries Orthopaedic Knowledge Update (OKU): sports medicine 4. Rosemont: American Academy of Orthopaedic Surgery (AAOS); 2009. p. 199–214.
22. Bell SJ, Mologne TS, Sitler DF, Cox JS. Twenty-six-year results after Brostrom procedure for chronic lateral ankle instability. *Am J Sports Med*. 2006;34:975–8.
23. Handoll HH, Rowe BH, Quinn KM, de Bie R. Interventions for preventing ankle ligament injuries. *Cochrane Database Syst Rev*. 2001;3:CD000018.
24. Sitler M, Ryan J, Wheeler B, et al. The efficacy of a semirigid ankle stabilizer to reduce acute ankle injuries in basketball. A randomized clinical study at West Point. *Am J Sports Med*. 1994;22:454–61.
25. Surve I, Schweltnus MP, Noakes T, Lombard C. A fivefold reduction in the incidence of recurrent ankle sprains in soccer players using the Sport-Stirrup orthosis. *Am J Sports Med*. 1994;22:601–6.
26. Metcalfe RC, Schlabach GA, Looney MA, Renehan EJ. A comparison of moleskin tape, linen tape, and lace-up brace on joint restriction and movement performance. *J Athl Train*. 1997;32:136–40.
27. Pope RP, Herbert RD, Kirwan JD, Graham BJ. A randomized trial of preexercise stretching for prevention of lower-limb injury. *Med Sci Sports Exerc*. 2000;32:271–7.
28. Barrett JR, Tanji JL, Drake C, Fuller D, Kawasaki RI, Fenton RM. High- versus low-top shoes for the prevention of ankle sprains in basketball players. A prospective randomized study. *Am J Sports Med*. 1993;21:582–5.
29. Willems T, Witvrouw E, Verstuyft J, Vaes P, De Clercq D. Proprioception and muscle strength in subjects with a history of ankle sprains and chronic instability. *J Athl Train*. 2002;37:487–93.
30. Verhagen E, van der Beek A, Twisk J, Bouter L, Bahr R, van Mechelen W. The effect of a proprioceptive balance board training program for the prevention of ankle sprains: a prospective controlled trial. *Am J Sports Med*. 2004;32:1385–93.
31. Smith RW, Reischl SF. Treatment of ankle sprains in young athletes. *Am J Sports Med*. 1986;14:465–71.
32. Bernot MP. Medical care of the Atlanta Hawks. *J Med Assoc Ga*. 2000;89:21–3.
33. Jones R. I. Fracture of the base of the fifth metatarsal bone by indirect violence. *Ann Surg*. 1902;35:697–700.2.
34. McDermott EP. Basketball injuries of the foot and ankle. *Clin Sports Med*. 1993;12:373–93.
35. Dameron Jr TB. Fractures and anatomical variations of the proximal portion of the fifth metatarsal. *J Bone Joint Surg Am*. 1975;57:788–92.
36. Lawrence SJ, Botte MJ. Jones' fractures and related fractures of the proximal fifth metatarsal. *Foot Ankle*. 1993;14:358–65.
37. Smith JW, Arnoczky SP, Hersh A. The intraosseous blood supply of the fifth metatarsal: implications for proximal fracture healing. *Foot Ankle*. 1992;13:143–52.
38. Meyer SA, Saltzman CL, Albright JP. Stress fractures of the foot and leg. *Clin Sports Med*. 1993;12:395–413.
39. Raikin SM, Slenker N, Ratigan B. The association of a varus hind-foot and fracture of the fifth metatarsal metaphyseal-diaphyseal junction: the Jones fracture. *Am J Sports Med*. 2008;36:1367–72.
40. Yu B, Preston JJ, Queen RM, et al. Effects of wearing foot orthosis with medial arch support on the fifth metatarsal loading and ankle inversion angle in selected basketball tasks. *J Orthop Sports Phys Ther*. 2007;37:186–91.
41. Chuckpaiwong B, Queen RM, Easley ME, Nunley JA. Distinguishing Jones and proximal diaphyseal fractures of the fifth metatarsal. *Clin Orthop Relat Res*. 2008;466:1966–70.
42. Petrisor BA, Ekrol I, Court-Brown C. The epidemiology of metatarsal fractures. *Foot Ankle Int*. 2006;27:172–4.
43. Fernandez Fairen M, Guillen J, Busto JM, Roura J. Fractures of the fifth metatarsal in basketball players. *Knee Surg Sports Traumatol Arthrosc Off J ESSKA*. 1999;7:373–7.
44. Iwamoto J, Takeda T. Stress fractures in athletes: review of 196 cases. *J Orthop Sci Off J Jpn Orthop Assoc*. 2003;8:273–8.
45. Kavanaugh JH, Brower TD, Mann RV. The Jones fracture revisited. *J Bone Joint Surg Am*. 1978;60:776–82.
46. DeLee JC, Evans JP, Julian J. Stress fracture of the fifth metatarsal. *Am J Sports Med*. 1983;11:349–53.
47. Torg JS, Balduini FC, Zelko RR, Pavlov H, Peff TC, Das M. 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*. 1984;66:209–14.
48. Clapper MF, O'Brien TJ, Lyons PM. Fractures of the fifth metatarsal. Analysis of a fracture registry. *Clin Orthop Relat Res*. 1995;315:238–41.



49. Zelko RR, Torg JS, Rachun A. Proximal diaphyseal fractures of the fifth metatarsal – treatment of the fractures and their complications in athletes. *Am J Sports Med.* 1979;7:95–101.
50. Glasgow MT, Naranja Jr RJ, Glasgow SG, Torg JS. Analysis of failed surgical management of fractures of the base of the fifth metatarsal distal to the tuberosity: the Jones fracture. *Foot Ankle Int.* 1996;17:449–57.
51. Wright RW, Fischer DA, Shively RA, Heidt Jr RS, Nuber GW. Refracture of proximal fifth metatarsal (Jones) fracture after intramedullary screw fixation in athletes. *Am J Sports Med.* 2000;28:732–6.
52. Pietropaoli MP, Wnorowski DC, Werner FW, Fortino MD. Intramedullary screw fixation of Jones fractures: a biomechanical study. *Foot Ankle Int.* 1999;20:560–3.
53. DeVries JG, Cutticia DJ, Hyer CF. Cannulated screw fixation of Jones fifth metatarsal fractures: a comparison of titanium and stainless steel screw fixation. *J Foot Ankle Surg Off Pub Am Coll Foot Ankle Surg.* 2011;50:207–12.
54. Roche AJ, Calder JD. Treatment and return to sport following a Jones fracture of the fifth metatarsal: a systematic review. *Knee Surg Sports Traumatol Arthrosc Off J ESSKA.* 2013;21:1307–15.
55. Josefsson PO, Karlsson M, Redlund-Johnell I, Wendeberg B. Jones fracture. Surgical versus nonsurgical treatment. *Clin Orthop Relat Res.* 1994;299:252–5.
56. Heckman DS, Gluck GS, Parekh SG. Tendon disorders of the foot and ankle, part 2: achilles tendon disorders. *Am J Sports Med.* 2009;37:1223–34.
57. Schepsis AA, Jones H, Haas AL. Achilles tendon disorders in athletes. *Am J Sports Med.* 2002;30:287–305.
58. Maffulli N, Longo UG, Maffulli GD, Khanna A, Denaro V. Achilles tendon ruptures in elite athletes. *Foot Ankle Int.* 2011;32:9–15.
59. Raikin SM, Garras DN, Krapchev PV. Achilles tendon injuries in a United States population. *Foot Ankle Int.* 2013;34:475–80.
60. Moller A, Astron M, Westlin N. Increasing incidence of Achilles tendon rupture. *Acta Orthop Scand.* 1996;67:479–81.
61. Chiodo CP, Glazebrook M, Bluman EM, et al. Diagnosis and treatment of acute Achilles tendon rupture. *J Am Acad Orthop Surg.* 2010;18:503–10.
62. Soroceanu A, Sidhwa F, Aarabi S, Kaufman A, Glazebrook M. Surgical versus nonsurgical treatment of acute Achilles tendon rupture: a meta-analysis of randomized trials. *J Bone Joint Surg Am.* 2012;94:2136–43.
63. Horstmann T, Lukas C, Merk J, Brauner T, Mundermann A. Deficits 10-years after Achilles tendon repair. *Int J Sports Med.* 2012;33:474–9.
64. Talusan PG, Milewski MD, Toy JO, Wall EJ. Osteochondritis dissecans of the talus: diagnosis and treatment in athletes. *Clin Sports Med.* 2014;33:267–84.
65. Alexander AH, Lichtman DM. Surgical treatment of transchondral talar-dome fractures (osteochondritis dissecans). Long-term follow-up. *J Bone Joint Surg Am.* 1980;62:646–52.
66. Flick AB, Gould N. Osteochondritis dissecans of the talus (transchondral fractures of the talus): review of the literature and new surgical approach for medial dome lesions. *Foot Ankle.* 1985;5:165–85.
67. Leontaritis N, Hinojosa L, Panchbhavi VK. Arthroscopically detected intra-articular lesions associated with acute ankle fractures. *J Bone Joint Surg Am.* 2009;91:333–9.
68. Hembree WC, Wittstein JR, Vinson EN, et al. Magnetic resonance imaging features of osteochondral lesions of the talus. *Foot Ankle Int.* 2012;33:591–7.
69. Elias I, Zoga AC, Morrison WB, Besser MP, Schweitzer ME, Raikin SM. Osteochondral lesions of the talus: localization and morphologic data from 424 patients using a novel anatomical grid scheme. *Foot Ankle Int.* 2007;28:154–61.
70. Easley ME, Latt LD, Santangelo JR, Merian-Genast M, Nunley 2nd JA. Osteochondral lesions of the talus. *J Am Acad Orthop Surg.* 2010;18:616–30.
71. Tol JL, van Dijk CN. Anterior ankle impingement. *Foot Ankle Clin.* 2006;11:297–310, vi.
72. Hess GW. Ankle impingement syndromes: a review of etiology and related implications. *Foot Ankle Spec.* 2011;4:290–7.
73. Akseki D, Pinar H, Yaldiz K, Akseki NG, Arman C. The anterior inferior tibiofibular ligament and talar impingement: a cadaveric study. *Knee Surg Sports Traumatol Arthrosc Off J ESSKA.* 2002;10:321–6.
74. van Dijk CN, Wessel RN, Tol JL, Maas M. Oblique radiograph for the detection of bone spurs in anterior ankle impingement. *Skeletal Radiol.* 2002;31:214–21.
75. Parma A, Buda R, Vannini F, et al. Arthroscopic treatment of ankle anterior bony impingement: the long-term clinical outcome. *Foot Ankle Int.* 2014;35:148–55.

Volker Schöffl

**Abstract**

Lower leg injuries make up to 50 % of acute climbing injuries. They mostly result from falling and hitting the wall or from ground falls. Strains and fractures to the foot are most common. Chronic climbing injuries in the feet result from the use of too small shoes which force the toes in an erect position.

**Keywords**

Rock climbing • Climbin injuries • Sport climbing • Feet injuries climbing

**Injuries**

Recent studies show up to 50% of acute injuries in rock climbing involving the lower extremities (foot, toe and ankle) [1, 2]. Killian et al. [3] showed a significant correlation between the incidence of ankle sprains and bouldering as well as ankle sprains and sport climbing. Besides the acute lower limb injuries the incidence of chronic feet problems increases in the higher levels of sport climbing [4]. Typical feet injuries in rock climbers are: contusions, calcaneus fractures, talus fractures, ankle fractures and ankle sprains with lateral ligament injuries [1]. Considering chronic injuries it is important to know that 87 % of climbers are willingly accepting pain within their climbing shoes to achieve a better performance [5]. Typical overstrain injuries are: chronic foot pain, callosity and pressure marks on the toes (Fig. 48.1), dead toe nails, broken and missing toe nails, nail bed infections, dermatomycosis, blisters, claw toes, subungual hematoma (Table 48.1) [1, 3, 6–8]. In the long term, using tight fit climbing shoes can also lead to the development of a hallux valgus deformity [6].

**Etiology and Pathomechanism**

Most acute injuries to the lower extremity in climbing are caused by a fall. Falling pathomechanisms show either a wall-collision fall or a ground fall [1, 9]. A wall-collision fall is one where the climber impacts the wall in a more or less vertical plane (caused by pull of the belay rope), while a ground fall is one where the climber impacts in more or less the horizontal plane [1]. Typical wall-collision injuries are contusions or compound fractures, while ground falls frequently lead to ankle fractures, ankle sprains or calcaneus fractures. The majority of chronic climbing foot injuries result from wearing climbing shoes unnaturally shaped or too small in size [3, 6]. Those shoes facilitate the ability to stand on friction with straight toes and on edges with bent toes with precision and proper contact. Most climbers wear their climbing shoes 2–3 sizes smaller than their street shoes [6]. This shoe size reduction forces the foot to conform the shoe and changes the biomechanical position of the foot within the shoe (Fig. 48.2) [1].

**Epidemiology**

Almost 50 % of acute injuries involve the leg and feet [2, 10, 11]. Acute injuries are either caused by ground falls or rock hit trauma during a fall. Most frequently strains, contusions

V. Schöffl, MHBA  
Department of Sportorthopedics, Sportsmedicine, Shoulder and  
Elbow Surgery, Klinikum Bamberg, Bugerstraße 80, 96049  
Bamberg, Federal Republic of Germany  
e-mail: [volker.schoeffl@me.com](mailto:volker.schoeffl@me.com)

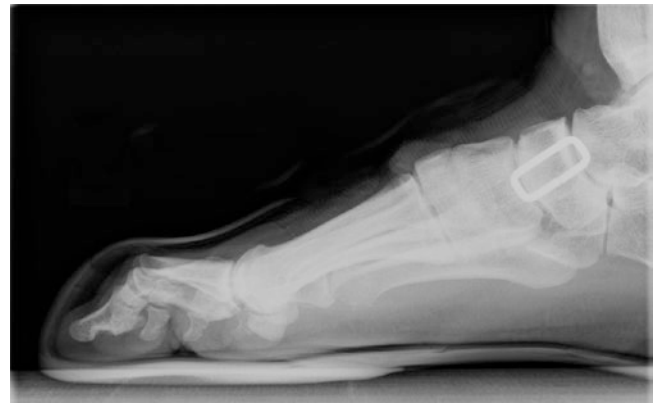


**Fig. 48.1** Pressure marks on the toes due to tight climbing shoes

**Table 48.1** Chronic feet injuries in long time high ability rock climbers [6]

Pathology	N
Pressure marks	30
Pressure tenderness	26
Hallux valgus	16
Subungual haematoma	9
Unguis incarnatus	6
Blisters	3
Broken nails	3
Dead nail	3
Mycosis	3
Nail bed infection	2
Achillodynie	1
Claw toes	1
Joint pain	1
Nail loss	1

and fractures of the calcaneus and talus. More rare injuries, as *e.g.*, osteochondral lesions of the talus demand a highly specialized care. The chronic use of tight climbing shoes leads to overstrain injuries also. As the tight fit of the shoes changes the biomechanics of the foot an increased stress load



**Fig. 48.2** X-ray analysis of a climbers foot in a tight shoe (lateral plane)

is applied to the fore-foot [4]. Up to 90% of high active climbers complain about feet pain within their climbing shoes [6].

## Therapy

### Acute Injuries

These typical injuries undergo standard orthopedic trauma guidelines and in case of displaced fractures mainly operative reconstruction (open reduction and internal fixation). Considering that climbers are mostly young and highly active patients we also favor a surgical repair in most talus and calcaneus fractures to achieve an optimal outcome.

### Chronic Injuries

These injuries should be avoided in the first place. Climbers should not use their shoes too tight and the shoe industry should work on more anatomical lasts for the shoes to minimize chronic conditions (Fig. 48.3). For training a larger seized shoes should be used. Hygienic and caring handling of callosities and nail bed infections are necessary [1].

## Prevention

With numbers of up to 90% of examined and questioned climbers with chronic feet conditions [6] more work needs to be done in this respect. To reduce these complaints climbers need to be advised not to wear their shoes too tight and have an additional loose fit training shoe. Also the industry producing climbing shoes must be involved, and new fitting strategies with less stretching outer materials and biomechanical adjusted constructions impaired [1]. New



**Fig. 48.3** Modern climbing shoe with anatomical last

shoes should have a inner lining to reduce bunions and callosity, while still guaranteeing a good perception of the rock [1]. To reduce the risk of wall-collision in acute injuries belay training for climbers as well as reasonable bolt and protection placement is essential [9]. To minimize ground fall injuries the use of “crash-pads” and “spotting” in bouldering is important [9]. Those movable mats should be standardized to guarantee certain impact loads, a procedure that the UIAA safety and medical commissions are currently undertaking. Other preventive measurements are the mandatory closure of gaps of the mats in climbing gyms and for competition [9].

## Evidence

In general maximal level of evidence II-III.

Level II for:	The use of tight climbing shoes leads to chronic conditions such as e.g. bunions, subungual haematoma and toe nail dystrophy
	Tight fit climbing shoes increase the incidence of a hallux valgus deformity
Grade B for:	Larger seized climbing shoes can reduce these risks.

## Summary

Lower leg injuries make up to 50% of acute climbing injuries. They mostly result from falling and hitting the wall or from ground falls. Strains and fractures to the foot are most common. Chronic climbing injuries in the feet result from the use of too small shoes which force the toes in an erect position. These result in bunions, claw toes, hematoma, nail bed infections and on the long term a hallux valgus deformity may occur.

## References

- Schöffl V, Küpper T. Feet injuries in rock climbers. *World J Orthop.* 2013;4(4):218–28.
- Schöffl V, Morrison A, Schöffl I, Küpper T. Epidemiology of injury in mountaineering, rock and iceclimbing. In: Caine D, Heggie T, editors. *Medicine and sport science – epidemiology of injury in adventure and extreme sports.* Karger; 2012. p. 17–43.
- Killian RB, Nishimoto GS, Page JC. Foot and ankle injuries related to rock climbing. The role of footwear. *J Am Podiatr Med Assoc.* 1998;88(8):365–74.
- van der Putten EP, Snijder CJ. Shoe design for prevention of injuries in sport climbing. *Appl Ergon.* 2001;32(4):379–87.
- Schöffl VR, Hochholzer T, Imhoff AB, Schoffl I. Radiographic adaptations to the stress of high-level rock climbing in junior athletes: a 5-year longitudinal study of the German junior national team and a group of recreational climbers. *Am J Sports Med.* 2007;35(1):86–92.
- Schöffl V, Winkelmann HP. [Footdeformations in sportclimbers] Fußdeformitäten bei Sportkletterern. *D Z Sportmed.* 1999;50: 73–6.
- Peters P. Orthopedic problems in sport climbing. *Wilderness Environ Med.* 2001;12(2):100–10.
- Buda R, Di Caprio F, Bedetti L, Mosca M, Giannini S. Foot overuse diseases in rock climbing: an epidemiologic study. *J Am Podiatr Med Assoc.* 2013;103(2):113–20.
- Hochholzer T, Schöffl V. *One move too many.* 2nd ed. Ebenhausen: Lochner Verlag; 2006.
- Neuhof A, Hennig FF, Schöffl I, Schöffl V. Injury risk evaluation in sport climbing. *Int J Sports Med.* 2011;32(10):794–800.
- Schöffl V, Morrison AB, Schwarz U, Schöffl I, Küpper T. Evaluation of injury and fatality risk in rock and ice climbing. *Sports Med.* 2010;40(8):657–79.

Andreas Gösele-Koppenburg

**Abstract**

In general, recreational cyclists are not at high risk for injuries when compared to other sports. Cycling injuries can be categorized in overuse injuries and acute traumatic injuries. Overuse injuries involve mostly the surface of contact between bike and human body, but also knee, neck and lower back can be affected. Acute injuries are mainly caused by accidents, often resulting in skin lesions and broken bones caused by high velocity and high impact forces. Special attention should be given to the foot because in cycling the entire energy is transferred from the foot to the bike. The most common acute foot injury in cycling is the ankle sprain. Overuse foot injuries include metatarsalgia and ingrown toenails from improper footwear and achilles tendon injuries often caused by excessive saddle height. For overuse injuries it is important to detect the etiology of the underlying mechanisms. This can be done through biomechanical bike fitting analyses or functional tests of joint mobility and strength of the involved muscle groups.

**Keywords**

Cycling • Foot problems • Skin lesions • Ankle injuries • Metatarsalgia • Plantar neuropathy • Achilles tendon lesions • Ingrowing nail

**Introduction**

The popularity of cycling has grown over the last few years. Disciplines can be distinguished, which differ remarkably according to terrain, sports equipment (bike), also the components and the sports-specific liability and effort in cycling. Therefore the stress for the musculoskeletal system and the risk of injury are very diverse and incomparable with each other.

Overall, the risk of injury in cycling is much lower compared to team sports such as soccer or handball.

We distinguish between acute injury/trauma and injuries caused by overstraining. The relation of these subdivisions varies from study to study. It depends on the discipline and also the degree of activity in each study group.

Whilst professional road racers have an almost balanced relation between acute injuries and overuse injuries [1, 2], recreational riders show a majority of up to 85 % in injuries caused by overuse. However, we mainly find injuries of the upper extremities (16 %) and the shoulder girdle (34 %) between competitive riders. Especially the collarbone has an injury incidence over 20 % [1]. In addition, competitive road racers show contusions and skin injuries caused by high-velocity and high-impact trauma. Acute foot injuries occur uncommonly (4 %).

Regarding the overuse injuries, the lower extremities (67.9 %) are mainly affected. In the foreground are knee problems (31.1 %) followed by muscle pathologies (26.4 %). The foot problems are mainly Achilles tendon disorders (9.6 %).

Amateur cyclists show a totally different pattern of problems. The issues occurring here are problems of the neck (48.80 %), the lumbo-sacral junction (30.3 %) and the knee (41.1 %). Gender and age-specific differences can be noticed [3]. Injuries of the foot are very rare in this group [4].

A. Gösele-Koppenburg, MD  
Crossklinik, Swiss Olympic Medical Center,  
Bundesstrasse 1, 4009 Basel, Switzerland  
e-mail: [a.goesele@mac.com](mailto:a.goesele@mac.com); [goesele@crossklinik.ch](mailto:goesele@crossklinik.ch)

Overuse injuries often affect the “contact points” between the rider and the bike (hands, buttocks, feet). One of the reasons for the small number of overuse injuries is certainly the fact that the load factors on the musculoskeletal system are very low in cycling.

Whilst sitting on the bike the load is about 50% of the bodyweight, it can rise up to three to four times the body weight when standing up during climbing.

In running however, we can find a lot higher loading forces (up to eightfold body weight).

## Traumatic Injuries

Foot problems in cycling are differentiated in acute traumatic injuries and overuse injuries. Acute traumas can include ankle sprains, skin lesions and bruises.

## Skin Lesions

Contusions issued by falling can cause skin injuries in the foot area. The injuries are usually limited to the ankle region as the shoe protects the foot. Most injuries are caused by a combination of pressure and friction and can affect all layers of the skin. We can't see any bleeding if just the epidermis is affected. Pure epidermal lesions are rare. Usually combinations of superficial (epidermis) and deep (dermis) lesions appear (Fig. 49.1).

Diagnostically it is important to determine the gravity and extent of skin injuries. Fractures must be excluded. The therapy depends entirely on the severity. Dirt and stones often contaminate the wounds. An extensive cleaning with water or saline is one of the most important therapeutic steps. Intensive mechanical cleaning with sponges and brushes should be avoided.



**Fig. 49.1** Superficial and deep skin lesion of the ankle after bike crash

Subsequently a non-adherent dressing should cover the lesion. Especially in the first few days after the accident the lesions should be checked every day. The tetanus protection has to be provided. After the lesions are healed up they should be looked after for several weeks. Sunscreen with UV protection is very important, as is moisturizing with certain ointments.

## Ankle Injuries

Ankle sprains are one of the most common musculoskeletal injuries. The rate of ankle sprains ranges from 15 to 20% in all sport injuries. Ankle sprains are not as common in cycling than in other sports such as soccer or basketball.

The most common injury mechanism is caused by a combination of inversion and adduction of the foot in a plantar flexion (supination). This injury mechanism can cause damage to the ankle ligaments and in rare cases to the bone (fractures). Injury of the anterior talofibular ligament with intact medial ligaments leads to anterolateral rotary instability. In addition the transection of the calcaneofibular ligament brings a tilting of the talus (talar tilt).

Ankle ligament sprains are usually graded on the basis of severity. Grade I is a slight stretching of the ligaments without macroscopic rupture or joint instability. Grade II (moderate) is a partial rupture of the ligament with moderate pain and swelling. The results are functional limitations so as a slight to moderate instability. Typically, patients present with problems in weight bearing. Grade III (severe) is a complete ligament rupture with marked pain, swelling, hematoma and pain (Fig. 49.2). Injuries of Grade III are marked by a great deal of annoyance, which is shown by instability.

Biological ligament healing can be divided into three different phases: (1) inflammatory phase (day 1 to 5 after trauma), (2) the proliferation phase (day 5–28 after trauma)



**Fig. 49.2** Typical distribution of swelling and hematoma after ankle sprain

and (3) the remodelling or maturation phase (day 28–42 or more after trauma). The duration of the different phases can vary individually [5].

Many options of treatment have been suggested: surgery, immobilization, functional treatment with bandages, tape or different braces, balance training. Today the majority of the authors recommend a non-surgical treatment for lateral ankle sprains.

Nevertheless, many studies have shown that ankle sprains are more serious than generally thought since many patients develop chronic problems after an injury. The symptoms include chronic pain, recurrent swelling, and chronic instability. We also have strong evidence that within the first year after the injury the risk of a recurrent ankle sprain is twice as high for athletes.

Overlooked associated lesions, such as syndesmosis or cartilage injuries, might explain the high rate of failure after ankle sprain treatment. Another cause for that could be an inappropriate considering the different injury grades and healing phases.

Comparing the advantages and disadvantages of surgical and non-surgical treatment you will find that the majority of lateral ankle ligament ruptures can be managed without surgery in the grades I, II and III.

This is why the decision for surgical repair should always be made on an individual basis. Types I and II injuries should be treated with a semi-rigid brace. Systematic review show that for grade III injuries, a phase adapted non-surgical treatment of acute ankle sprains with a short-term immobilization followed by a semi-rigid brace is the best treatment strategy.

Neuromuscular training should support functional rehabilitation after ankle sprain. Balance training is effective for the prevention of resprains of athletes with previous sprains. Braces are also effective for the prevention of ankle sprains in athletes.

## Overuse Injuries

Looking at chronic overuse injuries of the foot you can find problems like inflammatory changes in the achilles tendon, irritation of the skin by pressure of the shoes, nail problems and a general foot pain syndrome called metatarsalgia.

## Metatarsalgia and Plantar Neuropathy

Burning feet, undefined general mid- and forefoot pain, numbness and electrifying pain are the typical clinical pictures of plantar neuropathy. These are mostly caused by tight shoes, which leads to an excessive compression of the middle and forefoot. As a result for that pain can occur during

cycling since the foot volume increases after a while. The pain is caused by irritation/compression of the interdigital nerves (II-V).

Usually the plantar branch is affected. The outcome of the clinical examination often shows a forefoot-compression-pain and a click phenomenon called Mulder's Sign.

In addition to the clinical examination, ultrasonography and MRI are used to confirm the diagnosis. The MRI examination often shows a neural and perineural swelling with fluid accumulation. In differential diagnosis a capsulitis and bursitis have to be differentiated. A test infiltration with a local anesthetic can be used before a cycling test.

The treatment is primarily conservative. Bigger but especially wider shoes with a wide toe box are usually sufficient. Shifting back the cleat, loosening the straps and using an insole to improve the pressure distribution in the forefoot can be helpful to prevent these problems [6]. If these procedures are not sufficient, an infiltration therapy can be attempted with a steroid injection. This happens after previous infiltration with a local anesthetic. Injections with steroids should be used sparingly, since they cause a certainly useful, swelling effect on the nerve, though also interrupt the surrounding tissue sustainably in their metabolism. A surgical excision (neurolysis/neurectomy) is hardly necessary (Fig. 49.3).

## Achilles Tendon Injuries

Complaints in the area of the Achilles tendon can be divided into: Insertional- and Mid-Portion-Lesions. However, in cycling you will hardly ever find problems in the musculotendinous junction. In a longitudinal study, which lasted over 4 years 51 cyclists showed an appearance of 10%. Other studies showed a frequency of 5–8%. The main reason for



**Fig. 49.3** Surgical excision of Morton neuroma



**Fig. 49.4** Placement of markers for 3-D bike fitting

that is an incorrect seating position. Having the saddle on a too high position causes excessive plantar flexion, a too low saddle however causes an excessive dorsiflexion. This results in an increased “Ankling” which can be considered as a cause of inflammatory changes.

Rotation blockades by certain shoe cleat-pedal combinations are also accepted as local pressure from the heel of the shoe.

The rate of affected athletes is a lot higher for sport beginners than experienced athletes. “Too much too soon” is usually the reason for the problem.

Clinically we find a painful thickening of the Achilles tendon in the mid-portion or in the area of the insertion. The insertional problems are often found in addition to a bursitis under the achilles tendon (subachilles bursa). The diagnosis is made clinically and confirmed with ultrasound or MRI.

The focus of therapy contains analysing the cause of the problem which is used as the basis of a causal therapy. A correction of the seating position in the context of a professional bike fitting (Fig. 49.4) is usually the first step to a successful treatment. Currently, the combination of shock wave therapy and eccentric strength training seems to achieve the best results. Peritendinous injections are rarely used. Only in rare cases, surgery (tenolysis or bursectomy) is necessary.

### **Ingrowing Nail**

A common problem in sports medicine are ingrown toenails, especially effecting the big toe. The reason is an anatomical predisposition combined with narrow shoes or unprofessional nail care. The diagnosis is usually not a problem. The treatment is primarily conservative. Foot care, ointment dressings, disinfectants, antibiotics and local professional nail care are helpful.

## **Rehabilitation and Back-to-Sports**

The rehabilitation after foot and ankle injuries in cycling depends on several factors. In general acute traumatic injuries for example, ankle sprains are treated like in other sports by conservative treatment. Back to sports depends on the severity of injury as well as on the location and concomitant factors like age and the level of sports (recreational vs professional). Cycling is a “non contact, non weight bearing sport” with almost no eccentric forces. Therefore rehabilitation and back to sports are in general faster than in other sports especially than in contact sports. Easy training after ankle sprains is possible after 7–10 days. In case of an overuse injury a cause analysis is the most important factor. Back to sports and rehabilitation time can vary from days to months.

### **Prevention**

Acute foot and ankle trauma in cycling are not easy to prevent. Overuse injuries can be prevented by a perfect seating position, insoles and balanced mixture of training and regeneration. Flexibility, coordination and whole body fitness are the basis of success not only under sportive aspects but also under the aspect of overuse prevention.

### **Evidence**

Literature concerning foot and ankle problems in cycling is rare. Most of the studies are Level III or IV studies.

### **Summary**

- Cycling injuries can be categorized in overuse injuries and acute traumatic injuries. Recreational cyclists are not at high risk for injuries when compared to other sports.
- Overuse injuries involve mostly the surface of contact between bike and human body, but also knee, neck and lower back can be affected.
- Acute injuries are mainly caused by accidents, often resulting in skin lesions and broken bones caused by high velocity and high impact forces.
- The most common acute foot injury in cycling is the ankle sprain.
- Overuse foot injuries include metatarsalgia and ingrown toenails from improper footwear and achilles tendon injuries often caused by excessive saddle height.
- For overuse injuries it is important to detect the etiology of the underlying mechanisms. This can be done through biomechanical bike fitting analyses or functional tests of joint mobility and strength of the involved muscle groups.



## References

1. De Bernardo N, Barrios C, Vera P, Laíz C, Hadala M. Incidence and risk for traumatic and overuse injuries in top-level road cyclists. *J Sports Sci.* 2012;30(10):1047–53.
2. Silberman MR. Bicycling injuries. *Curr Sports Med Rep.* 2013;12(5):337–45.
3. Wilber CA, Holland GJ, Madison RE, Loy SF. An epidemiological analysis of overuse injuries among recreational cyclists. *Int J Sports Med.* 1995;16(3):201–6.
4. Dahlquist M, Leisz M-C, Finkelstein M. The club-level road cyclist: injury, pain, and performance. *Clin J Sport Med.* 2014;25(2):88–94.
5. Petersen W, Rembitzki IV, Gösele-Koppenburg A, Ellermann A, Liebau C, Brüggemann GP, et al. Treatment of acute ankle ligament injuries: a systematic review. *Arch Orthop Trauma Surg Springer Berlin Heidelberg.* 2013;133(8):1129–41.
6. Bousie JA, Blanch P, McPoil TG, Vicenzino B. Contoured in-shoe foot orthoses increase mid-foot plantar contact area when compared with a flat insert during cycling. *J Sci Med Sport.* 2013; 16(1):60–4.

Martin Wiewiorski and Christie-Joy Cunningham

**Abstract**

In addition to being artists, ballet dancers can be seen as highly trained professional athletes. They suffer frequent injuries to the lower extremities, particularly the foot and ankle. Unfortunately, most injuries are not treated adequately, due to poor access to treatment, rehabilitation, and the pressure from the companies to continue rehearsing and performing.

**Keywords**

Ballet • Foot • Ankle • Dance

**Introduction**

Dancers are high performance athletes suffering from great numbers of injuries due to the demanding nature of their rehearsal and performance regimens. Dancers themselves (and their companies) see themselves rather as artists, not athletes. This is one of the reasons why only few companies and theaters offer adequate medical and physiotherapeutic care. Frequent relocation of dancers due to temporary contract work and touring complicates consequent treatment attempts. Additionally, dancers feel often pressured to ignore injuries and delay treatment in the effort to stay employed by a dance company or to stay in rehearsals. Rehearsing and performing through pain is common and leads to worsening of symptoms and prolongation of recovery.

The most widely known and studied dance genre is classical ballet, with ballet dancers suffering injuries especially around the foot and ankle joints. During dancing, the ankle and first metatarsophalangeal joints are placed in maximal

dorsal and plantar flexion hundreds of times a day. Extreme range of ankle motion required of dancers, especially in classical ballet where the en-pointe and demi-pointe positions are common.

The following chapter covers the most common foot and ankle injuries and treatment in classical ballet dancers.

**Injuries****Ankle Sprain/Ankle Joint Instability**

Ankle sprains are the most common foot and ankle injury in dancers [1]. Inversion is the most common pathomechanism resulting in injury of the anterior fibular ligament complex (41%) [2]. The majority of ankle sprains (75%) occur in dancers who are 26 years of age or younger. Arendt et al. found persistent instability and repetitive ankle sprains in 75% of ballet dancers suffering from a previous ankle sprain. Recurrent sprains, pain, giving way, and swelling are common symptoms. Acute lateral ankle sprains are graded from 1 to 4 [3]. Swelling only present on examination in grade 1 (few fibers of the lateral ligaments are stretched or torn). A lateral hematoma can be found in grade 2 sprains (complete tear of the ATFL and CFL). In grade 3, lateral and medial hematoma can be found, and typically weight bearing is not possible due to pain (lateral and medial ligaments are torn).

M. Wiewiorski, MD (✉)  
Osteoarthritis Research Center, Basel, Switzerland

Department of Orthopedics and Traumatology, Kantonsspital  
Winterthur, Brauerstrasse 15, 8401 Winterthur, Switzerland  
e-mail: [wiewiorskim@gmail.com](mailto:wiewiorskim@gmail.com)

C.-J. Cunningham, BA  
Osteoarthritis Research Center, Basel, Switzerland  
e-mail: [christiecunningham0@gmail.com](mailto:christiecunningham0@gmail.com)

Stage 4 is associated with additional injuries: fractures, synovial lesions, and/or chondral/osteochondral lesions.

The goal of treatment after an acute ankle sprain is to reduce the risk of recurring ankle sprains, and chronic ankle instability. Conservative treatment is the management of choice for grade 1–3 ankle sprains in all dancers. Early symptomatic treatment aims to reduce pain and swelling (Protection, Rest, Ice, Compression, Elevation [PRICE]), and should be followed by a good stability protection (brace or walker depending on the grade of sprain), early Range of motion (ROM) exercises, strengthening, proprioception, and activity-specific training under physiotherapeutic supervision. The time taken for a return to rehearsing and performing varies from 1 to 6 weeks. Proprioception training and peroneal strengthening must be initiated early in the rehabilitation of ankle sprains, with emphasis on attaining full mobility of the subtalar and transverse tarsal joints and the ankle joint [4]. Additional immobilization measures like taping can be helpful. However, limiting the ROM with taping, also for aesthetic reasons, is usually not accepted by most dancers. Dancers who had sustained an prior ankle sprain have altered sensorimotor control compared with those without prior injury, despite having returned to full-time performing and completing proprioception training [5].

Surgical treatment is recommended for grade 4 ankle sprain. Our preferred method for ligament reconstruction is direct anatomic ligament reinsertion with a modified Brostrom/Gould procedure [6]. Arendt et al. report about excellent outcome after ligament reconstruction in chronic ankle instability in two dancers [7].

### Anterior Ankle Impingement Syndrome

Dancers suffering from anterior ankle impingement typically complain about reduced dorsal flexion of the ankle joint limiting their ability to perform a demi-plié (dancer bends the knees with the hips turned-out), and pain on landings. Pain of the anterior ankle joint line can be provoked on clinical examination by palpation or forced dorsal extension [4]. ROM is typically limited in comparison of the healthy side. Conventional radiographs show osteophyte formation at the tibial and talar neck on weight bearing lateral radiographs. Conservative treatment fails due to the mechanical cause of the symptoms. The osteophytes are frequently found anteromedially and can be debrided through a mini-open incision or arthroscopically [8].

### Posterior Impingement Syndrome

Ballet dancers perform repetitive extreme ankle flexion during demi-pointe and en-pointe position. This can lead to com-

pression of the posterior structures of the ankle joint, and can result in pain [9]. The most common cause of posterior impingement are an os trigonum, or a protruding lateral process of the talus (Stieda process) [10]. The reported incidence of os trigonum varies from 7 to 24%. Dancers complain about loss of ROM and pain at the posterolateral part of the ankle joint. Pain can increase during exercise. Demi-pointe and en-pointe positions should be performed on clinical examination, to determine whether pain is triggered when the dancer rises onto the ball of the foot (relevé). Radiological diagnosis includes standard weightbearing lateral radiographs of the whole foot and mortise view. A supplemental radiograph in demi-pointe position can be performed. Additional MRI can help assessing additional soft tissue pathologies.

Another cause of posterior ankle pain can be tendinitis of the flexor hallucis longus (FHL) tendon. The tendon of it is at risk of impingement because it travels in the groove between the medial and lateral posterior talar tubercle. The maneuver of relevé can predispose the FDL tendon for tendinitis. A biomechanical study demonstrated that the muscles crossing the metatarsophalangeal joints work 2.5–3 times harder than those crossing just the ankle joint in dancers rising on to the full pointe position, placing these muscles and tendons (FHL, flexor digitorum longus [FDL]) at risk for overuse injuries. Symptoms in the FHL and the os trigonum are commonly reported in combination in ballet dancers.

Conservative treatment of posterior ankle impingement should include exercises to strengthen the deep muscles of the leg with the purpose of decreasing the action of the gastrocnemius muscles. Surgical treatment is recommended after failure of conservative treatment in cases of recurrent/persisting symptoms accompanied by reduced plantarflexion.

Albiseti et al. evaluated the medical records of 186 trainee ballet dancers [11]. Of those, 12 suffered posterior ankle impingement, and only three underwent surgical excision of an os trigonum after failed conservative treatment of 6 months. Hamilton et al. reviewed the outcome of open debridement in 37 dancer's (41 ankles) with posterior impingement syndrome and/or FHL-tendinitis. Of those 31 ankles had a good or excellent result [12]. Willits et al. performed posterior ankle arthroscopy for symptomatic os trigonum (11 ankles) and FHL tendinitis (5 ankles) and found a good outcome with a mean AOFAS score of 91 after a mean followup of 32 months (range, 6–74) [13]. No significant complications were encountered.

### Achilles Tendinitis

Achillodynia may be seen in male and female dancers, with potential causes being Achilles tendinitis (acute overuse,

systemic reasons, and chronic degeneration), midportion tendinopathy (degeneration), insertion tendinopathy (with possible bony spurs, through overuse), malalignment of the ankle joint, or Haglund's deformity. Failure of the dancer to land with his or her heels on the ground from jumps also can contribute to shortening of the Achilles tendon and risk for injury [4]. Most tendinopathies can initially be treated conservatively with excentric exercises, therapeutic ultrasound, adaptation of training intensity, choice of shoes, PRP infiltrations, and other [14]. Adjustment to shoes can be made, like less tight ribbons or use of elastic sewn over the Achilles tendon. Acute ruptures can happen upon landing with hyperdorsiflexed ankle joint, or eccentric loading of the foot during push-off. Surgical repair is mandatory in cases of Achilles tendon ruptures.

### Metatarsal Stress Fractures

The unique anatomic configuration of the second tarsometatarsal joint combined with major stresses at the base of second metatarsal in pointe position can result in stress fractures. Those are more common in ballerinas, then male dancers or other athletes. Biomechanical experiments have shown that both the pointe shoe and Lisfranc ligaments are important stabilizers of the Lisfrancs joint [15].

Dancers usually complain of pain creeping in over several days or weeks, which initially, occurs only during activity and resolves on resting [16]. If dancing is continued, the pain can become chronic after 7–20 days [17]. Stress fracture at the base of the second metatarsal has been recognized as a difficult injury because of delayed diagnosis [18]. Clinical examination is often inconclusive due to minimal swelling and ill-defined pain. Conventional radiographs show usually no apparent changes. MRI should be considered early on suspicion and is the gold standard in confirming the diagnosis [7]. Nutritional history, training program, type of shoes and dancing surface, recent trauma and menstrual patterns should be assessed [16]. Conservative treatment consist of rest (3–5 weeks) without immobilization of the foot, and stretching exercises and isometric exertion without weight bearing. Gradual return to full dance activity is recommended. Albisetti et al. treated 19 dancers with metatarsal stress fractures with a period of rest and external shock-wave therapy [16]. At 2.2 years mean follow-up (range 1.3–3.3 years) all dancers healed without any midfoot pain. O'Malley et al. reported about treatment of 51 ballet dancers with 64 metatarsal stress fractures. Conservative treatment consisted of rest a short leg walking cast for six patients, and a wooden shoe and symptomatic treatment for the remainder of the dancers. Dancers returned to performance at an average of 6.2 weeks following diagnosis.

### Hallux Valgus

Hallux valgus is found more frequently in female (86 %) than male (33 %) ballet dancers. Some authors assume that hallux valgus deformity is promoted by the incorrect placement of the foot during en-point position. Some dancers stand incorrectly more on the first toe during en-point (rolling the foot). Ideally the pressure should be distributed evenly between the first to third toes. Other factors for hallux valgus are a relatively longer first ray (Egyptian foot) and increased forefoot pressure with consecutive splayfoot. Though commonly assumed, that hallux valgus is more common among the dance community, a study by Einarsdottir et al. could not show a higher frequency than in non-dancers. Hallux valgus in ballet dancers rarely needs surgical treatment and should be managed conservatively. Padding over the prominent medial MTP joint, toe spacers, and intrinsic muscle strengthening exercises can help to make dancing on pointe more comfortable for those dancers with bunions [19]. Fitting of the pointe shoe with a higher vamp to better support the hallux MTP joint may reduce symptoms. Surgical correction may lead to loss of ROM at the metatarsophalangeal joint, therefore surgery is recommended only after retirement from the dance career [4].

### Osteoarthritis

In dancing, the foot and ankle joints are placed in maximal dorsal and plantar flexion hundreds of times a day. The cause of osteoarthritis is probably an accumulation of microtraumas. Van Dijk et al. examined 19 former professional female dancers aged 50–70 years and found a statistically significant increase in osteoarthritis of the ankle, subtalar, and first metatarsophalangeal joints in the ballet group compared with a control group [20]. Interestingly, all former dancers who showed signs of degenerative joint disease on conventional radiographs were asymptomatic. Ambré et al. examined 20 ballet dancer with a mean age of 28 years, who were dancing since their childhood [21]. No significant changes in the ROM of the first metatarsal-phalangeal joint was found in comparison with a control group of non-dancers. Radiographs showed only slight signs of degeneration, which didn't correlate with clinical symptoms. Anderson et al. examined 44 retired ballet dancers [22]. Of those 24 dancers (54%) had radiologic signs of osteoarthritis in the first metatarsal-phalangeal joint: all were asymptomatic. Brodelius examined a group of ballet dancers (13 females and 3 males) were age 18–39 years, dancing for 3–30 years [23]. All but two dancers showed osteoarthritis of the ankle joint (not radiologically classified, no data about clinical symptoms).

In conclusion, the presence of radiological signs of osteoarthritis in the foot and ankle joints in ballet dancers has probably no relevance to clinical practice.

## Etiology and Pathomechanism

The exaggerated external rotation of the hips (turn out) from the hips is needed for correct position in classical ballet. Lack of flexibility and external rotation can be compensated by the knee and ankle/foot joint. This can cause major strain and increase the risk for instability, tendinitis, and early joint degeneration [2]. Other factors have been claimed to predispose dancers to lower leg injuries: performing on a non-sprung hard floor, a cold studio or theater, or dancing without sufficient warm up. Overwork and poor occupational health and safety conditions increase risk of injury. A survey among dancers showed that dancers think that their injuries are due to feeling overtired, run down, overworked, and under strain and pressure [24]. Other perceived causes were dancing on unsuitable stages and flooring; dancing in cold, draughty environments and being insufficiently warmed up, the demands of difficult choreography, and the continual repetition of difficult movements' during rehearsals [24].

## Epidemiology

Overall, dancers have a high incidence and prevalence of injuries [25]. A survey of injuries to 141 dancers commissioned by the British National Organisation of Dance and Mime showed that 67 (47%) had experienced a chronic injury and 59 (42%) an injury in the previous 6 months that had affected their dancing [24]. Of the injuries occurring in the previous 6 months, 19 (32%) happened during a performance, 16 (28%) during rehearsals, and nine (16%) during classes. Dancers describe discomfort in the lumbar spine (88%), the knee (80.5%), and the ankle (74%) [2]. The foot and ankle joints are the location of 46% injuries in men and 62% in women. In men 40% of injuries were traumatic, such as distortions; in women 35% were traumatic and 65% were from overuse, with a subacute onset [1].

## Therapy (On the Field, Conservative, Surgical)

For specific injury therapy, please see injuries subsections.

## Prevention

Teachers and choreographers should be more aware of a dancer's limitations and of dancers' needs to rest as soon as injuries occur; and dancers need immediate access to adequate treatment [24]. Many ballet companies in the United

**Table 50.1** Overview of relevant publications on foot/ankle injuries in ballet dance

Author	Topic	Level of evidence
Bronner et al. [27]	Comprehensive case management reduces injury incidence and time loss	2
Nilsson et al. [1]	Prospective assessment of injuries in a single ballet company over 5 years	4
Kadel et al. [4]	Review of foot/ankle injuries in dance	4
Albisetti et al. [11]	Treatment of posterior impingement in dancers	4
Albisetti et al. [16]	Metatarsal stress fractures in dancers	4

States have instituted in-house medical and therapy services to reduce the physical and financial impact of injuries on the dancer and company. In one ballet company of 70 dancers, this resulted in a decrease of annual injuries from 94 to 75% and savings in excess of \$1.2 million over a 5-year period [26]. Bronner et al. suggested a comprehensive management program in order to decrease the amount of workers compensation cases and days lost from work. This included primary prevention (dance-specific annual screenings, technique modification, cross-training, and treatment of minor complaints and secondary prevention (triage to determine the need for medical referral, treatment, and rehabilitation after injury) [27]. Additional services like medical coverage and on-site physical therapy intervention during rehearsal periods and before performances were provided. Through this management program, the annual number of new workers' compensation cases was dramatically reduced from a 81 to 17%, and the number of days lost from work decreased by 60%.

## Evidence (Description of Highest Evidence with Mentioning the Level and Grade of Evidence)

For literature and evidence see Table 50.1.

## Summary

Five most important points in one to two sentences each.

Dancers are high performance athletes and artist, who are at high risk for acute and overuse foot and ankle joint injury. Most of the injuries can be treated conservatively, the indication for surgical treatment is rare. Delayed diagnosis, and poor access to medical and physiotherapeutical treatment extend the recovery time unnecessarily. A comprehensive case management service provided by companies would be favorable to reduce the number of new injuries, and fasten return to rehearsal and performing.

## References

1. Nilsson C, et al. The injury panorama in a Swedish professional ballet company. *Knee Surg Sports Traumatol Arthrosc.* 2001;9(4):242–6.
2. Arendt Y, Kerschbaumer F. Injury and overuse pattern in professional ballet dancers. *Zeitschrift für Orthopädie und ihre Grenzgebiete.* 2002;141(3):349–56.
3. Valderrabano V. Akute und chronische OSG-Instabilität. GOTS Expertenmeeting 2012 Sprunggelenksinstabilität. 2012; p. 43–55.
4. Kadel NJ. Foot and ankle injuries in dance. *Phys Med Rehabil Clin N Am.* 2006;17(4):813–26.
5. Hiller CE, Refshauge KM, Beard DJ. Sensorimotor control is impaired in dancers with functional ankle instability. *Am J Sports Med.* 2004;32(1):216–23.
6. Gould N, Seligson D, Gassman J. Early and late repair of lateral ligament of the ankle. *Foot Ankle.* 1980;1(2):84–9.
7. Arendt EA, Griffiths HJ. The use of MR imaging in the assessment and clinical management of stress reactions of bone in high-performance athletes. *Clin Sports Med.* 1997;16(2):291–306.
8. Jerosch J, et al. Arthroscopic treatment of anterior synovitis of the ankle in athletes. *Knee Surg Sports Traumatol Arthrosc.* 1994;2(3):176–81.
9. Marotta JJ, Micheli LJ. Os trigonum impingement in dancers. *Am J Sports Med.* 1992;20(5):533–6.
10. Russell JA, et al. Pathoanatomy of posterior ankle impingement in ballet dancers. *Clin Anat.* 2010;23(6):613–21.
11. Albisetti W, et al. Clinical evaluation and treatment of posterior impingement in dancers. *Am J Phys Med Rehabil.* 2009;88(5):349–54.
12. Hamilton WG, Geppert MJ, Thompson FM. Pain in the posterior aspect of the ankle in dancers. Differential diagnosis and operative treatment. *J Bone Joint Surg Am.* 1996;78(10):1491–500.
13. Willits K, et al. Outcome of posterior ankle arthroscopy for hind-foot impingement. *Arthroscopy.* 2008;24(2):196–202.
14. Hall MP, et al. Platelet-rich plasma: current concepts and application in sports medicine. *J Am Acad Orthop Surg.* 2009;17(10):602–8.
15. Kadel N, et al. Stability of Lisfranc joints in ballet pointe position. *Foot Ankle Int.* 2005;26(5):394–400.
16. Albisetti W, et al. Stress fractures of the base of the metatarsal bones in young trainee ballet dancers. *Int Orthop.* 2010;34(1):51–5.
17. Morris G, Nix K, Goldman FD. Fracture of the second metatarsal base. An overlooked cause of chronic midfoot pain. *J Am Podiatr Med Assoc.* 2003;93(1):6–10.
18. Harrington T, Crichton KJ, Anderson IF. Overuse ballet injury of the base of the second metatarsal. A diagnostic problem. *Am J Sports Med.* 1993;21(4):591–8.
19. Hamilton WG. Foot and ankle injuries in dancers. *Clin Sports Med.* 1988;7(1):143–73.
20. van Dijk CN, et al. Degenerative joint disease in female ballet dancers. *Am J Sports Med.* 1995;23(3):295–300.
21. Ambré T, Nilsson BE. Degenerative changes in the first metatarsophalangeal joint of ballet dancers. *Acta Orthop.* 1978;49(3):317–9.
22. Andersson S, et al. Degenerative joint disease in ballet dancers. *Clin Orthop Relat Res.* 1989;238:233–6.
23. Brodelius Å. Osteoarthritis of the talar joints in footballers and ballet dancers. *Acta Orthop.* 1961;30(1–4):309–14.
24. Bowling A. Injuries to dancers: prevalence, treatment, and perceptions of causes. *BMJ.* 1989;298(6675):731–4.
25. Leanderson C, et al. Musculoskeletal injuries in young ballet dancers. *Knee Surg Sports Traumatol Arthrosc.* 2011;19(9):1531–5.
26. Solomon R, et al. The “cost” of injuries in a professional ballet company. *Med Probl Perform Art.* 1995;10:3–10.
27. Bronner S, Ojofeitimi S, Rose D. Injuries in a modern dance company: effect of comprehensive management on injury incidence and time loss. *Am J Sports Med.* 2003;31(3):365–73.

Monika Horisberger, Martin Wiewiorski, and Alexej Barg

**Abstract**

Statistically, riding is a dangerous sport despite many prevention campaigns and improved safety gear. This is – and always will be – because equitation is about the interaction between a human and an unpredictable large animal. Most injuries sustained in equitation are of minor severity and most of them probably do not lead to a doctor's visit. However, those that are admitted to hospital are severe and have a poor outcome. Concerning specific foot injuries in equitation, literature is limited to just a few case reports. The most common foot injuries are soft tissue injuries, such as bruises and contusions from horses standing on the feet or kicks with hoofs. Injuries to ligaments and bones can result from falls from the horse or due to trapping the foot under the horses' body in falls with the horse.

**Keywords**

Equitation • Horse riding • Safety stirrup • Midfoot • Lisfranc • Concussion • Bruise

**Injuries**

Around 3 million people in Great Britain and about 30 million Americans are active riders in various disciplines such as dressage, show jumping, eventing, western riding, racing or polo [1, 2] (Fig. 51.1). Especially girls and young women are very much involved in equitation. However, riding is a dangerous sport despite many prevention campaigns and improved safety gear. This is – and always will be – because equitation is about the interaction between a human and an animal that weights up to 500 kg, kicks with a force of more than 1 ton [3] and can run at a speed of up to 65 km/h [4].

---

M. Horisberger, MD (✉)

Orthopaedic Department, University Hospital Basel,  
Basel, Switzerland

e-mail: [Monika.Horisberger@usb.ch](mailto:Monika.Horisberger@usb.ch)

M. Wiewiorski, MD

Department of Orthopedics and Traumatology, Kantonsspital  
Winterthur, Brauerstrasse 15, 8401 Winterthur, Switzerland

e-mail: [martin.wiewiorski@ksw.ch](mailto:martin.wiewiorski@ksw.ch)

A. Barg, MD

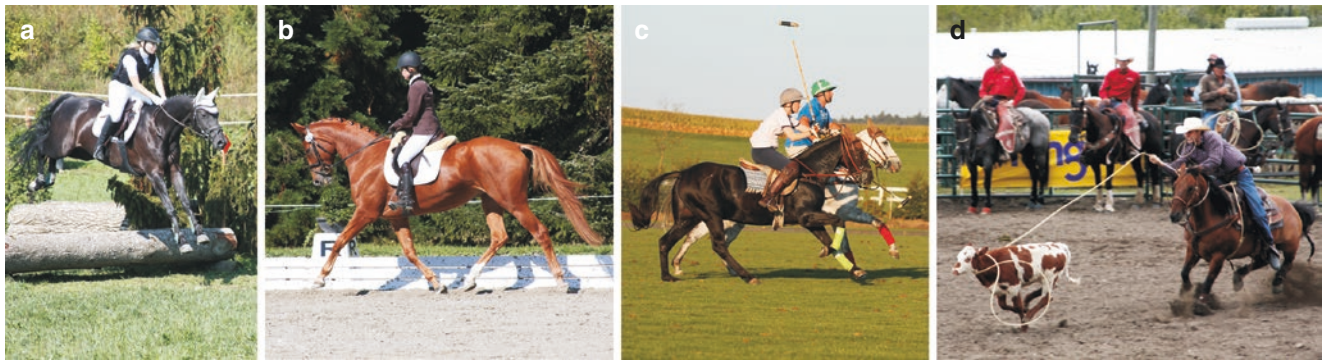
Department of Orthopaedics, University of Utah,  
590 Wakara Way, Salt Lake City, UT 84108, USA

e-mail: [alexej.barg@hsc.utah.edu](mailto:alexej.barg@hsc.utah.edu)

When sitting on a horse the riders head is at a height of up to 3 m [4]. Horses are sometimes unpredictable and – despite a centuries-long domestication – will always follow its basic instincts of a gregarious animal which flies in case of danger. Most accidents in riding are related to the particular behaviour of horses and possible mis-assessment or misunderstanding of such behaviour by the rider.

Most injuries sustained in equitation are of minor severity and most of them probably do not lead to a doctor's visit [5]. However, those that are admitted to hospital are severe and have a poor outcome. Equitation has the highest mortality of all sports [6]. Papachristos et al. for example retrospectively analysed 172 patients that were seen in an ER department within 5 years. They report on 48% head injuries, 24% admittance to ICU and 18% need for mechanical ventilation. Even after 6 months, 35% of the patients suffered of severe pain and disability and half of the patients in the cohort took more than 6 months to get back to work [7].

Most published studies of equestrian injuries report acute injuries only [5]. Very few papers take “overuse injuries” into account but do not specify the type of lesion more precise. Ekberg et al. report on overuse injuries in eventing, which concern mostly the back and the knees [8]. A paper on



**Fig. 51.1** Equestrian disciplines. Equestrian includes a wide variety of disciplines, such as (a) eventing/show jumping, (b) dressage, (c) polo, (d) western riding

polo injuries report overuse injuries such as hip adductor tendinitis and rotator cuff problems. However, this seems not to be specific to equestrian but rather related to the one-handed throwing movement which is necessary to push the ball forward while steering the horse [9]. There are no studies reporting on overuse injuries related to the foot and ankle.

The number and quality of studies on injuries in equestrian are poor. Most studies report on statistical data of insurances and case numbers from emergency rooms [10–14]. Concerning specific foot injuries in equestrian, literature is limited to just a few case reports.

## Etiology and Pathomechanism

There is no typical pathomechanism of foot injuries in equestrian. It rather is important to assess the circumstances that led to the injury: injuries sustained due to a fall from the horse, fall with the horse or while getting entangled in parts of obstacles or the stirrups lead to entirely different types of lesions than injuries sustained while handling a horse from the ground.

The most common foot injuries are soft tissue injuries [15], such as bruises and contusions or minor fractures from horses standing on the feet or kicks with hoofs (Figs. 51.2 and 51.3). Typically, such injuries are not admitted to hospital and may be under-represented in published studies [5].

Apart from that, injuries to ligaments and bones can result from falls from the horse or due to trapping the foot under the horses' body in falls with the horse (Fig. 51.4.). In such falls, injury often is the result of forced abduction of the mid- and forefoot, often in combination with an axial force when the foot gets caught under the horses' body because the stirrups act as a fulcrum [16]. Depending on the exact mechanism of fall, fractures with very variable characteristics can be found. The resulting injury depends on the exact point of force transmission and leads to nutcracker type of fractures of the cuboid bone, severe lesions of lisfranc joint line with dislocation or



**Fig. 51.2** Soft tissue injury. A horse jumped on the dorsum of the foot in this 25 years old female patient. Despite the patient was wearing appropriate riding boots severe soft tissue concussion with gross swelling was the result. Fractures have been excluded by radiography. Conservative treatment (RICE) has been applied. The rider was back to training within 2 weeks

multiple fractures of the metatarsals [16]. Ceroni et al. present four nutcracker type fractures of the cuboid in young riders and observed that in all cases they were associated with other severe fractures of the midfoot [16].



## Epidemiology

There is little detailed information about the general demographics of equestrian injuries. This might on one hand be because many minor injuries do not lead to hospital admission [17]. Studies therefore may have an inclusion bias. On the other hand, most of papers existing lack clear information about exposure [5]. Moreover, there seem to be substantial differences in reported incidences and injury patterns among countries, regions and equestrian disciplines being studies [18].

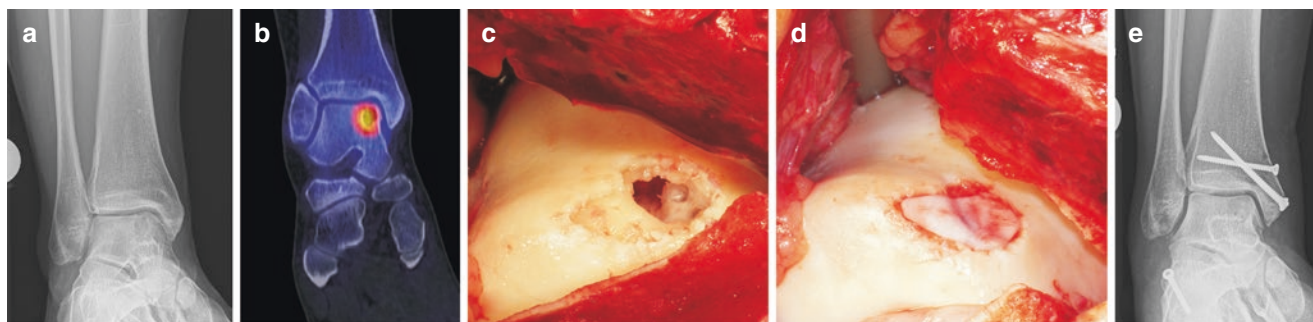


**Fig. 51.3** Phalangeal fracture. A shod horse was standing on this patient's great toe. X-ray revealed multi fragment fracture of the distal phalanx (a, ap, b, oblique view). Conservative treatment with a stiff sole for 6 weeks was successful

Several studies have shown that a higher degree of experience in riding might lead to a lower injury rate [19]. However, a study by Papachristos et al. has shown that the rate of injuries was lower in more experienced riders but that injury severity was higher [7] and Kiss et al, have also shown that increasing riding experience leads to more severe accidents [14]. However, since these study do not state an exposure time, increased amount of hours spent in equitation in more experienced as well as involvement in different disciplines of riding might bias these results.

In a large German study with advanced English style riders (dressage, show jumping and eventing) it was shown that more than 50% of all accidents happened while handling a horse from the ground and not while riding. About 6% of these injuries are related to the foot. In a study by Kiss et al. on children involved in equitation, only 23.2% of injuries occurred while handling a horse and 76.8% resulted from falls [14]. This difference might be the result of different level of riding experience and involvement in handling horses. In the Kiss et al. study 12% (injuries when riding a horse) and 15% (injuries due to handling a horse), respectively, affected the foot and ankle. However, the type of foot injury was not specified. In a study on injuries in professional riders in Japan breeding farms it was shown that 18.9% of fractures were at the foot and 92% of cases happened by being kicked by a horse [15]. In a study focusing on riding injuries, the percentage of fractures around the foot was 14.5% of all fractures [20]. Injuries sustained while handling a horse from the ground are usually less severe than those resulting from falls, but concern the foot in a higher percentage [14, 21, 22]. In a study on jockey injuries, 10.7% of all injuries affected the foot. The majority of those injuries happened during the start of the race and were the result of the jockeys getting crushed in the crowd [23].

The majority of injuries occur during leisure riding rather than in competition [24].



**Fig. 51.4** Ankle sprain and osteochondral lesion. Thirty-five years old female rider which got entangled in the stirrup when falling from the horse. Initial swelling, hematoma around the medial malleolus and tenderness was treated conservatively. She complained about persistent pain when moving her ankle and when weight-bearing. Conventional

x-ray showed suspicion of traumatic osteochondral lesion (a) which has been proven by SPECT-CT (b). Consecutive AMIC-procedure through a medial malleolar osteotomy has been performed. Intraoperatively, the lesion could be clearly seen and debrided (c) and was filled up with AMIC-plasty over autograft spongiosa (d). Postoperative ap x-ray (e)

## Therapy

Injuries in equitation do not follow a stereotype pattern because many different mechanisms can be involved. Therefore, each foot injury needs to be evaluated carefully. In minor trauma general treatment rules for bruises (RICE: Rest, Ice, Compression, Elevation) can be applied and riders mostly are back in the saddle within a couple of days. However, given the large forces involved in riding accidents a high level of suspicion for more severe soft tissue lesions as well as bone/joint injuries is mandatory. Any suspicion should lead to further imaging diagnostics (CT-Scan, MRI, ultrasound). In severe midfoot injuries, many fractures are only detected in CT scan [16]. Moreover, soft tissues need careful management and compartment syndrome of the foot has to be considered in severe concussion.

Then, an individualized conservative or operative treatment is warranted in order to restore anatomical position of joints, reduction of fractures and, reconstruct soft tissue lesions in order to avoid long-term sequela in these mostly young patients.

---

## Rehabilitation and Back-to-Sports

Working with horses always implies to take the typical behavior of horses into account. Anytime, an unforeseeable reaction of a horse can ask for full attention of the rider and all his mental and physical strength and flexibility to handle a situation. Therefore, there is no “slow return” to equitation after an injury. Lesions need to heal completely, basic fitness needs to be regained in a patient-centered rehab program without the horse. Only then, patients should return to work with horses. Taking the particular character of each horse into consideration, it might be worth re-starting on horses that are rather easy to handle. Professional riders should stop working with horses for an adequate time period – there is no such thing as partial working ability around horses.

---

## Prevention

There are no published data on injury prevention or risk factor analysis that have been subjected to formal analysis [5]. Despite many prevention programmes, lethal injuries in equitation still account for up to 2–2.5 % of hospital admissions [7, 25]. According to literature, 25 % of all sports accidents with consecutive death happen in equitation [12, 25, 26].

Of the non-life threatening injuries, foot and ankle lesions are a substantial percentage and have severe long-term sequela influencing working ability and life quality. Moreover, getting entangled in the stirrups while falling is

an important injury mechanism not just for direct lesions to the foot but also for related even more severe lesions of inner organs and the brain. Therefore, the foot is of a two-fold interest in prevention: first, foot injuries should be the aim of prevention campaigns themselves because they are frequent and can be avoided by awareness in many cases. Secondly, the foot as a connection between the rider and the saddle and therewith the horse should be the target for preventive measures which aim to reduce overall morbidity and mortality.

In several studies for the United States, Canada and Australia between 38 and 63 % of injured retrospectively stated their accident would have been preventable by sticking to general security measures [7, 27, 28]. Unfortunately, in a German study, only one third of injured riders changed their security behavior after an accident [21]. There is a study from 1998 stating that 65 % of riders never used a safety stirrup and 20 % of riders never wore riding boots [21].

Therefore, primary prevention strategies must focus on education about the special behaviour of horses and security measures must be deduced from this knowledge [14, 26]. As almost no other sports, equitation is a team sport involving two species. Therefore, injury patterns are never the result of an isolated act of one involved being, but always the result of the interaction between the rider and his horse.

Empirically, many injuries in equitation – especially those that happen while handling a horse from the ground [16] – can be avoided by wearing security gear, i.e. stable shoes with adequate toe protection [1, 18, 29] and careful analysis of possibly dangerous situations before they even occur [30–32].

For safety stirrups which are sold for many years already, there is no clinical study which would prove that either or the other design really reduces the number of falls with entangling the foot in stirrups or the amount/severity of foot injuries or general injury severity [5, 29, 33–36] (Fig. 51.5).

The same is true for riding boots and riding shoes. So far, there are no studies about mechanical properties of riding shoes and boots. When looking at the complex midfoot injuries that occur in falls from or with the horse, one can guess that shoes with a better resistance against axial and longitudinal forces across the midfoot area might help to reduce the severity of resultant injuries [16]. A heel is important to prevent the rider’s foot from slipping through the stirrup and being dragged behind the horse [18, 29].

---

## Evidence

The number and quality of studies on injuries in equitation are poor. Most studies report on statistical data of insurances or case numbers from emergency rooms and do not specify the type of foot and ankle injuries. Concerning specific foot



**Fig. 51.5** Safety stirrup designs. Several types of safety stirrups are on the market. They all aim to immediately release the foot in case of a fall. (a) Standard stainless steel stirrup. If the foot gets too deep inside the stirrup, the foot cannot be released in a fall. (b) Toe basket which

avoid that the foot gets too deep into the stirrups and would not slip out in case of a fall. (c) Deformable stirrup due to several mechanical joints that are integrated into the sides of the stirrups

injuries in equitation, literature is limited to just a few case reports.

- Kiss K. et al. Analysis of horse-related injuries in children. *Pediatr Surg Int*. 24, 1165–1169. 2008. Level IV
- Iba K. et al. Horse-related injuries in a thoroughbred stabling area in Japan. *Arch Orthop Trauma Surg*. 121, 501–504. 2001. Level IV
- Moss PS et al. A changing pattern of injuries to horse riders. *Emerg. Med. J*. 19, 412–414. 2002. Level IV
- Heitkamp H-C et al. Reitverletzungen und Verletzungen beim Umgang mit Pferden bei erfahrenen Reitern. *Unfallchirurg* 101, 122–128. 1998. Level IV
- Thomas KE et al. Non-fatal horse related injuries treated in emergency departments in the United States, 2001–2003. *Br. J.Sports Med* 40, 619–626. 2006. Level IV
- Ceroni D et al. The Importance of Proper Shoe Gear and Safety Stirrups in the Prevention of Equestrian Foot Injuries. *J Foot Ankle Surg* 46, 32–39. 2007. Level IV

## Summary

Riding is a dangerous sport despite many prevention campaigns and improved safety gear. The most severe injuries are related to the head, back and chest. Injuries to the foot do not have such a devastating impact, however, injuries to the foot and ankle are very common in equitation. On one hand, they result from the horse standing or jumping on the foot of the rider while handling it from the ground. On the other hand, the foot is at risk when riders are falling from or with the horse and the foot might get entangled in the stirrups. Here, the stirrup acts as a fulcrum and typically, severe lesions to the midfoot, i.e. nutcracker type fractures of the cuboid and lisfranc fractures are found.

All injuries to the foot and ankle need careful soft tissue assessment and necessitate – because of the high forces involved – a high index of suspicion for compartment syndrome. Fractures around the midfoot might not be obvious in conventional x-ray and should be searched for by CT scan if in doubt. Given the variable aspect of injuries around the foot and ankle in equitation, an individualized treatment plan needs to be set up. This might include conservative or operative measures.

Prevention should focus on the wear of appropriate shoe gear as well as the use of safety stirrups. However, no formal studies on the efficacy of such safety gear have been performed so far.

## References

1. Chitnavis J, Gibbons C, Hirigoyen M, Lloyd Parry J, Simpson A. Accidents with horses: what has changed in 20 years? *Injury*. 1996;27:103–5.
2. Hobbs G, Yealy D, Rivas J. Equestrian injuries: a 5-year review. *J Emerg Med*. 1994;12:143–5.
3. Nelson D, Bixby-Hammett D. Equestrian injuries in children and young adults. *Am J Dis Child*. 1992;146:611–4.
4. Sorli J. Equestrian injuries: a five year review of hospital admissions in British Columbia, Canada. *BMJ*. 2000;6:59.
5. McCrory P, Turner M. Equestrian injuries. *Med Sport Sci*. 2005;48:8–17.
6. Paix B. Rider injury rates and emergency medical services at equestrian events. *Br J Sports Med*. 1999;33:46–8.
7. Papachristos A, Edwards E, Dowrick A, Gosling C. A description of the severity of equestrian-related injuries (ERIs) using clinical parameters and patient-reported outcomes. *Injury*. 2014;45(9):1484–7.
8. Ekberg J, Timpka T, Ramel H, Valter L. Injury rates and risk-factors associated with eventing: a total cohort study of injury events among adult Swedish eventing athletes. *Med Sci Sports Int J Inj Contr Saf Promot*. 2011;18:261.
9. Costa-Paz M, Aponte-Tinao L, Muscolo D. Injuries to polo riders: a prospective evaluation. *Br J Sports Med*. 1999;33:329–31.

10. Blümel J, Pfeifer G. Unfälle durch den Umgang mit Pferden und ihre Auswirkungen im Bereich des Gesichtsschädels. *Unfallheilkunde*. 1977;80:27–30.
11. Dittmer H, Wübbena J. Eine Analyse von 367 Reiterunfällen. *Unfallheilkunde*. 1977;80:21–6.
12. Giebel G, Braun K, Mittelmeier W. Unfälle beim Pferdesport. Unfallhergang, Verletzungen und Prävention. *Hefte Z Unfallchir*. 1994;244:151–5.
13. Steinbrück K. Wirbelsäulenverletzungen beim Reiten. *Unfallheilkunde*. 1980;83:366–72.
14. Kiss K, Swatek P, Swatek P, Lénárt I, Mayr J, Schmidt B, et al. Analysis of horse-related injuries in children. *Pediatr Surg Int*. 2008;24:1165–9.
15. Iba K, Wada T, Kawaguchi S, Fujisaki T, Yamashita T, Ishii S. Horse-related injuries in a thoroughbred stabling area in Japan. *Arch Orthop Trauma Surg*. 2001;121:501–4.
16. Ceroni D, De Rosa V, De Coulon G, Kaelin A. The importance of proper shoe gear and safety stirrups in the prevention of equestrian foot injuries. *J Foot Ankle Surg*. 2007;46(1):32–9.
17. Bixby-Hammett D, Brooks W. Common injuries in horseback riding: a review. *Sports Med*. 1990;9:36–47.
18. Havlik H. Equestrian sport-related injuries: a review of current literature. *Curr Sports Med Rep*. 2010;9:299–302.
19. Mayberry J, Pearson T, Wiger K, et al. Equestrian injury prevention efforts need more attention to novice riders. *J Trauma*. 2007;62:735–9.
20. Moss PS, Wan A, Whitlock MR. A changing pattern of injuries to horse riders. *Emerg Med J*. 2002;19:412–4.
21. Heitkamp H-C, Horstmann T, Hillgeris D. Reitverletzungen und Verletzungen beim Umgang mit Pferden bei erfahrenen Reitern. *Unfallchirurg*. 1998;101:122–8.
22. Thomas KE, Annett JL, Gilchrist J, Bixby-Hammett DM. Non-fatal horse related injuries treated in emergency departments in the United States, 2001–2003. *Br J Sports Med*. 2006;40:619–26.
23. Waller A, Daniels J, Weaver N, Robinson P. Jockey injuries in the United States. *JAMA*. 2000;283:1326–8.
24. Whitlock M, Whitlock J, Johnston B. Equestrian injuries: a comparison of professional and amateur injuries in Berkshire. *Br J Sports Med*. 1987;21:25–6.
25. Kricke E. Der tödliche Reitunfall. *Unfallheilkunde*. 1980;83:606–8.
26. Ingemarson H, Grevsten S, Thoren L. Lethal horse-riding injuries. *J Trauma*. 1998;29:25–30.
27. Newton AM, Nielsen AM. A review of horse-related injuries in a rural Colorado hospital: implications for outreach education. *J Emerg Nurs*. 2005;31(5):442–6.
28. Ball C, Ball J, Kirkpatrick A, Mulloy R. Equestrian injuries: incidence, injury patterns, and risk factors for 10 years of major traumatic injuries. *Am J Surg*. 2007;193:636–40.
29. Jagodzinski T, DeMuri G. Horse-related injuries in children: a review. *WMJ*. 2005;104:50–4.
30. Barber H. Horse-play: survey of accidents with horses. *Br Med J*. 1973;3:532–4.
31. Grossman G, Kulund D, Miller C, Winn R, Hodge Jr R. Equestrian injuries: results of a prospective study. *JAMA*. 1978;240:1881–2.
32. Pounder D. The grave yawns for the horseman. Equestrian deaths in South Australia 1973–1983. *Med J Aust*. 1984;141:632–5.
33. Gierup J, Larsson M, Lennquist S. Incidence and nature of horse-riding injuries: a one-year prospective study. *Acta Chir Scand*. 1976;142:57–61.
34. Watt G, Finch C. Preventing equestrian injuries: locking the stable door. *Sports Med*. 1996;22:187–97.
35. Robson S. Some factors in the prevention of equestrian injuries. *Br J Sports Med*. 1979;13:33–5.
36. Firth J. Equestrian. In: Fu F, Stone D, editors. *Sports injuries: mechanisms, prevention, treatment*. Baltimore: Williams & Wilkins; 1994. p. 315–31.

Martin Wiewiorski, Markus Wurm, Alexej Barg,  
Markus Weber, and Victor Valderrabano

### Abstract

At the beginning of the 21st century, FIFA estimated 250 million soccer player worldwide, which makes soccer one of the world's most popular sports. Soccer is being played on various surfaces: grass, turf, dirt, sand or concrete. The lower extremity, and particularly the ankle joint are prone to injury. Mechanism of injury can be of direct force (tackling), or indirect (sprain, overuse). Injuries can be severe and in the worst case be career ending. Long term sequela of repetitive ankle injuries, like osteoarthritis, can have an impact on the athletes life quality beyond his/her sports career. The focus should be not only on treatment of the acute and chronic injuries, but especially on injury prevention.

### Keywords

Soccer • Ankle sprain • Osteochondral lesion • Ankle instability • Anterior ankle impingement

## Introduction

Soccer is one of the world's most popular sports with approximately 200.000 professional soccer players and around 240 million amateur soccer players. Complex, quickly changing movements make soccer one of the highest demand sports.

M. Wiewiorski, MD (✉)  
Department of Orthopaedics and Traumatology,  
Kantonsspital Winterthur, Winterthur, Switzerland  
e-mail: [wiewiorskim@gmail.com](mailto:wiewiorskim@gmail.com)

M. Wurm, MD  
Department of Orthopaedics and Traumatology,  
University Hospital Basel, Basel, Switzerland  
e-mail: [wurmarkus@gmail.com](mailto:wurmarkus@gmail.com)

A. Barg, MD  
Department of Orthopaedics, University of Utah,  
Salt Lake City, UT, USA  
e-mail: [alexej.barg@hsc.utah.edu](mailto:alexej.barg@hsc.utah.edu)

M. Weber  
Club Physician FC Basel 1893, Praxis Neumatt, Aesch, Switzerland  
e-mail: [markus.weber@hin.ch](mailto:markus.weber@hin.ch)

V. Valderrabano, MD, PhD  
Orthopaedic Department, SWISS ORTHO CENTER,  
Schmerzklinik Basel, Swiss Medical Network, Basel, Switzerland  
e-mail: [vvalderrabano@gsmn.ch](mailto:vvalderrabano@gsmn.ch)

Good coordination and body control are needed for walking, running, sprinting, sudden changes in direction, jumping, and body contact. With rising popularity comes an increased incidence of injuries. These present a wide variety of musculo-skeletal problems to the orthopaedic surgeon. Most injuries are localized in the lower extremity with 65 % being foot and ankle joints most affected [1–3]. The high injury rate among soccer players in general, and female players in particular constitutes a considerable problem for the player, the team, the club, and given the popularity of soccer, for society at large. Injuries are not only short-term consequences (loss of practice/competition time), but also long-term effects (career ending injury) on the players careers and lives.

## Injuries

### Ankle Sprains

Ankle sprains account for about 40 % of all sport injuries and up to 80 % of all soccer injuries [4]. Around 85 % of all ankle sprains are due to an inversion trauma with the foot in various degrees of plantar flexion. The ligaments most commonly involved are the anterior talofibular (ATFL) and calcaneal

fibular ligament (CFL). Ankle joint instability can be found on the medial and lateral side. A combined mediolateral instability is called a rotational instability. Recurrent sprains, pain, giving way, and insecurity on uneven grounds are common symptoms. The player's history (previous sprains, feeling of instability) is equally important for the diagnosis as physical and radiographic examination.

Acute lateral ankle sprains are graded from 1 to 4 [5]. Swelling only present on examination in grade 1 (few fibers of the lateral ligaments are stretched or torn). A lateral hematoma can be found in grade 2 sprains (complete tear of the ATFL and CFL). In grade 3, lateral and medial hematoma can be found, and typically weight bearing is not possible due to pain (lateral and medial ligaments are torn). Stage 4 is associated with additional injuries: fractures, syndesmotic lesions, and/or chondral/osteochondral lesions [6].

The goal of treatment after an acute ankle sprain is to reduce the risk of recurring ankle sprains, and chronic ankle instability. Conservative treatment is the management of choice for grade 1–3 ankle sprains in all patients. Early symptomatic treatment aims to reduce pain and swelling (Rest, Ice, Compression, Elevation [RICE]), and should be followed by a good stability protection (brace or walker depending on the grade of sprain), early ROM exercises, strengthening, proprioception, and activity-specific training under physiotherapeutic supervision. The time taken for a return to sports specific training and return to competition is dependent upon the athlete's ability to perform the functional requirements of the sport and varies from 1 to 6 weeks. Additional stabilizing measures in the game situation like taping and orthotics can be helpful. Surgical treatment is recommended for grade 4 ankle sprain. The surgery should start with an arthroscopical joint inspection to assess the extent of intraarticular damage. Ligament reconstruction has to be performed due to anatomical conditions. One option is direct anatomic ligament reinsertion with a modified Brostrom/Gould procedure [7]. Platelet rich plasma can be added on the repair site, to augment ligament healing [8]. Syndesmotic injuries need to be stabilized (screws, non-resorbable rope). Various stabilization devices ie screws or tight rope can be used. Chondral or osteochondral lesions have to be debrided or fixated.

## Bruises and Contusions

Contusions to the shins and thigh are among the most common injuries in soccer [9, 10]. Though most contusions are minor in nature, severe injuries and complications can occur. Most bruises are acquired by player's contact with an opponent's foot, knee or elbow. Pain, swelling, and reduced muscle-function are typical symptoms. Opening examination can reveal a variable amount of swelling and tenderness. Fractures are rare, but need to be ruled out by radiographs if suspected on clinical examination. Initial treatment includes ice and compression with restriction from activity dependent on the severity of the injury. Analgesics may be necessary. Intensity of physical therapy should account

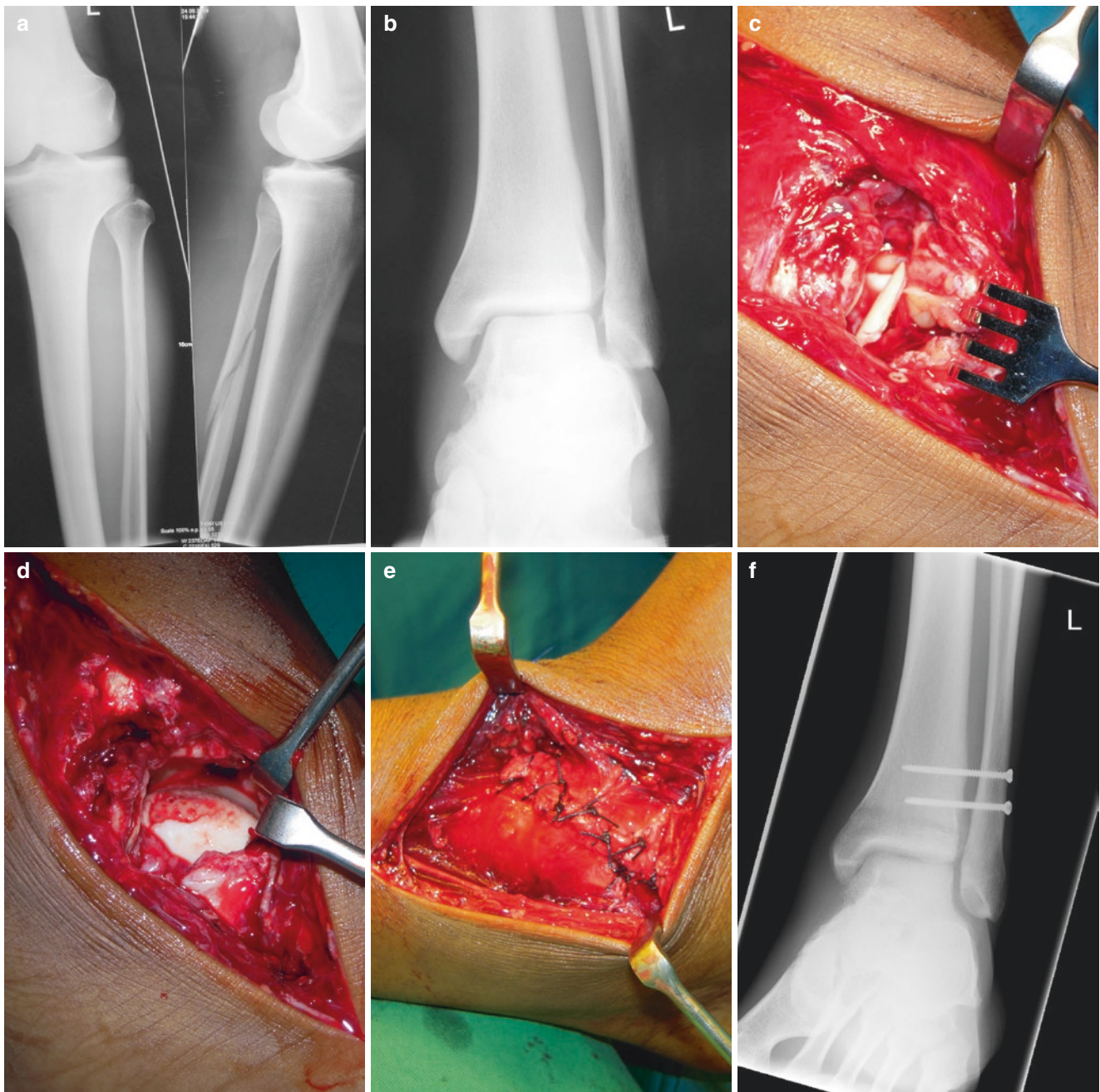
for severity of the symptoms: careful stretching of the affected muscle group followed by gradual strengthening and progressive functional activity. Before returning to play, full range of motion full range of motion, symmetrical strength and a demonstrated ability to perform the functional requirements of the soccer have to be reached. Possible complications include muscle tears and ruptures, compartment syndrome and myositis ossificans.

## Fractures

The kinetic energy generated by kicking the soccer ball, is usually is usually dispersed by the ball itself, and the lower extremity during the follow through movement. Miskicks or slide tackles are the source of severe injuries. Fractures may result by energy transfer from an opponent's lower leg. The incidence of fractures in soccer has been reported to range from 1 to 9.7% of all injuries [11]. Most of those (75%) occur during competition [12]. Of all lower leg fractures, ankle fractures are the most common (36%), followed by fractures of the foot (33%) and the tibia (22%). McCarroll and el assessed 4018 youth soccer players and found a fracture rate of 9.7% of all injuries. Of the 17 documented fractures, only 4 were found in the lower extremity [9]. Hoff and coworkers found a 2% incidence rate for fractures in outdoor soccer, and 8% for indoor soccer [6]. To our knowledge, there is no data on the exact classification and morphology of ankle fractures in soccer. Overall, a supination-external rotation force is the most common cause of ankle fractures in sports. Other less common fracture in soccer are metatarsal fractures and lisfranc fractures/lesions. In soccer players, stress fractures of the fifth metatarsal account for 78% of all stress fractures [13]. Those fractures can cause long absences from training and competition for more than 3 months.

## Chondral/Osteochondral Lesions

Osteochondral lesions describe the presence of a cartilage-bone defect, usually located at the talus. Acute lesions can occur in soccer due to ankle sprains or ankle fractures. In professional soccer players is important to do in severe ankle sprains an extensive imaging with conventional radiographs and MRI (here if possible: Arthro-MRI). In ankle fractures the diagnostic ankle arthroscopy at the time of fracture fixation visualizes possible hidden chondral or osteochondral lesions. Missed acute osteochondral lesions have tendency not to heal properly and become chronic lesions, causing pain and play disability. Synovial fluid is pressed into the fracture fissure, which theoretically prevents bony union and can lead to cystic degeneration of the subchondral bone [15]. Ankle joint instability, especially lateral instability, increases joint pressure on the medial talus, where most lesions are located. It is unclear whether lateral instability is a etiologic factor of osteochondral lesions, or if increases the symptoms of an preexisting lesion [14]. Symptoms are pain, swelling and reduced range of motion. If an osteochondral lesion



**Fig. 52.1** Severe eversion trauma. This professional soccer player sustained severe eversion trauma with luxation of the ankle joint, while kicking a ball blocked by the opponent's foot, resulting in a syndesmotom injury, rupture of the interosseous membrane and high fibular fracture (Weber C) (a, b). Open exposure revealed disrupted chondral

fragments on the medial talar edge, and a deltoid and syndesmotom rupture (c). The chondral flakes were removed, microfracturing of the medial talus edge lesion was performed (d), and the deltoid and spring ligaments were reconstructed anatomically (e). Two syndesmotom screws were inserted (f), which were removed 8 weeks postoperatively

is suspected on clinical examination, further radiological diagnostics involving MRI and/or CT is recommended. Surgical reconstruction depends on the size and location of the lesion, and can be performed either in acute lesions as arthroscopic debridement, microfracturing, or refixation, or in chronic lesions as one-step repair (microfracturing, AMIC, OATS) or two-step repair (ACI, MACI) (Fig. 52.1) [16]. Additional biological healing augmentation with platelet rich plasma (PRP) has been described for treatment of cartilage lesion [17].

### Tendon Lesions

The tendons most prone to injury in soccer are: Achilles tendon, peroneal tendons, posterior tibial tendon, flexor hallucis longus tendon, posterior tibial tendon [18]. Achillodynia can be commonly found, with potential causes being Achilles tendinitis (acute overuse or equipment problems, systemic reasons, chronic degeneration), midportion tendinopathy (degeneration), insertion tendinopathy (with possible bony

spurs, through overuse), malalignment of the ankle joint, or Haglund's deformity. Most tendinopathies can initially be treated conservatively with excentric exercises, therapeutic ultrasound, adaptation of training intensity, choice of shoes, PRP infiltrations, and other [19]. Another frequent issue are the Achilles tendon ruptures which can be frequently found in soccer players. Nine of 100.000 Achilles tendon ruptures can be attributed to soccer [20]. The risk for rupture is higher with increased training intensity. Interestingly, the risk of rupture is three times higher before than during competition season [21]. The typical location of rupture is approximately 2–6 cm proximal of the insertion in the tuber calcanei. Vascularity of this area decreases with increasing age, therefore healing potential of this particular region is reduced [22]. Clinical examination shows a pathologic Thompson-test and a palpable gap in the tendon. MRI and ultrasound are helpful adjuncts to confirm the clinical diagnosis. Conservative treatment is not recommended in high demand athletes [23]. Our preferred surgical technique is a mini-open technique with anatomic reconstruction of the soleus muscle attachment. Postoperatively a plantar flexion shoe with a 30° plantar flexion wedge for week 1 and 2, a 15° wedge for week 3 and 4, and plantigrade after week 5 is necessary to avoid pathological tendon overlength, which causes plantar flexion force reduction and therefore reduced sprint power. An individual adapted rehabilitation program in cooperation with team physiotherapist and conditional trainer is essential.

### Anterior Ankle Impingement

The anterior ankle impingement syndrome (soccer ankle) is caused by anterior osteophytes on the anterior tibia and talar neck resulting in soft-tissue or bony impingement on ankle dorsiflexion. Anterior ankle pain with reduced and painful dorsiflexion, catching, and subjective feelings of giving way is typical. Up to 60% of professional soccer players are affected [24]. Lateral ankle radiographs reveal "kissing osteophytes". The formation of the osteophytes is not fully understood. However bony spur formation may be due to traction from repetitive micro-trauma (damage to anterior ankle cartilage, and forced plantar or dorsiflexion causing traction on the joint capsule) [25]. If conservative treatment fails most of these lesions can be burred away arthroscopically, and in cases of more extensive bony formations by minimal open excision.

### Etiology and Pathomechanism

Generally one can differentiate intrinsic and extrinsic factors. Intrinsic factors are such concerning the particular athlete ie joint flexibility, ligamentous laxity, muscle tightness, functional instability, previous injuries, inadequate rehabilitation. Load of exercise, inadequate equipment, field conditions and foul play are being considered extrinsic on the other hand



**Fig. 52.2** Foul Play: Lesion of a soccer player during offensive contact

[26]. Intrinsic factors are hard to quantify, and can be underestimated. Most important individual factors for high risk of injury are a previous injury, and inadequate rehabilitation after injury [27]. Simple injuries can be followed by severe injuries [28]. Persistent symptoms after injuries can be a precursor of further injuries in the future. General ligament laxity, previous strain, ligament instability and isokinetic ankle force seem not to be associated with the risk of injury [29]. However, a muscular misbalance, e.g. between eversion and inversion force of the foot has been shown to be a risk factor [29]. Slow reaction time was shown to be an additional risk factor for injury [30]. The most important extrinsic risk factor seems to be foul play, which is committing an unfair act by a player against another, usually involving illegal body contact (Fig. 52.2). This makes up to 23–33% of all injuries [31]. Certain forms of engaging the opponent, like tackling, where a player attempts to take the ball away from an opposing player by deliberately leaving his feet and sliding along the ground with one leg extended to push the ball away from the opposing player, can cause severe injuries. Injuries can be also caused due to collision when jumping for a header, or landing. However, many of the injuries are caused without direct body contact. Soccer shoes are equipped with cleats for better grip on the turf. Getting stuck on turf leads to unusually high load and torque in the knee and ankle joint. The composition of the soccer playground might influence the injury rate. It has been suggested that soccer players are at greater risk of sustaining ankle sprains on artificial turf, wherein less muscle strains are expected [32].

### Epidemiology

Soccer is to be considered one of world's most popular sports, with participants being women and men of all age groups and skill levels. Overall, soccer injuries are more frequent with higher age of the participants, while incidence of injury in preadolescent players is low [33, 34]. The incidence of



**Table 52.1** Overview of publication on foot/ankle injuries in soccer

Author	Topic	Level of evidence
Yard et al. [36]	High school soccer injury patterns	3
Soederman et al. [37]	Injury prevention in female soccer players	2
Cloke et al. [38]	Ankle injuries in football academies	3
Soligard et al. [39]	Role of warm-up in injury prevention in young female footballers	2
Nielsen et al. [40]	Epidemiology and traumatology of injuries in soccer	3
Hawkins et al. [41]	Epidemiology of soccer injuries	3
Vanlommel et al. [12]	Fractures of lower leg in soccer	4
Elias [2]	10 year follow up, USA soccer cup	1

injuries during practice is lower than during competition (about 1:2) [35]. Amateur soccer players have less injuries than professional soccer players [1]. The most common site of injury in soccer player is the lower extremity. Breaking it down further, the most injured joint is the ankle joint (0.17–6.52 per 1000 person hours), followed by the knee and the thigh [1]. Of all foot and ankle injuries, ankle **sprains** are the most common (80%), then **bruises** (9–49%), and **tendon lesions** (2–23%) (Table 52.1). With 1% of all ankle injuries in soccer, fractures are very rare [42].

### Therapy (On the Field, Conservative, Surgical)

For specific injury therapy, please see the following injuries subsections.

### Prevention

Prevention measures are needed not only to prevent acute injuries, but also long term effects of injuries, like osteoarthritis [43]. Important prerequisites for preventing injuries is a structured warm up training (FIFA 11+ program) [39] and professional coaching [40]. Shooting exercises can lead to muscle strains, if adequate warm up is not performed [28]. Athletes starting playing soccer in a club before the age of 6 years, are less prone to injuries. Players with long careers are usually more talented and have more endurance, which reduces the risk of injury [44].

Shin guards reduce the impact forces to the leg, thereby preventing or reducing the severity of soft-tissue injuries and fractures. Load forces were reduced 41.2–77.1% at the ankle and knee with shin guards [45]. Boden et al. performed a retrospective review of 31 athletes who sustained a fracture of the lower leg from direct impact while playing soccer. Of those fractures, 26 (90%) occurred while the player was wearing a shin guards [46].

Long enough rehabilitation time is needed after injury. Players returning to exercising with not sufficiently healed injuries, have a higher risk of re-injury [47]. Taping has to be seen critically, because they give players an illusion of safety. The use of an orthosis after a sustained ankle sprain proofed to be beneficial in terms of reducing the re-injury rate [48]. In soccer players

without previous history of sprains, there is no decrease in risk. Balance board training showed not to be useful in prevention of severe knee injuries in female soccer players [37].

### Evidence (Description of Highest Evidence with Mentioning the Level and Grade of Evidence)

For literature and evidence see Table 52.1.

### Summary

- Soccer is a physically demanding and highly competitive sport with a high rate of injuries.
- The foot and the ankle joint are especially at danger.
- Risk factors of foot and ankle injuries in soccer players are: lack of structured warm up training, neuromuscular deficits, inadequate training, chronic fatigue, previous injuries, foul play, artificial turf, and others.
- While the most common injury is the ankle sprain, complex injuries like fractures, ligament tears, and cartilage damage can occur. Such injuries have a high impact on the player's career. Professional soccer players are pressured to meet the expectations of the clubs, and the public.
- While most injuries to the foot and ankle joint can be treated conservatively, complex injuries require anatomic reconstruction to allow for quick rehabilitation and return to play earliest possible.

### References

1. Wong P, Hong Y. Soccer injury in the lower extremities. *Br J Sports Med.* 2005;39(8):473–82.
2. Elias SR. 10-year trend in USA Cup soccer injuries: 1988–1997. *Med Sci Sports Exerc.* 2001;33(3):359–67.
3. Inklaar H. Soccer injuries. I: incidence and severity. *Sports Med.* 1994;18(1):55–73.
4. Giza E, Mithofer K, Farrell L, Zarins B, Gill T. Injuries in women's professional soccer. *Br J Sports Med.* 2005;39(4):212–6; discussion –6.
5. Valderrabano V. Akute und chronische OSG-Instabilitaet. GOTS Expertenmeeting 2012 Sprunggelenksinstabilitaet. 2012; p. 43–55.

6. Ueblacker P, Hänsel L, Seebauer L, Müller-Wohlfahrt H-W. Die isolierte Ruptur der vorderen Syndesmose – eine häufig übersehene Verletzung. Darstellung eines Falles aus der Fußball-Europameisterschaft 2008 mit aktueller Literaturübersicht. *Sport-Orthopädie – Sport-Traumatologie – Sports Orthop Traumatol.* 2009;25(4):311–5.
7. Gould N, Seligson D, Gassman J. Early and late repair of lateral ligament of the ankle. *Foot Ankle.* 1980;1(2):84–9.
8. Mishra A, Woodall J, Vieira A. Treatment of tendon and muscle using platelet-rich plasma. *Clin Sports Med.* 2009;28(1):113–25.
9. Tucker AM. Common soccer injuries. Diagnosis, treatment and rehabilitation. *Sports Med.* 1997;23(1):21–32.
10. Batt ME. Shin splints – a review of terminology. *Clin J Sports Med Off J Can Acad Sports Med.* 1995;5(1):53–7.
11. Engström B, Johansson C, Tornkvist H. Soccer injuries among elite female players. *Am J Sports Med.* 1991;19(4):372–5.
12. Vanlommel L, Vanlommel J, Bollars P, Quisquater L, Van Crombrugge K, Corten K, et al. Incidence and risk factors of lower leg fractures in Belgian soccer players. *Injury.* 2013;44(12):1847–50.
13. Ekstrand J, Torstveit MK. Stress fractures in elite male football players. *Scand J Med Sci Sports.* 2012;22(3):341–6.
14. Valderrabano V, Miska M, Leumann A, Wiewiorski M. Reconstruction of osteochondral lesions of the talus with autologous spongiosa grafts and autologous matrix-induced chondrogenesis. *Am J Sports Med.* 2013;41(3):519–27.
15. van Dijk CN, Reilingh ML, Zengerink M, van Bergen CJ. Osteochondral defects in the ankle: why painful? *Knee Surg Sports Traumatol Arthrosc.* 2010;18(5):570–80.
16. Wiewiorski M, Barg A, Valderrabano V. Chondral and osteochondral reconstruction of local ankle degeneration. *Foot Ankle Clin.* 2013;18(3):543–54.
17. Smyth NA, Fansa AM, Murawski CD, Kennedy JG. Platelet-rich plasma as a biological adjunct to the surgical treatment of osteochondral lesions of the talus. *Tech Foot Ankle Surg.* 2012;11(1):18–25. doi:10.1097/BTF.0b013e3182463ca1.
18. Oztekin HH, Boya H, Ozcan O, Zeren B, Pinar P. Foot and ankle injuries and time lost from play in professional soccer players. *Foot.* 2009;19(1):22–8.
19. Hall MP, Band PA, Meislin RJ, Jazrawi LM, Cardone DA. Platelet-rich plasma: current concepts and application in sports medicine. *J Am Acad Orthop Surg.* 2009;17(10):602–8.
20. Cretnik A, Frank A. Incidence and outcome of rupture of the Achilles tendon. *Wien Klin Wochenschr.* 2004;116 Suppl 2:33–8.
21. Woods C, Hawkins R, Hulse M, Hodson A. The Football Association Medical Research Programme: an audit of injuries in professional football-analysis of preseason injuries. *Br J Sports Med.* 2002;36(6):436–41; discussion 41.
22. Lagergren C, Lindholm A. Vascular distribution in the Achilles tendon: an angiographic and microangiographic study. *Acta Chir Scand.* 1959;116:491.
23. Carden DG, Noble J, Chalmers J, Lunn P, Ellis J. Rupture of the calcaneal tendon. The early and late management. *J Bone Joint Surg Br.* 1987;69(3):416–20.
24. Massada JL. Ankle overuse injuries in soccer players. Morphological adaptation of the talus in the anterior impingement. *J Sports Med Phys Fitness.* 1991;31(3):447–51.
25. Walcher MG, Leumann A, Wiewiorski M, Pagenstert G, Valderrabano V. Sprunggelenks-und Fussserkrankungen bei Fussballern. *Schweizerische Zeitschrift für Sportmedizin und Sporttraumatologie.* 2010;58(2):44.
26. Inklaar H. Soccer injuries. II: aetiology and prevention. *Sports Med.* 1994;18(2):81–93.
27. Dvorak J, Junge A, Chomiak J, Graf-Baumann T, Peterson L, Rosch D, et al. Risk factor analysis for injuries in football players. Possibilities for a prevention program. *Am J Sports Med.* 2000;28(5 Suppl):S69–74.
28. Ekstrand J, Gillquist J, Moller M, Oberg B, Liljedahl SO. Incidence of soccer injuries and their relation to training and team success. *Am J Sports Med.* 1983;11(2):63–7.
29. Baumhauer JF, Alosa DM, Renstrom AF, Trevino S, Beynonn B. A prospective study of ankle injury risk factors. *Am J Sports Med.* 1995;23(5):564–70.
30. Taimela S. Relation between speed of reaction and psychometric tests of mental ability in musculoskeletal injury-prone subjects. *Percept Mot Skills.* 1990;70(1):155–61.
31. Hawkins RD, Fuller CW. Risk assessment in professional football: an examination of accidents and incidents in the 1994 World Cup finals. *Br J Sports Med.* 1996;30(2):165–70.
32. Ekstrand J, Hägglund M, Fuller C. Comparison of injuries sustained on artificial turf and grass by male and female elite football players. *Scand J Med Sci Sports.* 2011;21(6):824–32.
33. Sullivan JA, Gross RH, Grana WA, Garcia-Moral CA. Evaluation of injuries in youth soccer. *Am J Sports Med.* 1980;8(5):325–7.
34. Dvorak J, Junge A, Graf-Baumann T, Peterson L. Football is the most popular sport worldwide. *Am J Sports Med.* 2004;32(1 Suppl):3S–4.
35. Ekstrand J, Gillquist J. Soccer injuries and their mechanisms. *Med Sci Sports Exerc.* 1983;15:267–70.
36. Yard EE, Schroeder MJ, Fields SK, Collins CL, Comstock RD. The epidemiology of United States high school soccer injuries, 2005–2007. *Am J Sports Med.* 2008;36(10):1930–7.
37. Soderman K, Werner S, Pietila T, Engstrom B, Alfredson H. Balance board training: prevention of traumatic injuries of the lower extremities in female soccer players? A prospective randomized intervention study. *Knee Surg Sports Traumatol Arthrosc.* 2000;8(6):356–63.
38. Cloke DJ, Ansell P, Avery P, Deehan D. Ankle injuries in football academies: a three-centre prospective study. *Br J Sports Med.* 2011;45(9):702–8.
39. Soligard T, Myklebust G, Steffen K, Holme I, Silvers H, Bizzini M, et al. Comprehensive warm-up programme to prevent injuries in young female footballers: cluster randomised controlled trial. *BMJ.* 2008;337:a2469.
40. Nielsen AB, Yde J. Epidemiology and traumatology of injuries in soccer. *Am J Sports Med.* 1989;17(6):803–7.
41. Hawkins RD, Fuller CW. A prospective epidemiological study of injuries in four English professional football clubs. *Br J Sports Med.* 1999;33(3):196–203.
42. Fong DT, Hong Y, Chan LK, Yung PS, Chan KM. A systematic review on ankle injury and ankle sprain in sports. *Sports Med.* 2007;37(1):73–94.
43. Valderrabano V, Hintermann B, Horisberger M, Fung TS. Ligamentous posttraumatic ankle osteoarthritis. *Am J Sports Med.* 2006;34(4):612–20.
44. Peterson L, Junge A, Chomiak J, Graf-Baumann T, Dvorak J. Incidence of football injuries and complaints in different age groups and skill-level groups. *Am J Sports Med.* 2000;28(5 Suppl):S51–7.
45. Bir CA, Cassatta SJ, Janda DH. An analysis and comparison of soccer shin guards. *Clin J Sport Med.* 1995;5(2):95–9.
46. Boden B, Lohnes JH, Nunley JA, Garrett Jr WE. Tibia and fibula fractures in soccer players. *Knee Surg Sports Traumatol Arthrosc.* 1999;7(4):262–6.
47. Freiwald J, Baumgart C, Hoppe MW, Slomka M, Brexendorf B, Partenheimer A, et al. Return to Sport nach Verletzungen im Hochleistungsfußball – was ist dazu notwendig? *Sport-Orthopädie – Sport-Traumatologie – Sports Orthop Traumatol.* 2013;29(1):4–12.
48. Surve I, Schweltnus MP, Noakes T, Lombard C. A fivefold reduction in the incidence of recurrent ankle sprains in soccer players using the Sport-Stirrup orthosis. *Am J Sports Med.* 1994;22(5):601–6.

Markus Wurm and T. Schlemmer

**Abstract**

Floorball is a relatively young sport (founded in 1986) compared to others like soccer or ice hockey however has a rapid growing community. In 1998 there were 151,547 registered floor ball players in 19 countries. In 2014 records revealed 300,133 registered players in 58 countries. Nordic countries together with Czech Republic and Switzerland exhibit the biggest communities.

Floorball is played on an indoor rink measuring 40×20 m enclosed by 50 cm high boarders.

A standard floorball (approx. 23 g) comprises 26 holes, is composed of plastic and shows a hollow design. It further provides dimples to reduce air resistance which allows top speeds up to 200 km/h [1].

Knee and ankle joint are two of the most affected injury sites besides head and neck. Collisions with opponents, predisposing factors, equipment (stick, ball) and overuse are the most common reasons for injuries in floorball [2].

Neuromuscular training can prevent sports related injuries in floorball.

**Keywords**

Floorball • Ankle sprain • Collision • Neuromuscular • Proprioception • Rehabilitation

**Introduction**

Floorball is a relatively young sport (founded in 1986) compared to others like soccer or ice hockey however has a rapid growing community. In 1998 there were 151,547 registered floor ball players in 19 countries. In 2014 records revealed 300,133 registered players in 58 countries. Nordic countries together with Czech Republic and Switzerland exhibit the biggest communities.

Floorball is played on an indoor rink measuring 40×20 m enclosed by 50 cm high boarders.

A standard floorball (approx. 23 g) comprises 26 holes, is composed of plastic and shows a hollow design. It further provides dimples to reduce air resistance which allows top speeds up to 200 km/h [1].

Knee and ankle joint are 2 of the most affected injury sites besides head and neck. Collisions with opponents, predisposing factors, equipment (stick, ball) and overuse are the most common reasons for injuries in floorball [2].

Neuromuscular training can prevent sports related injuries in floorball.

---

M. Wurm, MD (✉)  
Department of Orthopaedics and Traumatology, University  
Hospital Basel, Spitalstrasse 21, Basel 4031, Switzerland  
e-mail: [markus.wurm@usb.ch](mailto:markus.wurm@usb.ch)

T. Schlemmer, MD  
Orthopaedic Department, University Hospital Basel,  
Basel, Switzerland  
e-mail: [thomas.schlemmer@usb.ch](mailto:thomas.schlemmer@usb.ch)

## Injuries

Ankle sprains are the most common injuries in floorball since it is marked of fast directional changes. These can lead to ligamentous strain or even tear besides fractures. Snellman et al. showed 62 % of injuries on lower extremities during a one-year prospective observational period in 2000. 20 % of recorded injuries concerning the ankle joint were acute sprains. Ankle damage was subdivided into sprains, soft tissue overuse injuries, muscle strains, ligament tears and fractures [2].

There is neither big nor strong data on foot and ankle injuries in literature regarding floorball; however, Ullius showed 58 % of injuries concerning the foot and ankle [3].

Pasanen et al. reported on 29 % acute ankle injuries whereas 95 % were ligament injuries. On the other hand they encountered 22 % overuse injuries on calf/shin [4].

Traumatic ankle injuries in both sexes occurred in 27.5 % with a higher count in women according to Traanaeus et al. in 2016. They presented a prospective cohort study and injury profile of 12 Swedish elite floorball teams. Overuse injuries were most common in female ankles compared to back pain in male [5].

## Etiology and Pathomechanism

Floorball is one fast indoor sport and subsists by quick directional changes. Sticky ground and abrupt breaks are reasons for ligament injuries of the lower extremity. Causes for injuries of the foot and ankle are collisions with an opponent, predisposing extrinsic and intrinsic factors, overuse, stick and sudden movements. Furthermore Pasanan et al. illustrated 45 % of traumatic injuries arising without rival contact. Acute injuries (83 %) are far more common than overuse injuries (17 %) [2]. Lower extremity injuries seem to be more common and severe than in upper extremity. Most injuries occurring in floorball can be treated outpatient and hospital admission is rarely needed [4].

Exposure, training (amount, frequency, intensity and type) as well as environment and equipment are ranked among extrinsic factors. Physical characteristics, i.e. age, gender, previous injuries, joint mobility (laxity), ligamentous instability and psychological profile are considered intrinsic factors [6].

Reasons for relatively high count on non-contact injuries remain unclear yet possible factors may be friction between shoes and (sticky rubber-) floor, lack of proprioception as well as condition training. Injury rate in training is relatively low compared to game hours despite a quite high amount on non-contact injuries, which is presumed less during practice [2, 4].

## Epidemiology

Snellmann et al. showed an overall injury incidence of 1–23.7/1000 floorball hours whereat the numbers were drastically higher during game time compared to training time [2].

Recent studies showed equal data between 2.6/exposure hours in men and 3.9 in women. They further distinguished preseason injuries (14 %) and game season injuries (42 %) which indicates a higher risk for game play than training [5]. Men's handball reveals comparable numbers of injuries between 0.6/1000 h during practice and 14.3 during game time [7]. Soccer injuries occur between 7.6/1000 practice hours and 16.9/1000 game hours [8]. Lower extremity is affected in 62 % of floorball injuries whereas 20 % are being omitted to the ankle joint [2, 9].

## Therapy (on the Field, Conservative, Surgical)

Ankle sprains should be treated with special attention since adolescent players can ultimately develop ankle instability with demoralizing results on their future career.

The acquainted RICE-routine is one option being applied within acute injury (Rest, Immobilization, Compression, Elevation).

Mild injuries (ligament strains, sprains without tears) should be treated functionally and adapted to pain levels. External stabilization (i.e., taping, external braces, orthotics) can be applied to ensure no further spraining. Moderate and severe injuries should be immobilized since partial tears or even tears need 6–8 weeks healing time.

It is important to distinguish between recreational and elite athlete. Furthermore talking to the athlete and gaining an overall idea is crucial to elucidate the need for a fast return to sports. In the first phase balance and neuromuscular training (without maximal degree of utilization) followed by agility are feasible concepts. These should always be accompanied by endurance training to keep stamina levels high.

A steady proprioceptive training beside sports specific motion patterns can additionally be implemented. Of course physiotherapeutic measures are one mainstay of a fast return to sports.

Surgical treatment has to be adapted to athlete's needs and the particular injury as well as injury pattern.

Overuse injuries may be treated with a short downtime for athletes. Sometimes it is enough to only keep athletes from game play for a short period. Furthermore special adapted measures (ie rest, physiotherapy or swimming) can achieve potent results [9].

## Rehabilitation and Return-to-Sports

Tranaeus et al. presented an injury profile in Swedish elite floorball players. Results displayed majority of occurred damages being categorized as mild. The ankle joint showed highest numbers in mild female injuries (14.5%) yet to be on top of moderate injuries in male players (3.5%). Injuries were classified on number of days absent from floorball whereas mild was to be defined ranging from 1 to 7 days. Further distinctions were moderate 8–30 days and severe with more than 30 days [5, 10].

It is notable that female athletes have a higher risk on “permanent medical impairment” injuries than their male correlates [11].

Psychological components are an important factor, which is respected shabby to date. Of course physical stress factors have to be minimized yet the psychological ones often do not even find consideration. After repetitive injuries it is even more important to attend and supervise the athlete with regards to this aspects during potential long routes until return to sports [12].

## Prevention

Controlled trials revealed clear consent on a feasible reduction on sports injuries due to prevention programs. Strengthening of the lower extremity, plyometric, agility and endurance training besides protective equipment can help preventing acute and overuse injuries. Swift speed up and deceleration trainings can furthermore adapt ligaments and muscles to optimal preload and minimize ligament laxity [6].

Avramakis et al. conducted a study focused on sideward stability in floorball with regards of footwear. Subjects were asked to perform a defined sideward cutting movement with low-cut and high-cut shoe as well as barefoot style. They stated less supination in high-cut shoes than low-cut shoes, which appears to be a stabilizing factor [3].

Composition of the ground appears to influence the incidence on injuries as well. Hard rubber floors reveals higher injury counts than parquet floors [4].

Neuromuscular training (NT) programs showed effectiveness with regards of preventing acute non-contact injuries in female floorball players [13]. Furthermore NT improves sideways jumping speed and static balance [14].

## Evidence

Author(s), year	Journal	Focus	Level of evidence
Pasanan et al. (2008)	<i>Scand J Med Sci</i>	Elite, Finland Women	III

Author(s), year	Journal	Focus	Level of evidence
Snellman et al. (2001)	<i>Ort &amp; Clin Sci</i>	Mixed, Men/Women Finland	III
Sobhani et al. (2012)	<i>Scan J Med Sci</i>	Systematic review foot and ankle injuries	II
Tranaeus et al. (2015)	<i>KSSTA</i>	Elite, Sweden Female	II

## Summary

- Floorball is a fast growing indoor sport, which is characterized by sudden directional as well as acceleration and decelerating changes.
- Ligament and muscle injuries of the lower extremity are common seen violations. Lower extremities are more often affected than upper extremities.
- Injury rate is approximately equal compared to other team impact sports like handball, basketball or soccer.
- The ankle joint is one of the most affected injury sites besides knee joint, head and neck. Downtime for athletes is relatively low due to a mostly rather mild course of injuries.
- Neuromuscular training showed effectiveness in preventing floorball related injuries.

## References

1. Floorball.org. IFF today and history in short. [http://www.floorball.org/default.asp?id\\_sivu=252014](http://www.floorball.org/default.asp?id_sivu=252014).
2. Snellman K, Parkkari J, Kannus P, Leppala J, Vuori I, Jarvinen M. Sports injuries in floorball: a prospective one-year follow-up study. *Int J Sports Med*. 2001;22(7):531–6.
3. Avramakis E, Stakoff A, Stussi E. Effect of shoe shaft and shoe sole height on the upper ankle joint in lateral movements in floorball (uni-hockey). *Sportverletzung Sportschaden: Organ der Gesellschaft für Orthopädisch-Traumatologische Sportmedizin*. 2000;14(3):98–106.
4. Pasanen K, Parkkari J, Kannus P, Rossi L, Palvanen M, Natri A, et al. Injury risk in female floorball: a prospective one-season follow-up. *Scand J Med Sci Sports*. 2008;18(1):49–54.
5. Tranaeus U, Gotesson E, Werner S. Injury profile in Swedish Elite Floorball: a prospective cohort study of 12 teams. *Sports Health*. 2016.
6. Parkkari J, Kujala UM, Kannus P. Is it possible to prevent sports injuries? Review of controlled clinical trials and recommendations for future work. *Sports Med*. 2001;31(14):985–95.
7. Seil R, Rupp S, Tempelhof S, Kohn D. Sports injuries in team handball. A one-year prospective study of sixteen men's senior teams of a superior nonprofessional level. *Am J Sports Med*. 1998;26(5):681–7.
8. Ekstrand J, Gillquist J. Soccer injuries and their mechanisms: a prospective study. *Med Sci Sports Exerc*. 1983;15(3):267–70.

9. Sobhani S, Dekker R, Postema K, Dijkstra PU. Epidemiology of ankle and foot overuse injuries in sports: a systematic review. *Scand J Med Sci Sports*. 2013;23(6):669–86.
10. Tranaeus U, Johnson U, Ivarsson A, Engstrom B, Skillgate E, Werner S. Sports injury prevention in Swedish elite floorball players: evaluation of two consecutive floorball seasons. *Knee Surg Sports Traumatol Arthrosc*. 2015;23(3):899–905.
11. Aman M, Forssblad M, Henriksson-Larsen K. Incidence and severity of reported acute sports injuries in 35 sports using insurance registry data. *Scand J Med Sci Sports*. 2015.
12. Wiese-Bjornstal DM. Psychology and socioculture affect injury risk, response, and recovery in high-intensity athletes: a consensus statement. *Scand J Med Sci Sports*. 2010;20 Suppl 2:103–11.
13. Pasanen K, Parkkari J, Pasanen M, Hiilloskorpi H, Makiinen T, Jarvinen M, et al. Neuromuscular training and the risk of leg injuries in female floorball players: cluster randomised controlled study. *BMJ*. 2008;337:a295.
14. Pasanen K, Parkkari J, Pasanen M, Kannus P. Effect of a neuromuscular warm-up programme on muscle power, balance, speed and agility: a randomised controlled study. *Br J Sports Med*. 2009;43(13):1073–8.

Erik C. Nilssen and William K. Whiteside

**Abstract**

Ankle and foot injuries are relatively rare injuries in golf. When they occur they are usually due to overuse or the swing mechanism with the most common being lateral ankle sprains and plantar fasciitis. Both lateral ankle sprains and plantar fasciitis are treated initially with conservative measures and typically resolve. If chronic lateral instability develops, surgical options such as the modified Brostrum exist with good long term results. Plantar fasciitis is treated with stretching of the gastrocnemius and plantar fascia specific stretches with good success. Surgical options for plantar fasciitis continue to evolve from the standard open partial plantar fascia release for those with chronic symptoms.

**Keywords**

Golf • Plantar fasciitis • Ankle sprain • Lateral ankle instability

**Introduction**

In comparison to contact sports, golf is not considered a strenuous sport but injuries still occur. The most common musculoskeletal injury in golf is to the low back and upper extremity with ankle/foot comprising approximately 5% of injuries among recreational to professional golfers [1–3]. Most injuries are typically caused by either the swing mechanism or chronic overuse with the most common being lateral ankle sprains and plantar fasciitis.

**Injuries**

Although not specific to golf, lateral ankle sprains and plantar fasciitis are among the most common injuries.

**Etiology and Pathomechanism**

Lateral ankle sprains are caused by an inversion injury that result in either attenuation or rupture to one or more of the ligaments on the lateral ankle. Typically, the Anterior Talofibular Ligament (ATFL) is the first ligament injured followed by the Calcaneofibular ligament (CFL). A grading system is used to describe the ligament injuries with Grade I being a stretch injury; II, partial tearing; and III, complete rupture [3]. In golf this can be caused by either an inversion injury by walking on uneven terrain such as sand in the bunker, a hill in the rough, or can occur during the swing phase. As the front foot moves from a pronated position during the backwing to an inverted position in the downswing and follow through, body weight is shifted as the club contacts the ball which can result in an injury to the lateral ankle of the front foot.

Plantar fasciitis is the result of repetitive microtearing at the origin of the central band of the plantar aponeurosis at the medial tuberosity of the os calcis. This continuous trauma causes inflammation and pain most notably during the first steps in the morning as one gets out of bed. There may also be a component of traction neuritis on the first branch of the lateral plantar nerve as described by Baxter [4]. The walking

E.C. Nilssen, MD • W.K. Whiteside, MD (✉)  
Andrews Research and Education Institute,  
1040 Gulf Breeze Pky, Ste 210, Gulf Breeze, FL 32561, USA  
e-mail: [nilssenmd@yahoo.com](mailto:nilssenmd@yahoo.com); [will.whiteside@yahoo.com](mailto:will.whiteside@yahoo.com)

nature of golf plays a roll in the development of plantar fasciitis and the fact that many athletes including golfers have tight gastrocnemius muscles [5].

---

## Epidemiology

Lateral ankle sprains are the most common musculoskeletal injury in the general population. They represent approximately 45% of all sports related injuries and the incidence has been reported around 25,000 people per day [6]. Plantar fasciitis is also quite common with an incidence of 10% in the general population [7].

---

## Therapy

Most lateral ankle sprains do well with nonoperative measures. If an acute lateral ankle sprain occurs during a round of golf and the player cannot walk on the injured foot then they should be removed from the game. Usually keeping the sock and shoe in place can help with swelling. The well known RICE procedure should be initiated. The Ottawa ankle rules regarding radiographs can be applied in the field. However, in our practice, we routinely obtain radiographs but MRI is not usually necessary unless there is a history of chronic instability where a peroneal tendon or osteochondral injury is suspected. Some period of immobilization in a CAM boot or low ankle brace may be necessary depending on the severity. Some Grade I injuries can be treated with immediate rehabilitation, however this is patient dependent. Typically, Grade II injuries respond to 1 week of rest prior to physical therapy, and Grade III injuries may need up to 2 weeks of rest prior to rehabilitation. Surgical options such as the modified Brostrum lateral ankle ligament repair are considered if a patient fails 3–6 months of treatment and has persistent functional and mechanical instability.

Similar to lateral ankle sprains, nearly 90% of plantar fasciitis patients do well with nonoperative management [7]. It would be rare to have an acute flare up during a golf round since plantar fasciitis is a chronic overuse condition. Relative rest, stretching of the Achilles and plantar fascia, ice and NSAIDs can be initiated at any point for plantar fasciitis. Any number of physical therapy options exists including whirlpool, iontophoresis, phonophoresis, cryotherapy, and the Graston technique. A night splint can also be used which may prevent start up pain in the morning. Corticosteroid injection can also be considered. Radiographs are typically obtained but MRI rarely needed. EMG/NCV studies can be considered if the physical exam is concerning for nerve entrapment. Surgical options include open partial plantar fascia release with or without a tarsal tunnel release, gastrocnemius recession, and minimally invasive ultrasound guided

release such as the Tenex procedure. Extracorporeal shock wave therapy (ESWT) can also be considered.

---

## Rehabilitation and Back-to-Sports

Physical therapy should be the initial treatment for patients with lateral ankle instability after a brief period of rest. Early proprioceptive training and peroneal tendon strengthening programs are necessary. This usually involves balance boards and Theraband conditioning against resistance. Taping or bracing can also be helpful during recovery. Once the golfers swelling and pain are diminished return to sport can be initiated typically with a brace. Home exercises can help decrease the risk of recurrence and speed up the rehabilitation process. Once daily activities cause no pain or instability return to sport can be considered depending on the patient as well as the Grade of injury. Return to sport can take up to 3 months after an acute ankle sprain. Luckily, golf does not required cutting maneuvers and return to this sport can be quicker as a result.

Plantar fasciitis typically does not require a prolonged period out of sport. Since this is a chronic overuse condition, as long as the golfer can play through the pain there is no harm in returning to golf. A consistent stretching program is the mainstay to plantar fasciitis treatment and this can be done during a round of golf.

---

## Prevention

The best prevention for ankle sprains is to wear good supportive shoes. An orthotic may also help depending on the foot posture. If one has a cavovarus foot lateral posting in an orthotic may help reduce lateral ankle instability. Also strengthening the peroneal tendons and supportive structures around the ankle can be helpful. This can be done at home with Theraband exercises and proprioception training. A lace up ankle brace can also be used to keep the foot and ankle in a stable posture if the golfer has a history of ankle instability. Ensuring the golfer's foot makes solid contact with the ground when hitting off uneven terrain can help prevent ankle injuries.

Plantar fasciitis can be prevented by a stretching program focusing on the triceps surae. There are several protocols that involve both eccentric exercises for the Achilles tendon and plantar fascia specific stretches. Inexpensive over the counter orthotics with a cushioned heel or heel cups alone can help. These can typically be placed in a good supportive golf shoe.

General guidelines for prevention of ankle/foot injuries in golf include warming up before playing with controlled calf stretching/strengthening exercises and ankle range of motion. Before beginning a round of golf, take practice swings to roll



through your feet and ankles in a controlled manner. The pain of plantar fasciitis can be controlled by stretching during the golf round such as after every hole, or waiting for the next shot. Consideration of a golf cart may be necessary if one has foot or ankle problems. If you must ride, keeping the foot and ankle inside the cart is essential to prevent fractures, and degloving injuries which could occur when the extremity is hanging out of the cart.

## Evidence

There is limited if any Level I evidence with Grade A recommendations on treating specifically golf foot and ankle injuries. There are several Level II and III studies regarding the modified Brostrum procedure in general. Messer et al. conducted a Level III study that found fourteen of the sixteen patients who underwent the modified Brostrum procedure had no evidence of instability on physical examination or on stress radiographs at 34 months. However, five of the 16 patients had generalized ligamentous laxity [8]. More recently, Li et al in a case series (Level IV) found that 94% of high level athletes with Grade III ankle sprain that failed 6 months of conservative measures were able to return to their preinjury functional level at two years [9]. Based on the evidence available, a Grade B recommendation exists for surgical stabilization with a modified Brostrum. In regards to nonoperative measures, Hupperets et al in a Level I randomized control trial found that proprioceptive training program after usual care of an ankle sprain is effective for the prevention of self reported recurrences, a Grade B recommendation [10].

In regards to plantar fasciitis, DiGiovanni et al compared two stretching protocols and showed that heel pain was eliminated or improved at 8 weeks in 52% of patients treated with the plantar fascia specific stretching program versus only 22% of patients in the Achilles tendon stretching program. At 2-year follow-up, the study reported no difference between the two groups with 92% of all patients reporting satisfaction (Level III) [11]. A case-control study by Riddle et al found that reduced ankle dorsiflexion was the strongest independent risk factor for development of plantar fasciitis (Level III) [5] suggesting that stretching can be efficacious. For symptom duration less than 4 months, tissue-specific stretching was favored over anti-inflammatory or orthotic modalities based on a survey study of AOFAS members by DiGiovanni. Once more than 10 months have lapsed, nearly 75% of surgeons would recommend a variety of surgical procedures or shock wave therapy for chronic plantar fasciitis [12]. Based on the evidence, there is a Grade B recommendation for conservative measures with stretching as treatment for plantar fasciitis. As for surgical intervention, this is opinion only and no Grade

can be assigned based on current evidence and the variety of surgical procedures available.

## Summary

- Although not specific to golf, lateral ankle sprains and plantar fasciitis are the most common foot and ankle condition a clinician may encounter.
- Lateral ankle sprains typically resolve with conservative measures.
- If chronic lateral instability develops, surgical options exist with good long term results.
- Plantar fasciitis typically resolves with conservative measures with the cornerstone being calf stretching.
- Surgical options do exist for plantar fasciitis and continue to evolve from the standard open partial plantar fascia release.

## References

1. Batt ME. A survey of golf injuries in amateur golfers. *Br J Sports Med.* 1992;26(1):63–5.
2. Gosheger G, Liem D, Ludwig K, Greshake O, Winkelmann W. Injuries and overuse syndromes in golf. *Am J Sports Med.* 2003;31(3):438–43.
3. McHardy A, Pollard H, Luo K. One-year follow-up study on golf injuries in Australian amateur golfers. *Am J Sports Med.* 2007;35(8):1354–60.
4. Baxter DE, Pfeffer GB. Treatment of chronic heel pain by surgical release of the first branch of the lateral plantar nerve. *Clin Orthop Relat Res.* 1992;279:229–36.
5. Riddle DL, Pulisic M, Pidcoe P. Risk factors for plantar fasciitis: a matched case-control study. *J Bone Joint Surg Am.* 2003;85:872–7.
6. American Academy of Orthopaedic Surgeons. Sprained ankle. 2014. [www.orthoinfo.aaos.org](http://www.orthoinfo.aaos.org).
7. DeLee JC, Drez Jr D, Miller MD. *Orthopaedic sports medicine.* 2nd ed. Philadelphia: WB Saunders; 2009.
8. Messer TM, Cummins CA, Ahn J, Kelikian AS. Outcome of the modified Broström procedure for chronic lateral ankle instability using suture anchors. *Foot Ankle Int.* 2000;21(12):996–1003.
9. Li X, Killie H, Guerrero P, Busconi BD. Anatomical reconstruction for chronic lateral ankle instability in the high-demand athlete: functional outcomes after the modified Broström repair using suture anchors. *Am J Sports Med.* 2009;37(3):488–94.
10. Hupperets MD, Verhagen EA, van Mechelen W. Effect of unsupervised home based proprioceptive training on recurrences of ankle sprain: randomised controlled trial. *BMJ.* 2009;339:b2684.
11. DiGiovanni BF, Nawoczenski DA, Malay DP, Graci PA, Williams TT, Wilding GE, Baumhauer JF. Plantar fascia-specific stretching exercise improves outcomes in patients with chronic plantar fasciitis. A prospective clinical trial with two-year follow-up. *J Bone Joint Surg Am.* 2006;88(8):1775–81.
12. DiGiovanni BF, Moore AM, Zlotnicki JP, Pinney SJ. Preferred management of recalcitrant plantar fasciitis among orthopaedic foot and ankle surgeons. *Foot Ankle Int.* 2012;33(6):507–12.

Larry Nassar

**Abstract**

Artistic gymnastics is a sport in which the foot and ankle is the most commonly injured body area. The forces on the foot and ankle are very high in this barefoot sport. Simple inversion ankle sprains are the most frequent injury but more significant severe injuries can occur. Ottawa Ankle Rules are beneficial but remember their growth plates may remain open longer than usual since their maturation may be delayed. Rehabilitation needs to be done barefooted since it is a barefoot sport. Joint play of the foot and ankle is of therapeutic benefit. Progressions of force by performing a jump program may be an asset to assisting the gymnast back to safe performance. Skills progression needs to be done in manner in which a gradual progression of forces is reintroduced after an injury. Proper use of training aids can be a significant asset for injury prevention. Open and closed chain exercises, intrinsic and extrinsic muscle strength as well as static and dynamic proprioception/kinesthetic awareness exercises are all vital for a proper injury prevention program to have the best outcome.

**Keywords**

Gymnastics • Artistic • Foot • Ankle • Rehabilitation • Prevention • Manipulation • Training • Forces • Injury

**Introduction**

Gymnastics is composed of several different sub groups within the sport. Trampoline and tumbling are one discipline and the trampoline component has recently been added to the Olympics. Acrobatics is another subset where partner stunting is involved and it has not been added to the Olympics yet. Rhythmic gymnastics is either done in a group as a team or as an individual and both are Olympic sports. Men's and Women's artistic gymnastics are the most popular Olympic sports. All of these sports have foot and ankle as one of their most commonly injured joints. This chapter is focused primarily on the artistic gymnast.

---

L. Nassar, DO, FAOASM  
Department of Radiology, Division of Sports Medicine,  
Michigan State University,  
4660 South Hagadorn Road Suite 420, East Lansing,  
MI 48823, USA  
e-mail: [Larry.Nassar@gmail.com](mailto:Larry.Nassar@gmail.com)

**Injuries**

Artistic gymnasts sustain a variety of foot and ankle injuries. Common injuries include:

1. Anterior ankle impingement and bone spurs and high ankle sprains
2. Posterior ankle impingement
3. Medial ankle sprain
4. Lateral ankle sprain
5. Lisfrank joint sprain
6. Severs, calcaneal apophysitis
7. Achilles tendonopathy
8. 1st MTP sprain
9. Sesamoiditis
10. Somatic dysfunction of the foot and ankle
11. Stress fracture of the foot (cuboid, navicular, and metatarsals)
12. Traumatic fractures to the foot and ankle

## Etiology and Pathomechanism

The forces through the foot and ankle in artistic gymnastics are very high. The Achilles tendon sustains a force of 9000 N (about 15 times body weight) when the gymnast takes off from the floor to perform a double back salto [1]. When landing the double back salto the Achilles tendon sustains a force of 25,000 N [2]. Fortunately, due to appropriate progressive training over the years, the tendinous tissue of a trained gymnast is able to adapt and sustain these excessive forces.

On the balance beam, the female artistic gymnast sustains up to 8000 N with landings at the talonavicular joint. For the vault, the basic handspring landing has a peak impact ground reaction force of 10.3 times body weight [3]. Overall, from the research that has been done in gymnastics, it can be summarized that, in general, the ground reaction forces during tumbling take offs from the floor exercise mat range from 10 to 14 times body weight depending on the various tumbling skills.

The gymnast is able to train through these high forces with the use of training mats (sting mats) that can help decrease these forces. The real issue is when a gymnast performs these skills and they mistakenly place their ankle/foot in an inversion/supinated and everted/pronated position, the forces become even more excessive and injury may then occur. When a gymnast lands partially off a mat or in a space between two mats, the forces become overwhelming and significant injury occurs.

## Epidemiology

Artistic gymnastics is a sport in which every 4 years the rules change. In addition, there are relatively frequent changes in equipment. This changes the nature of the epidemiology of the sport basically every 4 years. This may explain why there has been no published prevalence data on lower extremity injuries affecting the female artistic gymnast. However, the foot and ankle always are the number one area of injury, in both men and women artistic gymnastics. When epidemiology studies are done, the inversion ankle sprain being the most frequent lower extremity injury, followed by calcaneal apophysitis [4, 5].

## Therapy

When a gymnast sprains her foot/ankle, it is important to use compression to help reduce the amount of swelling. In addition to the compression, ice may be applied, as well as elevation. These are the classic ways to treat acute swelling. In addition, the use of kinesio tape for swelling reduction may be applied first to the skin in a fashion to help with lymphatic drainage of the area.

Follow up imaging after initial injury is important. The use of the Ottawa Ankle Rules [6] are beneficial. However, as

many gymnasts still have open growth plates, always assess the growth plates for injury. When radiographs are performed, it is best to have comparison films done on the opposite side to best assess for growth plate injury. In addition, with anterior and posterior ankle impingement radiographs of the ankle are beneficial to look for bone spurs, bone fragments and an enlarged posterior talar process or os trigonum. For Lisfranc injury, weight bearing radiographs are important to assess the gapping at the Lisfranc joint.

The use of musculoskeletal ultrasound is beneficial in assessing ligament, tendon and bone pathology. It is becoming more common for this imaging device to be present on site at competitions and is of benefit to assess the acute injury. For impingement, dynamic musculo-skeletal ultrasound may prove of benefit to actually see the impingement as the ankle is dorsi flexed or plantar flexed.

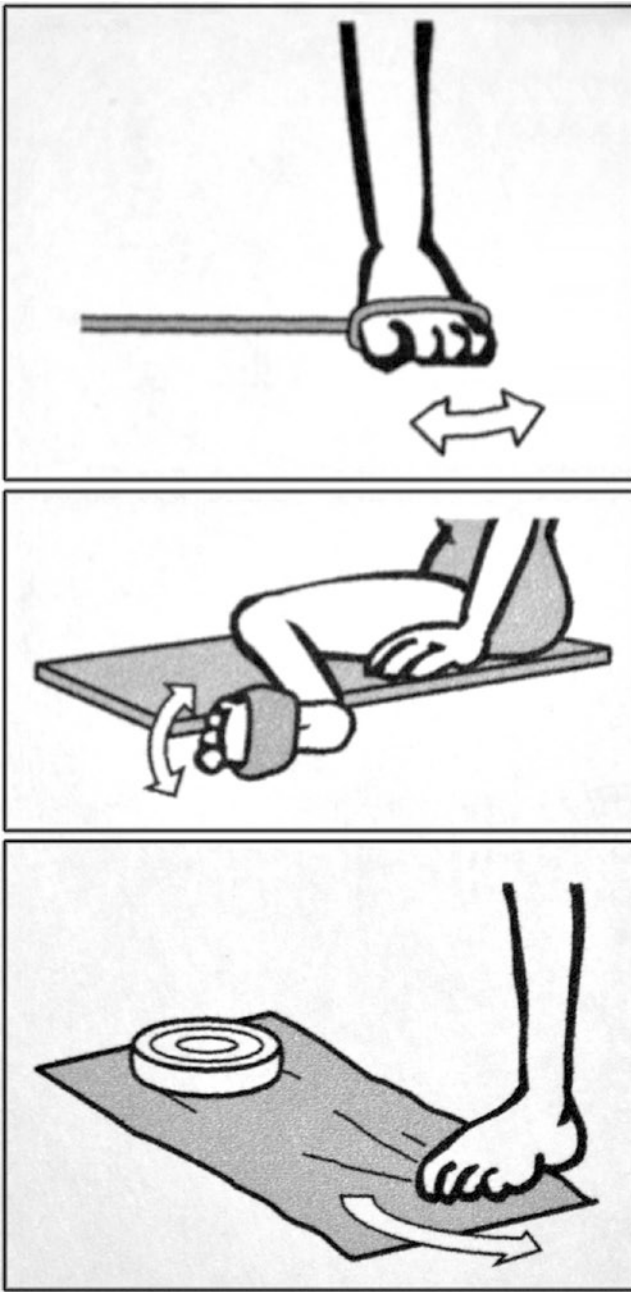
The use of platelet rich plasma (PRP) and prolotherapy have become more commonly employed to assist in ligament/tendon healing. The prolotherapy may assist in scarring of the ligaments to provide some stability to the foot/ankle. The PRP may assist with the healing of tissue. For sesamoid bone injury, it is important to perform imaging studies to see if there is a fracture. For sesamoid pain and 1st MTP sprain a hard shoe insert with a cut out for the 1st MTP may be of benefit, either with a hard shoe or a walking boot.

## Rehabilitation and Back to Sports

Rehabilitation of the foot/ankle for the artistic gymnast needs to be done in bare feet as they are a barefooted athlete. Work the extrinsic as well as the intrinsic muscle groups (Fig. 55.1). Static and dynamic proprioceptive/kinesthetic awareness and stability exercises are vital in the rehabilitation process.

Joint play to enhance talar tibial and tibial talar motions as well as the fibula on the talus [7]. This helps with proper plantar and dorsi flexion. The fibula frequently is anteriorly displaced when the gymnast sprains her ankle [8]. There then can be a restriction of ankle dorsiflexion of the talus on the tibia and the tibia on the talus with an anterior lateral ankle impingement. The treatment is to do joint mobilization to first ensure the fibula is mobilized back posteriorly to its neutral position relative to the tibia and talus (Fig. 55.2). This mobilization may best be done in both the open and closed chain positions. The proximal tibial fibular articulation needs to be assessed to ensure its proper position and motion too.

The high impact forces may create a depressed cuboid, pronated navicular, and restricted 1st metatarsal phalangeal joint motion. In addition the subtalar joint motion needs to be assessed. Joint play manipulation to these articulations may enhance the function and recovery of the injured gymnast.



**Fig. 55.1** Ankle eversion using therabands and weights for extrinsic muscle strength and towel toe crunches for intrinsic muscle strength

In addition to joint play, acupuncture is frequently employed with the care of the gymnasts on the USA Gymnastics National Team as an adjunctive therapeutic treatment. This therapeutic intervention has assisted these gymnasts in recovery from injury and sustained world class performance.

The lisfranc joint sprain has a multiple classification system. Surgery is indicated in many cases. For the more minor sprain of the lisfranc joint that is non-surgical, there are therapeutic interventions that can help. Leukotape P® taping of the 1st metatarsal plantarward is key. Do not lift with the medial arch.

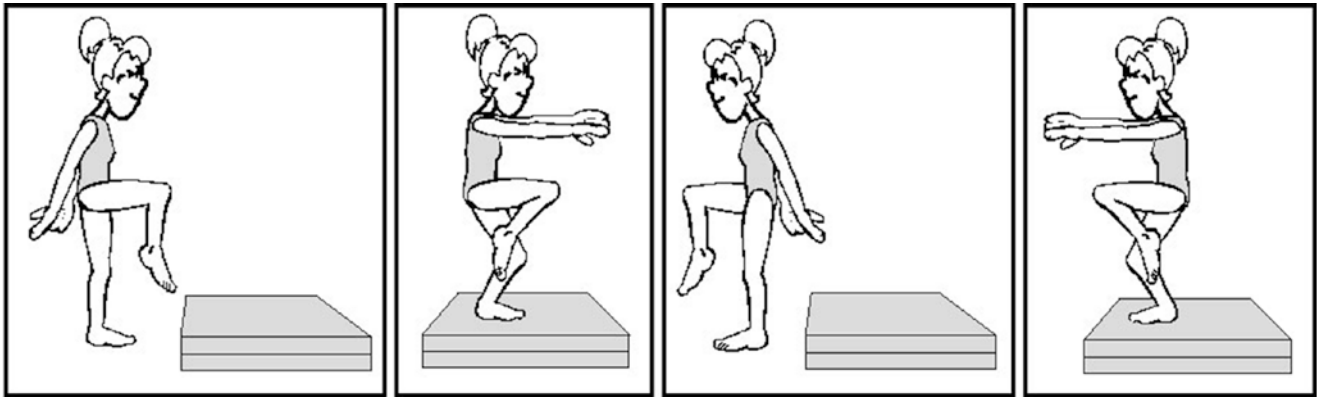


**Fig. 55.2** Anterior lateral ankle impingement located at the position of the “Lightning Bolt” may be caused from an anteriorly displaced fibula as the result of an inversion ankle sprain. Mobilization of the distal fibula head from anterior to posterior in the direction of the arrow may help resolve the impingement

Return to gymnastics is best done with a sample outline of progressions that can be modified to fit the specific needs of the gymnast. Here is an example of this sample outline:

1. Strengthen the foot and ankle intrinsic and extrinsic muscles
2. Strengthen in both the open and closed chain positions
3. Static and then dynamic proprioception/kinesthetic awareness
4. Return to closed chain impact jogging and progress to jump rope as the gymnast proceeds to the jumping progression
5. Jumping progression drills to allow for progression back to gymnastics skills.

The jumping progression consists of an incremental increase of height of jumps. Gymnasts perform mainly in forward and backward movements so the progression has jumps forward and backwards. It is important for them not to compensate so single leg jumps need to be incorporated. Separate



**Fig. 55.3** Single leg jump up/down forward and backwards

the jumps into both phases - jump up and jump down. This helps isolate the ability to perform the push through the foot and ankle upwards off the ground first. Then the ability to land and absorb the impact force of landing. It is important to also ensure good form is used for both phases of the jump and by separating these, it is easier for the gymnast to accomplish this goal. Finally, based upon the gymnast's ability to jump, skills can be added back to their training.

Jumping progression sample (Fig. 55.3):

1. Jump up double legged forward 6 in.
2. Jump up double legged backwards 6 in.
3. Jump up double legged forward/backwards 12 in. and jump down double legged forward/backwards 6 in.
4. Jump up double legged 18 in.<sup>1</sup> forward and backwards and jump down double legged forward and backwards 12 in. and then add single leg jump up forward/backward 6 in..
5. Jump down 24 in. two legged forward/backwards, jump up single legged 12 in. forward and backwards and jump down 6 in. single legged forward/backwards.
6. Jump down 36 in. two legged forward/backwards, jump down single legged 12 in. forward and backwards
7. Jump down 48 in. (the height of the high beam) double legged forward/backwards and jump down single legged 18 in. forward/backwards

To help with the progression back to gymnastics skills, it is recommended to use the Tumble Trak® aids that are commonly found in the gymnastics clubs. By following the Jump Progression, the gymnast builds confidence in her injured extremity and the coach is able to assess the gymnast's power and form in a more safe and controlled set of drills than by just attempting gymnastics skills. By performing these jump drills, it then becomes safer to return to the gymnastics skills.

<sup>1</sup>Jump up 18 in. is typically as high as needed, but the jump down can increase to help with the landing forces.

### Sample Skill Progression

- Once able to do the 12 in. jump up with both legs. The gymnast should be able to do jumps on the trampoline (tramp).
- Once able to do 18 in. double leg jump ups, 12 in. double leg jump down and 6 in. single leg jump up, she should be able to do leaps on the tramp, jumps on the floor exercise mat and start basic tumbling on the Tumble Trak® landing into a safety pit.
- Once able to do the 12 in. jump up single leg and 6 in. jump down single leg, they should be able to try leaps involving the injured leg on the floor exercise mat and jumps on the beam. Sometimes the gymnast is able to tumble before doing leaps since the leap may require more force through the injured extremity than the tumbling.
- Once able to do 24 in. double leg jump ups and 18 in. double leg jump downs, then start basic tumbling passes on the floor exercise mat and she may be able to start leaps on beam.
- It is actually more difficult in many cases to do standing back handsprings as opposed to doing round-off back handsprings. The standing back handspring requires more strength and power to explode off the mat from a stance than what is needed to do the round-off back handsprings.
- The round off, however, sometimes is more difficult to return to than the back handspring. The lead leg in the round off may place more force on the anterior ankle which may create more discomfort. So this depends on the dominance of the leg in their skills.
- Front tumbling can be attempted at the same time as back tumbling. Depending on the skill of the gymnast and the injury, some gymnasts are able to do front tumbling with less difficulty than back tumbling especially if the injury is an anterior ankle problem.
- The progression of the leaps and jumps to the beam is more difficult for the gymnast to perform than the same skill on the floor exercise. This is because the beam is

more rigid than the floor, requires more range of motion to fit the feet on the beam properly, and requires better balance/proprioception than on the floor exercise mat.

- If a Tumble Trak® Air floor is available in the gymnastics club, then progress the tumbling from the Tumble Trak® to the Tumble Trak® Air Floor and then to the regular floor exercise mat.
- Once able to perform tumble basics on the floor exercise mat, vault drills may be started.
- Tumbling on the balance beam is started with first tumbling the skills on the floor, then the low beam and then finally on the high beam.
- Dismounts should always be done into a safety pit at first.

---

## Prevention

Gymnasts need to work on injury prevention for the foot and ankle throughout the gymnastics season. Incorporated into each practice a variety of foot and ankle exercises may be included and progressed throughout the season. As mentioned earlier, open and closed chain exercises, intrinsic and extrinsic muscle strength, and finally static and dynamic proprioception/kinesthetic awareness exercises need to be incorporated. In addition, proper upkeep of the gymnastics equipment is vital for injury prevention. The safety landing mats, floor exercise mats, and the vault runway must all be replaced once they are worn out. The use of training aids like “sting” mats, trampolines/Tumble Traks®, air floors, beam pads, and landing pits all can help reduce the forces on the gymnasts feet/ankles and aid in injury prevention when used properly.

---

## Summary

1. The forces endured by the artistic gymnast are very high and proper positioning is vital to decrease injury.
2. The rules in the sport of artistic gymnastics change significantly every 4 year Olympic cycle. This creates

difficulty in performing epidemiology and comparing injuries. However, it is accepted that the foot and ankle are the most commonly injured joints.

3. The use of proper training aids and proper maintenance of equipment is important for reduction of forces and injury prevention.
4. A multifactorial approach to therapy is recommended. Ensuring proper joint play, muscle strength and proprioception/kinesthetic awareness all help enhance the recovery of the injured gymnast.
5. The performance of jumping drills assists with the progression of the injured gymnast back to her skills.

---

## References

1. Brueggemann GP. Mechanical load of the Achilles tendon during rapid sport movements. In: Perren SM, Scheider M, editors. *Biomechanics: current interdisciplinary research*. Dordrecht: Martinus, Nijhoff; 1985.
2. Panzer VP. Dynamic assessment of lower extremity load characteristics during landing [PhD]. University of Oregon; 1987. Dufek, JS, Bates BT. Biomechanical factors associated with injury during landing in jump sports. *Sports Medicine*. 1991; 12(5): 326–337.
3. Hall SJ. Mechanical contributions to lumbar stress injuries in female gymnasts. *Med Sci Sports Exerc*. 1986;18:599–602.
4. Dixon M, Fricker P. Injuries in elite gymnasts over 10 years. *Med Sci Sports Exerc*. 1993;25:1322–9.
5. Mackie SJ, Taunton JE. Injuries in female gymnasts. Trends Suggest Prevent Tactics. 1994;22:40–5.
6. Stiell IG, Greenberg GH, McKnight RD, Nair RC, McDowell I, Reardon M. Decision rules for the use of radiography in acute ankle injuries. Refinement and prospective validation. *JAMA*. 1993;269: 1127–32.
7. Mennell JM. *Joint pain*. Boston: Little, Brown and Company; 1964.
8. Kavanagh J. Is there a positional fault at the inferior tibiofibular joint in patients with acute or chronic ankle sprains compared to normal. *Man Ther*. 1999;4(1):19–24.

Christian Stelzenbach and Jochen Paul

**Abstract**

Team handball is one of the most popular sports worldwide and injuries of foot and ankle are common. Most of the injuries are ankle sprains and ligament injuries, tendon injuries, muscle injuries but even fractures might occur. Injuries can be divided into contact and non-contact injuries and the non-contact ones are more frequent. Probably as a result of fatigue. The trauma mechanism seems to be turning, landing after jumping and changing the direction with the feet in eversion and supine position. Contact injuries occur commonly while getting tackled by an opponent player. The injuries can be treated conservatively or with surgical procedures depending on the severity of the injury and concomitant lesions. The clinical examination is essential for choosing the best treatment option. An accompanying functional rehabilitation program is recommended for a fast return to sports. Proprioceptive training and coordination exercises seem to be quite important to reduce the frequency of recurrent ankle sprains and to decrease the risk of re-injuries.

**Keywords**

Ankle sprain • Ankle instability • Return to sports • Handball

**Injuries**

Team handball is one of the most popular sports worldwide. Injuries can happen in handball like in other team activities and most injuries appear to be located on the lower extremities (54%), especially at the ankle and knee [1–3]. Most of foot and ankle injuries are ankle sprains [1, 4–6]. In general, contusions, ligament injuries (beside ankle: talonavicular ligament, Lisfranc ligament,...), tendon injuries (as Achilles tendon), and fractures (e.g. Malleoli, Metatarsalia, Talus, etc) might occur in handball [3, 4, 7].

---

C. Stelzenbach, MD  
Orthopaedic Department, University Hospital, University of Basel,  
Basel, Switzerland  
e-mail: [christian.stelzenbach@usb.ch](mailto:christian.stelzenbach@usb.ch)

J. Paul, MD (✉)  
Rennbahnklinik, Kriegackerstrasse 100,  
Basel CH-4132, Muttenz, Switzerland  
e-mail: [jochen.paul@rennbahnklinik.ch](mailto:jochen.paul@rennbahnklinik.ch)

**Etiology and Pathomechanism**

Sport injuries of the foot and ankle occur frequently and about 70% of severe injuries are related to the lower extremities [8]. Ankle sprains seem to be the most frequent diagnosis leading to a handball player dropped out of a game [3]. Many of the injuries (30–50%) during competition are due to overuse and a quite high number of injuries occur in the second half of a handball game [2, 3, 8, 9]. On the one hand that might be caused by muscle exhaustion, lack of carbohydrates and the central neural fatigue. On the other hand it might be caused by the attempt of players to change the final result and they might take more risk [2, 8]. Furthermore the etiology of injuries can be divided in contact and non-contact injuries. Giza et al. (2003) reported that 35% of the injuries were in consequence of contact with an opponent player while 65% happened without contact [9]. Another study group showed that 69% of the injuries of the lower extremities happened without contact to an opponent player [8]. At the end of a game the non-contact-injuries were incurred (58%) as a result

of fatigue like above described [3]. Loss of body control while getting tackled seems to be the most common reason for a contact injury [2]. Furthermore the position of the handball players appears to be relevant. The higher injury incidence of the back players (41 %) (followed by center players (19%), goalkeepers (16%), wing players (12%) and line players (11%)) can be attributed to burst movement, quick change of direction and more severe and numerous collisions with the opponent players with the consequence of exhaustion [8, 10]. The trauma mechanism of injuries seem to be changing direction, turning and landing after a jump (feet are in a supine position and in eversion) [2, 8, 9, 11]. Piry et al. (2011) described that plant and cutting, (which means rotating your body while your foot is still on the ground) was the main mechanism (28.5 %) leading to injuries [8].

## Epidemiology

In a recent review Fong et al. (2007) considered articles published over 29 years, from 1977 to 2005 [4]. They described, that prevalence of ankle injuries has been ranked as the most injured body site in most studies (24 sports, 34.3 %) [4]. But in this cases only the frequency of occurrence is described and conclusions of the severity of injuries cannot be drawn [4]. This results were confirmed by another study which showed that the highest incidence of injuries in handball happens at the ankle (23.8 %), followed by knee injuries (15.9 %) [8]. Within the 43 sports providing information about ankle injuries, in 33 sports ankle sprains were the most common injury. In handball the reported ankle injuries were all ankle sprains. The incidence rate of all injuries during handball training and competition per 1000 person-hours was 1.59 and during competition only the incidence rate per 1000 person-hours was measured with 1.32 [2, 6]. Especially the incidence rate of ankle and foot injuries was measured with 0.8 (0.5–1.1)/1000 exposure hours in that prospective study with players on a superior nonprofessional level [6]. Piry et al. (2011) described, that the incidence of injury per 1000 h of training was 21.5 times less than the incidence of injuries per 1000 h in competition, which might be due to the higher intensity (physical and psychological) during the matches or be reflective by the nature of competition [8, 10]. In a review about overuse of foot and ankle in sports Sobhani et al. (2012) reported, that stress fractures, Achilles tendinopathis and plantar fasciitis were the most commonly reported problems due to overuse which is explained by repetitive component of the lower extremity [12]. However, in that review the studies were focused only on elite athletes and studies about amateur groups were rare (4 %). So injury rates between amateur and elite athletes cannot be compared. The measured results of injury incidence are heterogeneous. They vary between 4.7 [13] and 114 [14] injuries per 1000 h of competition such as 0.4 [13] and 0.96 [3] injuries per 1000 h of training.

## Therapy

Acute therapy focuses on controlling pain and swelling. Therefore, first of all on the field the injured foot and/or ankle should be elevated and cooled. An early compression prevents swelling. The first line treatment is also known as the PRICE scheme: Protection, Rest, Ice, Compression and Elevation. It is also reported, that applying ice and using non-inflammatory drugs improve healing and speeds recovery [15, 16]. Beside these acute treatment there are three more treatment options: Protection of the ankle with orthotics, bandages, walkers or braces (according to grade of distortion), functional therapy with physiotherapy and operative treatment [17]. The exact clinical examination is essential to choose the sufficient treatment option. Without concomitant injuries a conservative therapy is initially aspired. The functional treatment after injuries of the foot and ankle shows better results in comparison to immobilization. A higher percentage of patients returned to sport, the time to return to work was shorter, and fewer patients suffered from objective instability and persistent swelling. In addition the functionally treated patients were more satisfied [18].

In convalescence handball specific training with high impact forces (e.g. changing direction, jumping and uncontrolled movements while getting attacked by an opponent player) should be avoided during this time. Accompanying physiotherapy with lymphatic drainage can be performed. Functional treatment is possible and recommended to avoid the well documented degenerative effects of immobilization on bone, ligaments, joint cartilage, muscles and tendons [19]. Proprioceptive training with a tilt board should be started as soon as possible, usually after 2–4 weeks to improve the neurovascular control and balance of the ankle. The efficacy of tilt board training is described in some studies, were it improves compensatory mechanisms and therefore another distortion ankle trauma can be avoided [20–22]. Additional mobility training with muscle exercises, especially the peroneus muscle strengthening is recommended to reduce the period of disability [22].

Operative treatment is rare and depends on the severity of injury and the requirement profile (e.g. professional handball players vs. amateur players) [17]. Indications for an operative treatment are eversion injuries with medial ligamentous injury, non-stable syndesmotic injuries, symptomatic osteochondral lesions of the talus and osseous ligamentous ruptures, or fractures. In professional sports an early operative treatment might bring the athlete faster back to a high level of sport and functionally therapy [23]. In some cases chronic ankle instability beside persistent synovitis or tendinitis, ankle stiffness, pain and swelling can develop. In that case a surgical repair might be appropriate. The treatment option depends on the special kind of injuries and are described in the appropriate chapters in this book.



## Rehabilitation and Back-to-Sports

Rehabilitation after injuries at the musculoskeletal system, respectively in foot and ankle, is very important, because the risk of re-injury is high. Often the next injury happens to athletes while they are not being totally recovered. Various foot and ankle scoring systems exist, but none of them have been validated the return to sport decisions [24]. Therefore, it is difficult to determine the critical point at which the athlete can start playing handball or return to competition again. Functional rehabilitation - like strengthening exercises, proprioceptive training and motion restoration, like above mentioned, - is preferred [15]. Clinical tests during the rehabilitation (tests of proprioception, range of motion, agility, and strength) assess physical readiness and can help with decision making of physical activity easier [24]. Miyamoto et al. (2014) showed, that athletes treated by an accelerated rehabilitation program after anterior talofibular ligament (ATFL) reconstruction returned 5 weeks earlier to full athletic activity than those ones treated by traditional rehabilitation with immobilization [25]. In patients receiving surgical treatment for ankle instability, a return to sports activities is allowed approximately three months after surgery and a mechanical ankle support may be needed for about 6 months [22].

More information about rehabilitation and back to sports is given in the specific chapter in this book.

---

## Prevention

Injuries in team sports are common. Therefore, programs with the aim of injury prevention are important. To our knowledge there are no specific prevention programs for handball, but recently published studies showed that it is possible to prevent lower extremity injuries in handball [13, 26, 27]. They described, that the incidence of injury could be reduced significantly by implementing a structured warm-up program to improve running, cutting and landing techniques as well as neuromuscular control, balance and strength [13]. The peroneal muscle group plays a large role in dynamically stabilizing the lateral ankle-foot complex against an injurious moment [28–30]. In a comparison study of various ankle support devices with respect to inversion they demonstrated that taping and semirigid orthosis had a greater restriction than softshell or lace-up style brace [31]. Proprioceptive training and coordination practices seem to be important to reduce the frequency of recurrent ankle sprains [32]. Using appropriate techniques in changing the direction, landing and turning, can effectively decrease injuries of non-contact type [8]. Adaptation of the applied handball rules, training and implementing preventive plans seem to decrease the injuries - especially the severe ones [8]. In addition, more intensive and coordinated exercise for the muscles of the lower

extremity and taping could reduce the incidence of ankle sprain [33]. However, better primary treatment and programs for rehabilitation after instructions from physicians might reduce the incidence of re-injury [2]. Fong et al. (2007) also recommended, that ankle sprain prevention should be implemented in sports like handball [4]. Close cooperation with the referees might help to make handball a safer sport [3]. Also the floor type might have an influence of the incidence of injuries. Already it has been shown, that the risk of injuries increases on artificial floor (with generally higher friction) compared to wooden floor like parquet (with generally lower friction) [33]. Like above mentioned, proprioceptive exercises should be a part of every handball training. Moreover, adapting the recent handball rules like more breaks during competition or complete prohibition of attacking an athlete while jumping can help to decrease injuries. Thus, more evidence in the field of the type of floor, training methods and modulation of competition rules is necessary to prevent more injuries. However, by all means, all injuries cannot be avoided in sports.

## Case Report: Handball Player with Chronic Lateral Ankle Instability

A 24-year-old non-professional handball player suffered a distortion of his left ankle during a handball game. Immediately he felt pain, his ankle was swollen and a few days later there was a hematoma around the ankle. He started a conservative therapy with physiotherapy and local application of NSAID. Half a year later he complained about instability and persistent pain. He described a pressure pain in the area of the ATFL and the ventral ankle. Clinically the ankle was unstable lateral (lateral tilt ++) and the anterior-draw-test was positive. We did not find any osseous lesions in standard radiographs (Fig. 56.1). As the conservative therapy was unsuccessfully after another 3 months and symptoms persisted we recommended a surgical treatment. First we performed an arthroscopy of the ankle to determine intraarticular pathologies. A lateral instability with an anterior subluxation of the talus and scarred, lacerated lateral ligaments were observed. Therefore, we proceeded to open surgery. The ATFL was found to be elongated, the CFL well preserved but the connection of ATFL and CFL was divided. The ATFL and CFL were mobilized and cut at the insertion of the fibula. After preparation of the fibula with a rongeur we reinserted the ATFL and CFL with two transosseous sutures onto the fibula. Postoperatively the patient was mobilized in an Aircast-Walker with 15 kg weight bearing for 4 weeks and afterwards he began to raise the weight bearing slowly. Accompanying he got physiotherapy with proprioceptive training and could return to sport (jogging) after three months and to handball after five months.

**Fig. 56.1** Case report – Handball player with chronic lateral ankle instability. Radiographs of the ankle, Mortise and lateral view without any osseous injuries



## Summary

- Team handball is one of the most popular sports worldwide and injuries of foot and ankle are common.
- Most of the injuries are ankle sprains as well as contusions, ligament injuries, tendon lesions (Achilles), muscle injuries, and fractures.
- Injuries can be divided in contact and non-contact ones.
- Injuries can be treated conservative or surgical depending on the severity and concomitant injuries.
- A functional rehabilitation program is recommended to return to sport and prophylactic proprioceptive exercises should be performed to decrease the risk of re-injury.

## References

1. Dirx M, Bouter LM, de Geus GH. Aetiology of handball injuries: a case-control study. *Br J Sports Med.* 1992;26(3):121–4.
2. Nielsen A, Yde J. An epidemiologic and traumatologic study of injuries in handball. *Int J Sports Med.* 1988;09(05):341–4.
3. Langevoort G, Myklebust G, Dvorak J, Junge A. Handball injuries during major international tournaments. *Scand J Med Sci Sports.* 2007;17(4):400–7.
4. Fong DT-P, Hong Y, Chan L-K, Yung PS-H, Chan K-M. A systematic review on ankle injury and ankle sprain in sports. *Sports Med Auckl NZ.* 2007;37(1):73–94.
5. Yde J, Nielsen AB. Sports injuries in adolescents' ball games: soccer, handball and basketball. *Br J Sports Med.* 1990;24(1):51–4.
6. Seil R, Rupp S, Tempelhof S, Kohn D. Sports injuries in team handball a one-year prospective study of sixteen men's senior teams of a superior nonprofessional level. *Am J Sports Med.* 1998;26(5):681–7.
7. Valderrabano V, Perren T, Ryf C, Rillmann P, Hintermann B. Snowboarder's talus fracture treatment outcome of 20 cases after 3.5 years. *Am J Sports Med.* 2005;33(6):871–80.
8. Piry H, Fallahi A, Kordi R, Rajabi R, Rahimi M, Yosefi M. Handball injuries in Elite Asian Players. *Piry. World Appl Sci J.* 2011;14(10):1559–64.
9. Giza E, Fuller C, Junge A, Dvorak J. Mechanisms of foot and ankle injuries in soccer. *Am J Sports Med.* 2003;31(4):550–4.
10. Wedderkopp N, Kaltoft M, Lundgaard B, Rosendahl M, Froberg K. Injuries in young female players in European team handball. *Scand J Med Sci Sports.* 1997;7(6):342–7.
11. Olsen O-E, Myklebust G, Engebretsen L, Bahr R. Injury pattern in youth team handball: a comparison of two prospective registration methods. *Scand J Med Sci Sports.* 2006;16(6):426–32.
12. Sobhani S, Dekker R, Postema K, Dijkstra PU. Epidemiology of ankle and foot overuse injuries in sports: a systematic review. *Scand J Med Sci Sports.* 2013;23(6):669–86.
13. Olsen O-E, Myklebust G, Engebretsen L, Holme I, Bahr R. Exercises to prevent lower limb injuries in youth sports: cluster randomised controlled trial. *BMJ.* 2005;330(7489):449.
14. Junge A, Langevoort G, Pipe A, Peytavin A, Wong F, Mountjoy M, et al. Injuries in team sport tournaments during the 2004 Olympic games. *Am J Sports Med.* 2006;34(4):565–76.
15. Ivins D. Acute ankle sprain: an update. *Am Fam Physician.* 2006;74(10):1714.
16. Ogilvie-Harris DJ, Gilbert M. Treatment modalities for soft tissue injuries of the ankle. *Clin J Sport Med.* 1995;5(3):175–86.
17. Kerkhoffs GM, Handoll HH, de Bie R, Rowe BH, Struijs PA. Surgical versus conservative treatment for acute injuries of the lateral ligament complex of the ankle in adults. *Cochrane Database Syst Rev* 2007 Apr 18;(2).

18. Kerkhoffs GM, Rowe BH, Assendelft WJ, Kelly KD, Struijs PA, van Dijk CN. Immobilisation and functional treatment for acute lateral ankle ligament injuries in adults. *Cochrane Database Syst Rev* 2002(3).
19. Jozsa L, Thöring J, Järvinen M, Kannus P, Lehto M, Kvist M. Quantitative alterations in intramuscular connective tissue following immobilization: an experimental study in the rat calf muscles. *Exp Mol Pathol*. 1988;49(2):267–78.
20. Gauffin H, Tropp H, Odenrick P. Effect of ankle disk training on postural control in patients with functional instability of the ankle joint. *Int J Sports Med*. 1988;09(02):141–4.
21. Freeman MAR. Treatment of ruptures of the lateral ligament of the ankle. *J Bone Joint Surg Br*. 1965;47-B(4):661–8.
22. Lynch SA, DPAFH Renström. Treatment of acute lateral ankle ligament rupture in the athlete. *Sports Med*. 1999;27(1):61–71.
23. Coughlin MJ. Mann's surgery of the foot and ankle, 2-Volume Set 9th edition. UK Elsevier Health Bookshop [Internet]. ISBN: 9780323072427. [Cited 2014 Oct 21]. Available from: <http://www.eu.elsevierhealth.com/isbn/9780323033053/Medicine/Surgery-of-the-Foot-and-Ankle/Coughlin-Mann-and-Saltzman>.
24. Clanton TO, Matheny LM, Jarvis HC, Jeronimus AB. Return to play in athletes following ankle injuries. *Sports Health Multidiscip Approach*. 2012;11:1941738112463347.
25. Miyamoto W, Takao M, Yamada K, Matsushita T. Accelerated versus traditional rehabilitation after anterior talofibular ligament reconstruction for chronic lateral instability of the ankle in athletes. *Am J Sports Med*. 2014;42(6):1441–7.
26. Wedderkopp N, Kalltoft M, Holm R, Froberg K. Comparison of two intervention programmes in young female players in European handball – with and without ankle disc. *Scand J Med Sci Sports*. 2003;13(6):371–5.
27. Petersen W, Zantop T, Steensen M, Hypa A, Wessolowski T, Hassenpflug J. Prävention von Verletzungen der unteren Extremität im Handball: Erste Ergebnisse des Kieler Handball-Verletzungs-Präventionsprogrammes. *Sportverletz Sportschaden*. 2002;16(3):122–6.
28. Isakov E, Mizrahi J. Is balance impaired by recurrent sprained ankle? *Br J Sports Med*. 1997;31(1):65–7.
29. Konradsen L, Voigt M, Hojsgaard C. Ankle inversion injuries the role of the dynamic defense mechanism. *Am J Sports Med*. 1997;25(1):54–8.
30. Cordova ML, Ingersoll CD, Palmieri RM. Efficacy of Prophylactic Ankle Support: An Experimental Perspective. *J Athl Train*. 2002;37(4):446–57.
31. Comparison of Support Provided by Ankle Taping and Semirigid Orthosis. *J Orthop Sports Phys Ther*. 1987;9(1):33–9.
32. Tropp H, Askling C, Gillquist J. Prevention of ankle sprains. *Am J Sports Med*. 1985;13(4):259–62.
33. Olsen OE, Myklebust G, Engebretsen L, Holme I, Bahr R. Relationship between floor type and risk of ACL injury in team handball. *Scand J Med Sci Sports*. 2003;13(5):299–304.

Thomas Schlemmer, Andreas von Roll, Markus Wurm,  
and Victor Valderrabano

## Abstract

Hiking and mountaineering is one of the most popular sports worldwide. Despite hiking does not seem to be an activity with a high risk of injury the incidence is surprisingly high. Possible reasons are the unfamiliar environment in high altitudes and the overestimation of one's own capabilities. This chapter will show the risk factors of hiking and mountaineering, possible injuries, their treatment and prevention.

## Keywords

Hiking • Mountaineering • Frostbite • Blisters • Stress fracture • Hiking poles • Equipment

## Etiology, Risk Factors and Pathomechanisms

Severe injuries at the ankle and foot occur due to falling and spraining, often as a consequence of physical exhaustion or inadequate technique or equipment (Fig. 57.1). In high altitudes over 1500 m above sea level an acclimatization, even in young and healthy people is necessary to maintain physiological function. If there is too little time to adapt, high altitude sickness with cerebral edema and edema of the lung can occur. In high altitudes dehydration and hypovolemia can lead to loss of concentration and as a consequence injuries. Also the function of the visual system can be impaired in high altitudes due to ultraviolet radiation and low level of oxygen, this can also lead to missteps and injury [1].

Hypothermia in wet and cold environment is also a risk factor for injury [2]. And of course improper equipment as

inappropriate shoes and clothes can lead to injuries and death.

## Epidemiology

The typical hiker is male, between 30 and 40 years old [3, 4]. Most of the accidents happen at the weekend and in the afternoon [5]. 4 out of 100.000 hikers die every year, 37 % of the fatalities are related to falling [6, 7]. The numbers of hikers and mountaineers over the age of 50 years was increasing from 1975 until 2010 when 22 % of injured persons in hiking have been older than 50 years in the USA [8]. 56 % of all injuries are lower limb injuries, 9–34 % are fractures or dislocations [3, 4]. After distortions of the knee (17.5 %), ankle sprains are the second most frequent injury (15 %) [9].

## Acute Pathologies

### Blisters

Blistering is a frequent problem in hiking. Up to 57 % of participants report from blistering during hiking [10]. Blisters are a result of frictional forces that separate the epidermal cells at the level of the stratum spinosum [11]. Blisters occur when friction force is applied over a longer

T. Schlemmer, MD • M. Wurm, MD  
Orthopedic Department, University Hospital Basel,  
Basel, Switzerland  
e-mail: [thomas.schlemmer@usb.ch](mailto:thomas.schlemmer@usb.ch); [markus.wurm@usb.ch](mailto:markus.wurm@usb.ch)

A. von Roll, MD  
Orthopaedics Solothurn, Solothurn, Switzerland  
e-mail: [vonroll@orthopaedie-solothurn.ch](mailto:vonroll@orthopaedie-solothurn.ch)

V. Valderrabano, MD, PhD (✉)  
Orthopaedic Department, SWISS ORTHO CENTER,  
Schmerzklinik Basel, Swiss Medical Network, Basel, Switzerland  
e-mail: [vvalderrabano@gsmn.ch](mailto:vvalderrabano@gsmn.ch)

period, the higher the force the less cycles of stress on the skin are necessary for the development of blisters. Moist skin increases the friction force, very wet or very dry skin reduces the friction force [12]. As a result blistering occurs after more cycles.

The treatment of blisters is to keep the blisters intact whenever possible, otherwise the risk of secondary infection rises. To prevent infection, unroofing of accidental opened blisters is recommended. The unroofed blisters are best covered with hydrocolloid dressings. This kind of dressing decreases the pain and provides an ideal condition for healing [11].

So to prevent blisters it is necessary to reduce friction forces at the skin. Keeping foot dry is one option, wearing



**Fig. 57.1** Canyoning – An increasing sports causing foot and ankle injuries

proper socks another. Best results are found with socks made of acrylic fibers [13]. In general socks with low friction against skin and insole seem to be the best choice [14]. Antiperspirant solutions might bring an advantage but study results are mixed, it might reduce blistering but frequent skin irritation is seen [15]. Taping of endangered areas can also prevent blisters. Under repeated low intensity stress on the skin it comes to epidermal thickening and as a consequence less blistering [11].

## Sprains

As mentioned above the ankle sprain is the second most frequent injury in hiking activities [9]. Despite the often assumed, misleading minor severity of an ankle sprain it has a high potential for severe injuries with progression to a chronic condition. The ligaments most commonly involved are the anterior talofibular (ATFL) and calcaneofibular ligament (CFL). Misdiagnosed or undertreated ligament injuries can lead to chronic ankle instability, which is an acknowledged risk factor for the development of ligamentous post-traumatic ankle osteoarthritis [16]. Cartilage lesions are often seen in ankle instability. Up to 98 % of ankles with injuries of the deltoid ligament show cartilage damage, up to 66 % when the lateral ligaments are injured [17].

So in cases of ankle sprains with prolonged pain a chondral or osteochondral lesion or a chronic ligament instability should be considered. In fractures with higher force involved the risk is even bigger and an arthroscopy of the tibiotalar joint should be part of every operative fracture treatment at the ankle.

*For further detail please see Chap. 27 on acute ankle instability and Chap. 28 on chronic ankle instability.*

## Frostbites

Of all mountain injuries 22 % are related to hypothermia. Of all areas the feet are affected in 57 % of all cases, mainly the first toe [18]. Skin freeze occurs as soon as the temperature of the skin sinks under  $-4$  °C [19]. Frostbites are described in Grade 1–4 (Table 57.1). The on the field therapy includes rewarming in warm water of 37–39° Celcius for half an hour or until the color of the extremity turns into red or purple [20]. Rewarming should not be done if there is a possibility of refreezing until the arrival at a hospital, this can cause

**Table 57.1** Classification of frostbites

Grade 1	superficial, pallor, hypesthesia, edema
Grade 2	superficial, large blisters, filled with clear fluid, edema and erythema
Grade 3	deep, hemorrhagic blisters, black eschar
Grade 4	deep, muscles and bone involved, tissue necrosis, mummification

further damage. Rubbing will also produce more tissue damage [20]. Due to hypoesthesia a rewarming over open fire or electric heaters is not recommended because of the inability to feel skin burns [21, 22].

During the painful rewarming process adequate analgesia is necessary. Additional to the peripheral rewarming a systemic rewarming should be the aim. In cases with low core body temperature it should be avoided to move the extremities more than necessary to prevent the flow of cold peripheral blood into the body core, leading to further hypothermia which might be lethal. The sentence “Life before limb” should be remembered.

The best diagnostic tool to predict the amount of necrosis is the angiography and the technetium bone scan [23]. Too early surgical therapy with amputation should be avoided, the final expansion of necrosis can increase up to 45 days after exposure to the cold [18]. The only reason to amputate earlier is wet gangrene, infection and sepsis [24].

Conservative treatment includes several possibilities. Hyperbaric oxygen therapy show mixed results, animal studies show no benefit, in human case series good results were seen [23].

In severe cases who present within 24 h after onset of symptoms, infusion with iloprost or thrombolysis with rtPA (recombinant tissue plasminogen activator) should be considered [25]. Contraindication is a high risk of bleeding complication. In severe cases antibiotic therapy should be

considered. Tetanus vaccination should be done when necessary.

## Chronic Pathologies

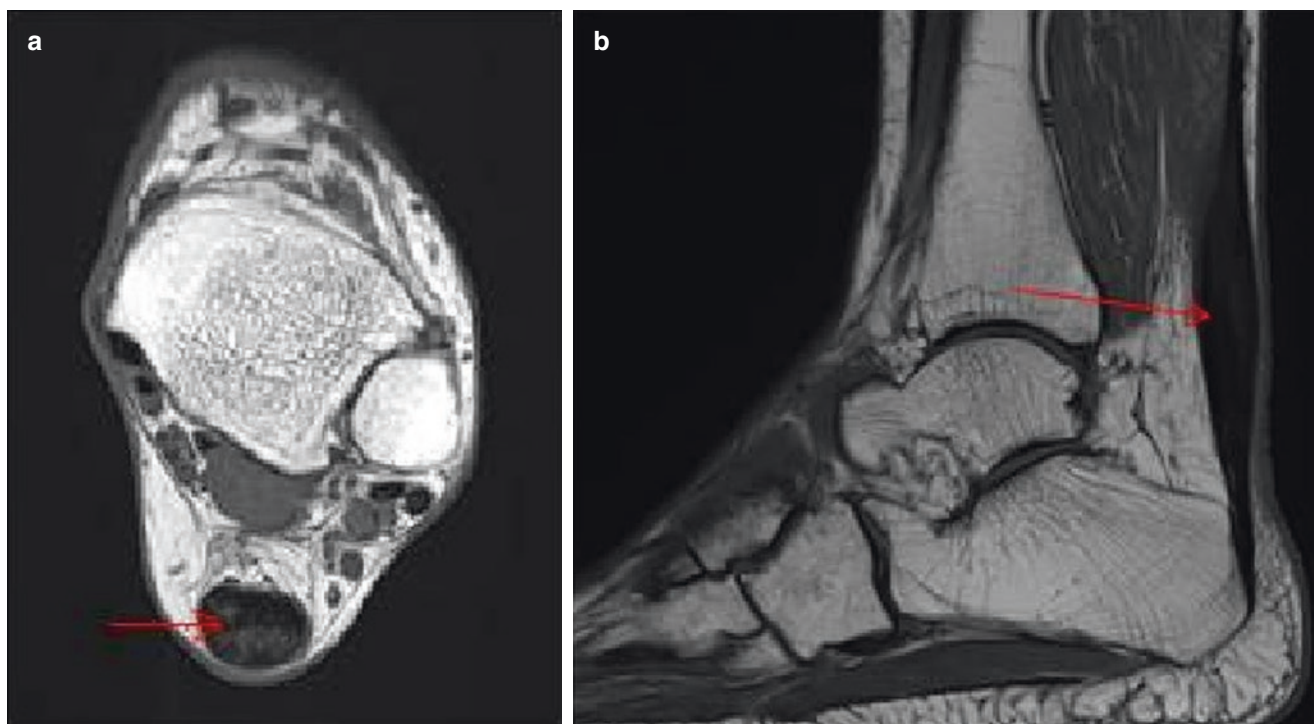
### Stress Fractures

Stress fractures in endurance athletes are most likely localized in the tibia (49.1%), followed by the tarsal bones (25.3%), the metatarsal bones (8.8%), the fibula (6.6%) and the sesamoids (0.9%). Bilateral stress fractures are seen in 16.6% [26]. Tarsal stress fractures are more frequent in older patients, stress fractures at the tibia and fibula are more likely in younger patients [26]. Calcaneal stress fractures are less common, due to this they are often misdiagnosed and treated as achillodynia or plantar fasciitis [27]. The average time for recovery is around 12 weeks, tarsal bones need the longest time to recover.

*Further information please see Chap. 17.*

### Achillodynia and Plantar Fasciitis

Hiking is a highly repetitive kind of sports. Over the time tendons and fasciae are under stress. This can lead to degeneration and ultimately rupturing (Fig. 57.2). Despite no exact



**Fig. 57.2** Achilles tendon degeneration. (a) Chronic degeneration of the achilles tendon in cross sectional MRI. (b) Chronic degeneration with thickening of the achilles tendon in sagittal MRI

numbers concerning the prevalence of achillodynia or plantar fasciitis in hiking are reported in the recent literature we know that in long distance running with similar movement patterns and also long duration of load the prevalence is high [28, 29].

*Further information please see Chaps. 20 and 21.*

## Paraesthesia

Paraesthesia is common in long distance hikers. Up to 34 % report from paraesthesia in general. Concerning the foot, most frequently the digitalgia paresthetica (7.5 %) and the tarsal tunnel syndrome (2.1 %) are seen [30]. These neuropathies are normally self-limiting and resolve when patients stop hiking [30]. In high altitude neuropathic pain and/or paraesthesia can occur. In a Himalayan expedition 4.8 % of European participants reported from these symptoms [31]. These are not related to frostbite which is also seen more often in high altitudes. The symptoms resolved spontaneously 4–8 weeks after return to accustomed altitudes. The pathomechanism is not yet clear [31].

## Prevention

Using proper equipment and avoiding the overestimation of one's own capabilities is the best prevention for acute and degenerative pathologies in hiking, mountaineering and canyoning.

Prevention of hypothermia is of course using proper equipment and avoiding to wear wet clothes whenever possible.

Hiking boots with good fit and if necessary custom made insoles can prevent chronic pathologies like achillodynia and plantar fasciitis. A rigid boot which covers the ankle and has a tight fit at the distal lower leg can prevent sprains. Otherwise with the increasing rigidity of hiking shoes the risk of paraesthesia is increasing [32]. So a perfect fit of hiking shoes is important.

To keep the feet dry is not only prevention for blistering but also a prevention of interdigital mycosis.

Using hiking poles leads to less exertion at least for short time [33]. Further it decreases sagittal movement in all joints of the lower extremity [34] and increases lateral stability, especially when carrying heavy loads like a backpack [35]. So the joints of the lower extremity are under less stress and the risk of falling in rough alpine terrain is lower due to better balance.

## Rehabilitation and Return to Sports

Depending on the kind and severity of the injury rehabilitation can take significant time.

Especially chronic pathologies as plantar fasciitis and achillodynia can lead to a long absence in sporting activities.

As mentioned above, paraesthesia normally disappears when the triggering activity is ceased, but without proper modification of the equipment the risk of a return is given.

In sprains and fractures it is depending on the severity and kind of treatment when the hiking activity can be started again. As in every sport a slow rehabilitation with continuous increase of load and duration is recommended.

Concerning blisters a complete healing with complete formation of healthy skin should be awaited. Hiking the first time after healing, taping of the skin areas at risk can prevent from early new blister formation due to thin epidermis after blister healing.

In frostbites a steady state of necrosis should be awaited. In case of amputation the complete healing of the amputation stump and if necessary an adaptation of the shoes is recommended.

## Level of Evidence

Blisters (socks, shoes)	Level I–II
Frostbite	Level II–III
Fractures	Level II–III
Distorsions	Level II
Equipment (hiking poles)	Level II
Paraesthesia	Level III

## Summary

- Acclimatization to unfamiliar environment in high altitudes
- Do not overestimate your capabilities
- Do not underestimate the simple ankle sprain (OCL, chronic instability and their consequences)
- Use of appropriate equipment, especially shoes and warm clothes. Care taking of feet with regular nurturing and use of correct insoles if necessary
- Using hiking sticks reduces the strain on the lower limb joints, especially when carrying external loads

## References

1. Gibson A, McKenna M. The effect of high altitude on the visual system. *J R Army Med Corps.* 2011;157(1):49–52.
2. Fischer R. Hazards of mountain climbing and hiking. *MMW Fortschr Med.* 2005;147(38):28–30, 32.
3. Johnson RM, Huettl B, Kocsis V, Chan SB, Kordick MF. Injuries sustained at Yellowstone National Park requiring emergency

- medical system activation. *Wilderness Environ Med.* 2007;18(3):186–9.
4. Ela GK. Epidemiology of wilderness search and rescue in New Hampshire, 1999–2001. *Wilderness Environ Med.* 2004;15(1):11–7.
  5. Roberts DJ, Ouellet JF, McBeth PB, Kirkpatrick AW, Dixon E, Ball CG. The “weekend warrior”: fact or fiction for major trauma? *Can J Surg.* 2014;57(3):E62–8.
  6. Burtscher M, Pachinger O, Schocke MF, Ulmer H. Risk factor profile for sudden cardiac death during mountain hiking. *Int J Sports Med.* 2007;28(7):621–4.
  7. Stephens BD, Diekema DS, Klein EJ. Recreational injuries in Washington state national parks. *Wilderness Environ Med.* 2005;16(4):192–7.
  8. Platts-Mills TF, Hunold KM. Increase in older adults reporting mountaineering-related injury or illness in the United States, 1973–2010. *Wilderness Environ Med.* 2013;24(1):86–8.
  9. Leemon D, Schimelpfenig T. Wilderness injury, illness, and evacuation: National Outdoor Leadership School’s incident profiles, 1999–2002. *Wilderness Environ Med.* 2003;14(3):174–82.
  10. Van Tiggelen D, Wickes S, Coorevits P, Dumalin M, Witvrouw E. Sock systems to prevent foot blisters and the impact on overuse injuries of the knee joint. *Mil Med.* 2009;174(2):183–9.
  11. Knapik JJ, Reynolds KL, Duplantis KL, Jones BH. Friction blisters. Pathophysiology, prevention and treatment. *Sports Med.* 1995;20(3):136–47.
  12. Knapik JJ. Prevention of foot blisters. *J Spec Oper Med.* 2014;14(2):95–7.
  13. Herring KM, Richie Jr DH. Friction blisters and sock fiber composition. A double-blind study. *J Am Podiatr Med Assoc.* 1990;80(2):63–71.
  14. Dai XQ, Li Y, Zhang M, Cheung JT. Effect of sock on biomechanical responses of foot during walking. *Clin Biomech (Bristol, Avon).* 2006;21(3):314–21.
  15. Knapik JJ, Reynolds K, Barson J. Influence of an antiperspirant on foot blister incidence during cross-country hiking. *J Am Acad Dermatol.* 1998;39(2 Pt 1):202–6.
  16. Egloff C, Hügler T, Valderrabano V. Biomechanics and pathomechanisms of osteoarthritis. *Swiss Med Wkly.* 2012;142:w13583.
  17. Hintermann B, Boss A, Schäfer D. Arthroscopic findings in patients with chronic ankle instability. *Am J Sports Med.* 2002;30(3):402–9.
  18. Foray J. Mountain frostbite. Current trends in prognosis and treatment (from results concerning 1261 cases). *Int J Sports Med.* 1992;13 Suppl 1:S193–6.
  19. Mazur P. Cryobiology: the freezing of biological systems. *Science.* 1970;168(3934):939–49.
  20. McIntosh SE, Hamonko M, Freer L, Grissom CK, Auerbach PS, Rodway GW, Cochran A, Giesbrecht G, McDevitt M, Imray CH, Johnson E, Dow J, Hackett PH, Wilderness Medical Society. Wilderness Medical Society practice guidelines for the prevention and treatment of frostbite. *Wilderness Environ Med.* 2011;22(2):156–66.
  21. Murphy JV, Banwell PE, Roberts AH, McGrouther DA. Frostbite: pathogenesis and treatment. *J Trauma.* 2000;48(1):171–8.
  22. Biem J, Koehncke N, Classen D, Dosman J. Out of the cold: management of hypothermia and frostbite. *CMAJ.* 2003;168(3):305–11.
  23. Gross EA, Moore JC. Using thrombolytics in frostbite injury. *J Emerg Trauma Shock.* 2012;5(3):267–71.
  24. Mills Jr WJ. Frostbite. A discussion of the problem and a review of the Alaskan experience. 1973. *Alaska Med.* 1993;35(1):29–40.
  25. Handford C, Buxton P, Russell K, Imray CE, McIntosh SE, Freer L, Cochran A, Imray CH. Frostbite: a practical approach to hospital management. *Extrem Physiol Med.* 2014;3:7.
  26. Matheson GO, Clement DB, McKenzie DC, Taunton JE, Lloyd-Smith DR, MacIntyre JG. Stress fractures in athletes. A study of 320 cases. *Am J Sports Med.* 1987;15(1):46–58.
  27. Weber JM, Vidt LG, Gehl RS, Montgomery T. Calcaneal stress fractures. *Clin Podiatr Med Surg.* 2005;22(1):45–54.
  28. Lopes AD, Hespanhol Júnior LC, Yeung SS, Costa LO. What are the main running-related musculoskeletal injuries? *A Syst Rev Sports Med.* 2012;42(10):891–905.
  29. Sobhani S, Dekker R, Postema K, Dijkstra PU. Epidemiology of ankle and foot overuse injuries in sports: a systematic review. *Scand J Med Sci Sports.* 2013;23(6):669–86.
  30. Boulware DR. Backpacking-induced paresthesias. *Wilderness Environ Med.* 2003;14(3):161–6.
  31. Ricart de Mesones A, Turón Sans J, Misiego M, Onaga Pueyo H, Real Soriano R, Botella de Maglia J. Neuropathic pain and dysesthesia of the feet after Himalayan expeditions. *High Alt Med Biol.* 2002;3(4):395–9.
  32. Anderson Jr LS, Rebholz CM, White LF, Mitchell P, Curcio 3rd EP, Feldman JA, Kahn JH. The impact of footwear and packweight on injury and illness among long-distance hikers. *Wilderness Environ Med.* 2009;20(3):250–6.
  33. Jacobson BH, Wright T. A field test comparison of hiking stick use on heart rate and rating of perceived exertion. *Percept Mot Skills.* 1998;87(2):435–8.
  34. Bohne M, Abendroth-Smith J. Effects of hiking downhill using trekking poles while carrying external loads. *Med Sci Sports Exerc.* 2007;39(1):177–83.
  35. Jacobson BH, Caldwell B, Kulling FA. Comparison of hiking stick use on lateral stability while balancing with and without a load. *Percept Mot Skills.* 1997;85(1):347–50.



Lukas Weisskopf, Julian Röhm, and Thomas Hesse

**Abstract**

Ice hockey is one of the most exciting and fast-paced team-sports played, however it also involves a high risk of injuries. Although injuries of the lower limb are common, injuries of the foot and the ankle are relatively rare, yet due to the long period of recovery not neglectable. Eleven to twelve percent of all injuries documented in ice hockey affect foot and ankle. Injury of the lateral ligament complex is seldom due to the protective design of the ice skates and because the typical pathomechanism of plantarflexion and inversion are lacking. In contrast, the syndesmotic injury (“high ankle sprain”) is a typical injury in ice hockey due to the common pathomechanism of dorsiflexion, eversion and external rotation. If the radiograph proves evidence of syndesmosis widening, operative treatment is indicated. Usually high ankle sprains need up to 12 weeks to heal and cause the longest absence from practice and game due to injury in ice hockey. Bruises of foot and ankle are second most common after ligament injuries in ice hockey. Especially exposed bony parts like the malleoli as well as the navicular and the base of the fifth metatarsal bone are at risk for bruises and fractures. The skate bite (also known as “lace bite”) is characteristic for ice hockey. It is an inflammation of the anterior aspect of the ankle due to wrong lacing technique or lack of padding. The latter and so called “boot-top-injury” can easily be prevented by wearing proper protection equipment.

**Keywords**

Ice hockey • Injury • Foot • Ankle • Syndesmosis • Skate bite

**Injuries**

Ice hockey is one of the most exciting and fast-paced team-sports played [1], however it also involves a high risk of injuries. Although injuries of the lower limb are common,

injuries of the foot and the ankle are relatively rare, yet due to the long period of recovery not neglectable [1–7].

L. Weisskopf (✉) • T. Hesse  
ALTIUS Swiss Sportmed Center & Sehnenzentrum Schweiz SZS,  
Habich-Dietschy-Strasse 5a, Rheinfelden CH 4310, Aargau,  
Switzerland  
e-mail: [lukas.weisskopf@altius.ag](mailto:lukas.weisskopf@altius.ag); [thomas.hesse@altius.ag](mailto:thomas.hesse@altius.ag)

J. Röhm  
Department of Orthopaedic Surgery and Traumatology,  
Kantonsspital Baselland, Switzerland, Rheinstrasse 26,  
Liestal CH 4410, Switzerland  
e-mail: [julian.roehm@gmail.com](mailto:julian.roehm@gmail.com)

**Ankle Sprains/Syndesmosis**

Due to the configuration and the rigidity of the ice skates up to supramalleolar level, injuries of the foot and ankle are rare compared to other ball sports and martial arts [8]. However the rigid fixation of the foot in the ice skates leads to a specific force distribution and consecutively to an accumulation of relatively rare injury patterns.

This makes injuries to the lateral ligament complex seldom, due to the protective and supporting function of the ice skates and the lack of the typical pathomechanism of plantarflexion



**Fig. 58.1** (a, b) Typical pathomechanism for syndesmosis injuries with dorsiflexion, eversion and external rotation

and inversion. In contrast dorsiflexion, eversion and external rotation is more likely to occur, e.g. if the blade gets stuck in the ice and forces the ankle in external rotation and eversion (Figs. 58.1a and 58.2). A second injury mechanism, which can lead to syndesmotic injuries, is a fall over the tiptoe-position in combination with external rotation and dorsiflexion (Fig. 58.1b). The deltoid ligament and the syndesmosis are therefore at risk of injuries in ice hockey [3, 5, 9]. The current clinical diagnostic tests such as palpation of the tibiofibular ligaments, external rotation stress test and squeeze test as well as dorsiflexion range of motion (ROM) show low diagnostic accuracy. MRI or arthroscopy help to make a final diagnosis whether, and to what extent, a syndesmosis injury is present [10, 11]. In ice hockey 50–74% of the ligament injuries of the ankle joint are syndesmotic injuries and compared to other sport more common (e.g. American Football 1.1–18.4%) [12]. The average time loss for this kind of injury is the longest in ice hockey with 15 practices and 5.4 games missed [5].

### Bruises/Fractures

Bruises of foot and ankle are second most common after ligament injuries in ice hockey. Especially exposed bony parts like the malleoli as well as the navicular and the base of



**Fig. 58.2** Typical pronation trauma with stress on the syndesmosis; © Weisskopf

the fifth metatarsal bone are at risk for bruises [6] (Fig. 58.3). Fractures of these bones are seldom but possible when getting hit by the stick or puck, which travels with a speed up to 170 km/h and creates pressures of about 25 bar/10 cm [2, 13]. Toe fractures are less common since the ice skates give good protection.



**Fig. 58.3** Contusion of the medial malleolus through puck; © Weisskopf

### Injury to the Tendons, Vessels and Nerves

Ice skates are commonly laced up to the top allowing different tension application at different parts of the lacing to allow extra flexibility. This and when new or old, inflexible ice skates are used so called lace bite (also called “skate bite”) can occur [14]. Due to the high pressure and friction of the skate tongue on the anterior aspect of the ankle during the repeated dorsiflexion the tendons of toe extensors as well as the tibialis anterior tendon, which glide underneath the retinaculum, may get irritated [3]. Seldom thrombophlebitis may appear.

Injuries, like cuts or lacerations of the extensor tendons as well as the vessel-nerve bundle just above the shoe through direct trauma (“boot-top”-injury), are rare and can be prevented by proper protective equipment. Case reports with complete laceration of the anterior tibial tendon in ice hockey have been published [15–17]. Also a traumatic complete rupture of the posterior tibial tendon is described [18].

### Skin

In ice skates warm and humid conditions in combination with constant friction, shear forces, chronic pressure and collisions with surfaces promote critical skin conditions with fungal infections and inflammatory dermatoses. Maintaining good personal hygiene is very helpful in preventing transmission. Mechanical skin irritations can best be prevented by using well-fitted skates, proper lacing techniques and orthotic devices [19–21].

### Etiology and Pathomechanism

Ice hockey is one of the fastest sports played with players skating up to 45 km/h. The partly allowed body checking and the hard ice surface framed with rigid boards, the hockey puck, which is a disc made of vulcanized rubber, which weighs 170 g and can reach speeds up to 170 km/h are all factors which can potentially increase the risk of injury [13]. All of these factors lead to different forces which act on the players and can cause different types of injuries. In ice hockey injuries can generally be divided into two types of pathomechanism:

High-speed – low mass injury (collision of body with puck or stick)

Low-speed – high mass injury (colliding with another player, the boards or ice surface)

### Epidemiology

The risk of an injury during a championship match in general is up to eight times greater than during training [22, 23]. The injury pattern is mostly direct body contact (50%) followed by collision with the boards, ice surface, puck or stick (40%). Only 10% of the injuries observed occur without contact [22, 23]. Eleven to twelve percent of all injuries documented in ice hockey affect foot and ankle [2, 5]. Ankle- and ligament-injuries appear in 4–4.5%, contusions to the foot in 1.6–7.2% of all ice hockey related injuries.

### Therapy (on the Field, Conservative, Surgical)

On-field therapy of the foot and ankle is difficult due to the protection gear and ice skating boots. Nevertheless, treatment should be started with compression, ice and elevation as soon as possible.

Ligament injuries of the ankle will heal most of the time when treated conservatively. Syndesmotic ruptures with mortise widening, observed on weight bearing x-ray (increased total clear space), are exceptions. These injuries

are severe and need to be treated by operative stabilization [24–26]. In high ankle sprains with a normal mortise even when stressed a staged conservative treatment can be performed: reduction of pain and swelling, then return of motion followed by proprioception training as well as ankle strengthening. Once full weight bearing is tolerated the player can proceed with off-ice training. Finally return to normal sport levels can be up to 12 weeks after the injury [27–30].

Chronic irritations of the extensor tendons (“skate bite”) are treated conservatively with anti-inflammatory medication and mechanical unloading (e.g. padding) in the skating boot. Breaking in the tongue of the skate will also decrease the pressure on the anterior aspect of the ankle. Traumatic boot-top injuries usually have to be treated operatively within a narrow time frame. Non-displaced fractures of the toes can be treated conservatively by splinting to the neighboring toe. Dislocated fractures should be reduced and need osteosynthesis.

## Rehabilitation and Back-to-Sports

Since in ice hockey twisting and turning is compulsory, high ankle sprains with syndesmosis injuries require a longer time to return to the on-ice activities compared to other sports. After the swelling is gone and strength is appropriate a functional on-ice evaluation should be performed. Returning to the game despite pain raises the risk for chronic instability and re-injury. Usually high ankle sprains need up to 12 weeks to heal [3, 14].

Since the ice skates support and protect the lateral ligaments low ankle sprains need a significant shorter amount of time for a player to return to practice and game. Usually return to play can be achieved within a week however the complete ligament healing may take 6–12 weeks [3, 31].

## Prevention

Wearing proper protective equipment, not only for the foot and ankle region, is the most important prevention aspect in ice hockey [2, 3, 5, 13]. Injuries of the foot and ankle through direct trauma can be prevented by special padding and shields on top of the boot. In general, attention should be paid to well fitted, not worn-out high-quality equipment. Since the risk for lacerations due to sharp blades is increased in ice hockey, cut resistant equipment is available. Low and high ankle sprains can be prevented by taping [32].

## Evidence

Most of the cited studies are retrospective and Level IV of evidence. There are only few Level III studies concerning the diagnostic and treatment of syndesmotic injuries [28, 30,

33]. Prospective, randomized studies are, to our knowledge, not available yet.

## Summary

- Ice hockey is one of the most exciting and fast-paced team-sports played [1], however it also involves a high risk of injuries.
- Although injuries of the lower limb are common, injuries of the foot and the ankle are relatively rare, yet due to the long period of recovery not neglectable. Eleven to twelve percent of all injuries documented in ice hockey affect foot and ankle.
- Injury of the lateral ligament complex is seldom due to the protective design of the ice skates and because the typical pathomechanism of plantarflexion and inversion are lacking. In contrast, the syndesmotic injury (“high ankle sprain”) is a typical injury in ice hockey due to the common pathomechanism of dorsiflexion, eversion and external rotation. If the radiograph proves evidence of syndesmosis widening, operative treatment is indicated. Usually high ankle sprains need up to 12 weeks to heal and cause the longest absence from practice and game due to injury in ice hockey.
- Bruises of foot and ankle are second most common after ligament injuries in ice hockey. Especially exposed bony parts like the malleoli as well as the navicular and the base of the fifth metatarsal bone are at risk for bruises and fractures.
- The skate bite (also known as “lace bite”) is characteristic for ice hockey. It is an inflammation of the anterior aspect of the ankle due to wrong lacing technique or lack of padding. The latter and so called “boot-top-injury” can easily be prevented by wearing proper protection equipment.

## References

1. Luke D. Characteristics of ice hockey-related injuries treated in US emergency departments, 2001–2002. *Pediatrics*. 2005;115–5:1448–9; author reply 9.
2. Deits J, Yard EE, Collins CL, Fields SK, Comstock RD. Patients with ice hockey injuries presenting to US emergency departments, 1990–2006. *J Athl Train*. 2010;45–5:467–74.
3. Laprade RF, Surowiec RK, Sochanska AN, Hentkowski BS, Martin BM, Engebretsen L, Wijdicks CA. Epidemiology, identification, treatment and return to play of musculoskeletal-based ice hockey injuries. *Br J Sports Med*. 2014;48–1:4–10.
4. Benson BW, Meeuwisse WH. Ice hockey injuries. *Med Sport Sci*. 2005;49:86–119.
5. Flik K, Lyman S, Marx RG. American collegiate men’s ice hockey: an analysis of injuries. *Am J Sports Med*. 2005;33–2:183–7.
6. Weisskopf L. Kapitel 3.8 Eishockey. In: *Aerzte-Verlag; Fuss, Sprunggelenk und Sport – Empfehlungen von Sportarten aus orthopädischer und sportmedizinischer Sicht*. Deutscher Aerzte-Verlag, Köln; 2008. p. 268–70.

7. Weisskopf L. Verletzungen des Bewegungsapparates im Eishockey. *Schweizerische Zeitschrift für Sportmedizin und Sporttraumatologie*. 2010;58(2):52–5.
8. Kujala UM, Taimela S, Antti-Poika I, Orava S, Tuominen R, Myllynen P. Acute injuries in soccer, ice hockey, volleyball, basketball, judo, and karate: analysis of national registry data. *BMJ*. 1995;311–7018:1465–8.
9. Wright RW, Barile RJ, Surprenant DA, Matava MJ. Ankle syndesmosis sprains in national hockey league players. *Am J Sports Med*. 2004;32–8:1941–5.
10. Mak MF, Gartner L, Pearce CJ. Management of syndesmosis injuries in the elite athlete. *Foot Ankle Clin*. 2013;18–2:195–214.
11. Sman AD, Hiller CE, Refshauge KM. Diagnostic accuracy of clinical tests for diagnosis of ankle syndesmosis injury: a systematic review. *Br J Sports Med*. 2012;47–10:620–8.
12. Hopkinson WJ, St Pierre P, Ryan JB, Wheeler JH. Syndesmosis sprains of the ankle. *Foot Ankle*. 1990;10–6:325–30.
13. Groger A. Ten years of ice hockey-related-injuries in the German Ice Hockey Federation – A Ten Year Prospective Study/523 International Games. *Sportverletz Sportschaden*. 2001;15–4:82–6.
14. LaPrade RF, Wijdicks CA, Griffith CJ. Division I intercollegiate ice hockey team coverage. *Br J Sports Med*. 2009;43–13:1000–5.
15. DiDomenico LA, Blasko GA, Cane L, Cross DJ. Repair of lacerated anterior tibial tendon with acellular tissue graft augmentation. *J Foot Ankle Surg*. 2012;51–5:642–4.
16. Hovelius L, Palmgren H. Laceration of tibial tendons and vessels in ice hockey players. Three case histories of a skate boot top injury. *Am J Sports Med*. 1979;7–5:297–8.
17. Simonet WT, Sim L. Boot-top tendon lacerations in ice hockey. *J Trauma*. 1995;38–1:30–1.
18. Jacoby SM, Slauterbeck JR, Raikin SM. Acute posterior tibial tendon tear in an ice-hockey player: a case report. *Foot Ankle Int*. 2008;29–10:1045–8.
19. Mohrenschlager M, Seidl HP, Schnopp C, Ring J, Abeck D. Professional ice hockey players: a high-risk group for fungal infection of the foot? *Dermatology*. 2001;203–3:271.
20. Tloutan BE, Mancini AJ, Mandell JA, Cohen DE, Sanchez MR. Skin conditions in figure skaters, ice-hockey players and speed skaters: part II – cold-induced, infectious and inflammatory dermatoses. *Sports Med*. 2011;41–11:967–84.
21. Tloutan BE, Mancini AJ, Mandell JA, Cohen DE, Sanchez MR. Skin conditions in figure skaters, ice-hockey players and speed skaters: part I – mechanical dermatoses. *Sports Med*. 2011;41–9:709–19.
22. Agel J, Dick R, Nelson B, Marshall SW, Dompier TP. Descriptive epidemiology of collegiate women’s ice hockey injuries: National Collegiate Athletic Association Injury Surveillance System, 2000–2001 through 2003–2004. *J Athl Train*. 2007;42–2:249–54.
23. Agel J, Dompier TP, Dick R, Marshall SW. Descriptive epidemiology of collegiate men’s ice hockey injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 through 2003–2004. *J Athl Train*. 2007;42–2:241–8.
24. Molinari A, Stolley M, Amendola A. High ankle sprains (syndesmotom) in athletes: diagnostic challenges and review of the literature. *Iowa Orthop J*. 2009;29:130–8.
25. Williams GN, Jones MH, Amendola A. Syndesmotom ankle sprains in athletes. *Am J Sports Med*. 2007;35–7:1197–207.
26. Amendola A, Williams G, Foster D. Evidence-based approach to treatment of acute traumatic syndesmosis (high ankle) sprains. *Sports Med Arthrosc*. 2006;14–4:232–6.
27. Brosky T, Nyland J, Nitz A, Caborn DN. The ankle ligaments: consideration of syndesmotom injury and implications for rehabilitation. *J Orthop Sports Phys Ther*. 1995;21–4:197–205.
28. Nussbaum ED, Hosea TM, Sieler SD, Incremona BR, Kessler DE. Prospective evaluation of syndesmotom ankle sprains without diastasis. *Am J Sports Med*. 2001;29–1:31–5.
29. Mulligan EP. Evaluation and management of ankle syndesmosis injuries. *Phys Ther Sport*. 2011;12–2:57–69.
30. McCollum GA, van den Bekerom MP, Kerkhoffs GM, Calder JD, van Dijk CN. Syndesmosis and deltoid ligament injuries in the athlete. *Knee Surg Sports Traumatol Arthrosc*. 2012;21–6:1328–37.
31. Medina McKeon JM, Bush HM, Reed A, Whittington A, Uhl TL, McKeon PO. Return-to-play probabilities following new versus recurrent ankle sprains in high school athletes. *J Sci Med Sport*. 2013;17–1:23–8.
32. Karlsson J, Sward L, Andreasson GO. The effect of taping on ankle stability. Practical implications. *Sports Med*. 1993;16–3:210–5.
33. Jones MH, Amendola A. Syndesmosis sprains of the ankle: a systematic review. *Clin Orthop Relat Res*. 2007;455:173–5.

Christopher E. Gross and James K. DeOrio

## Abstract

At the height of their popularity, roller skating and in-line skating were a major public health concern. Roughly \$4 billion dollars were spent in 1997 treating injuries related to these recreational activities. While most of these sports-specific injuries tend to be of the upper extremity, ankle injuries tend to be more severe that require operative treatment.

## Keywords

Skating • Sprains • Fractures • Injuries

## Injuries

The first reported use of roller skates was in 1743 on a London stage by an unknown inventor. Its modern four-wheeled counter part, the quad skate, was designed and patented by James Leonard Plimpton in 1863. Similarly, a primitive in-line skate was invented by John Joseph Merlin in 1760. This type of skate saw a resurgence in popularity in 1987 when the Olson brothers started marketing the “Rollerblade” (Rollerblade, Inc., Minneapolis, MN) as a fitness product [1].

The roller skate is inherently an unstable device that is designed for speed and maneuverability. They are designed with large polyurethane wheels that have bearings to improve roll and speed [2]. In-line skates are designed with a single row of four or five low-friction wheels that have greater maneuverability and speed than traditional roller skates. The skaters can achieve speeds up to 30 miles-per-hour [3].

Since 2000 however, a new iteration of wheeled shoes has become popular. These shoes have wheels in the heel (Heelys, HSL, Carrollton, TX) and allow a user to switch from walking to rolling by shifting the body weight backwards. In 2006, the producing company reported a 60 country worldwide dis-

tribution of 4.5 million pairs [4]. Another variation is the Street Gliders (Glowgadgets Ltd, Bristol, England) in which wheels are strapped to regular athletic shoes to combine walking with rolling only by shifting body weight.

The sports of roller and in-line skating have steadily declined since their peak in popularity in the late 1990s. In 2012, the National Sporting Goods Association reported that 7,851,000 and 7,451,000 participated in in-line and roller skating, respectively [5]. This is in steep decline from the 29.1 million participants of in-line skating in 1997 [6].

The Consumer Products Safety Commission’s National Electronic Injury Surveillance System (NEISS) reported that the estimated rate of product-related injuries per 100,000 population in the United States and Territories that were treated in emergency departments was 4.1/100,000 and 24.7/100,000 for in-line and roller skating, respectively [7]. Taken in context, there were 102,820 estimated injuries in 1996 and 12,828 injuries in 2012.

## Etiology and Pathomechanism

### Roller Skating

Ferkel et. al. reviewed 202 roller skating injuries in during a 6 month period in 1979 [2]. 186 patients; 202 injuries. The average patient age was 25.3 years. The wrist was the most commonly injured bone (47%), while the ankle was the 3rd

C.E. Gross, MD (✉) • J.K. DeOrio, MD  
Department of Orthopaedic Surgery, Duke University Medical Center,  
4709 Creekstone Drive, 2nd Floor, Durham, NC 27703, USA  
e-mail: [cgross144@gmail.com](mailto:cgross144@gmail.com); [james.deorio@duke.edu](mailto:james.deorio@duke.edu)

most common injury at 10%. While upper extremity injuries were more common, the lower extremity injuries were more severe. Ankle fractures were of particular significance since they accounted for 46% of all surgical cases. Based on the fracture pattern, the ankle was fractured via a rotational mechanism as no pronation abduction or supination adduction injuries were seen. The posterior malleolus was involved 75% of the time. Supination external rotation injuries were more prevalent in high top versus low top skates. The most frequent explanation for injuries was a loss of balance (61%). Interestingly, more experienced skaters tended to need surgery more so than the inexperienced ones.

Another study reported on the incidence of roller skating injuries [8]. At the time of the study (1983), roller skating injuries cost the United States health care system over \$100 million. Kvidera and Frankel looked at 35 fractures secondary to roller skating seen during a 6.5 month period in 1979. The average patient age was 25 years. Wrist fractures accounted for 77% of the injuries. However, the one ankle fracture in the study required operative fixation.

Nayeem, et al. looked at the cost of the introduction of roller skating rink to the local emergency department [9]. During the 14 months of the study, 384 patients were treated with an injury. The foot and ankle were injured 11% of the time. Out of the 32 ankle injuries, the ankle was fractured 38% of the time. 17% of the ankle fractures required open reduction internal fixation.

One case report looked at a 9-year-old Australian junior female roller-skating champion [10]. The patient suffered from a medial malleolus stress fracture after a training regimen of 10 km around a 100 m track 3 nights a week in the same direction. An x-ray confirmed the stress fracture. The patient was successfully treated with activity modification over 3 months.

## In-Line Skating

The spectrum of in-line skating injuries range from cuts and scrapes to fatal head trauma. During the height of in-line skating's popularity, the estimated annual cost of treatment for these related injuries was \$4 billion in 1997 [11]. Males accounted for a majority of those injured [11]. Sixty percent of the injured involved 10–14 year olds. The following risk factors have been implicated in increasing the odds of an in-line skating injury: inexperienced skaters, those performing tricks, skating in locations with railings, ledges, and ramps, and increased skating time per week [11, 12].

Most patients injure themselves with losing their balance and colliding or trying to avoid a collision with a stationary or moving object [12, 13]. In his study of in-line skating injuries in Singapore, Goh et al. [14] described three mechanisms of injury. In the first pattern, the patient falls forward with an outstretched hand and arm, resulting in an upper

extremity injury. The second pattern, backwards, injures the head. The third mechanism results in a twisting injury to the lower extremity.

Injuries to the upper extremities (mostly the wrist) occur between 57 and 77% of all injuries [15]. Of all wrist injuries, 62% are fractures [11]. The lower extremity accounts for roughly 19% of all injuries [11]. In a study which documented 9 years of in-line skating injury data, foot and ankle injuries accounted for 4.5% of all injuries. The ankle was fractured 83% of the time the ankle was injured.

One study evaluated the difference of types of injuries seen in a emergency rom in Denmark in 1997 [16]. Of the 300 in-line skaters and 107 roller skaters treated, 60.4% had minor injuries such as sprains and abrasions and 39.6% fractures. Between skater groups, there was a statistically significant difference in the types of injury. The most common serious injury was a distal radius fracture, which occurred about 25% in both skater groups. Ankle fractures were aggregated into the lower extremity fractures (5.5%: in-line and 7.5%: roller skate) and therefore, specifics about the foot and ankle were unable to be determined.

## Heelys and Street Gliders

The number of injuries involving Heelys and Street Gliders has caused most American school districts and one town in England to ban the shoes [17]. The most recent study of the literature presented in this chapter comments on the epidemiology of Heelys and Street Gliders injuries [4]. This study reviewed the burden of Heelys injuries over a 2.5 month period at one emergency department in Ireland. 67 children were treated for fractures with an average age of 9.9 years. Heelys and Street Gliders injuries represented 8% of the orthopaedic workload during the duration of the capture period. Six patients sustained foot and ankle fractures (9.0% of all injuries). The majority of trauma were distal radius fractures (87%). Most injuries occurred while the children were learning how to use the shoes.

---

## Therapy

If one suspects an ankle sprain or fracture, one should immediately go the local emergency room or orthopaedic urgent care center.

---

## Rehabilitation and Back to Sports

There are no data to suggest a particular skating-specific protocol. The authors of the medial malleolus stress fracture suggested returning the athlete when she no longer had any medial sided pain. They also recommended skating in the opposite direction in which she was training [10].

## Prevention

Personal protective equipment is highly encouraged. Safety gear includes: knee pads, elbow pads, wrist guards, and helmets. Unfortunately, only 7% of users actually use all four pieces of equipment [12]. In a study involving 161 patients [3], those who did not use wrist splints were 10.4 times likely to suffer a wrist injury compared to those who did. The absence of elbow pads contributed to a 9.5 odds ratio of increased chance of injury compared to those wearing elbow guards. Knee pads were not associated with a statistically significant difference in injuries. Safety equipment statistically decreases the risk of hospitalization from injury [18]. Though the risk of injury does not approach zero while wearing protective equipment, the severity and incidence of injuries is significantly decreased while wearing safety gear [11]. There is no literature to suggest that wearing safety equipment reduces the frequency of foot and ankle injuries.

## Evidence

The level of evidence in this chapter is Level IV.

## Summary

- While roller skating and in-line skating are declining in popularity, they will always exist as a health care burden as long as children and adults use them recreationally.
- Common injuries are ankle fractures and medial malleolus stress fractures.
- Personal protective equipment is essential in decreasing the frequency and severity of injuries.
- We recommend roller skates and in-line skates with good and rigid ankle support to help reduce the amount and severity of foot and ankle injuries.

## References

1. Calle SC, Eaton RG. Wheels-in-line roller skating injuries. *J Trauma*. 1993;35:946–51.
2. Ferkel RD, Mai LL, Ullis KC, Finerman GA. An analysis of roller skating injuries. *Am J Sports Med*. 1982;10:24–30.
3. Schieber RA, Branche-Dorsey CM, Ryan GW, Rutherford Jr GW, Stevens JA, O'Neil J. Risk factors for injuries from in-line skating and the effectiveness of safety gear. *N Engl J Med*. 1996;335:1630–5.
4. Vioreanu M, Sheehan E, Glynn A, Casidy N, Stephens M, McCormack D. Heelys and street gliders injuries: a new type of pediatric injury. *Pediatrics*. 2007;119:e1294–8.
5. Association SGM. 2012 sports, fitness and leisure activities topline participation report; 2012.
6. Osberg JS FS, Poole J, McHenry J. Skating: an emerging mode of transportation. transportation research board. 79th annual meeting; 2000.
7. Commission USCPS. NEISS data highlights 2012. Washington, DC; 2012.
8. Kvidera DJ, Frankel VH. Trauma on eight wheels. A study of roller skating injuries in Seattle. *Am J Sports Med*. 1983;11:38–41.
9. Nayeem N, Shires SE, Porter JE. Cost of a roller skating rink to the local accident and emergency department. *Br J Sports Med*. 1990;24:240–2.
10. Hitchen PR, Lyons WJ. Fatigue fracture of the medial malleolus in a junior roller skater. *Aust N Z J Surg*. 1996;66:265–6.
11. Tan V, Seldes RM, Daluiski A. In-line skating injuries. *Sports Med*. 2001;31:691–9.
12. Seldes RM, Grisso JA, Pavell JR, et al. Predictors of injury among adult recreational in-line skaters: a multicity study. *Am J Public Health*. 1999;89:238–41.
13. Commission USCPS. NEISS data highlights – 1997. Washington, DC; 1998.
14. Goh SH, Tan HK, Yong WS, Low BY. Spectrum of roller-blading injuries. *Ann Acad Med Singapore*. 1996;25:547–9.
15. Nguyen D, Letts M. In-line skating injuries in children: a 10-year review. *J Pediatr Orthop*. 2001;21:613–8.
16. Houshian S, Adersen HM. Comparison between in-line and roller-skating injury. *Scand J Med Sci Sports*. 1999;10:47–50.
17. S P. Hilly town decides Heels just too dangerous. *Daily Mail*; 2007.
18. Adams SL, Wyte CD, Paradise MS, del Castillo J. A prospective study of in-line skating: observational series and survey of active in-line skaters – injuries, protective equipment, and training. *Acad Emerg Med : Off J Soc Acad Emerg Med*. 1996;3:304–11.



Sebastian Mueller, Matthijs Jacxsens, and Claudio Rosso

## Abstract

Foot and ankle injuries are among the most common injuries in Martial Arts and account for a major part of loss of sports. Two factors are of utmost importance in dealing with foot and ankle injuries in contact combat sports. Firstly, injury prevention should already inhibit such injuries. Here, proprioceptive training is essential. Secondly, referees need to strictly adhere to the rules in order to prevent those injuries.

After such an injury, full muscle force and especially above-mentioned proprioceptive balance should be restored after multi-disciplinary discussion with the sports physician, the training staff and physiotherapists before returning to play.

## Keywords

Martial Arts • Karate • Combat sports • Turf toe

## Introduction

The term “Martial Arts” summarizes traditional combat sports like Judo, Taekwondo, Jiu-Jiutsu and Karate as well as more recent types such as Kick- or Thai-Boxing.

Since all of them are practiced barefoot, typical injury patterns, demanding specific therapies, occur.

Besides the “free-fight” (Kumite), the traditional Asian combat sports additionally includes shadow-fighting (Kata) and the so-called basic education (Kihon).

Kumite mostly is practiced according to semi-contact rules, where hits must be controlled and contacts to face and neck are penalized. This again, results in variable injury patterns. While athletes performing Kata and Kihon mostly experience injuries from overexertion, external influence

plays a more important role in Kumite. In this chapter, particular focus is set on the injury patterns sustained in the free-fight.

## Injury Patterns, Etiology and Pathomechanism

Due to the intensive training in **Kata**, overexertion symptoms, as sprains, similar to the ones seen in (ballet) dancers, occur. Small muscles in particular are affected, since they are exposed to very high proprioceptive stresses [1, 2]. In **Kumite**, the direct impact of the opponents’ foot in kicks or foot sweeps/lateral kicks (see Figs. 60.1 and 60.2) are responsible for the resulting contusions, lacerations and fractures.

In Martial arts the most common injuries are Achilles tendon ruptures, ankle sprains, fractures (ankle, metatarsal), Linsfranc injuries, turf toe, bruises, bone contusions.

S. Mueller, MD • M. Jacxsens, MD  
University Hospital Basel, University of Basel, Basel, Switzerland  
e-mail: [s.mueller@usb.ch](mailto:s.mueller@usb.ch); [matthijs.jacxsens@gmail.com](mailto:matthijs.jacxsens@gmail.com)

C. Rosso, MD, MSc, PD Dr. med. (✉)  
Shoulder and Elbow Center, Arthro Medics, Basel, Switzerland  
e-mail: [c.rosso@arthro.ch](mailto:c.rosso@arthro.ch)



**Fig. 60.1** Pathomechanism of lateral foot kick when encountering defense. The kicking foot is in plantar flexion and hereby exposes the complete mid-foot and the toes. The main pillar is exposed to strong rotational forces. Additionally, high kicks often result in sprains because of the strong forces interacting on the main pillar



**Fig. 60.2** Pathomechanism of foot sweeps. As a consequence a trauma of the lateral malleolus on one hand, and a sudden loss of stability and consecutive ankle sprain on the other hand may occur. Furthermore, contusions and fractures of the attacking fore- and midfoot are possible

## Epidemiology

The incidence of injuries due to chronic overexertion in the basic contact-free training techniques (Kata) should not be underestimated in daily exercise routine, although there are hardly any reports in current literature.



**Fig. 60.3** Turf toe of the left great toe. In modern sports Karate (Shobu-System), the great toe frequently suffers from hyper-plantar flexion

In the free-fight (Kumite), for example in the semi-contact Karate, injuries to face and neck are predominant with up to 91%, whereas in Taekwondo 65% of the injuries are sustained to the lower extremities [3]. According to literature, injuries to the lower extremity in total account for 10–65% [4]. An example can be seen in Fig. 60.3.

Injuries in competing professional athletes are far more common than during exercise in amateur athletes. This is due to the increased exercise intensity as well as exercise duration [4, 5]. In Karate, for instance, injuries are frequent but of smaller extent (contusions, abrasions) [4, 6].

Injuries around the foot and ankle are shown in Table 60.1. They account for a major part of injuries since martial arts are performed without footwear [7] [Figures: Swiss Accident Insurance Company, 2008–2012].

## Prevention

To avoid injuries resulting from overexertion, proprioceptive training on unstable surfaces as well as shorter exercise units are recommended [1].

Injury prevention with protective pads is discussed controversially: on the one hand these pads cushion the unwanted impact in semi-contact sports, on the other hand hits and kicks are performed with less control [8].

However, all authors agree that the consequent interpretation of the rules by the referee minimizes best the risk of injury [6, 9]. A solid and sound education and guidance of the athlete by the coach is fundamental. Additionally, surveillance and medical care by an experienced tournament doctor is essential.

**Table 60.1** Overview of Foot and Ankle Injuries in Martial Arts

Foot and ankle joint injuries- all combative sports	17 %
Foot and ankle sprains	7.9 %
Contusions (upper ankle joint and foot)	3.9 %
Toe fractures	2.1 %
Tarsal- and midfoot-fractures	0.8 %
Dislocations (ankle, Chopard, Lisfranc, toes)	0.15 %

## Therapy

Therapy of sprains comprise a rather unpopular training interruption or modifications in the intensity and type of exercise (proprioception), since the use of insoles have to be completely avoided.

The therapeutical options in an acute Trauma are usually conservative, following PRICE (pause, rest, ice cooling, compression and elevation of the affected limb), since these are mostly minor injuries.

Fractures and dislocation should be treated according to medical standards (see Sect. 2.1. Fractures).

Achilles tendon ruptures shall be treated typically surgically: see Sect. 2.6 Achilles tendon ruptures.

## Rehabilitation and Back-to-Sports

In consequence of the high, explosive exertions in martial arts, a return to training and competition is only recommended after complete consolidation and good sensomotoric rehabilitation. Here is the role of a team-decision: team physician, surgeon, physiotherapist, trainer, and athlete. The average time of out-of-competition can be from 1 to 2 weeks to half a year, depending on the injury.

## Evidence

All the literature in the area are Level V evidence.

See evidence in the other chapters.

## Summary

- The most common injuries in martial arts are sprains, contusions, toe fractures.
- Prevention is done by proprioceptive training on unstable surfaces as well as shorter exercise units.
- Return-to-sports is a team-decision, this by: team physician, surgeon, physiotherapist, trainer, and athlete

## References

1. Shan G. Comparison of repetitive movements between ballet dancers and martial artists: risk assessment of muscle overuse injuries and prevention strategies. *Res Sports Med.* 2005;13(1):63–76.
2. Khan K, et al. Overuse injuries in classical ballet. *Sports Med.* 1995;19(5):341–57.
3. Altarriba-Bartes A, et al. Epidemiology of injuries in elite taekwondo athletes: two Olympic periods cross-sectional retrospective study. *BMJ Open.* 2014;4(2):e004605.
4. Destombe C, et al. Incidence and nature of karate injuries. *Joint Bone Spine.* 2006;73(2):182–8.
5. Pocecco E, et al. Injuries in judo: a systematic literature review including suggestions for prevention. *Br J Sports Med.* 2013;47(18):1139–43.
6. Rosso C, et al. Karate-do – the path of the empty hand. *Sport-Orthopädie SportTraumatol Sports Orthopaedics Traumatol.* 2012;28(1):12–6.
7. Burks J, Satterfield K. Foot and ankle injuries among martial artists. Results of a survey. *J Am Podiatr Med Assoc.* 1998;88(6):268–78.
8. Müller-Rath R, Bolte S, Petersen P, Mommsen U. Injury profile in modern competitive karate-analysis of 1999 WKC-Karate World Championships Games in Bochum. *Sportverletz Sportschaden.* 2000;14:20–4.
9. Critchley G, Mannion S, Meredith C. Injury rates in Shotokan karate. *Br J Sports Med.* 1999;33:174–7.

Christian Stelzenbach and Victor Valderrabano

**Abstract**

Motorsport is a high-risk sport with the permanently increasing number of participants and therefore increasing number of foot and ankle injuries. The most common mechanism for loss of control of a motorcycle is a collision with an immovable object (e.g., trees or rocks). According to the trauma different types of injuries (fractures, ligamentous injuries and muscle strains) can appear. Severe and complex injuries like multiple fractures or combined lesions can be caused by high speed and high-energy impact forces. Intraarticular fractures as well as complex fractures require anatomic reduction and osteosynthetic fixation, while ligamentous injuries and non-dislocated fractures often can be treated conservatively. A functional rehabilitation program is recommended to return to sport as fast as possible.

**Keywords**

Motorsport • Sports • Motorcycle • Foot and ankle • Functional rehabilitation • Prevention

**Injuries in Motorsports**

Motorsport is a high-risk sport with the permanently increasing number of participating persons. Therefore the number of injuries may also increase. Different types of injuries can be related to motorsport including foot and ankle fractures, ligamentous injuries and muscular strains [1, 2].

**Etiology and Pathomechanism**

Motorsport is a popular sport which is practiced by millions of people worldwide. However, literature addressing foot and ankle injuries related to motorsport accidents is limited. While neck injuries including neck sprain are frequent in car

racing (45 % of all injuries), there is a lower risk for injuries of the lower limb (20 %) [3]. Nevertheless, high speed and the high-energy impact of force in combination with the lack of leg room especially in single seat cars can substantially contribute to ankle sprains and fractures of the lower limb [3].

The most common mechanism reported for loss of control of the motorcycle in a crash situation was collision with an immovable object (70 %), such as a tree or a rock [4]. This was followed by loss of traction (15 %), and collision with a movable object (6 %), such as a boundary marker or another motorcyclist. Loss of control while jumping and striking immovable objects, a condition where the motorcycle starts to oscillate and becomes difficult to steer, accounted for approximately 2 % of the total injuries. Thus, different terrain and riding conditions cause different types of injuries [4].

The main cause of injuries of the ankle in motocross accidents is the forced pronation and external rotation of grounded foot when taking a curve or direct impact by the bike on the leg. Even a direct trauma with weight of the motorcycle can cause injuries like fractures of the foot and/or ankle. Complex injuries with multiple fractures and ligamentous lesions may result from failed jumps. In such cases, bilateral injuries are not rare. Uncontrolled landing

C. Stelzenbach, MD (✉)  
Orthopaedic Department, University Hospital of Basel,  
Spitalstrasse 21, Basel CH-4031, Switzerland  
e-mail: [christian.stelzenbach@usb.ch](mailto:christian.stelzenbach@usb.ch)

V. Valderrabano, MD, PhD  
Orthopaedic Department, SWISS ORTHO CENTER,  
Schmerzklinik Basel, Swiss Medical Network, Basel, Switzerland  
e-mail: [vvalderrabano@gsmn.ch](mailto:vvalderrabano@gsmn.ch)

may cause lesions of the ligaments in foot and ankle after jumps, when the foot hits the ground or the footpeg. A forced dorsiflexion and eversion or (less commonly) inversion are the consequences. Interestingly, injuries on the left side are more common (65%) than on the right side. This can be partially explained by changing the gear during a jump with the left foot being in plantar flexion and the toes are pointed toward the ground [1]. In this context, a fracture of the processus lateralis tali should be excluded, as this injury may be often misdiagnosed [3]. The incidence of that type of injury has increased in the past years because of the popularity of snowboard riding. It is not easy to identify that lesion because of the unspecific clinical symptoms and inconspicuous conventional radiographs. When such a fracture is suspected a computed tomography should be considered [2, 5].

---

## Epidemiology

Injuries in motorsports are not rare. On the one hand, because of higher safety standards in motorsport competitions, substantially improved equipment, and safety measures around the route injuries can be reduced, on the other hand, drivers are willed to take more risks [4].

Colburn and Meyer showed an incidence of injury for motorcyclists in a professional competition of 2.7/1000 exposure hours [4]. Other sports like rugby have 18.3/1000 exposure hours, soccer 32/1000 exposure hours, football 59/1000 exposure hours and hockey 66/1000 exposure hours, respectively [4]. Other studies showed an injury rate/1000 exposure hours between 22.7 and 49.2 for motorsports [1, 6].

Tomida described injuries in elite motorcycle racing of ankle with 16.7% and foot with 1.7% [6].

A retrospective study including 1500 accidents in a period of 12 years revealed the overall incidence of accidents was 94.5‰ per year for motocross races, which was significantly lower than the 115‰ for road races [1]. Furthermore, the incidence of accidents in the outdoor competitions was significantly lower with 76‰ compared to the stadium cross competitions with 150‰ per year. From all injuries, 14.5% of ligamentous lesions concerned the ankle and 15% of them were treated surgically with open ligament repair. They also noted that fractures are significantly lower in road races than in motocross [1].

Minoyama and Tsuchida investigated injuries in a professional motor car racing [3]. They found out that forces that cause severe injuries, such as fractures and concussions, are around 5–10 G, which are produced by acute deceleration of about 150–200 km/h. The second most injury body site were the lower limbs (24% of all injuries) with 11 bruises and one ankle sprain, the most were injuries to the neck (34%). Overall injuries of the lower extremity appeared more often in single cars than in saloon cars, which can be explained by the lack of legroom in single cars.

## Therapy

First of all, on the field the injured lower extremity should be immobilized, elevated, and cooled. An early compression may prevent a posttraumatic swelling. If there is a luxation, the reposition of the ankle should be performed immediately to avoid or to minimize the soft tissue damage. Ligamentous injuries around the ankle often can be treated conservatively. For ankle stabilization we prefer a brace for 6 weeks, 24 h/day. Sports with high impact forces should be avoided during this time. Accompanying physiotherapy with lymphatic drainage can be performed. In patients with severe injuries, a partial weight bearing should be kept for 4–6 weeks. Non-dislocated fractures also can be treated conservatively in a brace or in a walker. Dislocated, intraarticular, and/or complex fractures require anatomic reduction and sufficient osteosynthetic fixation.

---

## Rehabilitation and Back-to-Sports

Various foot and ankle scoring systems exist (e.g., Star-Excursion Balance Test, The Dorsiflexion Lunge Test, Agility T-Test, Sargent/Vertical Jump Test) measuring range of motion, muscle power, static and dynamic balance, but none of them can determine the appropriate time point at which sports can be regained, because it depends on many different factors [7–10]. Clinical tests of proprioception, balancing, range of motion, agility and strength also can be measured during the rehabilitation and can show the progress of healing. Coupled with psychological assessment back to sports can be easier to decide [11]. Stress increases the risk of an athletic injury and athletes who demonstrate fear or anxiety are at a much greater risk of re-injury, and there is often a deleterious effect on athletic performance [12, 13]. Patients lacking adequate dorsiflexion are at increased risk for re-injury and have limitations in normal functional activities [8].

Especially for motorsports it is difficult to decide, when there is the appropriate time for the patients to go back to sports. Often there are unexpected impacts on foot and ankle while driving. Therefore we recommend not driving for at least 6 weeks. Depending on the severity of a ligamentous injury the patient can start early with functional training. A brace respectively a cast is obligatory for a minimum of 6 weeks. If surgery was necessary an individual rehabilitation program is required.

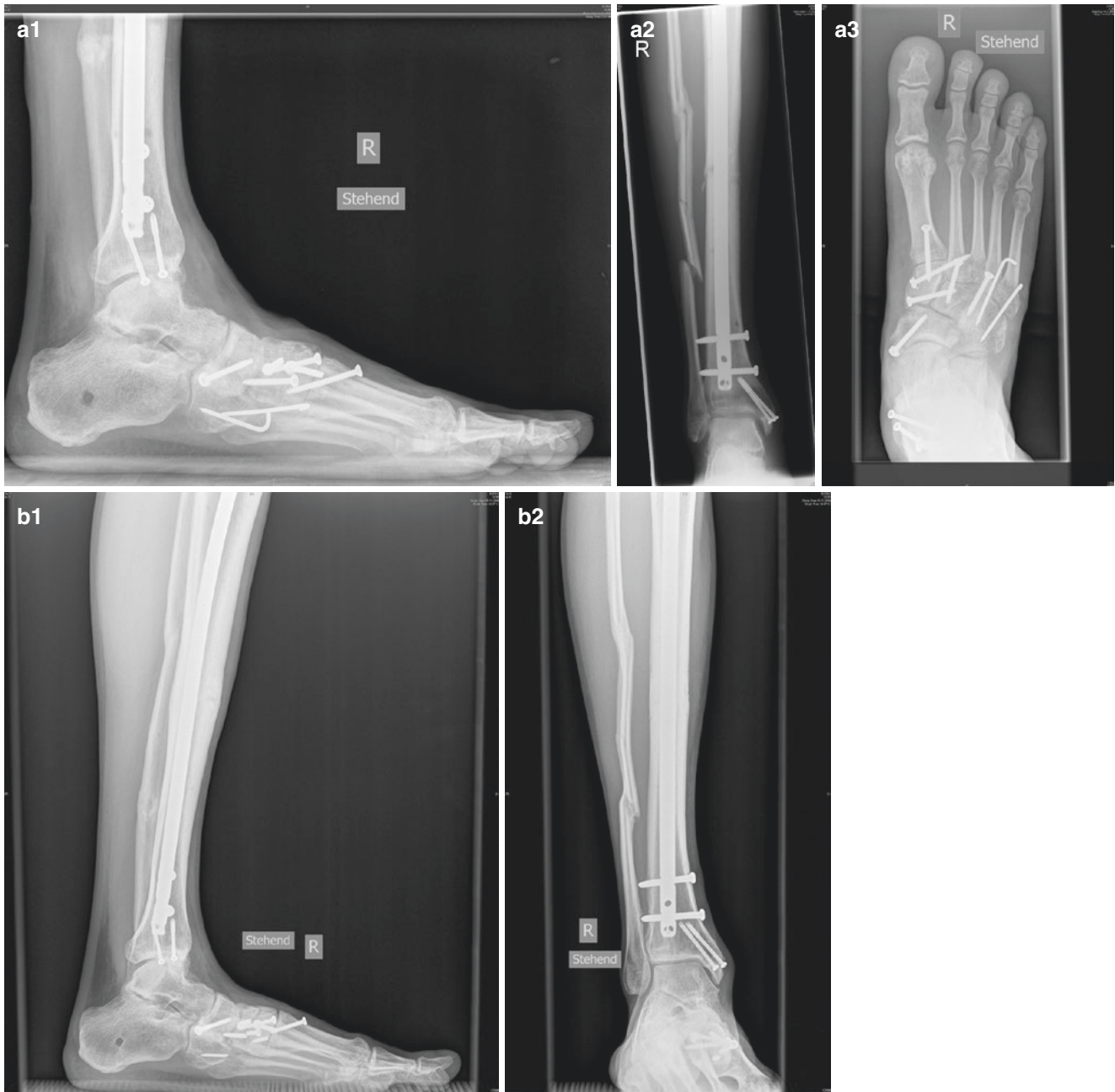
---

## Prevention

The most common foot and ankle injuries usually occur when riders are thrown from the motorcycle. So special attention should be paid to the manner of dropping.

Instruction on rolling techniques and proper falling may help to reduce injuries. Protective equipment (chest guard, kidney belt, trousers, boots, knee pads, knee brace, gauntlets), motorcycle design and helmet are important advancements [1, 4, 14]. In particular, the riders protect their foot and ankle with ankle cross shoes. Subsequently, injuries of the knee

may increase. Training experience, conditioning and skills of the riders seem to be effective strategies to avoid injuries in motorsports. In a study of Tomida it appeared that safety regulations and a better field did lower the incidence of serious injuries but did not reduce the injury rate [6]. Though complete prevention of injury cannot be expected.



**Fig. 61.1** Clinical case – professional car racer. Fractures of the tibia shaft, the medial malleolus, multilevel fractures of the fibula shaft, a non-displaced cuboid fracture, and an isolated first tarsometatarsal joint lesion with a fracture of basis of first metatarsal were diagnosed. (a) Open reduction and internal fixation (ORIF) was performed.

Radiographs at 3-month follow-up showed the consolidation process of the fractured bones and full-weight bearing could be started. (b) Two years after surgery a well alignment ankle and a painfree patient were documented

## Clinical Case

A 28-year old male professional car racer had an accident with 220 km/h and suffered a multiple trauma (Fig. 61.1). Beside other injuries, the CT-scan of the right lower leg and foot and ankle confirmed a fracture of the tibia shaft, multi-level fractures of the fibula shaft, a medial malleolar fracture, a non-displaced cuboid fracture, and an isolated first tarsometatarsal joint lesion with a fracture of basis of first metatarsal. Operative fixation and early mobilization was considered to be the best option to restore the patient concerning to the right foot and ankle. Therefore, after a temporary ExFix softtissue-stabilisation, an open reduction and internal fixation (ORIF) of the tibia with an intramedullary nail and of the medial malleolus with two screws (AO size 3.5) was performed. The syndesmosis was repaired and stabilized using a screw. Also, the Lisfranc joint was transfixed using screws (TMT 1–3) and K-wires (TMT 4–5). The fracture of the fibula was treated conservatively due to the soft-tissue situation. Due to other injuries, the mobilization was a bit complicated. Early functional physiotherapy begun with passive foot/ankle motion and a lymphatic drainage. Postoperative clinical and radiographic follow-ups showed appropriate clinical and osseous healing. One year postoperatively, partial hardware removal (tarsometatarsal) was performed. At the final follow-up of about 2 years, the patient was painfree with active ankle range of motion of 10/0/40°. Clinical evaluation revealed no ligament instability.

## Evidence

Level of evidence: III, a prospective study [4]. This study includes 1787 participants. Approximately 10% had injuries that required attention from a medical response unit. The injuries were graduated by the injury severity score (ISS) and the abbreviated injury scale (AIS). Most of them (85%) sustained mild injury and the most common types of injury were ligamentous (50%). The most common fractures were those of foot and ankle (36%). Loss of control while jumping and striking immovable objects were important risk determinants for serious injury. Speeds were below 50 km/h in the majority of accidents (80%), and were not statistically correlated with severity.

## Summary

- Motorsport is a high-risk sport with the permanently increasing number of participants and therefore increasing number of foot and ankle injuries.

- Due to the mechanism of the trauma different types of injuries (fractures, ligamentous injuries and muscle strains) can appear.
- Caused by high speed and high-energy impact severe and complex injuries like multiple fractures or combined lesions can occur.
- While ligamentous injuries and non-dislocated fractures often can be treated conservatively, dislocated and intra-articular ones as well as complex fractures require anatomic reduction and osteosynthetic fixation (ORIF).
- A functional rehabilitation program is recommended to return to sport. Fear and anxiety cause increased risk for reinjury, so the psychological aspect should be also respected.

## References

1. Gobbi A, Tuy B, Panuncialman I. The incidence of motocross injuries: a 12-year investigation. *Knee Surg Sports Traumatol Arthrosc.* 2004;12(6):574–80.
2. Barnett TM, Teasdall RD. Bilateral lateral process fracture of the talus in a motocross rider. *Foot Ankle Int.* 2008;29(2):245–7.
3. Minoyama O, Tsuchida H. Injuries in professional motor car racing drivers at a racing circuit between 1996 and 2000. *Br J Sports Med.* 2004;38(5):613–6.
4. Colburn NT, Meyer RD. Sports injury or trauma? Injuries of the competition off-road motorcyclist. *Injury.* 2003;34(3):207–14.
5. Valderrabano V. *Fuß & Sprunggelenk und Sport: Empfehlungen von Sportarten aus orthopädischer und sportmedizinischer Sicht.* Deutscher Ärzteverlag; 2009. p. 426.
6. Tomida Y, Hirata H, Fukuda A, Tsujii M, Kato K, Fujisawa K, et al. Injuries in elite motorcycle racing in Japan. *Br J Sports Med.* 2005;39(8):508–11.
7. Kinzey SJ, Armstrong CW. The reliability of the star-excursion test in assessing dynamic balance. *J Orthop Sports Phys Ther.* 1998;27(5):356–60.
8. Collins N, Teys P, Vicenzino B. The initial effects of a Mulligan's mobilization with movement technique on dorsiflexion and pain in subacute ankle sprains. *Man Ther.* 2004;9(2):77–82.
9. Sheppard JM, Young WB. Agility literature review: classifications, training and testing. *J Sports Sci.* 2006;24(9):919–32.
10. Munro AG, Herrington LC. Between-session reliability of four hop tests and the agility T-test. *J Strength Cond Res.* 2011;25(5):1470–7.
11. Clanton TO, Matheny LM, Jarvis HC, Jeronimus AB. Return to play in athletes following ankle injuries. *Sports Health Multidiscip Approach.* 2012;4(6):471–4. 1941738112463347.
12. Gribble PA, Hertel J. Considerations for normalizing measures of the star excursion balance test. *Meas Phys Educ Exerc Sci.* 2003;7(2):89–100.
13. Fong DT-P, Hong Y, Chan L-K, Yung PS-H, Chan K-M. A systematic review on ankle injury and ankle sprain in sports. *Sports Med Auckl NZ.* 2007;37(1):73–94.
14. Cordova ML, Ingersoll CD, Palmieri RM. Efficacy of prophylactic ankle support: an experimental perspective. *J Athl Train.* 2002;37(4):446–57.

Daniel Bianco

## Abstract

Orienteering is a sport that combines physical endurance and complex cognitive processes. It involves foot racing through unfamiliar wild terrain while reading a special map and a compass. Injuries caused by trips and collisions are very common, they affect the lower limb and the most frequent ones are ankle sprains of the lateral ligaments. Overuse injuries have been described related to changes in training routines. Proper gear helps prevent cuts and wounds. In case of acute ankle sprains functional management is the mainstay of treatment. After a period of rest, early mobilization and protective bracing during functional activities, facilitates recovery. Proprioceptive training enhances dynamic and functional joint stability and is effective as primary or secondary injury prevention.

## Keywords

Orienteering • Sports • Ankle sprains • Injuries • Protective bracing • Treatment

## Introduction

Orienteering is a sport that combines physical endurance and complex cognitive processes. It involves foot racing through unfamiliar wild terrain while reading a special map and a compass. The orienteer must find control locations in a determined sequence in order to complete the race. These locations are denoted by circles printed on the map, which is provided seconds before the race starts. The distance between control locations is termed a leg [1].

The first steps of this sport were in the nineteenth century under the supervision of the Swedish captain Enest Killander, who realized that using maps and compasses as a military training was an exceptional method to build up self-confidence, control and improve the ability to make decisions in difficult situations. Since 1942 orienteering was included in the Swedish national education program

and it had spread worldwide by the 1960s. Official world championships have been held biannually since 1966 and they can include different modalities besides foot-orienteering like orienteering by bike and cross-country skiing [2].

## Injuries: Etiology and Pathomechanism

In order to win the races orienteers need to complete all the legs in the shortest time possible. \*Therefore, they have to attend the map, ground underfoot and environment while running. Altogether this represents a true challenge and a short lack of attention can not only affect the running speed but also increase the risk of injuries caused by trips and collisions. Cuts and wounds can be found in any part of the body and blisters from footwear are not uncommon [3]. The most affected region of the body is the lower limb; acute injuries of the lateral ankle ligaments are most common, accounting up to 24% of all cases [4–10]. Since running is the main training activity in orienteering injuries associated with overuse are also common [11].

D. Bianco  
Orthopaedic Department, University Hospital of Basel,  
Spitalstrasse 21. 4031 Basel, Switzerland  
e-mail: [danbiancocot@gmail.com](mailto:danbiancocot@gmail.com)



## Acute and Chronic Ankle Instability

The anterior talofibular ligament is affected in most of the cases of ankle supination trauma, which can lead to anterior instability of the talus. This instability is increased in plantar flexion and if not treated properly it can overstress the calcaneofibular ligament and make it insufficient, generating a chronic ankle rotational instability over time. In some athletes, this instability leads to excessive internal rotation of the leg while running and also overloading of the medial side of the ankle [7]. This can often explain other pathologies besides ankle discomfort, like Achilles peritendinitis, tenosynovitis or ruptures of peroneal tendons [8].

Overuse injury of the shin area has been reported among orienteers [4, 12]. It often appears with a sudden increase in training intensity and duration affecting most commonly the posteromedial tibia. The symptoms are vague in early stages with diffuse pain along the middle-distal tibia at the beginning of their training that decreases while running. At later stages pain can persist during all training and even appear during daily activities. Repetitive stress over the bone surface can result in microfractures especially in the tibia [12]. Differential diagnosis sometimes is difficult and further tests like X-rays or triple phase bone scans may be needed to differentiate between overuse injuries like shin splint syndrome and stress fractures.

---

## Epidemiology

A prospective 1-year study from Denmark of 42 elite orienteers studied the types of injuries and their frequency [13]. They found 52% acute and 48% overuse injuries. Eighty two percent of the acute injuries were located in the lower extremity and acute ankle sprains were the most frequent type (37%). Overuse injuries were also observed in the ankle and knee, however they were more related to training season than competition period. Another prospective study in Sweden about injuries in elite orienteers over a year follow-up got similar results in 89 elite athletes [4]. The lower extremity was affected in 93.6% of male injuries and in all of the female injuries. Eighty percent of total injuries were minor or moderate. The most frequent traumatic injuries were ankle sprains up to 24%. Overuse injuries were common in both sexes and represented 57% of total injuries, 3% of stress fractures occurred. Recent studies from Finnish Jukola and Venla relay competitions [14] show that injuries occurred most commonly in the lower extremities (70%) and the most frequent were blisters in women and small wounds in men, followed by ankle sprains that accounted for 25% of all competition injuries. A Swiss study examined the Swiss National Orienteering during their annual check-up with a total of 43 athletes and found a prevalence of 86% for acute ankle sprains [8]. They found 57% of

the athletes to have chronic ankle instability and separated them into three groups of instability, mechanical in 29%, functional in 21% and combination of both in 7% of the cases.

---

## Therapy

Competition injuries are mostly traumatic and the majority of them vary from minor to moderate severity, which also means that they can be treated on the field and mainly in a conservative way. Cleaning and disinfection of all wounds is mandatory in order to minimize risk of infection. Stitching can be performed in first aid stations and tetanus prophylaxis should be available if needed. In case of acute ankle sprains functional management is the mainstay of treatment. After a period of rest, ice, compression and elevation of the extremity (RICE), early weight bearing and protective bracing during functional activities, facilitates early recovery compared to immobilization and non-weight bearing [15]. Once the pain and swelling cease and the athlete recuperates full range of motion, neuromuscular and proprioceptive training is the next step in therapy [16]. The time to return to practice depends on the grade of injury taking between 1 and 2 weeks for grade I-II sprains. In cases where a chronic instability is established and the compensation mechanism is insufficient to stabilize the joint from further degeneration, surgical repair may be considered [17]. In the case of overuse injuries the majority responds successfully to changes in the training routine, decreasing running distance, frequency and intensity by 50% are advised. Shoe modifications and the use of orthotics in some patients may also be required.

---

## Prevention

Wounds and contusions can easily be avoided by using adequate gear, e.g., leggings with soft pads over the knees may prevent scratches and abrasions from falls. Anti-tetanus immunization should be up to date in all the cases. Proper footwear is needed to adapt to the special demands of the terrain. Usually shoes with a deeper profile and less damping capacity facilitate better grip and proprioception. Strengthening of ankle evertor muscles, such as the peroneals, and prophylactic disk training should be advised to enhance dynamic and functional joint stability [16]. Taping or functional bracing for control of inversion and eversion is useful as primary or secondary injury prevention [8, 18, 19].

The intensity of training along the year must be controlled in order to prevent overuse injuries. High levels of aerobic fitness and strengthening of the legs is necessary to overcome the rough terrain. Strenuous training can also be related with impaired immune function that is why proper recovery time and nutrition is important to maintain high energy levels.

## Evidence

Author	Topic	Level of evidence
Folan (1982) [3]	Orienteering injuries	4
Linde (1986) [13]	Orienteering injuries	4
Johansson (1986) [4]	Injuries in elite orienteers	3
Hintermann and Hintermann (1992) [6]	Injuries in orienteering. Swiss 6-day orienteering event	4
Kujaka et al. (1995) [5]	Acute injuries in orienteers	3
Linko et al. (1997) [14]	Orienteering competition injuries	4
Leumann et al. (2010) [8]	Chronic ankle instability in Swiss Orienteering National Team	4
Leumann et al. (2010) [12]	Injuries in orienteering: ankle instability and overuse injuries	5

## Summary

- Orienteering is a safe sport with few severe injuries.
- Acute ankle sprains are the most frequent injuries.
- Overuse injuries are common and can be avoided by changes in training routine.
- Proper equipment is necessary to minimize wounds from falls.
- Proprioception training and strengthening of peroneal muscles is effective as primary and secondary prophylaxis for ankle sprains.

## References

1. Eccles D, Arsal G. How do they make it look easy? The expert's orienteer's cognitive advantage. *J Sports Sci.* 2015;33(6):609–15. doi:10.1080/02640414.2014.951953. Epub 2014 Aug 26.
2. Creagh U, Reilly T. Physiological and biomechanical aspects of orienteering. *Sports Med.* 1997;24(6):409–18.
3. Folan JM. Orienteering injuries. *Br J Sports Med.* 1982;16(4):236–40.
4. Johansson C. Injuries in elite orienteers. *Am J Sports Med.* 1986;14(5):410–5.
5. Kujala UM, Nylund T, Taimela S. Acute injuries in orienteers. *Int J Sports Med.* 1995;16(2):122–5.
6. Hintermann B, Hintermann M. Injuries in orienteering. A study of the 1991 Swiss 6-days orienteering event. *Sci J Orienteering.* 1992;8:72–8.
7. Hintermann B, Hintermann M. Ankle sprains in orienteering – a simple injury? *Sci J Orienteering.* 1992;8:79–86.
8. Leumann A, Zuest P, Valderrabano V, Geman C, Bernard M, Hintermann B. Chronic ankle instability in the Swiss Orienteering National Team. *Sportorthopädie und Sporttraumatologie.* 2010;26(1):20–8.
9. Ekstrand J, Roos H, Trop H. The incidence of ankle sprains in orienteering. *Sci J Orienteering.* 1990;6:3–9.
10. Tiki-Pui Fong D, Hong Y, Chan L, Yung P, Chan K. A systematic review on ankle injury and ankle sprain in sports. *Sports Med.* 1997;37(1):73–94.
11. Johansson C. Training, injury and disease in senior and junior elite orienteers. *Sci J Orienteering.* 1988;4:3–13.
12. Leumann A, Zuest P, Geman C, Valderrabano V. Injuries in orienteering: ankle instability and overuse injuries. *Sportmedizin und Sporttraumatologie.* 2010;58(2):49–51.
13. Linde F. Injuries in orienteering. *Br J Sports Med.* 1986;20(3):126–7.
14. Linko PE, Bolmberg HK, Frilander HM. Orienteering competition injuries: injuries incurred in the Finnish Jukola and Venla relay competitions. *Br J Sports Med.* 1997;31(3):205–8.
15. Digiovanni BF, Partal G, Baumhauer JF. Acute ankle injury and chronic lateral instability in the athlete. *Clin Sports Med.* 2004;23(1):1–19.
16. Lephart SM, Pincivero DM, Giraldo JL, Fu FH. The role of proprioception in the management and rehabilitation of athletic injuries. *Am J Sports Med.* 1997;25(1):130–7.
17. Karlsson J, Lansinger O. Chronic lateral instability of the ankle in athletes. *Sports Med.* 1993;16(5):355–65.
18. Janssen KW, van Mechelen W, Verhagen EA. Bracing superior to neuromuscular training for the prevention of self-reported recurrent ankle sprains: a three-arm randomised controlled trial. *Br J Sports Med.* 2014;48(16):1235–9.
19. Hintermann B, Valderrabano V. The effectiveness of rotational stabilization in the conservative treatment of severe ankle sprains: a long-term investigation. *Foot Ankle Surg.* 2001;7:235–9.

Elango Selvarajah and Timothy Schneider

**Abstract**

Rugby union is a popular contact team sport. It is played by 6.6 million players in 120 countries [1]. It can result in various injuries to players with the resultant loss of training and playing time. This chapter provides an evidenced based approach to the commonest foot and ankle injuries in rugby from the perspective of a sports physician. This chapter covers the injury mechanism, on field treatment, detailed assessment and definitive treatment, and evidence for preventive strategies are outlined. Treatment and rehabilitation with a view of early return to function are discussed.

**Keyword**

Rugby foot ankle injury

Rugby, also called Rugby Union is one of the most popular team contact sports in the world [1]. It is played by 6.6 million players in 120 countries [1]. Rugby is a collision sport played by two teams with 15 players each. The players are divided into forwards and backs. Forwards win the ball from the opposing team and the back use the ball to score points. Forward players use a scrum (Fig. 63.1) or ruck and maul (Fig. 63.2) or a line out (Fig. 63.3) to win the ball. Five points are awarded for grounding the ball (called a try) within the opposition defence line (try line) and two points are awarded for kicking the ball between the goal posts after a try and three points are awarded for a penalty kick between the goal posts.

Rugby has one of the highest rates of injury of all sports [2]. Players sustain injuries during match play and during training.

Most of the injuries occur in or during contact between players. However, injuries can occur during accelerating, pivoting,

jumping/landing or kicking without any contact. Studies report around 50% of match injuries involving the lower limb [3–7]. Out of the lower limb injuries 33% of injuries involve the foot and ankle in professional rugby players [3].

This chapter aims to provide an outline of these injuries and their management.

**Ankle Injuries**

Ankle injuries accounted for 11% of all match injuries and 15% of all training injuries. This resulted in 10% of loss from playing time and 13% loss of training time [8]. The commonest ankle injuries sustained during professional rugby are the lateral ligament complex injuries followed by syndesmototic injuries, Achilles tendon injuries, ankle cartilage injuries, ankle joint capsule sprains and deltoid ligamentous injuries [8, 9]. Bony injuries are not common but they are the most severe with lateral malleolar fractures being the commonest [8, 9]. Overall, ankle lateral ligamentous injuries (29%) and Achilles tendon injuries (27%) together accounted for more than half of the absence from training and match play due to ankle injuries [8].

---

E. Selvarajah • T. Schneider (✉)  
Melbourne Orthopaedic Group, 33 The Avenue,  
Windsor, VIC 3181, Australia  
e-mail: [elango\\_selvarajah@yahoo.com](mailto:elango_selvarajah@yahoo.com); [taschneid@optusnet.com.au](mailto:taschneid@optusnet.com.au)

**Fig. 63.1** 2011 rugby world cup champions New Zealand All Blacks contesting the ball against the Australian Wallabies in a rugby scrum (Getty images)



**Fig. 63.2** 2011 rugby world cup champions New Zealand All Blacks contesting the ball against the Australian Wallabies in a rugby maul (Getty images)



## Lateral Ligamentous Injuries

Main components of the lateral ligamentous complex involve the Anterior Talofibular Ligament (ATFL) and the Calcaneo Fibular Ligament (CFL). Lateral ligament complex injuries were the most common ankle injuries during training and match play among rugby players with an incidence of 2.4–4.2 injuries/1000 h of match play [8, 9]. The incidence has declined over subsequent seasons [9]. Indicating greater awareness and preventive strategies.

These injuries are caused by an inversion mechanism with an inverted, plantar flexed foot, with an internally rotated hind foot on which the leg externally rotates.

Players are likely to report an incident during contact or landing from a jump with a twisting and inversion trauma to the ankle. They are likely to have associated swelling, ecchymoses over the lateral hind foot and difficulty weight bearing.

On field therapy involves, rapid assessment with removing player from the field with assistance. Rest Ice Compression Elevation (RICE) treatment should be begun

**Fig. 63.3** 2011 rugby world cup champions New Zealand All Blacks contesting the ball against the Australian Wallabies in a rugby lineout (Getty images)



on the side lines of the playing field. Temporary splinting would be beneficial to rest and provide support to the ankle. A delayed detailed clinical assessment with radiological assessment as required should be planned.

Confirmed lateral ligamentous injuries should be treated with a functional rehabilitation protocol [10, 11]. It is important to exclude other lesions such as syndesmosis injury, or ankle chondral lesions as they may not respond to a functional treatment protocol, but may require other early intervention. Usually functional bracing is recommended for 3–4 weeks with progressive weight bearing in the brace as pain permits. Early non weight bearing range of motion exercises should be started. Once non protected weight bearing is pain free begin proprioceptive and strengthening exercises and progress onto agility training. Peroneal strengthening is key for recovery and prevention of these injuries. Functional therapy is recommended for 6 weeks prior to returning to sports. Return to sports is recommended when players can run and pivot without pain while the ankle is taped in a figure eight position [11]. Taping is recommended for up to 6 months after injury and gradually weaned off [11]. Up to 34–40% may have residual or recurrent symptoms [11, 12]. For these players a surgical opinion should be sought for a lateral ligament reconstruction.

Inappropriate foot wear, cavovarus feet, and poor playing technique can be addressed to prevent injuries. But evidence for this is limited.

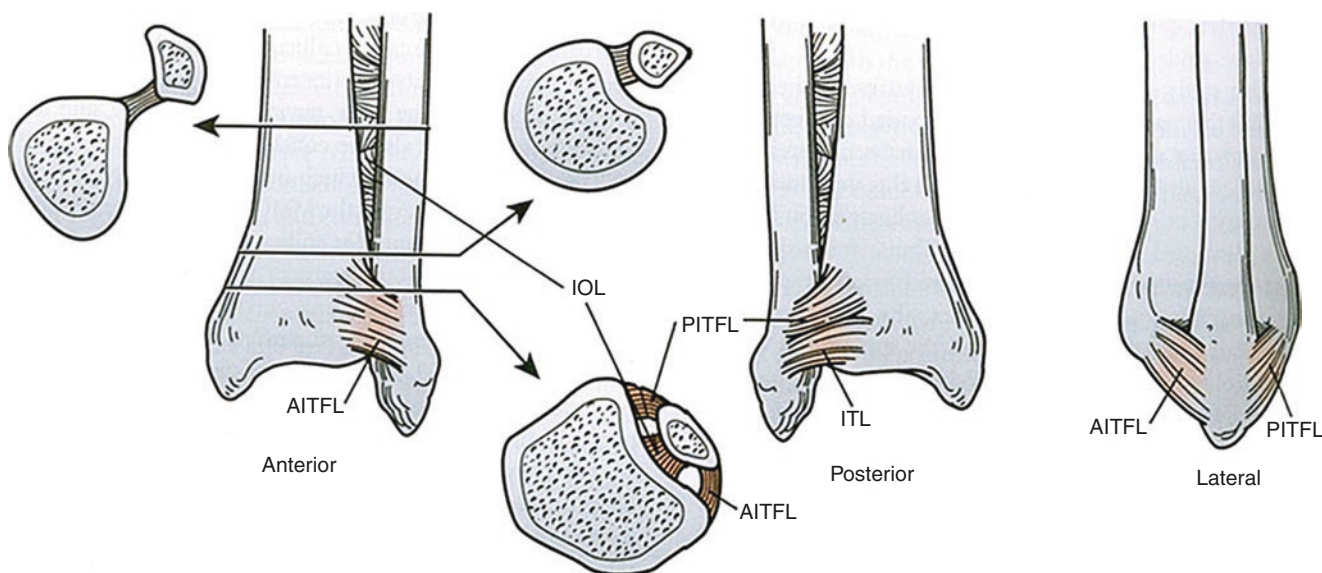
**Summary** Lateral ligament injuries can be treated with functional treatment with equally good results when compared to acute surgical repair.

**Evidence: Level 1** Pihlajamaki H, Visuri T et al: Surgical Versus Functional Treatment for Acute Ruptures of the Lateral Ligament Complex of the Ankle in Young Men: a randomized controlled trial. *Journal of Bone and Joint Surgery Am.* 2010;92(14):2367-2374.

### Ankle Syndesmotic Injuries

The incidence of syndesmotic injuries is 11% of all ankle injuries in professional rugby players [8]. The ankle syndesmosis consists of anterior inferior tibiofibular ligament (AITFL), the posterior inferior tibiofibular ligament (PITFL), the interosseous ligament (IOL) and the inferior transverse ligament (Fig. 63.4) [13]. The interosseous ligament is in continuity with the interosseous membrane proximally. The fibrocartilaginous inferior transverse ligament forms the distal portion of the posterior inferior tibiofibular ligament and can be considered as the same structure [13]. The AITFL (35%) PITFL (33%) contribute the most to ankle syndesmotic stability with the IOL (22%) contributing the rest [13, 14].

The syndesmotic ligaments are injured usually with an externally rotated and dorsiflexed foot and axial loading, but other mechanisms are possible [13]. With this mechanism AITFL is the first and commonest to be injured. PITFL is the last to be injured. If the entire syndesmotic ligamentous complex is injured then ankle diastasis will result. In the presence of ankle diastasis there is likely to be a high rate of associated injuries including fibular fractures, medial ligamentous injuries and medial malleolar fractures.



**Fig. 63.4** Ankle syndesmotism anatomy (Reproduced with permission from Davidovitch and Egol [38], Figure 57–12)

Players will have pain and swelling over the syndesmosis region and are unable to fully weight bear on the injured side. With rapid assessment players will need to be removed from the field and the RICE treatment should be begun with splinting until detailed clinical and radiological assessment can be completed [13].

Syndesmotism injuries without a fracture or ankle diastasis can be treated with non weight bearing and a moon boot until pain resolves. Early mobilisation and weight bearing as tolerated is recommended [15]. This usually takes 6–12 weeks to resolve. Impact sports should be avoided for at least 6 weeks.

Players with persistent symptoms, with ankle diastasis or a fracture are likely to require surgical intervention [13].

**Summary** Ankle syndesmotism injuries without Tibiofibular diastasis can be treated non surgically. Injuries with Tibiofibular diastasis will require surgical fixation.

**Evidence: Level 4** Heest TJ, Lafferty PM. Current Concept review: Injuries to the Ankle Syndesmosis. *The Journal of Bone and Joint Surgery (Am)*. 2014;96:603-613.

## Achilles Tendon Injuries

Achilles tendon injuries are the 16th commonest reason for loss of playing time overall in the 2011–12 season among English professional rugby players [9]. Achilles tendon injuries result in a significant amount of time lost to playing and training among rugby players [9].

Acute Achilles tendon ruptures result from an acute traumatic event where the patient feels as if there has been a kick in the back of the calf and unable to walk afterwards with

associated pain and swelling. In rugby this is usually the result of explosive play such as acceleration, running pivoting or tackling.

Examination will likely show ecchymosis with decreased resting tension in the foot. There will be a palpable gap in the tendon and calf squeeze will not elicit a response in the foot. Players will need rapid assessment on the field followed by assisted removal from the field. Icing on the field with application of a splint with the foot in equinus will be beneficial. Delayed complete clinical assessment should include the history and any chronic issues with the tendon as well as treatment to date.

Functional treatment of acute Achilles tendon ruptures have been shown to result in equally good outcomes when compared to surgical treatment [16, 17]. Non surgical treatment will negate any risks from surgery but studies show a higher rate of rerupture with non surgical treatment (8 vs 4 % for surgical group) [17, 18]. However, this has been challenged by other studies (Glazebrook et al.) [19].

Non surgical treatment will involve casting the injured limb in equinus for 2 weeks with non weight bearing mobilisation. At 2 weeks if clinical assessment confirms continuity of tendon then patient can be placed in to a moon boot locked in 20° of plantar flexion to unlimited plantar flexion [16, 17]. Patients are allowed to move the foot within this range. At 4 weeks from injury the brace should be set in neutral plantar flexed position permitting gentle motion from neutral to full plantar flexion. At 6 weeks if tendon is in continuity then patient can begin weight bearing in the brace in addition to gentle dorsiflexion out of brace. May begin supervised physiotherapy [16, 17]. Patients can wean off brace at 8 weeks from injury.

If at any time the tendon is felt to be not in continuity then surgical repair should be considered. Surgery can also be considered as the primary treatment method, with the rehabilitation similar to nonsurgical treatment protocol.

**Summary** Acute Achilles tendon ruptures can be treated surgically or with functional treatment, producing equally good outcomes. Functional treatment may have higher rates of rerupture but avoids all the risks associated with surgery.

**Evidence: Level 1** 1. Twaddle BC, Poon P: Early motion for Achilles tendon ruptures: Is surgery important? A randomized, prospective study. *Am J Sports Med* 2007; 35: 2033–2038.

2. Nilsson-Helander K, Silbernagel KG, Thomee R, et al: Acute Achilles tendon rupture: a randomized, controlled study comparing surgical and nonsurgical treatments using validated outcome measures. *Am J Sports Med* 2010; 38: 2186–2193.

3. Soroceanu A, Sidhwa F, Aarabi S, Kaufman A, Glazebrook M. Surgical versus nonsurgical treatment of acute Achilles tendon rupture: a meta-analysis of randomized trials. *J Bone Joint Surg Am* 2012;94:2136–2143.

---

## Chronic Achilles Tendinopathy

Chronic Achilles tendon dysfunction, which is another common complaint among rugby players has been divided into insertional and non insertional tendinopathies. Non insertional Achilles tendinopathy is often managed with nonsurgical treatment while insertional disease is managed by identifying coexisting pathology around the site of insertion and addressing those issues [20].

Non insertional Achilles tendinopathy can be treated initially with non surgical modalities. Rest, activity modification, eccentric stretching, shoe modifications and orthotics can be trialled initially [20]. Extra corporeal shock wave therapy can be trialled in conjunction with eccentric stretching if the initial treatment fails [21]. Evidence of benefit from other modalities such as non steroidal anti inflammatory, glyceryl trinitrate patches and Platelet rich plasma injections are limited and in fact there is evidence these modalities can be detrimental to the healing and overall quality of the tendon [20].

Surgery is likely to involve debridement of tendon as necessary, with repair if enough tendon is available, or if tendon quality is poor then reconstruction with FHL tendon [22, 23]. Post operative rehabilitation will be similar to the above outline with acute ruptures.

Treatment of insertional tendinopathy depends on surrounding pathology as well as treating the tendon itself. Nonsurgical therapy involves heel wedges, regular stretching exercises and extracorporeal shockwave therapy [23–25]. Again, similar to non insertional Achilles tendinopathy, no conclusive evidence is available in the literature for other modalities in the treatment of insertional Achilles tendinopathy.

Surgical treatment of insertional disease involves calcaneoplasty, debridement of retrocalcaneal bursa with or with-

out tendon repair or reconstruction with flexor hallucis longus tendon (FHL) [23, 26].

**Summary 1. Insertional Achilles tendinopathy:** Rest, activity modification and eccentric stretching with ECSW therapy is first line of treatment. Surgical debridement with repair or reconstruction can be considered for failure of non surgical treatment.

**Evidence: Level 1** Rompe JD, Furia J, Maffulli N. Eccentric loading versus eccentric loading plus shock-wave treatment for midportion achilles tendinopathy: a randomized controlled trial. *Am J Sports Med* 2009;37:463–470.

2. **Non insertional Achilles tendinopathy:** Rest, activity modification, regular stretching exercises, heel wedges and ECSW are initial non surgical modalities. Surgical debridement of retrocalcaneal bursa and calcaneal prominences with repair or reconstruction using FHL should be considered for cases of failed insertional disease.

**Evidence: Level 3** 1. Kearney R, Costa ML. Insertional achilles tendinopathy management: a systematic review. *Foot Ankle Int* 2010;31:689–694.

2. Roche AJ, Calder JDF. Achilles tendinopathy. A review of the current concepts of treatment. *Bone Joint J* 2013;95-B:1299–1307

---

## Medial Ankle Ligament Injuries

Rugby has one of the highest incidences of Medial ankle sprains among all sports [27]. Deltoid ligament has a deep and superficial layer and they are injured with an eversion type mechanism. These tend to be high energy injuries due to the strength and surface area of the ligament in addition to the inherent stability of the bony ankle to eversion.

The player is likely to complain of pain and swelling in the medial ligament region following a traumatic event, usually either in a tackle situation, a ruck and maul or scrum where there was a significant eversion injury to the ankle.

On field assessment should be followed by RICE treatment initially. A detailed clinical assessment including radiological studies should be completed. X-rays should also look for proximal fibular fractures to rule out a Maisonneuve injury and MRI scans to look at the medial ligamentous structures and the syndesmosis is recommended.

Isolated medial ligamentous injuries can be treated non surgically with outcomes similar to surgical repair [28, 29]. Treatment is in a locked moon boot with medial arch support to prevent eversion of the foot. Weight bearing as tolerated is permitted in the boot. The brace is to be worn for 24 h a day

or consider casting. The bracing is recommended for 6 weeks at which point physiotherapy can be started. Early immobilization is a high priority as the condition has the potential to cause significant long term morbidity.

Players can be weaned off the moon boot starting at 6 weeks from injury and begin graduated return to rugby specific exercises with a view of returning to rugby at 12 weeks from injury.

If there are ongoing symptoms of pain or instability surgical intervention including arthroscopic assessment and/or reconstruction should be considered [28, 30].

**Summary** Medial ankle ligament injuries can be treated non surgically with immobilisation initially, followed by physiotherapy.

**Evidence: Level 1** Stromsoe K, Hoqevold HE, Skjeldal S, Alho A: The repair of a ruptured deltoid ligament is not necessary in ankle fractures. *J Bone Joint Surg Br.* 77(6): 920–921, 1995.

---

## Lisfranc Injuries

These are rare injuries in rugby players with only 1% of all foot injuries in rugby union players make up lisfranc injuries [31]. These injuries are caused by the foot in a fixed plantar flexed or plantigrade position with a twisting force applied to it. These injuries can also occur from direct trauma to the dorsal aspect of the mid foot with tension failure over the plantar aspect of the foot [32, 33].

Players will commonly report a twisting injury to the foot either in a tackle situation or a ruck and maul. Players should be initially treated with RICE treatment pending a detailed clinical assessment. Clinical examination is likely to show ecchymoses on the dorsal and/or plantar aspect of the mid foot with localised tenderness in the lisfranc region. Inability to fully weight bear on the limb is a common finding. X-rays and MRIs can be used to confirm and grade the injury. Weight bearing x-rays with x-rays of contralateral side weight bearing x-rays can be very useful to make the diagnosis.

Lisfranc injuries or sprains with no displacement on weight bearing at the Lisfranc region can be considered for non surgical treatment [34]. Initial casting with non weight bearing mobilisation is recommended for 6 weeks [34]. Then removable orthosis, with a well moulded arch support, and gradual weight bearing is permitted depending on pain. Repeat weight bearing x-rays to look for displacement is critical at regular intervals and if there is ongoing pain or displacement at anytime then surgical opinion should be sought.

Lisfranc injuries with confirmed displacement at initial assessment will need early surgical intervention with open

reduction and internal fixation [35, 36]. Players will be kept non weight bearing on the limb for 6 weeks followed by ankle and hind foot range of motion exercises starting at 6 weeks. They need to wear a splint for further 6 weeks. Non impact aerobic exercises such as cycling or swimming can be started at 6 weeks. Metalware removal can be considered from 3 months onwards. Graduated return to rugby specific injuries can begin after metalware removal.

Lisfranc injuries can be severe injuries and players need to be counselled with regards to long term complications such as stiffness, residual pain, complex regional pain syndrome and arthritis [33, 37]. Midfoot arthrodesis can be considered as salvage for unresolved or complex lisfranc injuries [35].

**Summary** Lisfranc injuries without diastasis can be treated non surgically. However, lisfranc injuries with diastasis will require urgent open reduction and internal fixation.

**Evidence: Level 3** Rammelt S., Schneiders W., Schikore H., et al: Primary open reduction and fixation compared with delayed corrective arthrodesis in the treatment of tarsometatarsal (Lisfranc) fracture dislocation. *J Bone Joint Surg Br* 2008; 90: pp. 1499–1506.

---

## Foot and Ankle Fractures

Fractures of the foot and ankle region are uncommon injuries in rugby players with only 1.2% of all ankle injuries in rugby players involve lateral malleolar fractures. Of all the foot injuries in rugby players, 10% involve fractures in the foot [8, 31].

On field treatment includes removal of player with initial RICE treatment. If there is a strong suspicion of fracture then a temporary splint with radiological assessment is recommended. Fractures, in general need to be assessed with radiological imaging in addition to the associated soft tissue injuries. Treatment of acute fractures varies from neighbour strapping of toe injuries to surgical fixation as outlined elsewhere in this book.

---

## References

1. International Rugby Board. <http://www.irb.com/mm/Document/AboutIRB/IRBOrganisation/02/07/03/26/IRB010GlobalMembershipMapOnlinepdfv7.pdf>.
2. Nicoll JP, Coleman P, Williams BT. The epidemiology of sports and exercise related injury in the United Kingdom. *Br J Sports Med.* 1995;29:232–8.
3. Fuller CW, Sheerin K, Targett S. Rugby World Cup 2011: International Rugby Board Injury Surveillance Study. International Rugby Board Website; <http://irbplayerwelfare.com/?documentid=94>.
4. Bathgate A, Best JP, Craig G, Jamieson M, Wiley J. A prospective study of injuries to elite Australian rugby union players. *Br J Sports Med.* 2002;36(4):265–9.



5. Brooks JH, Fuller CW, Kemp SP, Reddin DB. Epidemiology of injuries in English professional rugby union. Part 1. Match injuries. *Br J Sports Med.* 2005;39:757–66.
6. Brooks JH, Fuller CW, Kemp SP, Reddin DB. Epidemiology of injuries in English professional rugby union. Part 2. Training Injuries. *Br J Sports Med.* 2005;39:767–75.
7. Jakoet I, Noakes TD. A high rate of injury during the 1995 Rugby World Cup. *S Afr Med J.* 1998;88:45–7.
8. Sankey RA, Brooks JHM, Kemp SPT, Haddad FS. The epidemiology of ankle injuries in professional rugby union players. *Am J Sports Med.* 2008;36(12):2415–24.
9. England professional rugby injury surveillance project: 2011–2012 season report. International Rugby Board website. <http://irbplayer-welfare.com/?documentid=109>.
10. Pihlajamaki H, Hietaniemi K, Paavola M, Visuri T, Mattila VM. Surgical versus functional treatment for acute ruptures of the lateral ligament complex of the ankle in young men: a randomized controlled trial. *J Bone Joint Surg Am.* 2010;92(14):2367–74.
11. Kerkhoffs GM, van den Bekerom M, Elders LAM, et al. Diagnosis, treatment and prevention of ankle sprains: an evidence-based clinical guideline. *Br J Sports Med.* 2012;46(12):854–60.
12. van Rijn RM, van Os AG, Bernsen RMD, Luijsterburg PA, Koes BW, Bierma-Zeinstra SMA. What is the clinical course of acute ankle sprains? A systematic literature review. *Am J Med.* 2008;121(4):324–31.
13. Heest TJ, Lafferty PM. Current concept review: injuries to the ankle syndesmosis. *J Bone Joint Surg (Am).* 2014;96:603–13.
14. Ogilvie-Harris DJ, Reed SC, Hedman TP. Disruption of the ankle syndesmosis: biomechanical study of the ligamentous restraints. *Arthroscopy.* 1994;10(5):558–60.
15. Williams GN, Jones MH, Amendola A. Syndesmotic ankle sprains in athletes. *Am J Sports Med.* 2007;35(7):1197–207.
16. Twaddle BC, Poon P. Early motion for Achilles tendon ruptures: is surgery important? A randomized, prospective study. *Am J Sports Med.* 2007;35:2033–8.
17. Nilsson-Helander K, Silbernagel KG, Thomee R, Faxen E, Olsson N, Eriksson BI, Karlsson J. Acute Achilles tendon rupture: a randomized, controlled study comparing surgical and nonsurgical treatments using validated outcome measures. *Am J Sports Med.* 2010;38(11):2186–93.
18. Wilkins R, Bisson LJ. Operative versus nonoperative management of acute Achilles tendon ruptures: a quantitative systematic review of randomized controlled trials. *Am J Sports Med.* 2012;40:2154–60.
19. Soroceanu A, Sidhwa F, Aarabi S, Kaufman A, Glazebrook M. Surgical versus nonsurgical treatment of acute Achilles tendon rupture: a meta-analysis of randomized trials. *J Bone Joint Surg Am.* 2012;94(23):2136–43.
20. Roche AJ, Calder JDF. Achilles tendinopathy. A review of the current concepts of treatment. *Bone Joint J.* 2013;95-B:1299–307.
21. Rompe JD, Furia J, Maffulli N. Eccentric loading versus eccentric loading plus shock-wave treatment for midportion achilles tendinopathy: a randomized controlled trial. *Am J Sports Med.* 2009;37:463–70.
22. Martin RL, Manning CM, Carcia CR, Conti SF. An outcome study of chronic Achilles tendinosis after excision of the Achilles tendon and flexor hallucis longus tendon transfer. *Foot Ankle Int.* 2005;26(9):691–7.
23. Paavola M, Kannus P, Orava S, Pasanen M, Järvinen M. Surgical treatment for chronic Achilles tendinopathy: a prospective seven month follow up study. *Br J Sports Med.* 2002;36(3):178–82.
24. Kearney R, Costa ML. Insertional achilles tendinopathy management: a systematic review. *Foot Ankle Int.* 2010;31(8):689–94.
25. Furia JP. High-energy extracorporeal shock wave therapy as a treatment for insertional Achilles tendinopathy. *Am J Sports Med.* 2006;34(5):733–40.
26. Johnson KW, Zalavras C, Thordarson DB. Surgical management of insertional calcific Achilles tendinosis with a central tendon splitting approach. *Foot Ankle Int.* 2006;27(4):245–50.
27. Waterman BR, Belmont PJ, Cameron KL, Svoboda SJ, Alitz CJ, Owens BD. Risk factors for syndesmotic and medial ankle sprain: role of sex, sport, and level of competition. *Am J Sports Med.* 2011;39(5):992–8.
28. Hintermann B, Knupp M, Pagenstert GI. Deltoid ligament injuries: diagnosis and management. *Foot Ankle Clin North Am.* 2006;11:625–37.
29. Stromsoe K, Hoqevold HE, Skjeldal S, Alho A. The repair of a ruptured deltoid ligament is not necessary in ankle fractures. *J Bone Joint Surg Br.* 1995;77(6):920–1.
30. Beals TC, Crim J, Nickisch F. Deltoid ligament injuries in athletes: techniques of repair and reconstruction. *Oper Tech Sports Med.* 2010;18(1):11–7.
31. Pearce CJ, Brooks JHM, Kemp SPT, Calder JDF. The epidemiology of foot injuries in professional rugby union players. *Foot Ankle Surg.* 2011;17:113–8.
32. Coetzee JC. Making sense of Lisfranc injuries. *Foot Ankle Clin North Am.* 2008;13:695–704.
33. Myerson MS, Fisher RT, Burgess AR, Kenzora JE. Fracture dislocations of the tarsometatarsal joints: end results correlated with pathology and treatment. *Foot Ankle.* 1986;6(5):225–42.
34. Nunley JA, Vertullo CJ. Classification, investigation, and management of midfoot sprains: Lisfranc injuries in the athlete. *Am J Sports Med.* 2002;30(6):871–8.
35. Mulier T, Reynders P, Dereymaeker G, Broos P. Severe Lisfranc injuries: primary arthrodesis or ORIF? *Foot Ankle Int.* 2002;23(10):902–5.
36. Rammelt S, Schneiders W, Schikore H, Holch M, Heineck J, Zwipp H. Primary open reduction and fixation compared with delayed corrective arthrodesis in the treatment of tarsometatarsal (Lisfranc) fracture dislocation. *J Bone Joint Surg Br.* 2008;90(11):1499–506.
37. Arntz CT, Veith RG, Hansen ST. Fractures and fracture-dislocations of the TMT joint. *J Bone Joint Surg Am.* 1988;70:173–81.
38. Davidovitch RI, Egol KA. Ankle fractures. In: Bucholz RW, Heckman JD, Court-Brown CM, Tornetta P, editors. *Rockwood and green's fractures in adults.* Philadelphia: Lippincott Williams & Wilkin; 2010.

Dave Santone and Timothy R. Daniels

**Abstract**

Ankle injuries specific to running or sports involving running are rare, but both soft tissue and bony injuries can occur in running athletes. Many running-related injuries are caused by repetitive impact stress in individuals who are either unprepared for the physical load, or have subtle to significant foot deformities. While the majority of these injuries are minor and often resolve with non-operative management, others can result in chronic pain, deformity and eventual loss of function and ability to partake in sport. Early identification and management are key. Once a running injury has occurred, cessation of the activity for an appropriate period of time is essential for healing and to enable the individual to return to running. It is important for the clinician to carefully assess the foot and ankle for deformity and/or instability; identifying the etiology is vital for developing an appropriate treatment pathway. Subtle foot deformities can often be managed by appropriate orthotic and shoe modifications. At times, the only long-term solution for repetitive or persistent running injuries is to permanently modify the exercise routine and/or duration of activity.

**Keywords**

Stress fractures • Sesamoid • Accessory navicular • Os Trigonum syndrome • Ankle sprains • Achilles tendinopathy • Plantar fasciitis

Ankle injuries specific to running or sports involving running are rare, but there has been a noted increase in their incidence. Both soft tissue and bony injuries can occur in running athletes. While the majority of these injuries are minor and often resolve with non-operative management, others can result in chronic pain, deformity and eventual loss of function with ability to partake in sport. Early identification and management are key [1].

D. Santone, MD, FRCSC  
Department of Orthopaedic Surgery, St. Michael's Hospital,  
Toronto, ON, Canada

238 Montebello Ave., Woodbridge, ON L4H 1L6, Canada  
e-mail: [dave.santone@gmail.com](mailto:dave.santone@gmail.com)

T.R. Daniels, MD, FRCSC (✉)  
Department of Orthopaedic Surgery, St. Michael's Hospital,  
Toronto, ON, Canada

Professor, University of Toronto, Toronto, ON, Canada  
e-mail: [danielst@smh.ca](mailto:danielst@smh.ca)

**Bony Injuries: Stress Fractures****Epidemiology**

Stress fractures are common in runners. The most commonly affected foot bones include the second metatarsal, navicular and third and fourth metatarsals. It has been suggested that metatarsal stress fractures comprise 10% of all sports overuse injuries [2].

**Etiology and Pathomechanism**

The cumulative effects of repetitive and substantial loads, combined with metabolic, anatomic, and environmental factors, are thought to precipitate stress fractures in the foot. The second and third metatarsals are the longest, most rigid forefoot bones and thus are prone to fracture; however, the fourth and fifth metatarsals are also susceptible. Established risk factors for stress frac-

tures include limb length inequality, cavus foot, excessive running, and menstrual irregularities [3]. An increased incidence of stress fractures occurs with sudden changes in the frequency, duration or intensity of activity (i.e., march fracture) or with specific anatomic configurations of the foot (i.e., pes cavus) [4].

### Therapy (On the Field, Conservative, Surgical)

Initial management of stress fractures includes rest, ice and immobilization. The vast majority of stress fractures can be treated non-operatively and typically heal within 6–8 weeks. A delay in healing may require additional therapeutic modalities such as bone stimulation, non-weight bearing, rigid external support, or prolonged immobilization. An aircast, well-moulded fiberglass cast or postoperative shoe may be considered. While surgery is rare, it may be required for displaced fractures or fracture non unions. In either case, a small longitudinal incision centered over the fracture site can be made, with a dorsal plate applied to the metatarsal with or without reduction. If surgery is indicated and bone grafting is also indicated, then a biologic augment may be considered [4]. All available evidence for stress fractures is Level III and Level IV.

### Rehabilitation and Back-to-Sports

Return to sport is gradual and is guided by radiographic and clinical exam, as well as pain. Running is typically avoided for 4–6 weeks, or until the patient is free of pain during daily activities for 2 weeks [4]. The patient should be warned that a stress fracture of the adjacent metatarsal may occur, especially if there is any dorsal angulation of the fractured metatarsal, as it will be under increased stress.

### Prevention

Individuals who start an exercise regimen after a period of inactivity should be counseled to begin gradually. They should be instructed to wear shoes with a supportive shank and a slight forefoot rocker. Many jogging runners are designed with these modifications. Orthotics with hindfoot posting and a metatarsal pad are optional if the patient has not had a previous metatarsal stress fracture, but with a history of stress fracture, they are recommended.

---

## Bony Injuries: Sesamoid Injury

### Epidemiology

Although small in structure, injury to the hallucal sesamoids often result in considerable pain, discomfort and inability to

partake in sporting activities. Due to its larger size, the tibial sesamoid occupies a larger portion of the plantar aspect of the distal first metatarsal and thus absorbs more weight-bearing forces during activities. This results in more frequent injuries to the tibial, relative to the fibular, sesamoid [5]. Most injuries to the sesamoids involve overuse injuries such as stress fracture (40%), sesamoiditis (30%), acute fracture (10%), osteochondritis (10%), osteoarthritis (5%) and bursitis (5%) [5].

### Etiology and Pathomechanism

The etiology depends on the type of sesamoid disorder. Etiologies include crush injury, hyperextension injury to the metatarsophalangeal joint, chondromalacia, impingement or repetitive overloading of the medial forefoot [3]. Patients with a subtle forefoot driven cavovarus deformity have mild forefoot pronation with plantarflexion of the first ray (peroneal overdrive). This places increased pressure under the first metatarsal head and can result in irritation of the soft tissues surrounding the sesamoids. The forefoot pronation deformity can be subtle and should be looked for on physical examination. Subtle contractures of the Achilles tendon should also be ruled out, particularly of the gastrocnemius (Silfverskiöld test). Assessment for Vitamin D deficiency is essential if clinical suspicion warrants, particularly in the presence of multiple or repetitive stress fractures with no obvious etiology.

### Therapy (On the Field, Conservative, Surgical)

Treatment is dependent on the type of sesamoid pathology. Fractures, osteochondrosis and osteonecrosis can initially be treated with orthoses, activity modification, footwear modification or casting, with the goal to avoid dorsiflexion of the toe [6]. Shoe modification may include a removable walking boot, or a shoe modified with a full length rigid shank and forefoot rocker, and/or an orthotic with lateral hindfoot and forefoot posting associated with a recess beneath the first metatarsophalangeal head (sesamoid region) [6].

Surgical treatment for sesamoid pathology is reserved for those who have exhausted conservative management and continue to have intractable pain. In the presence of a subtle cavovarus deformity and/or Achilles contracture, the focus should be on deformity correction and release of soft tissue contractures. Resection of the tibial sesamoid is reserved for pathologies specific to the sesamoid (fracture, unstable bipartite, or osteochondrosis). Resection should be approached with caution, as potential complications include first toe weakness, residual pain, or neuritic pain (injury to the plantar hallucal nerves). Either partial or complete resection of one or both sesamoids may be considered. However, complete resection of both sesamoids is generally avoided, as it can result in a rigid cocked-up first toe deformity.

## Rehabilitation and Back-to-Sports

Careful and gradual return to sport is generally undertaken over several weeks for patients treated non-operatively. The primary goal is to offload the area; no specific exercises are recommended. If there is clear evidence that the Achilles gastrocnemius complex is involved, appropriate stretching exercises should be initiated. In operatively treated patients, return to sport may not occur until 6 months postoperative [4].

## Prevention

Identification of the etiology will assist the treating physician to prevent further or persistent injury to the sesamoid complex. If a subtle foot deformity or soft tissue contractures are identified, attention should be paid to both extremities, as the deformities are often bilateral. A rehabilitation program focusing on soft tissue stretching, strengthening the peroneus brevis, and orthotic and shoe management can help prevent persistence and recurrence of the injury.

The available evidence for sesamoid injury is Level III and Level IV.

---

## Bony Injuries: Accessory Navicular

### Epidemiology

Accessory ossicles are common in the foot and ankle, with the accessory navicular being the most common ossicle (12–15% incidence) [7]. While the majority of accessory ossicles are asymptomatic and simply a radiographic finding, activities such as running can cause a small proportion to become symptomatic [8].

### Etiology and Pathomechanism

Repetitive trauma, as experienced during running, coupled with a pes planus deformity, increases medial pressure on the midfoot and causes pain and discomfort. This repetitive overuse activity results in injury to the synchondrosis [7]. The clinician should carefully assess the patient for a gastrocnemius contracture (Silfverskiöld test) and evaluate the flexibility of the flat foot deformity (fixed forefoot supination).

### Therapy (On the Field, Conservative, Surgical)

Conservative therapy is aimed at eliminating the strenuous insult, relieving pressure on the medial midfoot, and decreasing inflammation [7]. Custom orthoses, anti-inflammatory medications and occasional casting to eliminate the pain resulting from the pull of the posterior tibial tendon may be required [7].

Corticosteroid injections may be used with caution if other non-operative methods fail. Surgical intervention is seldom required. If surgery is considered, important points to take into account include: (1) Removal of the accessory navicular requires detachment of the posterior tibial tendon. Consequently, recovery time is substantial, with 4–6 weeks of non-weight bearing and 6 months until return to sports. (2) Correction of the pes planus deformity should be considered if it is associated with a symptomatic accessory navicular. (3) Large fragments can be considered for fusion to the navicular in younger patients. (4) When the posterior tibial tendon is reattached, it should be advanced anteriorly to improve its inversion function [7].

## Rehabilitation and Back-to-Sports

Return to sports, when managed conservatively, can be anywhere from 3 to 6 weeks, depending on the amount of time required to eliminate the inflammatory condition. Physiotherapy should focus on strengthening of the accessory invertors and flexors of the foot, including the intrinsic. Stretching of the Achilles gastrocnemius complex is indicated if there is clinical evidence of its contraction. A total contact orthosis with medial hindfoot posting, arch support and metatarsal pad is recommended [8]. For surgical cases, athletics should be avoided for up to 6 months.

## Prevention

Patients with a significant pes planus deformity should be consulted on proper shoe wear and orthotic management. A physiotherapy program to strengthen the invertors and stretch the gastrocnemius may be indicated and attempted before surgical intervention.

The available evidence for accessory navicular is Level III and Level IV.

---

## Bony Injuries: Os Trigonum Syndrome

### Epidemiology

The prevalence of os trigonum syndrome is greater in ballet dancers, soccer players and runners (both downhill and uphill) [9]. Radiographic studies have identified a prevalence of 14–25% in normal feet, whereas in ballet dancers it can be as high as 30% [9].

### Etiology and Pathomechanism

An os trigonum is an accessory bone off the posterolateral talus that develops from either a stress fracture of Steida's pro-

cess, or failed fusion of a secondary ossification center of the lateral tubercle of the talus, creating a synchondrosis [9]. Os trigonum syndrome is an overuse injury caused by repetitive plantarflexion stress. Symptoms occur as the soft tissues at the back of the ankle impinge with the tibia with ankle plantarflexion [8]. Inflammation of the flexor hallucis longus and tendinitis are common and should be assessed with MRI. In cases where the os trigonum is large, associated degenerative changes of the subtalar joint posterior facet may be observed. If this is present, patients undergoing surgical excision of the os trigonum should be warned that symptoms may persist.

### Therapy (On the Field, Conservative, Surgical)

Initial non-operative therapy includes rest, ice, non-steroidal anti-inflammatory drugs, and avoidance of aggravating activities [9]. Ultrasound-guided steroid injections may be helpful in recalcitrant cases. Surgical management is reserved for cases that have failed non-operative management and may include arthroscopic or open excision of the os, debridement and/or decompression of the flexor hallucis longus and debridement of the posterior facet of the subtalar joint [9].

### Rehabilitation and Back-to-Sports

For non-operatively treated patients, gradual return to sports is attempted over a 1–4 month period, depending on the ability to reduce the inflammatory response and control pain. If there is acute inflammation, physiotherapy modalities to help mitigate the inflammation should be implemented. In operatively treated cases, patients will not return to pre-operative sporting levels until 2–3 months postoperative.

### Prevention

Patients are counseled to avoid positions that aggravate their symptoms. Use of a shoe and/or orthotic with a slight heel rise may reduce posterior ankle impingement.

The available evidence for os trigonum syndrome is Level III and Level IV.

---

## Soft Tissue Injury: Ankle Sprains

### Epidemiology

In the United States, over two million acute ankle sprains occur annually [10]. Lateral ankle sprains make up 85% of all ankle sprains [11]. The lateral ligaments include the anterior talofibular ligament (ATFL), calcaneofibular ligament

(CFL) and the posterior talofibular ligament (PTFL). Each ligament stabilizes the ankle at various positions.

### Etiology and Pathomechanism

The most common injury pattern is a midsubstance tear of the ATFL, followed by a combination ATFL and CFL tear. Rarer injuries include combination ATFL, CFL and PTFL tears or isolated CFL or PTFL tears. Ligament tears can cause both mechanical and functional instability. Functional instability includes complaints of ankle instability without any change in ankle structure, whereas mechanical instability is noted on physical examination with increased ankle range of motion beyond physiologic ranges. Ankle sprains are commonly classified according to the AMA Standard Nomenclature System into three grades, where Grade 1 is a stretched ligament, grade 2 is a partially torn ligament, and grade 3 is a completely torn ligament.

### Therapy (On the Field, Conservative, Surgical)

Conservative treatment is the mainstay for ankle sprains. Initial treatment includes rest, ice and elevation. Gradual weight bearing is commenced after a short period of immobilization in either a below knee cast or an aircast. A recent level one study has demonstrated that a short period of immobilization in a below-knee cast results in superior clinical benefits when compared to tubular compression bandaging [12]. Once the patient is comfortable and swelling has decreased, gentle range of motion exercises are commenced, followed by proprioceptive and balance training commencing 10–14 days after the injury [8]. The time frame for commencing physiotherapy varies, depending on the severity of the sprain; it is generally accepted that the clinical criterion for initiating physiotherapy is that the patient needs to be comfortable and in minimal pain. Conservative treatment is successful in up to 90% of patients; however, if mechanical instability persists, surgical intervention for ligament reconstruction may be considered [8].

### Rehabilitation and Back-to-Sports

There is little evidence to guide return to sport after conservative or operative treatment of ankle sprains. Some authors recommend wearing a high-top boot while bracing or taping the ankle for 6 months after an ankle sprain, to increase the proprioceptive awareness of the ankle and to resist inversion forces. Others use more objective measures to determine readiness to return to sport, such as ability to do multiple single leg calf raises.

## Prevention

Taping, bracing, muscle strengthening, Achilles stretching and proprioceptive training have all been advocated to prevent ankle injuries.

The evidence for ankle sprains ranges from Level I [12] to Level IV.

---

## Soft Tissue Injury: Achilles Tendinopathy

### Epidemiology

Achilles tendinopathy includes insertional and non-insertional subtypes. Non-insertional tendinopathy affects up to 9% of recreational runners and causes approximately 5% of professional athletes to end their careers [13]. Overall, mid-substance or non-insertional Achilles tendinopathy accounts for 60–65% of Achilles disorders, whereas insertional tendinopathy accounts for 20–25% of cases [14].

### Etiology and Pathomechanism

Non-insertional tendinopathy has been postulated to occur secondary to a region of relatively poor blood supply located approximately 2–6 cm proximal to the calcaneal insertion [15]. This may predispose the tendon to chronic inflammation and eventual rupture. Insertional tendinopathy has established risk factors, including increasing age, inflammatory arthropathies, steroid use, diabetes, hypertension, obesity, gout, lipidemias and quinolone antibiotics [16].

The exact pathomechanism is not completely understood, but mucoid or lipoid degeneration is commonly seen in the tendinopathic Achilles [17]. While no true inflammatory cells have been identified in the tendinopathic Achilles, it is postulated that there may be an inflammatory cellular reaction in the paratenon in the acute phase, likely causing adhesions, crepitus and chronic changes with resultant circulatory impairment [16].

### Therapy (On the Field, Conservative, Surgical)

Conservative treatment is the initial management of choice. Conservative treatment constitutes rest, ice, activity modification, analgesics, shoe modifications, eccentric Achilles physiotherapy, shockwave therapy and possibly corticosteroid injections [16]. Surgical intervention includes debridement of the diseased portion of tendon and re-approximation of healthy tendon, with or without additional tendon transfer, and is reserved for patients who have failed a prolonged (i.e., longer than 6 month) course of conservative management [18].

## Rehabilitation and Back-to-Sports

Patients with conservatively managed Achilles disorders can return to sport on a gradual basis over days to weeks, dependent on the relief of pain and swelling. For operatively managed tendinopathy, patients begin eccentric strengthening by 4 weeks postoperative, gentle running by 6 weeks postoperative, and hill workouts and/or interval training by 3 months postoperative [17].

The available evidence for Achilles tendinopathy is Level III and Level IV.

---

## Soft tissue Injury: Plantar Fasciitis

### Epidemiology

Plantar fasciitis affects roughly two million people annually in the United States and is the most common cause of heel pain in adults [19]. It affects both athletes and non athletes, commonly between the ages of 40 and 60 years [20].

### Etiology and Pathomechanism

The plantar fascia is a multilayered fibrous aponeurosis that originates from the medial calcaneal tuberosity and inserts distally through several slips into the plantar plates of the metatarsophalangeal joints, flexor tendon sheaths, and the bases of the proximal phalanges of the toes [21]. While not a true inflammatory condition, histologically plantar fasciitis has been defined as myxoid degeneration with disorganized collagen fibers, angiofibroblastic hyperplasia and calcification [20].

Plantar fasciitis results from the degeneration of the plantar fascia on the sole of the foot; however, no clear consensus on etiology exists. Reduced ankle dorsiflexion, obesity (BMI > 30) and work related weight bearing have been shown to be independent risk factors for the development of plantar fasciitis [22].

### Therapy (On the Field, Conservative, Surgical)

Initial management is generally non-operative. Rest, ice, analgesics, orthoses, shoe modifications and avoidance of activities that incite the fasciitis are recommended, in addition to specific plantar fascia stretching programs [23]. Shoe modifications, including rocker soles, have been shown to be successful in treating plantar fasciitis [24]. A short period of immobilization and corticosteroid injections may be alternatives if the above mentioned modalities fail to relieve pain and discomfort.

Surgical management is reserved for cases that have failed non-operative management and includes either endoscopic or open partial plantar fascia release, with or without Achilles lengthening or gastrocnemius recession.

## Rehabilitation and Back-to-Sports

For conservatively managed patients, return to sport is dictated by pain relief and ability to train without plantar medial foot pain. In operatively treated cases, patients gradually perform plantar fascial and Achilles stretching exercises starting at 4 weeks postoperative. A return to full activities is expected by 3 months postoperative.

## Prevention

Proper pre-sport stretching regimens and avoidance of activities that result in repeated running and jumping are the optimal ways to avoid plantar fasciitis [21].

The evidence for ankle sprains ranges from Level II to Level IV.

## Summary

1. Many running related injuries are caused by repetitive impact stress in individuals who are either unprepared for the physical load or have subtle to significant foot deformities. It is important for the clinician to carefully assess the foot and ankle for deformity and/or instability; identifying the etiology is essential for developing an appropriate treatment pathway.
2. Once a running injury has occurred, cessation of the activity for an appropriate period of time is essential for healing and to enable the individual to return to running.
3. Subtle foot deformities can often be managed by appropriate orthotic and shoe modifications.
4. At times, the only long-term solution for repetitive or persistent running injuries is to permanently modify the exercise routine and/or duration of activity.
5. Although running injuries are common, very few require surgical management.

## References

1. Coughlin MJ, Saltzman CL, Anderson RB. Chapter 30. Athletic injuries to the soft tissue of the foot and ankle. In: Mann's surgery of the foot and ankle. 9th ed. Mosby: Elsevier; 2014.
2. Korpelainen R, Orava S, Karpakka J, Siira P, Hulkko A. Risk factors for recurrent stress fractures in athletes. *Am J Sports Med.* 2001;29(3):304–10. Epub 2001/06/08.
3. Hunt KJ, McCormick JJ, Anderson RB. Management of fore-foot injuries in the athlete. *Oper Tech Sports Med.* 2010;18:34–45.
4. Kindred J, Trubey C, Simons SM. Foot injuries in runners. *Curr Sports Med Rep.* 2011;10(5):249–54. Epub 2011/09/01.
5. Richardson EG. Hallucal sesamoid pain: causes and surgical treatment. *J Am Acad Orthop Surg.* 1999;7(4):270–8. Epub 1999/08/06.
6. Nihal A, Trepman E, Nag D. First ray disorders in athletes. *Sports Med Arthrosc.* 2009;17(3):160–6. Epub 2009/08/15.
7. Leonard ZC, Fortin PT. Adolescent accessory navicular. *Foot Ankle Clin.* 2010;15(2):337–47. Epub 2010/06/11.
8. Kennedy JG, Knowles B, Dolan M, Bohne W. Foot and ankle injuries in the adolescent runner. *Curr Opin Pediatr.* 2005;17(1):34–42. Epub 2005/01/22.
9. Nault ML, Kocher MS, Micheli LJ. Os trigonum syndrome. *J Am Acad Orthop Surg.* 2014;22(9):545–53. Epub 2014/08/27.
10. Soboroff SH, Pappius EM, Komaroff AL. Benefits, risks, and costs of alternative approaches to the evaluation and treatment of severe ankle sprain. *Clin Orthop Relat Res.* 1984;183:160–8. Epub 1984/03/01.
11. Maffulli N, Ferran NA. Management of acute and chronic ankle instability. *J Am Acad Orthop Surg.* 2008;16(10):608–15. Epub 2008/10/04.
12. Lamb SE, Marsh JL, Hutton JL, Nakash R, Cooke MW. Mechanical supports for acute, severe ankle sprain: a pragmatic, multicentre, randomised controlled trial. *Lancet.* 2009;373(9663):575–81. Epub 2009/02/17.
13. Lysholm J, Wiklander J. Injuries in runners. *Am J Sports Med.* 1987;15(2):168–71. Epub 1987/03/01.
14. Kvist M. Achilles tendon injuries in athletes. *Ann Chir Gynaecol.* 1991;80(2):188–201. Epub 1991/01/01.
15. Chen TM, Rozen WM, Pan WR, Ashton MW, Richardson MD, Taylor GI. The arterial anatomy of the Achilles tendon: anatomical study and clinical implications. *Clin Anat.* 2009;22(3):377–85. Epub 2009/01/29.
16. Roche AJ, Calder JD. Achilles tendinopathy: a review of the current concepts of treatment. *Bone Joint J.* 2013;95-B(10):1299–307. Epub 2013/10/01.
17. Zafar MS, Mahmood A, Maffulli N. Basic science and clinical aspects of achilles tendinopathy. *Sports Med Arthrosc.* 2009;17(3):190–7. Epub 2009/08/15.
18. Asplund CA, Best TM. Achilles tendon disorders. *BMJ.* 2013;346:f1262. Epub 2013/03/14.
19. Riddle DL, Schappert SM. Volume of ambulatory care visits and patterns of care for patients diagnosed with plantar fasciitis: a national study of medical doctors. *Foot Ankle Int.* 2004;25(5):303–10. Epub 2004/05/12.
20. Lareau CR, Sawyer GA, Wang JH, DiGiovanni CW. Plantar and medial heel pain: diagnosis and management. *J Am Acad Orthop Surg.* 2014;22(6):372–80. Epub 2014/05/27.
21. Coughlin MJ, Saltzman CL, Anderson RB. Chapter 13. Plantar heel pain. In: Mann's surgery of the foot and ankle. 9th ed. Mosby: Elsevier; 2014.
22. Riddle DL, Pulisic M, Pidcoe P, Johnson RE. Risk factors for Plantar fasciitis: a matched case-control study. *J Bone Joint Surg Am.* 2003;85-A(5):872–7. Epub 2003/05/03.
23. Rompe JD, Cacchio A, Weil Jr L, Furia JP, Haist J, Reiners V, et al. Plantar fascia-specific stretching versus radial shock-wave therapy as initial treatment of plantar fasciopathy. *J Bone Joint Surg Am.* 2010;92(15):2514–22. Epub 2010/11/05.
24. Lin SC, Chen CP, Tang SF, Wong AM, Hsieh JH, Chen WP. Changes in windlass effect in response to different shoe and insole designs during walking. *Gait Posture.* 2013;37(2):235–41. Epub 2012/08/14.

Jannis Sailer, Martin Majewski, Matthias Gilgien,  
and Victor Valderrabano

#### Abstract

Wintersports are popular activities and became in the past 20 years even more popular. The improvement of the equipment makes especially downhill skiing and snowboarding in general safer than in the past century. But due to higher speed and a trend to ride off the ski run courses, injuries itself get more severe. This can result in complex trauma and requires well trained surgeons to plan and perform therapy. The following should give an overview on the prevalence of Trauma in alpine sports and focus on basic injuries of the foot and ankle.

#### Keywords

Skiing • Wintersports • Alpine • Snowboarding • Pilon fracture • Haglund Exostosis • Weber Classification • Cross country skiing • Sports trauma

## Alpine Skiing

### Etiology and Pathomechanism

The most frequent injury types in competitive alpine skiing are joint and ligament injuries (44.0%), followed by fractures and bone stress (18.8%), contusions (12.0%) and muscle tendon injuries (10.5%) [1, 2].

J. Sailer, MD (✉)  
Orthopedic Department, University Hospital Basel,  
Basel, Switzerland

Department of Traumatology and Orthopedic Surgery,  
University Hospital Basel, Spitalstrasse 21,  
Basel, Baselstadt 4056, Switzerland  
e-mail: [jannis.sailer@usb.ch](mailto:jannis.sailer@usb.ch)

M. Majewski  
Orthopedic Department, University Hospital Basel,  
Basel, Switzerland  
e-mail: [martin.majewski@usb.ch](mailto:martin.majewski@usb.ch)

M. Gilgien  
Center for Alpine Sports Biomechanics, University Basel,  
Basel, Switzerland

V. Valderrabano, MD, PhD  
Orthopaedic Department, SWISS ORTHO CENTER,  
Schmerzklinik Basel, Swiss Medical Network, Basel, Switzerland  
e-mail: [vvalderrabano@gsmn.ch](mailto:vvalderrabano@gsmn.ch)

## Injuries

### Head and Spine

Incidence of craniocerebral injuries (CCI) is equal in recreational and professional skiing and represents 9% of all injuries [1, 3]. In most cases head traumata are CCI of variable severity, within that severe CCI define the mortality rate in skiing accidents. Generally the rate is of CCI declining over the past years, which is a result of increasing usage of helmets [1].

Trauma of the spine is rare and seems to have a correlation towards male gender [1] and high speed. Spine injuries combined with neurologic deficit occur each to one third in the cervical, thoracic and lumbar spine [4].

### Upper Extremity

In the beginning of the twenty-first century almost 1/3 of all injuries were located at the upper extremity [5]. The increase is connected to the popularity of carving skis and is associated with different curve radius, higher risk of falls and especially more forward and backward falls [3]. Professional athletes suffer more often from injuries of the thumb and hand. In contrast incidence of shoulder trauma is higher in recreational skiers compared to professionals. Reasons are higher speed and frequent snow contact [2, 3].



## Shoulder

Common injuries are lesions of the rotator cuff (24%), anterior glenohumeral instability (22%), acromioclavicular instability (20%), fractures of clavícula (11%), proximal humerus and tuberculum majus (10%) [6]. 1/3 of all shoulder dislocations showed also intraarticular fractures [7].

## Elbow, Wrist and Hand

More distal, fractures of distal radius, shaft and metacarpalia are common. Typical injury is the skier's thumb, which is a rupture of the ulnar collateral ligament or osseous rupture of the metacarpophalangeal (MCP) joint of the thumb and is the most common injury of the hand in skiing (62%) [8]. Injury mechanism is an atypical abduction in the MCP joint while holding a ski pole in case of fall. In severe cases the result is a Stener lesion with a dislocation of the distal ulnar collateral complex and interposition of the adductoraponeurosis [9].

## Lower Extremity

Due to the switch to buckled skiing boots in the 70s, location of injuries shifted from the ankle and distal leg towards the knee. Since then incidence of knee trauma is stable. In most of the cases capsula, mensicus and ligament lesions, especially ACL ruptures and medial collateral ligament lesions, occur. [10, 11].

## Injuries of the knee

Traumata of the knee are the most common in alpine sports. There is a gender difference where women suffer 3.3 times more likely from a ACL rupture than men [12, 13]. Reasons are anatomical differences (e.g. valgus alignment, smaller notch wideness), neuromuscular (insufficient quadriceps-hamstring coactivation) and gender specific joint laxity [14].

In recreational skiing ACL lesions happen because of a forward fall with a sudden "catch", while professional skiers incur a "slip-catch-mechanism" [15]. Additionally, concerning recreational athletes, in 96% of the patients with an ACL rupture ski binding was not released correctly [16]. Beside isolated ligament trauma, combined injuries occur. Multiligament- and also meniscal or even osteochondral lesions appear. The unhappy triad is quite rare [17].

## Lower Leg and Ankle

Beside soft tissue injuries, fractures of the tibia and the lower leg are typical in recreational and professional skiing. 62% of the fractures in recreational Skiing happen through rotational trauma while 59% of the cases are caused by a malfunction of the release mechanism of the binding [18]. The ski then acts as a lever arm. Professional skiers suffer more frequent of transvers and oblique fractures of the lower leg, especially at the edge of the skiing boot (boot-top-fractures) [19].

## Small Breaches

Blisters occur frequently, especially in active populations. It is a result from frictional forces that mechanically separate

epidermal cells at level of the stratum spinosum. Clinical experience suggests draining intact blisters and maintaining the blister roof. This results in the least discomfort to the patient and may reduce the possibility of secondary infection. Treating derroofed blisters with hydrocolloid dressings provides pain relief and may allow patients to continue physical activity if necessary. There is no evidence that antibiotics influence blister healing [20]. The best treatment is to prevent blisters. Blisters can be prevented by wearing properly sized boots, conditioning feet, wearing socks that reduce friction and moisture [21].

## Heel Injuries

In essence there are three afflictions about the ankle that result from skiing: rupture of the Achilles tendon, Achilles tendinitis, and dislocation of the peroneal tendon. The cause of Achilles tendinitis and Achilles tendon rupture is, in fact, pressure within the fascial compartment of the Achilles tendon, which is caused by a swollen distal soleus muscle, occluding the circulation and thereby producing avascular necrosis with subsequent tendinitis yielding to rupture [22]. Treatment of tendinitis should be conservatively. In case of Achilles Tendon rupture operative therapy should be preferred to gain optimal stability and lower rerupture risk.

Posterior heel pain can also be attributed to a Haglund deformity, a prominence of the calcaneus that may cause bursa inflammation between the calcaneus and Achilles tendon [23]. Extracorporeal shockwave therapy seems effective in patients with non-calcified insertional achilles tendinopathy. Although eccentric exercises resulted in a decrease in VAS score, full range of motion eccentric exercises shows a low patient satisfaction compared to floor level exercises and other conservative treatment modalities [24].

## Distal Fibula Fractures

Fibula fractures should be diagnosed quickly and depending on the findings, treatment needs to be determined whether nonoperative or operative therapy is required [25]. To classify these fractures the Weber or AO classification is used. In snowboarding nearly two-thirds of fractures are to the left limb whereas in skiers laterality is not typical. Most fractures in skiers are the result of falls while a greater proportion of snowboarders have jump-related injuries [26].

## Tibial Pilon Fractures

Complicated injuries are tibial pilon fractures. Typical is a rotational trauma leading to spiroid fractures (AO 43-C) associated with joint separation and modest articular and soft tissue damage. Open fractures from inside-out can occur. The elementary separations include the anterolateral pedicle bone on the anterior tibiofibular ligament (Chaput fracture), posterior the Volkmann fragment and medial. In a first step these injuries need to be stabilized, most often using an external fixation to restore the length and prepare for definitive fixation

by plating. Goal of the definitive treatment should be restoring the articular surface to prevent secondary joint degeneration best possible and avoid malpositioning of the foot. [27].

### Others

In contrast to lower leg fractures, femur or hip lesions are rare and a consequence of high-speed trauma or collisions [28]. Similar accidents can also cause blunt abdominal-, torso- and thoracic-trauma without involvement of the spine and occur isolated or combined with polytrauma with high “injury severity score” (ISS) [29].

## Epidemiology

### Competitive Alpine Skiing

Competitive Alpine Skiing is considered a sport with high injury rates. Every third athlete is injured each season and every sixth athlete suffers from an injury that leads to an absence from training and competition for more than 28 days [2]. In contrast to most other sports alpine skiing has a high proportion of time-loss and severe injuries [1, 2]. Males have a 1.24–1.42 higher relative injury risk than females. Ankle, foot and lower leg injury rate was also lower for females compared to males [30].

In competitive (World Cup) alpine skiing the most frequently injured body part is the knee (35.6–36.0 % of all injuries), followed by the lower leg and achilles tendon (11.1–11.5 %), the lower back region (11.1–11.5 %), hand (8.9–9.0 %), head and face (8.5–8.6 %) and shoulder (6.8–6.9 %). The ankle follows next with 5.2–5.3 %. Lower leg, foot, heel and toe injuries account for 1.6 % of all injuries [1, 2].

Injury types to the lower leg are fractures and bone stress (36.4 %), joint and ligament injuries (9.1 %), muscle and tendon injuries (9.1 %) and contusions (36.4 %). Injury types to the ankle are fractures or bone stress (30.0 %) joint and ligament injuries (70.0 %) [2].

Generally it has been observed that high-speed disciplines have a 3 times higher risk of injury compared to e.g. slalom [1].

### Recreational Alpine Skiing

Similar to competitive alpine skiing also recreational alpine skiing suffers from high injury risk compared to other sports [31]. Generally a reduction of injuries occurred in recreational alpine skiing in the past years compared to the reference season 1978/1980 [32]. This is due to more use of protection equipment, popularity of Carving skis and advanced slope preparation. 0.84/1000 alpine athletes, is the lowest incidence of inpatient treatments ever recorded in Germany. Reasons are a trend towards ambulant treatment, but can be interpreted as a lower severity of injuries [3].

In detail injuries of the knee joint are by far the most frequently with 37.2 % followed by shoulder injuries 20.1 %.

Head trauma occurs by 7.2 %. Findings of the past years showed a decrease of knee trauma and other body parts in contrast to upper extremity and shoulder [3]. Particular importance is the drop of craniocerebral injury as a result of acceptance of helmets.

Professional athletes have a risk of injury 4.1 per 1000 ski days in contrast to 1.1–3.2/1000 ski days in recreational riders, which is a two to three times higher risk [1].

## Therapy (On the Field, Conservative, Surgical)

Alpine skiing is a high-risk sport. Therefore a regional adapted rescue concept is required. If severity of injuries determines to continue the ride, treatment starts direct on the slope by specially trained paramedics. In cases of a life-threatening trauma or impossible to transport the patient on a sledge rescue is usually done by snow groomer or helicopter. In larger skiing areas hospitals for primary health care with radiologic facilities and trained specialists are able to diagnose and precise treatment. Therapy is depending on the injury. Smaller trauma can be treated ambulant while more severe cases need inpatient care. Fractures are handled conservative or if diagnosis requires an operation a definitive osteosynthesis or installation of a fixateur externe is performed. Rehabilitation and aftercare is done by the primary hospital and regional rehab centers itself or in case of tourists in their home countries. Due to limited availability of medical specialists in remote areas some patient need to be transferred to bigger hospitals to get the optimal treatment (e.g. intracerebral bleeding, intensive care monitoring, spine injuries with neurologic symptoms).

## Rehabilitation and Back-to-Sports

In general rehabilitation is based on the injury and its required treatment. Smaller trauma normally needs no specific aftercare beside enough rest to overcome the symptoms. More severe trauma needs longer after treatment. In situation with complex fractures or ligament lesions sometimes even stationary rehabilitation including daily physiotherapy is necessary. Regular clinical checkups should be performed by either a GP or a specialist to decide when the patient is ready to be back to sports. In professional sports the priority goal is to be back in training and/or competition. Therefore intensive physiotherapy, adaption of the training and competition schedule is mandatory. The moment of recovery is normally determined by a team doctor in consultation with the coaches, physiotherapists and of course the athlete. Important is to prevent a too early intensive load to avoid relapse or longer drop out especially during a season.

## Prevention

Due to the high injury risk in competitive alpine skiing, the International Ski Federation (FIS) has installed a permanent injury surveillance system [1, 2, 30, 33]. Accidents happen as a result of external (weather, snow condition, slope preparation) and internal factors (fitness level, skills) [34]. Specific training especially force in extensions and flexion of the lower extremity, core strength and technique to avoid lay-back position reduce the risk of injury. Another important role is the right choice for a suitable boot and binding combination [35].

In the past years more and more protective equipment found its way to the market and on the slopes. Their mechanism is based on passive stabilization and mechanical protections [32]. There is a variety of braces, vests and suits for ankle, knee and spine. Helmets gained acceptance throughout alpine sports. In professional disciplines is the use obliged [36] while in recreational skiing over 60% of the athletes protect themselves [37]. Helmets prevent or moderate head trauma between 22 and 60% [37–39].

The above section includes evidence level II.

## Summary

Alpine sports have a high risk to suffer injuries. The knee (fractures-, ligament injuries) is the most frequent affected body part. In the past years incidence of trauma that required medical treatment was reduced. Increasing numbers of athletes use protective helmets. Therapy is due to the injury and should be performed as soon as possible and with the goal of an early functional treatment.

---

## Snowboarding

### Etiology, Pathomechanism and Therapy

Causes of foot and ankle injuries are most likely falls or a result from jumps. There is no significant correlation between boot type (soft, hybrid, or hard) and overall foot or ankle injury rate. But there are significantly fewer ankle sprains in patients wearing hybrid boots and fewer fractures of the lateral process of the talus in patients wearing soft boots [40]. In total snowboarding has a higher incidence of injuries than downhill skiing [41].

### Fracture of the Processus Lateralis Tali

Many of these fractures are not visible on plain radiographs and require computed tomography (CT) imaging to be diagnosed. The physician should be very suspicious of anterolateral ankle pain in the snowboarder, where subtle fractures that

may require surgical intervention can be confused with anterior talofibular ligament sprains [40]. As the axial-loaded dorsiflexed foot becomes externally rotated and/or everted, fracture of the lateral process of the talus occurs. Primary surgical treatment may improve the outcome of this injury, reducing the risk of secondary subtalar joint osteoarthritis. In type II fractures, primary surgical treatment has led to achieving better outcomes, reducing sequelae, and allowing patients to regain the same sports activity level as before injury [42].

### Peroneal Tendon Injuries

Acute tears of the peroneus brevis, and less commonly the peroneus longus are often coexistent with peroneal instability. Subluxation typically occurs when the foot is in a dorsiflexed position and the peroneal muscles strongly contract, causing an eversion force simultaneously [43]. Most acute peroneus brevis tears are longitudinal, occur adjacent to the tip of the fibula and require surgical treatment. Acute peroneus longus tears more commonly occur at the level of the cuboid tunnel and may initially be managed nonoperatively. However, if associated with stenosing tendonitis, debridement and tenodesis may be required. Rarely, complete ruptures of both peronei occur and, if there is a significant defect, reconstructive procedures are required [44]. Techniques involves suturing using anchors, hamstring allograft reconstruction, the silicone rod technique, flexor digitorum longus transfer to the peroneus brevis, and treatment of associated pathology [43].

### Fractures of the Foot

Fractures of the foot are rare in alpine sports. Depending on the radiologic diagnosis treatment needs to be determined. Often CT scans are required to identify a fracture. Operative treatment should be performed in consideration of a wise soft tissue management.

### Epidemiology

A study from 12 Colorado ski resorts showed in total 3213 snowboarding injuries, 491 (15.3%) were ankle injuries and 58 (1.8%) were foot injuries. Ankle injuries included 216 (44%) fractures and 255 (52%) sprains. Thirty-three (57%) of the foot injuries were fractures and 16 (28%) were sprains. The remaining injuries were soft tissue injuries, contusions, or abrasions. An unexpectedly high number of fractures of the lateral process of the talus were noted. These fractures represented 2.3% of all snowboarding injuries, 15% of all ankle injuries, and 34% of the ankle fractures [40].

Similar to alpine downhill skiing injury patterns are different in competitive and recreational snowboarding. Elite-level snowboarders are often injured when performing difficult manoeuvres at high velocities and with amplified levels of

force to the lower limbs. Consequently, elite-level snowboarders suffer from injuries that are of higher severity and have decidedly greater lower extremity injury rates [45]. The risk of injuries is higher in snowboard cross than in halfpipe, big air and parallel slalom [46]. Compared to skiing foot or ankle injuries occur more likely with snowboarding [47].

## Therapy and Rehabilitation

Please refer section Rehabilitation and Back-to-Sports in the “Alpine Skiing” chapter.

## Cross-Country Skiing

### Injuries

The majority of injuries in cross-country skiing are overuse injuries compared to traumatology injuries [48, 49]. The types of injuries in competitive cross country skiing consist of muscle and tendon (37.5%), joint and ligament injuries (31.3%), contusions (14.6%), injuries of the nervous system (6.3), skin injuries (2.1%) and others [1]. The incidence is 0.2 injuries per 1000 km skied. All in all cross-country skiing has a low injury risk, especially when compared to alpine slalom, representing the alpine discipline with the lowest risk [50].

### Etiology and Pathomechanism

Most of the injuries in professional cross-country skiing are overuse ailment while 25% are caused by trauma. Most common is the medial tibial stress syndrome, achilles tendon problems and lower back pain especially in the age group between 16 and 20 years. Among traumatic injuries most frequently are ankle lesions (ligament and fractures), muscle ruptures and knee ligament sprains.

Shoulder dislocation, acromioclavicular separation, and rotator cuff tears are not infrequent. Lesions of the ulnar collateral ligament at the MCP (Stener’s lesion) is the most common injury of the upper extremity.

Back pain may result from repetitive hyperextension during the kick phase and recurring spinal flexion and extension during double poling phase.

Microtrauma in the musculotendinous units of the groin can be caused by repeated slipping on hard icy tracks. Tears of the medial collateral ligament and the anterior cruciate ligament (ACL) are typical injuries in falls and usually need surgical treatment and long rehabilitation. A twisting mechanism can conduct to a lateral patella dislocation, along with patella fractures and prepatellar bursitis through direct

trauma. Patellofemoral pain is a frequent problem especially on tracks with downhills because increased knee flexion angle requires greater force generated by the quadriceps muscles, which turns into greater stress between the patella and the femur.

Medial-tibial stress syndrome frequently occurs along the medial border of the tibia of professional skiers. Also an anterior compartment syndrome is observed especially in prolonged episodes of the skating technique.

Cross-country skiers also suffer frequently from achilles tendon problems and stress fractures in the foot [48].

## Epidemiology

Compared to competitive alpine skiing, cross country skiing has a much lower injury risk. For competitive cross country skiing 11.4 athletes per 100 athletes suffer an injury each season compared to 36.7 in competitive alpine skiing. The number of injuries which lead to absence from training and competition for more than 1 day is 6.3 compared to 29.8 in alpine skiing. 0.7 athletes in cross country skiing suffer a severe injury per season compared to 11.3 in alpine skiing [1]. Expressed in injuries per skiing day cross country skiers suffer an injury at 0.2 to 0.5 to 0.73 injuries per 1000 skiing days [48, 51].

The most frequently injured body part in competitive cross country skiing is the lower back, pelvis and sacrum (26.0% of all injuries), followed by the shoulder and clavicle (14.6%), lower leg and achilles tendon (10.4%), knee (8.3%) and elbow (8.3%), hip (6.3%) and ankle (6.3%). Foot, heel and toe injuries account for 4.2% of all injuries [1]. Beginners seem to be prone for upper body injuries [52]. Cross country skiing athletes suffer a large part of the acute injuries while executing sports other than cross country skiing [51].

### Focus: Lower Leg and Ankle

The most common overuse injuries include medial-tibial stress syndrome, arthritic changes in the great toe [53] and achilles tendon problems. Most common among traumatic injuries are ankle ligament sprains, fractures and muscle ruptures [48].

The early functional treatment of acute ankle sprains will return many skiers to activity quickly [54]. Dysfunction of the posterior tibial tendon or rupture of the peroneal tendons is rare, and dislocation cannot be diagnosed with certainty unless it is seen promptly or can be reproduced on examination. Most injuries in cross country skiing can be treated conservatively, and some can be avoided with adequate preparation and training [53]. Fractures, complex ligament ruptures and instability disorders usually need operative treatment.

A severe and often misdiagnosed injury is a lesion of the syndesmosis. External rotation and excessive dorsiflexion of the foot on the leg have been reported as the most common mechanisms. Early rigid immobilization and pain relief strategies, followed by strengthening and balance training are recommended. Heel lift and posterior splint intervention can be used to avoid separation of the distal syndesmosis induced by excessive dorsiflexion of the ankle joint. Surgical intervention is an option when a complete tear of the syndesmotic ligaments is present or when fractures are observed. Indication for a surgical intervention is also given by a rupture of the deltoid ligament, being an important part of maintaining medial stability to the ankle joint [55].

## Therapy

On the field medical services should be carried out in the manner of paramedics and in adaption to the situation. In professional sport involve the team doctor or physiotherapist. Sometimes competitions or training take place in remote areas, organize transportation and advanced medical help as soon as possible. Depending on the diagnosis a conservative or invasive treatment should be initiated. Surgical treatment includes osteosynthesis in alignment to the AO criteria and latest operational techniques e.g. in ACL reconstruction.

## Rehabilitation and Back-to-Sports

See section rehabilitation and back to sport in the alpine skiing chapter.

## Prevention

In recreational sports each athlete should be aware of their own skills and physical shape and adapt the sport load. Also equipment should be checked regularly and be prepared for outside conditions. In professional cross-country skiing taping and other symptoms preventions method are a mandatory part of the training and pre competition preparation. Also testing of the right equipment and sometime even custom made solutions are necessary.

Highest level of evidence in the above section is 3.

## Summary

Cross-country skiing is in recreational as well as in professional matter a low risk sport. Most problems occur in terms of an overuse (lower back pain, ventral knee pain, ankle sprains). Traumatic injuries a mostly caused by falls and can

lead into fractures and severe ligament injuries. Prevention involves adequate equipment and sport load in consideration of the individual physical performance.

## References

1. Flørenes TW, Nordsletten L, Heir S, Bahr R. Injuries among World Cup ski and snowboard athletes. *Scand J Med Sci Sports*. 2012;22(1):58–66.
2. Flørenes TW, Bere T, Nordsletten L, Heir S, Bahr R. Injuries among male and female World Cup alpine skiers. *Br J Sports Med*. 2009;43(13):973–8.
3. Schulz D. Unfälle und Verletzungen im alpinen Skisport. *Zahlen und Trend 2012/2013*. 2013.
4. Levy AS, Smith RH. Neurologic injuries in skiers and snowboarders. *Semin Neurol*. 2000;20(2):233–45.
5. Koehle MS, Lloyd-Smith R, Taunton JE. Alpine ski injuries and their prevention. *Sports Med*. 2002;32(12):785–93.
6. Kocher MS, Feagin JA. Shoulder injuries during alpine skiing. *Am J Sports Med*. 1996;24(5):665–9.
7. Ogawa H, Sumi H, Sumi Y, Shimizu K. Glenohumeral dislocations in snowboarding and skiing. *Injury*. 2011;42(11):1241–7.
8. Pechlaner S, Suckert K, Sailer R. Hand injuries in Alpine skiing. *Sportverletz Sportschaden*. 1987;1(4):171–6.
9. Adler T, Eisenbarth I, Hirschmann MT, Müller-Gerbl M, Fricker R. Can clinical examination cause a Stener lesion in patients with skier's thumb?: a cadaveric study. *Clin Anat*. 2012;25(6):762–6.
10. Pressman A, Johnson DH. A review of ski injuries resulting in combined injury to the anterior cruciate ligament and medial collateral ligaments. *Arthroscopy*. 2003;19(2):194–202.
11. Warme WJ, Feagin JA, King P, Lambert KL, Cunningham RR. Ski injury statistics, 1982 to 1993, Jackson Hole Ski Resort. *Am J Sports Med*. 1995;23(5):597–600.
12. Beynon B, Ettliger C, Johnson R. Understanding and preventing noncontact ACL injuries – American Orthopaedic Society for Sports Medicine. In: Griffin L, Hewett TE, Shultz SJ, editors. *Understanding and preventing noncontact ACL injuries*. Champaign: Human Kinematic. 2007. p. 183–8.
13. Johnson RJ, Ettliger CF, Shealy JF, Shealy. "Injury trends, risk factors involving ACL injuries in alpine skiing." Keynote Lecture during the 16th International Symposium on Ski Trauma and Skiing Safety, Vol. 17. 2005.
14. Hewett TE, Myer GD, Ford KR. Anterior cruciate ligament injuries in female athletes: part 1, mechanisms and risk factors. *Am J Sports Med*. 2006;34(2):299–311.
15. Ruedl G, Linortner I, Schranz A, Fink C, Schindelwig K, Nachbauer W, Burtscher M. Distribution of injury mechanisms and related factors in ACL-injured female carving skiers. *Knee Surg Sports Traumatol Arthrosc*. 2009;17(11):1393–8.
16. Urabe Y, Ochi M, Onari K, Ikuta Y. Anterior cruciate ligament injury in recreational alpine skiers: analysis of mechanisms and strategy for prevention. *J Orthop Sci*. 2002;7(1):1–5.
17. Barber FA. Snow skiing combined anterior cruciate ligament/medial collateral ligament disruptions. *Arthroscopy*. 1994;10(1):85–9.
18. A. Bürkner, H. Simmen. *Unterschenkelfrakturen beim alpinen Skisport—Einfluss von Skischuhen und Unfallmechanismus*. 2008.
19. Mückley T, Kruis C. *Die Unterschenkelfraktur im professionellen Skisport*. *Sportverletz Sportschaden*. 2004;18(1):22–7.
20. Knapik JJ, Reynolds KL, Duplantis KL, Jones BH. Friction blisters. *Sport Med*. 1995;20(3):136–47.
21. Knapik JJ. Prevention of foot blisters. *J Spec Oper Med*. 2014;14(2):95–7.

22. Oden RR. Tendon injuries about the ankle resulting from skiing. *Clin Orthop Relat Res.* 1987;216:63–9.
23. Priscilla T, Bytowski JR. Diagnosis of heel pain – American Family Physician. *Am Fam Physician.* 2011;84(8):909–16.
24. Wiegand JI, Kerkhoffs GM, van Sterkenburg MN, Siersevelt IN, van Dijk CN. Treatment for insertional Achilles tendinopathy: a systematic review. *Knee Surg Sports Traumatol Arthrosc.* 2013;21(6):1345–55.
25. Leach RE, Lower G. Ankle injuries in skiing. *Clin Orthop Relat Res.* 1985;198:127–33.
26. Patton A, Bourne J, Theis J-C. Patterns of lower limb fractures sustained during snowsports in Otago, New Zealand. *N Z Med J.* 2010;123(1316):20–5.
27. Dujardin F, Abdulmutalib H, Tobenas AC. Total fractures of the tibial pilon. *Orthop Traumatol Surg Res.* 2014;100(1 Suppl):S65–74.
28. Sterett WI, Krissoff WB. Femur fractures in Alpine skiing: classification and mechanisms of injury in 85 cases. *J Orthop Trauma.* 1994;8(4):310–4.
29. McBeth PB, Ball CG, Mulloy RH, Kirkpatrick AW. Alpine ski and snowboarding traumatic injuries: incidence, injury patterns, and risk factors for 10 years. *Am J Surg.* 2009;197(5):560–3.
30. Bere T, Flørenes TW, Nordsletten L, Bahr R. Sex differences in the risk of injury in World Cup alpine skiers: a 6-year cohort study. *Br J Sports Med.* 2014;48(1):36–40.
31. Pečina M. Injuries in downhill (alpine) skiing. *Croat Med J.* 2002;43(3):257–60.
32. Brucker PU, Katzmaier P, Olvermann M, Huber A, Waibel K, Imhoff AB, Spitzenpfel P. Recreational and competitive alpine skiing. Typical injury patterns and possibilities for prevention. *Unfallchirurg.* 2014;117(1):24–32.
33. Kröll J, Spörri J, Gilgien M, Chardonens J, Müller E. Verletzungsprävention innerhalb eines internationalen Sportverbandes: Eine Prozessbeschreibung am Beispiel des alpinen Skirennsports. *Schweiz Z Med Traumatol.* 2013;29:288–96.
34. van Mechelen W, Hlobil H, Kemper HC. Incidence, severity, aetiology and prevention of sports injuries. A review of concepts. *Sports Med.* 1992;14(2):82–99.
35. Hébert-Losier K, Holmberg H-C. What are the exercise-based injury prevention recommendations for recreational alpine skiing and snowboarding? A systematic review. *Sports Med.* 2013;43(5):355–66.
36. Fédération Internationale de Ski. Spezifikationen der Wettkampfausrüstung und kommerzielle Markenzeichen. Oberhofen Schweiz. 2013.
37. Ruedl G, Brunner F, Kopp M, Burtscher M. Impact of a ski helmet mandatory on helmet use on Austrian ski slopes. *J Trauma.* 2011;71(4):1085–7.
38. Ackery A, Hagel BE, Provvidenza C, Tator CH. An international review of head and spinal cord injuries in alpine skiing and snowboarding. *Inj Prev.* 2007;13(6):368–75.
39. Haider AH, Saleem T, Bilaniuk JW, Barraco RD. An evidence-based review: efficacy of safety helmets in the reduction of head injuries in recreational skiers and snowboarders. *J Trauma Acute Care Surg.* 2012;73(5):1340–7.
40. Kirkpatrick D, Hunter R. The snowboarder's foot and ankle. *Am J Sports Med.* 1998;26(2):271–7.
41. Kim S, Endres N. Snowboarding injuries trends over time and comparisons with alpine skiing injuries. *Am J Sports Med.* 2012;40(4):770–6.
42. Valderrabano V, Perren T. Snowboarder's talus fracture treatment outcome of 20 cases after 3.5 years. *Am J Sports Med.* 2005;33(6):871–80.
43. Cerrato R, Myerson M. Peroneal tendon tears, surgical management and its complications. *Foot Ankle Clin.* 2009;14.
44. Slater HK. Acute peroneal tendon tears. *Foot Ankle Clin.* 2007;12(4):659–74.
45. Wijdicks C, Rosenbach B. Injuries in elite and recreational snowboarders. *Br J Sports Med.* 2014;48(1):11–7.
46. Major D, Steenstrup S, Bere T. Injury rate and injury pattern among elite World Cup snowboarders: a 6-year cohort study. *Br J Sport Med.* 2014;48(1):18–22.
47. Abu-Laban R. Snowboarding injuries: an analysis and comparison with alpine skiing injuries. *C Can Med Assoc J.* 1991;145(9):1097–103.
48. Renstrom P, Johnson RJ. Cross-country skiing injuries and biomechanics. *Sport Med.* 1989;8(6):346–70.
49. Hemmingsson P, Ohlsen P. Injuries and diseases in elite cross country skiers. *J Swed Soc Sports Med.* 1987;2:14–5.
50. Flørenes T, Nordsletten L. Injuries to world cup nordic skiers and telemarkers—data from two seasons. *Br J Sports Med.* 2011;45:310.
51. Ristolainen L, Heinonen A. Type of sport is related to injury profile: a study on cross country skiers, swimmers, long-distance runners and soccer players. A retrospective 12-month study. *J Med.* 2010;20(3):384–93.
52. Howes J, Droog SJ, Evans J, Wood IM, Wood AM. The epidemiology of cross country skiing injuries. *Br J Sports Med.* 2011;45:A20.
53. Morris P, Hoffman D. Injuries in cross-country skiing. Trail markers for diagnosis and treatment. *Postgrad Med.* 1999;105(1):89–91, 95–8, 101.
54. Leach R, Lower G. Ankle injuries in skiing. *Clinical orthopaedics and related research*, 1985, 198. Jg., S. 127–133.
55. Lin C, Gross M, Weinholt P. Ankle syndesmosis injuries: anatomy, biomechanics, mechanism of injury, and clinical guidelines for diagnosis and intervention. *J Orthop Sport Phys.* 2006;36(6):372–84.

Karl-Heinz Kristen

---

## Abstract

Most doctors believe that water sports are safe and harmless concerning foot and ankle injuries. However, analyzing injury patterns in Windsurf, Kitesurf and wave surf, foot and ankle injuries rate within the most frequent. Collision with the board, obstacles in the water close to the beach and rotation trauma with the foot fixed in foot straps and bindings are the causes. Superficial wounds may cause dangerous infections especially in tropical water. Midfoot injuries tend to be overlooked and may cause painful chronic instability and arthritis. Surfers must be instructed in self-management of open wounds. Lisfranc joint Subluxation and subtalar injuries must be excluded after a fall with the foot caught in the foot strap.

---

## Keywords

Windsurf • Kitesurf • Surf • Lisfranc joint • Ankle • Injury • Foot injury • Infection

---

## Introduction

Surfing, Windsurfing and Kitesurfing are water sports performed by all age groups. Surfing areas are coastlines with waves. Kitesurfing and windsurfing areas are the sea and lakes all over the world with constant and strong winds. Competitions, professional world cup tours are pushing the level of surfing. Awards for the biggest surfed waves, high jump in kitesurfing, and storm surf in windsurfing are showing the extremes. But most of these fun sports are performed in easier and lighter conditions.

---

## Injuries

Although these sports are watersports, foot and ankle injuries are the most frequent injuries in Windsurf and Kitesurf athletes. As the team doctor at the world pro tour event of

Kitesurfing and windsurfing in Austria since the year 2000 there is a 14 years experience with injuries in Kite- and windsurfing. Every year since 2000 the world elite in windsurfing and kite surfing was questioned and examined. Ankle sprains are common. Skin lacerations especially on the feet are frequent and the feet of the athletes are usually full of scars. Limited range of motion in the ankle and subtalar joints is a frequent finding especially in freestyle windsurf athletes. In wave surfing, head injuries are the most frequent but also followed by ankle and foot injuries.

---

## Etiology and Pathomechanism

Why does a water sport cause such a high rate of foot and ankle injuries? The sport of surfing is mostly performed barefoot because of the better tactile feedback to the board. Neoprene boots are just used in cold water for cold protection. Sharp rocks, shells, sea urchin and poison fish but also beach pollution are frequent causes for cuts and skin lacerations – the most frequent foot injury in surfing sports. The own surfboard, especially the fiberglass or carbon fiber fins are the second frequent cause of injury. Open wounds need

---

K.-H. Kristen  
Department of Orthopaedic Sports Medicine, Sportklinik,  
Werdertorgasse 14/8, Wien 1010, Austria  
e-mail: [kristen@sportklinik.at](mailto:kristen@sportklinik.at)

1–2 weeks for proper healing. But the athletes wish to go back to surf. Therefore wounds are always wet, exposed to UV radiation and to bacteria. Deep wound infections can become a danger to life especially in tropic regions and after reef contact.

Foot straps are used in windsurfing and Kitesurfing for board control. Injuries occur if the foot stays in the foot strap during a fall. Rotation and hyper – extension or – flexion can cause ligament injuries, fractures and luxation around the Lisfranc joint. These injuries are similar to the injury described by Jacques Lisfranc in the 1800s. He observed Lisfranc joint luxation in horse riders falling from the horse and getting caught in the stirrup. If wide foot straps or – in Kitesurfing – wake board bindings are used, the foot has a better stability in the strap. But sliding out of the strap becomes more critical and subtalar or ankle joint injuries are more likely. Ankle ligament injuries – medial as well as lateral – may cause chronic ankle instability. Care must be taken in ankle compression & rotation injury. Talar neck fractures and subtalar fractures, snowboarder's ankle (see Chap. 68) are often overlooked injuries.

Jumps and falls are normal in these sports and most injuries occur landing the board after high jumps in the water. Kitesurfing presents a special risk as starting and landing the kite is by far the short period with the highest risk. Getting lifted up in the air on land without detaching from the power kite is critical.

## Epidemiology

### Kitesurf

In a recent review concerning Kitesurf injuries, Bourgois [1] concluded: Of all injuries, 45–70% occurred in the lower extremities. The ankle (64%;  $p < 0.01$ ) is the area of the lower extremities most affected by injury, followed by the foot (14%). Joint sprain (up to 40%); contusion (up to 34%); abrasion (up to 28%); muscle/tendon damage (up to 18%); and wounds, laceration, and cuts (up to 17%) were among the most frequent types of injuries, while fractures were responsible for 3–11% of all injuries.

Our personal experience [2] showed that the rate of injuries did not change over the last 14 years. We had in our collective of professional athletes 16% foot and ankle injuries – quite equal to shoulder with 17% (Fig. 66.1).

### Windsurf

Prymka [3] questioned 44 semi-professional windsurf competitors: The most common injuries were ligament ruptures of the lower leg (33%). Our own experience [4, 5] with pro-



**Fig. 66.1** X ray side view: foot of a professional kitesurfer with proximal second metatarsal fracture and ORIF



**Fig. 66.2** Windsurf footstrap

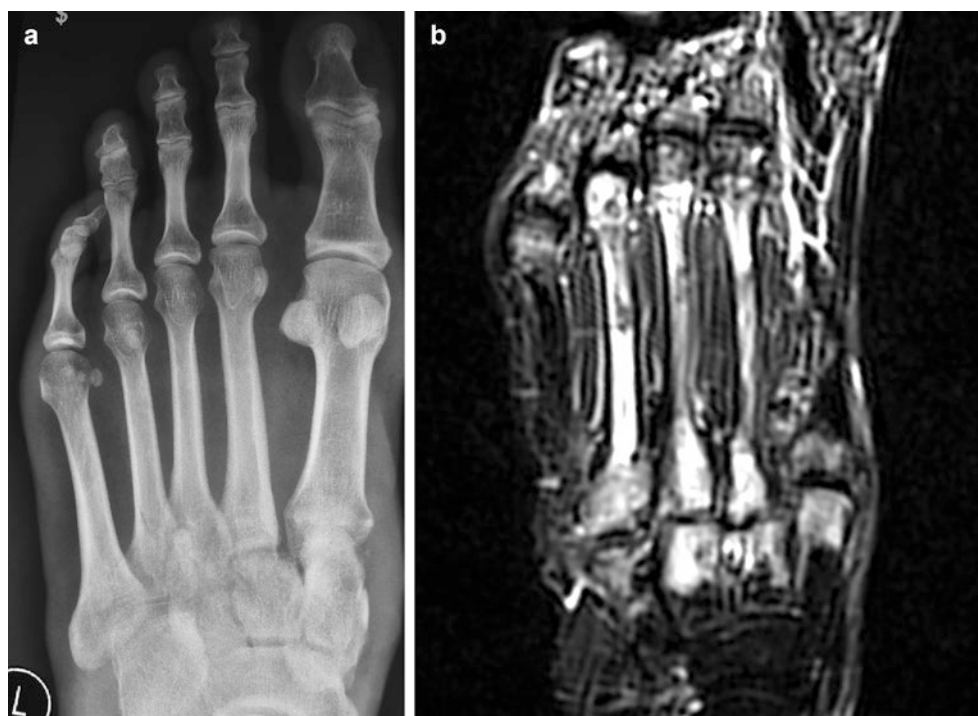
fessional windsurf athletes showed a rate of 38% lower leg injuries. Cuts and skin lacerations were the dominant injury. However, in the last years, freestyle windsurfing developed with spectacular rotation jumps. The foot has to stand deep in the foot straps in order not to lose the board during these jumps. The incidence of ankle ligament injuries has increased (Figs. 66.2, 66.3a, b and 66.4).

### Surf

Woodacre [6] analyzed Wave surf injuries: Head injuries were the most common (24%) followed by ankle (19%), knee (13%). Surfers collided most often with their own boards (31%). The other collisions were with rocks/coral (15%), the sea (11%), other surf boards (10%), the sea bed (7%), other water vessels (2%). cuts/lacerations (31%) were the commonest type of injury, followed by bruises/periorbital ecchymoses (24%), joint/ligament sprains (15%), muscle or tendon tears/ruptures (9%), concussion (5%), fractures (3%) (Fig. 66.5).



**Fig. 66.3** (a) X ray of a foot after distortion in the footstrap. (b) MRI of the foot showing bone marrow edema in the Lisfranc joint area



**Fig. 66.4** Windsurf freestyle maneuver



**Fig. 66.5** Superficial skin laceration of a surfer's foot

## Therapy

Cuts and open wounds need careful cleaning and removal of foreign bodies. Especially in tropical regions, cleaning, disinfection and taping has to be repeated every day. Be aware of infection.

For surfers travelling, an emergency bag should always contain Dressing and ev. Antibiotics.

Lisfranc joint and midfoot ligament and osseous injuries are often overlooked in normal X ray examination. Careful examination of the midfoot is recommended if a history of

“torn in the foot strap” is reported. CT scan or MRI is indicated in doubt. Lisfranc joint luxation and fracture often requires surgical reconstruction.

Ankle joint injuries may compromise the subtalar joint. Look out and ask for rotation and compression type of injury. Subtalar or talar neck fractures may require surgical reconstruction. Syndesmotomic injuries need rest for 6 weeks. Simple ankle lateral ligament injuries should be stabilized with orthoses. But take care: most ankle orthoses do not work in the water. Classic tape and kinesio tape are washed off in the water within minutes. Waterproof duck tape is often the only way to allow surfing after ankle sprain (Fig. 66.6).



**Fig. 66.6** Foot of a professional windsurfer with scars

---

## Rehabilitation and Back-to-Sports

Surfers – especially professionals – want to back in the water as soon as possible. Therefore rehab protocols should be adapted. Balance exercises can be done on SUP boards. In kite surfing, strapless boards can be an alternative after ankle sprains. However, Lisfranc-, subtalar und ankle fractures need long periods of rest.

---

## Prevention

Avoid sharp fins if possible. Wear shoes for protection in dangerous water conditions.

Choose and carefully select Bandage suitable for use in wild water conditions.

Sports equipment – foot straps must be adjusted to the feet of the rider carefully.

---

## Evidence

The level of evidence in this topic is Level IV.

---

## Summary

- Surfing sports have a high incidence of foot and ankle injuries.
- Cuts and open wounds are must be disinfected and treated carefully to avoid deep infections
- Lisfranc joint lesion may occur in foot straps and must be detected and treated.
- Ankle and subtalar joint injuries may include snowboarder's ankle and talar neck fractures.

---

## References

1. Bourgois JG, Boone J, Callewaert M, Tipton MJ, Tallir IB. Biomechanical and physiological demands of kitesurfing and epidemiology of injury among kitesurfers. *Sports Med.* 2014;44(1):55–66.
2. Kristen K, Syré S, Humenberger M. Kitesurfing – injuries and sports medical aspects. *Deutscher Ärzte-Verlag OUP.* 2014;3(6):306–11.
3. Prymka M, Plötz GM, Jerosch J. Injury mechanisms in windsurfing regatta. *Sportverletz Sportschaden.* 1999;13(4):107–11 [Article in German].
4. Kröner A, Kristen KH, Wlk MV, Engel A. Windsurf injuries (Verletzungen beim Windsurfen). *Österr J für Sportmedizin.* 2002;2:27–32.
5. Kristen K, Syre S, Kröner A. Windsurfing – sports medical aspects. *Sportorthopädie Sporttraumatologie.* 2007;23:98–104.
6. Woodacre T, Waydia SE, Wienand-Barnett S. Aetiology of injuries and the need for protective equipment for surfers in the UK. *Injury.* 2015;46(1):162–5.

Matthias D. Wimmer and Milena M. Ploeger

**Abstract**

Swimming and aquatics involve multiple and diverse single and team disciplines. Since swimming is a non weight bearing sport and water polo is the only discipline involving tackling, direct injury is rare. Eventually it may occur in diving disciplines. Overuse and monotonous training patterns should be avoided, especially when dry training in athletes. If an injury occurs, therapy should be individually adapted to the specific pattern after appropriate classification. Overall swimming and aquatics is probably the safest sport activity available regarding foot and ankle injuries. The way towards the pool on slippery ground is the most dangerous part of leisure and competitive aquatics in respect to foot and ankle injuries.

**Keywords**

Swimming • Diving • Water polo • Aquatics • Overuse • Dry training

**Injuries**

Swimming as a leisure activity or competitive sport is diverse. Multiple disciplines are part of the summer Olympics: freestyle, backstroke, breaststroke, butterfly, partially combined as medley and relay events in different ranges. Since 2008 a 10 km swim marathon is an official Olympic discipline as well. But swimming involves multiple other disciplines. Altogether they are usually summarized as “aquatics”. This involves synchronized swimming, water polo, and diving from different heights as a single or team competitions [1].

A sport physician treating athletes in aquatics should be familiar with the different techniques and physical demand of the different disciplines [2].

Swimming is a non weight bearing sport and thus the load and risk for foot and the ankle injuries are low. Subsequently

it is considered to be the optimal training and sport activity in athletes with prior lower extremity problems or injuries [3–5].

Swimming and diving rank among the disciplines with the fewest injuries during the summer Olympics 2008, and this is especially true for foot and ankle injuries [6, 7].

**Etiology and Pathomechanism**

Direct injury to the foot or ankle in aquatics is rare. It might be caused when flipping at the end of the pool. Direct contact to an opponent occurs in water polo or in training with multiple swimmers in one lane only. In diving a inadequate landing position might result in foot and ankle sprains or even in fractures [8].

In water polo tackling underwater can potentially result in ankle sprains and direct trauma, but since tackling in water polo is a low velocity trauma due to the water resistance trauma energy cannot be compared to non underwater sports. In athletes dry training such as running and fitness pose more risks of injuries to the foot and ankle system as described in the respective chapters of this book [9].

M.D. Wimmer, MD (✉) • M.M. Ploeger  
Department of Orthopaedics and Trauma Surgery, University  
Clinics of Bonn, Sigmund-Freud-Str. 25, Bonn 53125, Germany  
e-mail: [matthias.wimmer@ukb.uni-bonn.de](mailto:matthias.wimmer@ukb.uni-bonn.de); [milena.ploeger@ukb.uni-bonn.de](mailto:milena.ploeger@ukb.uni-bonn.de)

## Epidemiology

There is only very little scientific literature available reporting on swimming related foot and ankle injuries. A PUBMED search (Oct 10th 2014) for the terms (swimming AND (foot OR ankle)) revealed 374 results. The search terms (water AND polo AND (foot OR ankle)) produced 6 results only, a search for (diving AND (foot OR ankle)) revealed 75 publications. The term (aquatics AND (foot OR ankle)) produced 3 results. After exclusion of irrelevant publications only seven papers remain dealing with foot and ankle related problems and injuries specifically by aquatics.

## Therapy

Therapy should be adapted to the specific injury, to the individual injury pattern, and patients' needs.

## Rehabilitation and Back-to-Sports

As described above, the rehabilitation program and Back-to-Sports are usually adapted to individual patient needs depending on the injury pattern and the level of training [10].

## Prevention

The most dangerous part in aquatics is the way towards the pool on slippery ground and not the discipline itself. Pool edges and careless running should be avoided. Especially when training with children proper instructions and supervision by adults are important. For athletes discipline adapted training programs should be used to avoid monotonous movement and to minimize overuse, including extensor tendon inflammation over the dorsum of the foot [11].

A sufficient number of recovery days should be included in the training program, especially in older athletes [9, 12, 13].

## Evidence

There is no specific evidence available on foot and ankle injuries or their treatment of foot and ankle problems available in respect to EBM criteria.

## Summary

- Swimming and aquatics is probably the safest sport activity available regarding foot and ankle injuries.
- Direct injury is rare.
- Overuse and monotonous training patterns should be avoided.
- Therapy should be adapted to the individual injury.
- Overall the way towards the pool on slippery ground is the most dangerous part of leisure and competitive aquatics in respect to foot and ankle injuries.

## References

1. <http://www.olympics.org/sports>.
2. Fowler PJ, Regan WD. Swimming injuries of the knee, foot and ankle, elbow, and back. *Clin Sports Med.* 1986;5(1):139–48.
3. Asimenia G, Paraskevi M, Polina S, Anastasia B, Kyriakos T, Georgios G. Aquatic training for ankle instability. *Foot Ankle Spec.* 2013;6(5):346–51.
4. Bergamin M, Ermolao A, Tolomio S, Berton L, Sergi G, Zaccaria M. Water- versus land-based exercise in elderly subjects: effects on physical performance and body composition. *Clin Interv Aging.* 2013;8:1109–17.
5. Katsura Y, Yoshikawa T, Ueda SY, Usui T, Sotobayashi D, Nakao H, et al. Effects of aquatic exercise training using water-resistance equipment in elderly. *Eur J Appl Physiol.* 2010;108(5):957–64.
6. Junge A, Engebretsen L, Mountjoy ML, Alonso JM, Renstrom PA, Aubry MJ, et al. Sports injuries during the Summer Olympic Games 2008. *Am J Sports Med.* 2009;37(11):2165–72.
7. Mountjoy M, Junge A, Benjamin S, Boyd K, Diop M, Gerrard D, et al. Competing with injuries: injuries prior to and during the 15th FINA World Championships 2013 (aquatics). *Br J Sports Med.* 2015;49(1):37–43.
8. Marymont JV, Mizel MS. Fracture of the subtalar joint in springboard divers. A report of two cases. *Am J Sports Med.* 1996;24(1):123–4.
9. Mountjoy M, Junge A, Alonso JM, Engebretsen L, Dragan I, Gerrard D, et al. Sports injuries and illnesses in the 2009 FINA World Championships (Aquatics). *Br J Sports Med.* 2010;44(7):522–7.
10. Kenal KA, Knapp LD. Rehabilitation of injuries in competitive swimmers. *Sports Med.* 1996;22(5):337–47.
11. Johnson JE, Sim FH, Scott SG. Musculoskeletal injuries in competitive swimmers. *Mayo Clin Proc.* 1987;62(4):289–304.
12. Ristolainen L, Kettunen JA, Waller B, Heinonen A, Kujala UM. Training-related risk factors in the etiology of overuse injuries in endurance sports. *J Sports Med Phys Fitness.* 2014;54(1):78–87.
13. Eric S, Jennifer S. Sports injury prevention and rehabilitation. New York: McGraw-Hill Medical; 2001. p. 513.

Klaus Dann

**Abstract**

Snowboarding has become a very popular winter sport in North America, Asia, Europe and New Zealand. Around ten Million Boarders perform the sport worldwide and the sport disciplines are splitted in mainly Freeride/Freestyle orientated disciplines like Halfpipe, Snowboard Cross, Slopestyle and Alpine Racing with Parallel Slalom and Parallel Giant Slalom only performed in the FIS Alpine Snowboard World cup and all together at Olympic games like Sotschi 2014.

In general snowboarding moves from competitive formats towards freeriding in powder snow or fun parks. Most of the snowboarders prefer soft boots and soft bindings and 7 % of all fractures caused by snowboarding are fractures of the talus and the mid foot. The fracture of the lateral process of the talus (LPT), so called “snowboarder’s ankle”, is a typical lesion on the front foot in case of unexpected moguls, rollers and hard impact landings with the mechanism of dorsiflexion, hyperpronation and external rotation. This lesion is often ignored and treated as a lateral ligament ankle sprain. It is important to ask for special binding systems, which stance is used and ask whether the front foot is the affected one. Therefore it is necessary to perform a perfect clinical investigation in the hospitals. If a snowboarder reports pain 1 cm distal from the tip of the lateral malleolus and reports pain during weightbearing especially at movements in dorsiflexion and hyperpronation an X Ray/Brodén view and CT Scan are necessary to detect this lesion. All dislocated snowboarder’s ankle need an operative treatment, usually done with minimal invasive percutaneous osteosynthesis by cannulated titanium screws. If you miss the diagnosis the fragment of the LPT will be destroyed and must be resected. This procedure may lead to a posttraumatic osteoarthritis of the subtalar joint. Many of these snowboarders are not able to proceed with snowboarding.

Mid- and forefoot fractures are mostly caused by an open ankle strap of the softbinding systems. In case of dislocation operative treatment is necessary, if there is no dislocation a conservative treatment with a walkerboot is a good option.

---

K. Dann, MD  
Facharzt f Unfallchirurgie @ Sporttraumatologie, Sportarzt,  
Vienna, Austria

I.S.F. International Snowboard Federation,  
Snowboard World Championship Kreischberg, Austria

Zentrum f Traumatologie, Orthopädie, Plastische Chirurgie,  
Ordinationszentrum TOP-MED, Kinderspitalgasse 1/2/4,  
Vienna A-1090, Austria  
e-mail: [traumatology@top-med.at](mailto:traumatology@top-med.at); [k.dann@aon.at](mailto:k.dann@aon.at);  
<http://www.dann.at>; <http://www.top-med.at>

### Keywords

Snowboard history • Snowboard disciplines • Freeride/Freestyle versus Alpine Racing disciplines • Snowboard bindings • Epidemiology of snowboard related ankle injuries • “Snowboarder’s ankle” –fracture of the lateral process of the talus LPT • Treatment of LPT

## Introduction

Since the mid 1980s Snowboarding has become an enormous booming winter sport in North America and also Europe. Meanwhile we see ten Millions of snowboarders worldwide.

At the very beginning snowboarding was performed by boards without straps and bindings, directed only by a steering line from the tip of the shovel. These boards were called “snurfer”, a hybrid of ski and surfboard invented by Sherman Poppen in 1963 in USA.

Around 100 years before Austrian miners used similar wooden boards with leather straps during winter season to ride down from the gold mines in the Austrian alps around Heiligenblut and Mürzzuschlag. Some of these “miner,s horses”(“Knappenrösser”) are exhibited in the Museum of Mürzzuschlag, Styria and in the Museum of National History in Vienna, Austria.

Snowboarding has become an Olympic discipline in 1998 in Nagano with half pipe and GS Carving. Since the last Olympic games in Sotschi 2014 the snowboarding disciplines were held as PGS (Parallel Giant Slalom), PS (Parallel Slalom), HP (Half Pipe), SBX (Snowboard Cross) and SBS (Snowboard Slope Style) (Fig. 68.1).

## Snowboardbindings: Softbinding, Slip-in, Step-in and Plate Bindings

The Americans Jake Burton, Tom Sims and Dimitrij Milovich were snowboard pioneers in the USA and they started to develop bindings for snowboards for a better riding control on packed slopes.

The first bindings were just straps like windsurfing straps, you could only slip in with your boots without any fixation. In case of an accident, snowboarders slip out of the bindings with less risk to injure their ankle, the lower leg or the front foot. The disadvantage was that you are only able to surf on powder snow but not on packed and icy slopes.

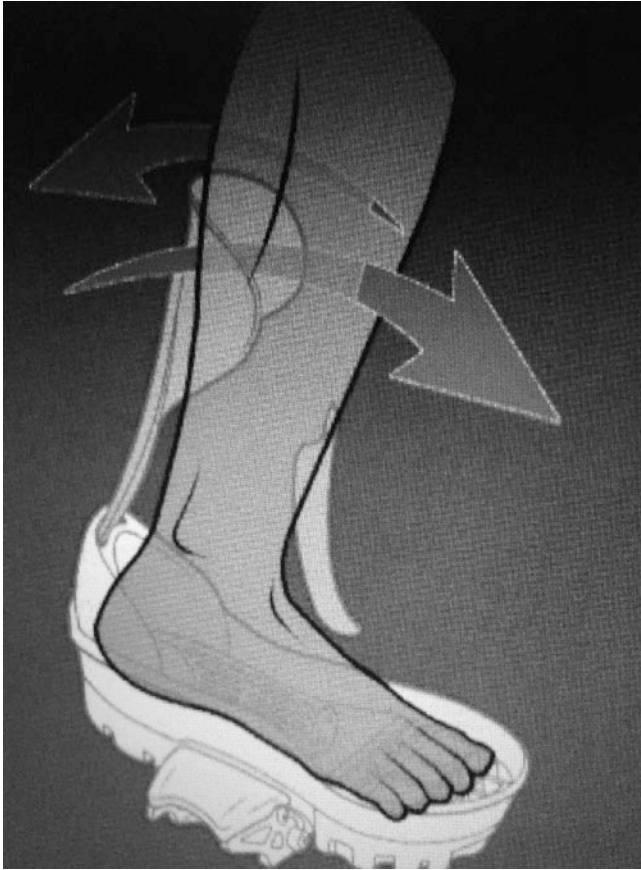
The development of snowboarding bindings shows two categories of bindings.

First the American style, with soft boots and soft bindings for freeriding and more freestyle orientated snowboarding and second the European style, race oriented hard boots and plate bindings for alpine racing like PS, PGS. Both binding systems have no possibility of release in case of sudden fall. It means snowboarders are fixed with both feet on the board. This circumstance produces typical lesions to the ankle and on the other hand protect the knee joints as we have seen the last three decades of snowboarding [1–6].



**Fig. 68.1** Snowboard cross (FIS, pic by Helmut Fritzer)

In the last 10 years the Snowboard industry developed more comfortable soft bindings systems with Step-in or Slip-in soft bindings. Step-in means you put your soft boot in



**Fig. 68.2** Soft binding step in/sole fixation, injury mechanism

a binding plate and its only fixed to the sole like a ski boot (Fig. 68.2).

Slip-in means that the high back and the ankle strap of the binding is combined open and wide and you slip in with your boot in the prepared toe strap and fix the whole system with closing the high back. This Slip-in system produces a perfect fit of the whole foot compared to the non fitting Step-in binding (Fig. 68.3).

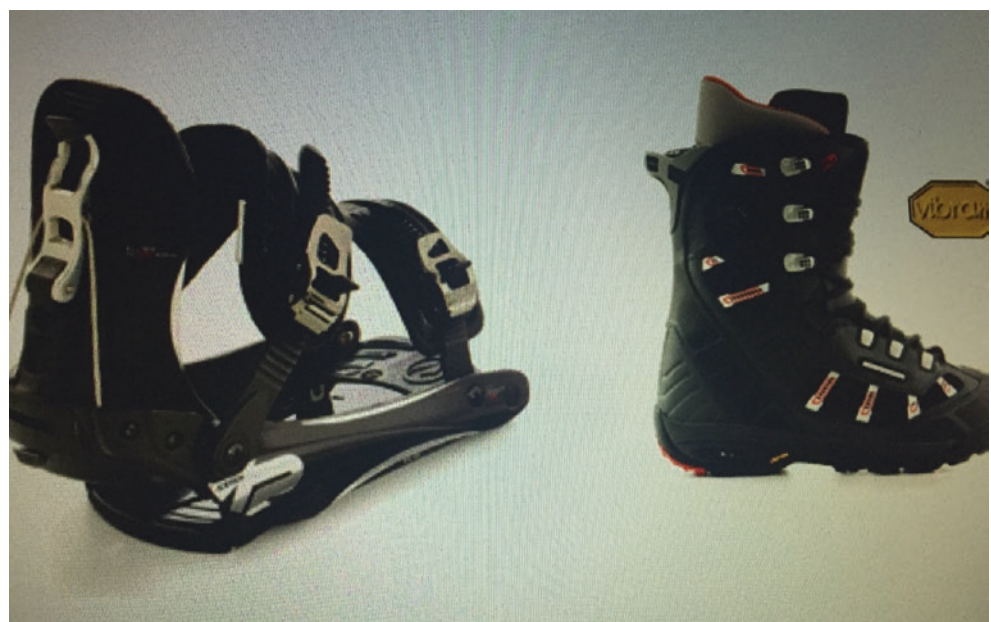
Nevertheless especially park snowboarders use the old fashioned high back bindings with separated ankle and toe straps which means you have to close both straps individually one after the other (Fig. 68.4).

The plate bindings are only used for Alpine Racing disciplines and extreme carving turns combined in racing with additional plates like shock absorbers and with hard boots similar to ski boots (Fig. 68.5).

### Epidemiology of Ankle Injuries

Snowboarding requires a permanent change from front to back-side, which leads to a strain on extensor and flexor muscles of lower limbs and also muscle and tendons around the ankle joint. There is a big difference compared with skiing, where you balance on both limbs and riding is energy saving [7].

Each snowboard discipline use different foot positions with different stances and angles to the longitudinal axis of the board. This leads to a better spread of body weight especially in freestyle and free ride disciplines therefore the stance is wide and up to duck foot (divergent position). Compared to alpine race disciplines, the stance is more closed and the foot direction of both feet is at the front



**Fig. 68.3** Soft binding slip in/soft boot system

**Fig. 68.4** Soft strap binding/soft boot



**Fig. 68.5** Plate bindings+plate system/Shockabsorber (By Sigi Grabner, SGS Boards, Austria)

60–50° and on the back foot around 40–50° to the longitudinal board axis.

The ankle of snowboarders seems to be well protected in hard boots combined with plate bindings and also soft boots with Slip-in bindings, less protected in soft boots combined with old fashioned soft binding systems and at least less protected by soft boots with Step-in bindings.

Step-in bindings fix the soft boot only to the sole and in case of unexpected moguls, rollers or worse landing situation the impact energy is most likely directed to the ankle which can cause hyperpronation and dorsiflexion of the front

foot. In general this leads to an injured front foot. In case of regular stance it is the left one and in case of goofy footers the right foot is in front position.

In former times the most frequent ankle injury of freestyle snowboarders was the fibular ligament sprain (50%) followed by 14% of malleolus lateralis ankle fractures with and without involvement of medial malleolus (2.8). Twenty-three percent of all injuries at the Olympic SBX (Snowboard Cross) in Sotschi 2014 were ankle affected injuries and caused a discharge of the athletes [8, 9].

### “Snowboarder’s Ankle”/Fracture of the Lateral Process of the Talus

Snowboarder’s ankle is often ignored but affects 15% of all ankle joint injuries after snowboard falls. Seven percent of all the fractures caused by snowboarding are fractures of the midfoot and the talus. Snowboarder’s ankle, the fracture of the lateral process of the talus (LPT) was first described in 1996 by Bladin and McCrory [10] and Kircpatrick et al. [11] in 1998. Estes et al [12] in 1999, Platz and Sommer [13] in 2000 even reported this kind of talus lesion as a typical snowboard affected trauma. This injury mainly occurs at the anterior board ankle in dorsiflexion, hyperpronation/eversion and external rotation caused in even landing manoeuvres or unexpected shocks to the front foot on the board by moguls and rollers as reported before [12, 14].



## Generally Classification + Epidemiology and Location of Talus Fractures

Classification by Hawkins	Type I–IV
Classification by Marti and Weber	Type I–IV
Central fractures	
Head	5%
Neck + Corpus	70%
Talar dome	5%
Periphereal fractures	
Processus lateralis tal “snowboarders ankle”+	
Processus posterior fractures	

### Clinical Signs

Snowboarders typical reported pain approximately 1 cm distal of the fibular tip and during fully weight bearing especially in case of hyper pronation [10].

### Diagnosis, Imaging Procedures

The visualization should be performed with a special tangential anterior-posterior projection by X ray, called Broden’s view and in case of a reported lesion further on with a CT scan plus 3 dimensional reconstruction to detect grade of dislocation and the size of the talar bone fragment. Additional a MRI to detect further lesions is an option (Fig. 68.6).

Often this injury is misinterpreted and treated as a lateral ligament ankle sprain. The clinical investigation of the

injured snowboarder should be done in a high sitting position (like an examination bed) in front of the doctor. It is important to palpate all bony and ligament structures, perform in- and eversion of the ankle joint, produce supination–pronation, dorsal – et plantar flexion and also anterior drawer test.

If we can not detect a dislocation of the LPT, snowboarders get a walker (as Vacoped, Oped Company, Germany) for 6 weeks. Three weeks in fixed 90° position and more 3 weeks in open position for a better walking mechanism. Afterwards an x- ray documentation of bone healing is necessary before fully weight bearing and sports activity can be done.

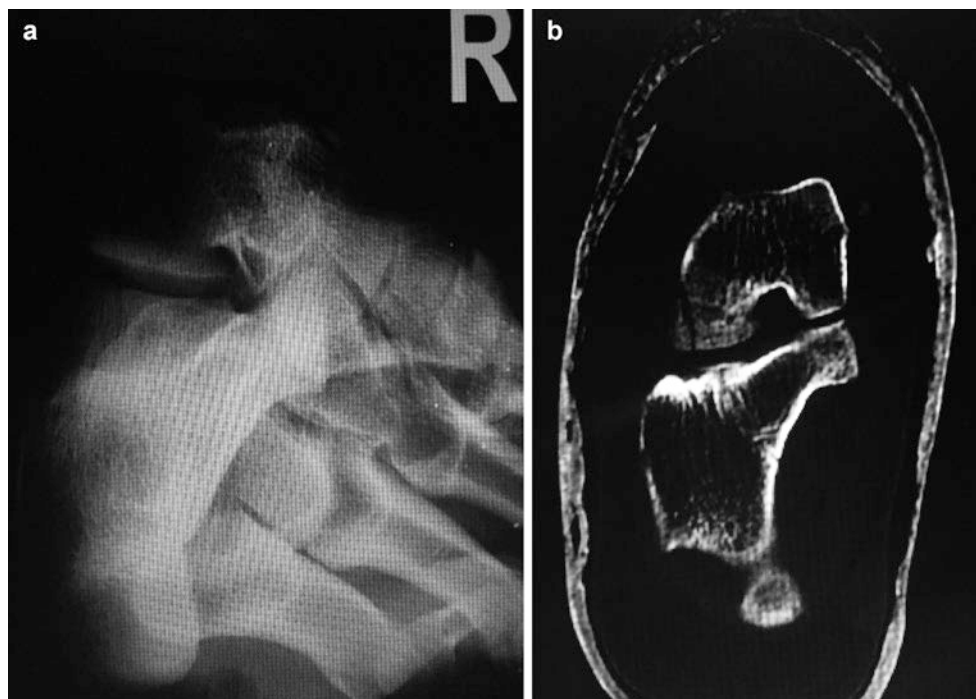
In case of fragment dislocation, the therapy of choice is the open reduction and internal fixation (ORIF) of the fragment via small incision and the fixation with a cannulated titanium screw (Fig. 68.7).

After ORIF, patient get a walker for 4 weeks in fixed position and further on with open walking mechanism for 2 weeks. Again an X ray is necessary to document bony healing of the fragment and the position of the screws. In case of doubt we perform again a CT scan do detect the fragment and the bony healing.

### Important

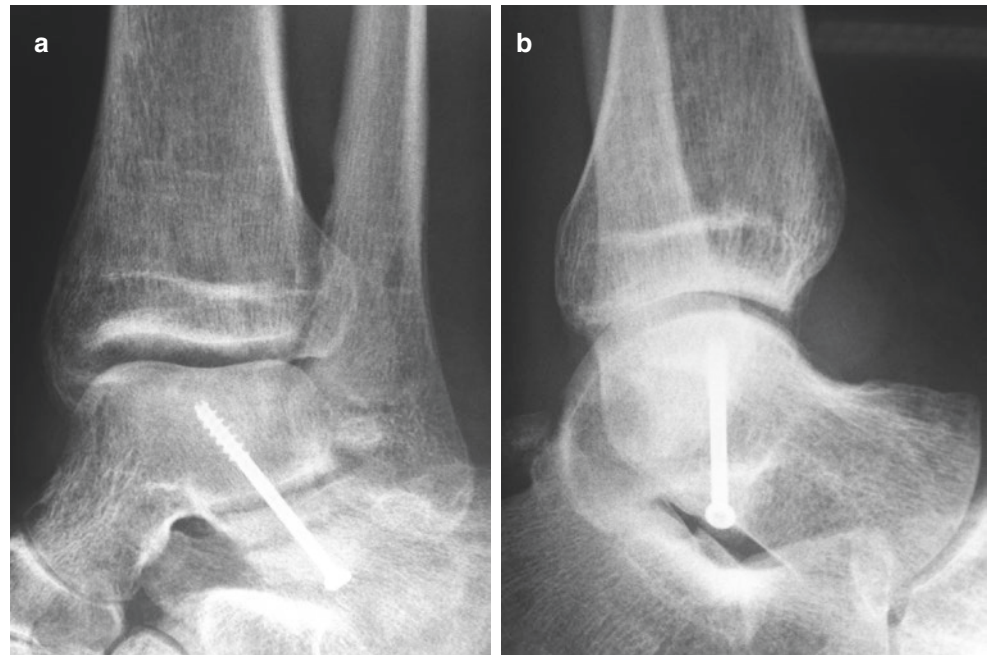
If you investigate a snowboarder with pain on the front foot, please ask for regular (left foot front) or goofy stance (right foot front), kind of boots and kind of bindings. Than palpate the tip of fibula bone, the LPT and think about “snowboarder’s ankle”.

Perform an X Ray and if Broden’s view detected a fracture of the LPT than a CT Scan with 3 dimensional reconstruction is necessary. Detect the grade of dislocation



**Fig. 68.6** Broden’s view Xray/CT scan

**Fig. 68.7** Snowboarder's ankle fixed with titanium screw



and fragment size to go on for an operative treatment with small cannulated titanium screws.

**Be aware about possible concomitant lesions like fractures of [3]**

Malleolar X	-15 %
Calcaneus X	-10 %
Cuneiforme bones, navicular, cuboid X	-7 %
Tarsalbone + Phanlanges X	-7 %

In case of concomitant calcaneus fracture please check the spine, maybe compression fracture of thoraco lumbal spine will exist.

Malleolar fractures, Pilon tibiale, are treated by the principles of the AO with anatomic reduction and osteosynthesis (ORIF). Displaced fractures of Cuneiformes, Cuboid, Naviculare and Tarsal bone are mainly caused by open or broken ankle straps of soft bindings.

First ray fractures are seldom and in case of dislocation, surgery is necessary.

After operative treatment in our hands, all boarders get a walker for 6–8 weeks with partial weight-bearing and physiotherapy.

Thirty-five percent of examined snowboarders complained about pain during boarding. The knee joint has been affected with 35% [15]. Snowboarders pain after ankle injury is triggered in 25% on front foot by non-union or necrosis of the dislocated fragment [10].

Many alpine racers reported shoe pressure problems like retrocalcaneal bursitis and inflammation of the skin caused by the heel lift in snowboard hard race boots [15–17] (Fig. 68.8).



**Fig. 68.8** Snowboarder's heel

## Evidence

All literature in this chapter is Level of Evidence four to five.

## Summary

- “Snowboarder’s ankle” is the fractures of the lateral process of the talus (LPT)
- This injury is often misinterpreted as ligament sprain
- Typically, patients report a local pain 1 cm distal to fibula tip
- The key diagnostics are: Tangential ap X Ray (Broden’s view), CT Scan + 3 D reconstruction; MRI might be also an option.
- Regarding the therapy: majority of the cases need operative treatment with a walker and partial weight-bearing

## References

1. Boldrino C, Furian G. Risikofaktoren beim Snowboarden. Eine empirische Studie. Institut. “Sicher Leben des österreichischen Kuratoriums für Schutz und Sicherheit”. 1999.
2. Campell L, Soklic P, Ziegler W, Matter P. Snowboardunfälle. Multizentrische schweizerische Snowboardstudie unter Mitwirkung der bfu. In: Matter P, Holzach P, Heim D, editors. 20 Jahre Wintersport und Sicherheit. Davos, 1992/93.
3. Dann K, Boldrino C, Kristen KH. Verletzungsrisiko und Risikofaktoren beim Snowboarden. TW Sport und Medizin. 1997;9:128–32.
4. Gabl M, Lang T, Pechlaner S, Sailer R. Snowboardverletzungen. Sportverletz Sportschaden. 1991;5(4):172.
5. Machold W, Kolonja A, Kwasny O, Fuchs M. Verletzungsrisiken beim Snowboarden. Sportverl Sportschad. 1999;13:1–7.
6. Zollinger H, Gorschewsky O, Cathrein P. Verletzungen beim Snowboardsport – eine prospektive Studie. Sportverl Sportschaden. 1994;31:7.
7. Knöringer M, Schaff PS, Rosemeyer B. Muscular dysbalance during snowboarding. EMG Video Anal Sport Orthop Traumatol. 1998;4:206–10.
8. Kusche H, Gutsfeld P, Bühren V. Halfpipe, Slopestyle und Snowboardcross- Spannung und Anforderung auf höchstem Niveau. Sport Orthop Traumatologie. 2014;29:276–82.
9. Kusche H, Schaller C, Dann K, Hörterer H. Gefahren in den neuen Schneesportarten Sportarten. Sport Orthop Traumatologie. 2014;30:306–11.
10. Bladin C, Mc Crory P. Fractures of the lateral process of the talus: a clinical review, “snowboarders ankle”. Clin Sports Med. 1996;6(5):124–8.
11. Kirkpatrick DP, Hunter RE, Janes PC, Mastrangelo J, Nicholas RA. The snowboarder’s foot and ankle. Am J Sports Med. 1998;26:271–7.
12. Estes M, Wang E, Hull ML. Analysis of ankle deflection during a forward fall in snowboarding. J Biomech Eng. 1999;121:243–7.
13. Platz A, Sommer C. Eine typische Snowboarderverletzung – die Fraktur des Processus lateralis tali. Therap Umschau. 2000;57:756–9.
14. Valderabano V, Perren T, Ryf C, Rillman P, Hintermann B. Snowboarder’talus fracture: treatment outcome of 20 cases after 3.5years. Am J Sports Med. 2005;33(6):871–80.
15. Dann K, Kristen KH, Boldrino C. Verletzungen von Snowboardprofis. Sportorthop Sporttraumatol. 1996;12(4):257–60.
16. Kristen KH, Dann K. The occurrence of retrocalcaneal bursitis at alpine snowboarding. Paper presentation, 2nd World Congress on Sport Trauma/Aossm 1996. 22nd Annual Meeting/Florida.
17. Kusche H, Gutsfeld P, Bühren V. Raceboarden-In reiner Carvingstechnik zur olympischen Medaille Sport Orthop. Traumatologie. 2014;29:270–5.

Christian Egloff and Victor Valderrabano

## Abstract

Racquet sports are amongst the most popular recreational sports activities in young and older people. The risk of injury is similar to other individual recreational sports without the catastrophic risk of collision. Patterns of injuries to the lower extremity differ between amateurs and professional players as well as between young and older players. Acute injuries tend to be more frequent in professional and young players whereas older and recreational players sustain more overuse injuries. Moreover there are distinct differences between tennis, badminton and squash.

This chapter discusses the epidemiology, incidence, pathomechanism and treatment strategies of injuries in racquet sports. Furthermore player specific factors like volume of play, age, sex, skill level and playing surface have an influence on the prevalence of injury. Lastly, recommendation for return to sport and treatment strategies are discussed in this chapter.

## Keywords

Foot and ankle • Tennis • Badminton • Squash • Injury • Racquet sports

## Introduction

Tennis, Badminton and Squash, summarized as racquet sports, have their origins in the nineteenth century with the first championships in Tennis organized in Wimbledon in 1877. Since then racquet sports have been one of the fastest growing sports during the last decades with a higher increase in number of participants than ice hockey, basketball or football. The United States Tennis association estimated 27 Million players in 2008 and more than 30 Million players in 2009 [1–3]. The ease of playing combined with the

widespread demographic range and many well-demonstrated health benefits contributed to the latest success of this sport. Moreover, with ongoing adaptation of rules, smaller court sizes for younger players (in Badminton) and slower tennis balls for older players the International Tennis Federation (ITF) tries to further expand the range of participating players. Racquet sports don't seem to be as dangerous as other impact sports but during a game players are subjected to repetitive stop and go stressors with muscle strains and unexpected events to the lower extremity [4, 5]. These observations have been linked to a variety of acute, subacute and chronic type of injuries in all major body regions [4–8]. In this chapter we focus on injuries to the lower limb especially foot and ankle. We will discuss types of injury in different player categories, pathomechanisms, therapeutical options and preventions of such injuries. The literature on racquet sports injuries is very limited with the biggest amount of data resulting from epidemiological studies from tennis players. Equivalent data from squash or badminton are scares and based on small unrepresentative samples [9–11].

---

C. Egloff, MD (✉)  
Department of Orthopaedic Surgery, University Hospital Basel,  
Spitalstrasse 21, Basel CH-4056, Switzerland  
e-mail: [Christian.egloff@usb.ch](mailto:Christian.egloff@usb.ch)

V. Valderrabano, MD, PhD  
Orthopaedic Department, SWISS ORTHO CENTER,  
Schmerzklinik Basel, Swiss Medical Network, Basel, Switzerland  
e-mail: [vvalderrabano@gsmn.ch](mailto:vvalderrabano@gsmn.ch)

## Injuries

Recent studies have shown that injury patterns in racquet sports distinctively differ between player categories [8]. We therefore have to sub-divide players into junior and elite categories versus adult recreational players in order to analyze types of injury, relative risks and giving play recommendation. Adolescent and professional players may tolerate higher training and match intensities, they have more acute and lower extremity injuries, and sustain more serious overuse stress injuries. Recreational players usually have less training- and match-hours and tend to have more upperextremity injuries and more degenerative lesions. Professional and elite tennis players develop more asymmetric musculoskeletal adaptations, which may result in an increase of pathological joint loading, abnormal biomechanics and overuse injuries [4]. In squash and badminton the literature provides less data, but the current knowledge suggests for more acute and traumatic injuries and less overuse injuries than in tennis [7, 10, 11]. Chronic injuries in racquet sports have a higher incidence in elite players. They present themselves as fatigue fractures of the metatarsal, tarsal, navicular and tibial bone, with an incidence of 12.9% [12]. Also plantar fasciitis, tendinopathies of the Achilles tendon and flexor hallucis tendon as well as Morton's neuroma occur in professional players. A rather specific pathology among tennis players is the "tennis leg", a strain at the musculo-tendinous junction of the medial head of the gastrocnemius muscle, first described by Golding in 1969 [13]. The classic tennis toe describes a subungual hematoma mostly of the great toe due to sudden stopping maneuvers and axial force on the great toe.

In badminton and squash acute injuries represent the majority of lesions especially concerning the lower extremity. A very typical injury pattern is the acute ankle sprain accompanied with acute ankle instability and rarely osteochondral lesions [7, 10]. As mentioned before we have to differentiate acute from chronic lesions. Lesions which need surgical treatment are most often resulting from chronic overuse such as achillodynia or achilles tendinitis [9, 14, 15]. The most prevalent acute injuries that require an emergency medicine consultation are ankle sprains, achilles tendon rupture and muscle strains. According to Goh et al. achilles tendon ruptures were responsible for 13–35% of badminton players who were hospitalized [10, 14]. Fractures in the foot and ankle are rarely seen in badminton or squash.

## Etiology and Pathomechanisms

In racquet sports the players inquire repetitive as well as abrupt high-energy loads during acceleration and stopping maneuvers [4]. Due to the sprint position the plantar pressure is often higher in the forefoot and central foot. This overload

may predispose fatigue fractures and tendinopathies as described earlier.

The mean pathomechanism of injury is often a sudden change of direction (for example from backward to forward sprint) or fast acceleration and deceleration maneuvers after overhead clear shots or smashes. This movement leads to an enormous eccentric load to the lower extremity and puts the ankle joint at risk for strains, sprains or ligament rupture [14].

Nonspecific mechanisms seem to be the leading cause of accidents to the lower extremity followed by twists and falls. Interestingly, more than 80% of all lesions happen in the second half or near the end of a match [9]. This suggests a correlation between muscle fatigue and increasing risk of injury.

A special focus has to be made regarding the playing surface. It has been shown that slower surfaces like clay the players endure longer sliding movements because of the lower friction coefficient. This leads to longer contact times and a higher incidence of muscle strains and tendinosis (Ref. Lynch SA, Renstrom P. Foot problems in tennis. In: Renstrom P, eds. Tennis. Oxford, UK: Blackwell, 2002:155–64.). Typical examples are strains of the tibialis posterior muscle and plantar aponeurosis.

On the other hand surfaces like hard court provide a higher friction coefficient, which increases the speed of the game but also enhances the acceleration force and torque during change of direction. This may lead to a greater likelihood of potential muscle fatigue. The repetitive stress placed on the foot is much higher on hardcourt than on other surfaces. It has been shown that the higher loading forces measured on hardcourt may be a risk factor for blocking type injuries like ankle strains, achilles tendonitis or metatarsalgia. Tendon ruptures of the flexor hallucis longus or the achilles tendon are also more likely on fast surfaces because of the higher peak forces during acceleration. (Ref Trepman E, Mizel MS, Newberg AH. Partial rupture of the flexor hallucis longus tendon in a tennis player: a case report. Foot Ankle Int 1995;) Therefore it is not surprising that there are less injuries on clay surface than on Hardcourt in professional players [5]

## Epidemiology

Tennis, Badminton and Squash have similar rates of injury compared to other individual recreational sports and junior competitive sports, without the catastrophic risk of contact/collision injuries with other players. In surveys, over 90% of the interviewed recreational tennis players reported to have sustained an injury to the lower limb. However, most of those accidents were trivial injuries not requiring medical treatment [16]. The risk of injury is positively correlated with increasing age as well as with sex. Winge and colleagues

**Table 69.1** Distribution of Racquet sport injuries to the lower limb, adapted from [8, 14, 18]

	Tennis (%)	Badminton (%)	Squash (%)
Injuries to the lower limb	42.2	82.9	68.0
Upper leg	2.3	2.8	5.0
Knee	19.5	11.5	24.0
Lower leg	15.8	14.3	5.0
Ankle	47.2	44.2	26.0
Foot	13.7	10.1	8.0

found a significant higher injury rate in men than in women (2.7 versus 1.1 injuries per 1000 h) [17]. Moreover, professional and elite players seem to sustain more serious injuries, which needed medical treatment [8]. A recent study by Gaw et al. documented almost half a million tennis related injuries in the United States from 1990–2011. Here 42.2% of all injuries affected the lower extremity with the ankle as the most affected site (47.2%) (Table 69.1) [8]. Chard et al. found similar numbers in an older study. They reported that 45% of all tennis injuries were involving the lower extremity [18]. In squash, injuries to the lower limb were even more frequent (58%). Here the knee and ankle were most affected (sprains) with mainly acute injuries (Table 69.1) [18]. The incidence of injuries while playing badminton described in the literature varies between 1.2 and 5% [9, 10, 14]. However, the lower limb is most likely to be affected in Badminton (Table 69.1) [10]. Recreational players and beginners are more prone to injuries than professional players. Also, men suffer much more injuries than women [9, 14].

## Therapy

Most of the injuries occurring in racquet sports, which lead to a visit at the emergency department, can be treated conservatively. The P.R.I.C.E principle has been shown to be effective during the first 24–72 h. This acronym stands for: [19]

- Protection
- Rest
- Ice
- Compression
- Elevation

The goal during this time frame is to control the amount of swelling, prevent further injury and reduce pain [20]. In case of acute ligament lesions or ankle sprains cryotherapy was found to be significantly more effective to decrease swelling 3–5 days after injury than heat therapy or exercise alone [21]. Temporary abstention of sport, a stabilizing orthotic device, taping and physiotherapy have shown to reduce the incidence and the severity of re-injuries [22, 23].

Regarding the treatment options for acute Achilles tendon rupture or fractures of the foot and ankle the same guidelines apply to tennis or badminton players as for the general population [12, 24]. For a concise decision regarding surgical and conservative treatment a thorough and integral analysis of the patients demands, sports activity level and personal perceptions are necessary to provide the best possible outcome.

In chronic pain situations like achillodynia or plantar fasciitis, eccentric strength and coordination training together with nonsteroidal anti-inflammatory drugs (NSAID's) and ultrasound or shockwave therapy have a high success rate [22, 25].

## Rehabilitation and Prevention

Important factors to consider for back to sports rehabilitation protocols are court surfaces. As described previously clay court surfaces have lower friction coefficient and less peak stress forces and may be better for the rehabilitation program after a foot and ankle injury. In addition, structured game specific on court exercises and progression is recommended during return to play protocols. To date, we could not find any intervention-studies on tennis injuries. Kibler et al. and Herbert and Gabriel looked at stretching protocols and their influence on the rate of injury [22, 26]. They showed a significant increase in the range of motion but there was no reduction of injury risk. Also, the current literature lacks on evidence on the effect of warm up and stretching habits regarding the incidence of Achilles tendon rupture [27]. However, it would be worthwhile to investigate the effects of specific prevention programs on the occurrence of injuries in racquet sports.

Predisposing factors for injuries to the foot and ankle in racquet sports are sex, age, weight, equipment and training intensity. Despite the remarkable increase of 46% in active tennis participants over the last decade, the number of tennis related injuries presenting to US emergency departments decreased by more than 40%. Moreover the study by Gaw et al. found a significant decrease in sprains and strains to the ankle [8]. This decrease might be due to better prevention strategies, proper game instructions and play settings. Regarding the equipment the players should watch for appropriate shoes with elevated heel and adequate cushioning

effect. To countervail serious injuries like achilles tendon ruptures, as one of the most prevalent injuries in badminton, specific endurance, strength and coordination training with eccentric training of the triceps surae muscle are crucial [9, 14]. These neuromuscular training sessions (balance, coordination, stretching and agility) were analysed in a recent meta-analysis and suggested that such training protocols could be effective in preventing foot and ankle injuries [25]. Recently FIFA presented that preseason and intraseason programs may reduce injury in up to 21 % of cases (FIFA eleven). This program consists of ten exercises and a commitment to fair play and sportsmanship [28]. Although this program is specified for soccer players it also might be a suitable recommendation for racquet sports player.

## Summary

Injuries to the foot and ankle in racquets sports are frequent and vary based on training intensities, age of the player, level of competition. Overuse injuries tend to occur more in elite player whereas acute injuries are more frequent in recreational players. Also Squash and Badminton have more acute injuries and less overuse injuries than Tennis.

Return to play recommendation should be based on the ability level and the personal expectation of the player. Rehabilitation protocols should include core stabilization, neuromuscular and proprioceptive training, kinetic chain integration and functional strengthening. Most injuries allow a successful return to play with conservative treatments.

## References

- America USTATfgsi. Tennis fastest growing sport in America [United States Tennis Association website]. 2009. 2013 [Cited 2014].
- Association TI. Tennis participation jumps 46 % from 2000 through 2010 [Tennis Industry Association website]. 2011. 2011 [Cited 2014 August].
- M. B. Forty to love: tennis takes the title in sports participation growth <http://www.forbes.com/sites/morganbrennan/2010/09/03/forty-to-love-tennis-takes-the-title-in-sports-participation-growth/>. 2010.
- Elliott B. Biomechanics and tennis. *Br J Sports Med*. 2006;40(5):392–6.
- Stiles VH, Dixon SJ. The influence of different playing surfaces on the biomechanics of a tennis running forehand foot plant. *J Appl Biomech*. 2006;22(1):14–24.
- Bylak J, Hutchinson MR. Common sports injuries in young tennis players. *Sports Med*. 1998;26(2):119–32.
- Jayanthi N, Esser S. Racket sports. *Curr Sports Med Rep*. 2013;12(5):329–36.
- Gaw CE, Chounthirath T, Smith GA. Tennis-related injuries treated in United States emergency departments, 1990 to 2011. *Clin J Sport Med Off J Can Academy Sport Med*. 2014;24(3):226–32.
- Fahlstrom M, Bjornstig U, Lorentzon R. Acute badminton injuries. *Scand J Med Sci Sports*. 1998;8(3):145–8.
- Kroner K, Schmidt SA, Nielsen AB, Yde J, Jakobsen BW, Moller-Madsen B, et al. Badminton injuries. *Br J Sports Med*. 1990;24(3):169–72.
- Eime R, Zazryn T, Finch C. Epidemiology of squash injuries requiring hospital treatment. *Inj Control Saf Promot*. 2003;10(4):243–5.
- Maquirriain J, Ghisi JP. The incidence and distribution of stress fractures in elite tennis players. *Br J Sports Med*. 2006;40(5):454–9. discussion 9.
- Golding D. Tennis leg. *Br Med J*. 1969;4(5677):234.
- Goh SL, Mokhtar AH, Mohamad Ali MR. Badminton injuries in youth competitive players. *J Sports Med Phys Fitness*. 2013;53(1):65–70.
- Jorgensen U, Winge S. Injuries in badminton. *Sports Med*. 1990;10(1):59–64.
- Pluim BM, Staal JB, Windler GE, Jayanthi N. Tennis injuries: occurrence, aetiology, and prevention. *Br J Sports Med*. 2006;40(5):415–23.
- Winge S, Jorgensen U, Lassen NA. Epidemiology of injuries in Danish championship tennis. *Int J Sports Med*. 1989;10(5):368–71.
- Chard MD, Lachmann SM. Racquet sports – patterns of injury presenting to a sports injury clinic. *Br J Sports Med*. 1987;21(4):150–3.
- Anderson M, Hall S, Martin M. (2000). *Sports Injury Management*. (2nd Ed.). Lippincott Williams & Wilkins: Philadelphia, PA.
- Bleakley CM, O'Connor S, Tully MA, Rocke LG, Macauley DC, McDonough SM. The PRICE study (Protection Rest Ice Compression Elevation): design of a randomised controlled trial comparing standard versus cryokinetic ice applications in the management of acute ankle sprain [ISRCTN13903946]. *BMC Musculoskelet Disord*. 2007;8:125.
- Cote DJ, Prentice Jr WE, Hooker DN, Shields EW. Comparison of three treatment procedures for minimizing ankle sprain swelling. *Phys Ther*. 1988;68(7):1072–6.
- Herbert RD, Gabriel M. Effects of stretching before and after exercising on muscle soreness and risk of injury: systematic review. *BMJ*. 2002;325(7362):468.
- Callaghan MJ. Role of ankle taping and bracing in the athlete. *Br J Sports Med*. 1997;31(2):102–8.
- Soroceanu A, Sidhwa F, Aarabi S, Kaufman A, Glazebrook M. Surgical versus nonsurgical treatment of acute Achilles tendon rupture: a meta-analysis of randomized trials. *J Bone Joint Surg Am*. 2012;94(23):2136–43.
- Hubscher M, Zech A, Pfeifer K, Hansel F, Vogt L, Banzer W. Neuromuscular training for sports injury prevention: a systematic review. *Med Sci Sports Exerc*. 2010;42(3):413–21.
- Kibler WB, Chandler TJ. Range of motion in junior tennis players participating in an injury risk modification program. *J Sc Med Sport Sports Med Aus*. 2003;6(1):51–62.
- Park DY, Chou L. Stretching for prevention of Achilles tendon injuries: a review of the literature. *Foot Ankle Int Am Orthopaedic Foot Ankle Soc Swiss Foot Ankle Soc*. 2006;27(12):1086–95.
- Junge A, Lamprecht M, Stamm H, Hasler H, Bizzini M, Tschopp M, et al. Countrywide campaign to prevent soccer injuries in Swiss amateur players. *Am J Sports Med*. 2011;39(1):57–63.

Oliver Miltner and Markus Wurm

**Abstract**

Volleyball shows a relative small number on injuries compared to other team-impact sports like ice hockey, handball or soccer. Most frequent affected sites are spine as well as the shoulder, knee and ankle joint. Acute trauma concerning the ankle joint as well as the foot is spraining with ligament-elongation or even tear. Furthermore rupture of the Achilles tendon and injuries to the metatarsophalangeal joint as well as damage due to overuse are commonly seen injuries.

Most of these injuries occur due to sprains during the jump or landing phase, typically due to a landing on a teammates foot.

Concise inspection, palpation and a careful functional examination should, after consideration of assessment parameters (i.e. limited range of motion, pain, instability, load capacity and athletes self assessment), ultimately lead to decision of return to play.

After acute ankle sprain an external stabilization should be established to prevent consequential damage as well as downtime for the athlete.

**Keywords**

Volleyball • Jumping sports • External stabilisation • Ligament injury • Turf toe • Tendinopathy

**Injuries****Acute Injuries**

1. *Ligament injuries of the ankle joint*
2. *Achilles tendon rupture*
3. *1st MTP joint injury (turf toe)*

---

O. Miltner (✉)  
DocOrtho – Die Bewegungsprofis,  
Friedrichstr.94, Berlin 10117, Germany  
e-mail: [miltner@docortho.de](mailto:miltner@docortho.de)

M. Wurm  
Orthopaedic Department, University Hospital Basel,  
Basel, Switzerland  
e-mail: [wurmarkus@gmail.com](mailto:wurmarkus@gmail.com)

**Stress-Injuries**

1. *Stress fractures of the foot*
2. *Achilles tendinopathy*
3. *Peroneal tendinopathy*

**Etiology and Pathomechanism**

Volleyball shows a relative low count on injuries compared to other team-impact sports like ice hockey, handball or soccer. The most frequent affected sites are spine as well as shoulder, knee and ankle joint. Acute trauma concerning the ankle joint as well as the foot is spraining with ligament-elongation or even rupture. Furthermore rupture of achilles tendon and injuries to the metatarsophalangeal joint (turf toe) as well as damage due to overuse (stress fractures of the



foot, achilles tendinopathy or peroneal tendinopathy) are common seen complaints.

Pro- and Supination trauma of the ankle joint are a common source for ligament tears concerning the medial and lateral side respectively.

Most of these sprains occur during the jump or landing phase, stereotypically because of landing on a teammates foot after spiking or blocking.

Recurring eccentric tensile loading can lead to chronic irritation (micro tears of tendon) and achilles tendinopathy. Besides this, deviation of leg axis and shortening of ischio-crural muscles/triceps surae muscle accompanied by repetitive jumping activity can ultimately end in Achilles tendon rupture due to an acute on chronic mechanism.

Due to altered course deformities, i.e. pes cavovarus, as well as ankle instability can lead to peroneal tendinopathy. Subluxation can ultimately lead to a tear of peroneal tendons due to recurrent damage.

Chronic traumatic hyperextension of the big toe while jumping and landing can lead to a so-called turf toe injury. It is characterized by a progression from a chronic capsulitis, synovialitis to an eventually tear of the short flexor tendon of the great toe. Arthritis of the 1st MTP (Hallux rigidus) is another irritating manifestation of repetitive trauma in this region.

Muscular fatigue due to repetitive jumping can to eventually lead to stress fractures of the foot and the tibia.

---

## Epidemiology

Most injuries are seen related to spine, shoulder, knee, finger besides the foot and ankle joint whereby the ankle sprain is to be considered the most frequent acute injury [1–7]. Pastor et al. found an overall injury incidence of 4.38/1000 played hours volleyball (3.3 acute injuries, 1.08 overuse injuries), which was somewhat higher than compared to former observed results [2]. They furthermore distinguished injury incidence levels between acute (1.94) and overuse injuries (0.64) per player. Outside attackers were most often affected position with 42.47%. Downtime for players was 14.74 days in acute and 2.17 days in overuse injury. Ankle joint was the third most often affected region with 11.29%. 6/13 major injuries where with regards to the ankle whereas “major” was stated being 4 or more weeks of downtime [6].

Agel et al. showed the ankle joint to be harmed in 44% while competition and 29.4% in training phase over a 16-year lasting observation period in female first league college volleyball players [1].

Slightly inverted forefoot position during landing phase or impact of another player are only two of manifold reasons for ankle distortions, which can lead to ligamentous strain of the foot and ankle.

Overuse injuries (especially shoulder and knee) and acute injuries seem to occur with equal incidence levels. Aagaard et al. also reported on almost equal distributed injury pattern between indoor and beach volleyball yet they found a longer caused disability after lower extremity trauma [2].

---

## Therapy (On Field, Conservative, Surgical)

### Acute Injuries

Functional testing followed by a concise diagnosis and adjacent therapy are the prerequisites for a proper management of injuries to keep downtime for the athlete as short as possible. It is crucial to perform a proper inspection and palpation, which should be shadowed by a talk/conversation with the athlete to find out if a return to the field is worthwhile and moreover possible. First on field actions should i.e. in a ankle sprain be conform to the RICE – scheme which is assembled by rest, ice, compression and elevation [8]. Different additional variations of this principle i.e. “HI-RICE” (H<sub>y</sub>dration, I**l**uprofen) are known today. Ice water- soaked sponges combined with compression (i.e. taping) showed to be effective tools. Adapted to severity of injury early immobilization in lower extremity injuries is important and should reduce swelling and pain levels [9]. Conservative as well as operative treatment of acute injuries of the foot (i.e. turf toe) and ankle (i.e. instability) should be according to individual stage [10, 11].

### Overuse Injuries

Achilles tendinopathy is primarily treated in a conservative fashion. Reduction of jumping, eccentric training of calf musculature and volleyball-specific landing patterns should be implemented during convalescence and in subsequent training program.

To address peroneal tendinopathy a reduction of jumping frequency and a forced technique training should be accompanied by a strengthening of pronator muscle and proprioceptive exercise. Passive physiotherapeutic actions should be performed besides these active approaches.

In stress fractures (high vs. low risk fracture) a specific treatment algorithm referring to the characteristic and manifestation location of the fracture is crucial [12].

## Rehabilitation and Return to Sports

Main goal of the convalescence and rehabilitation process after foot and ankle sprains is the prevention of consequential damage, recurrence of sprains and the recovery to full performance levels. Balance board training (eyes opened/shut) is a useful tool and training program incorporating inter alia proprioception for stabilization of the ankle joint [2]. A fully restored former injured region and volleyball-specific resilience is the cornerstone of a return to sports at full performance levels [6].

## Prevention

Literature shows a need of high athletic, tactical as well as technical requirements besides a very well established physical fitness level to prevent volleyball specific injuries [13]. In addition preventive procedures are needed to reduce frequency and severity of injuries [14]. Assessment of extrinsic (so-called environmental-related) and intrinsic (person-related) risk factors is crucial for proper injury prevention. Conducted level of sports (volleyball is to be considered risk category nine according to Ankle Activity Score by Halasi) exercise load, player's position are just some external risk factors [15–17]. Middle blocker i.e. have a higher incidence on foot and ankle injuries compared to their teammates [7].

Amongst others reduced proprioception as well as sense of balance besides previous injuries are some important internal risk factors which can lead to foot and ankle harm. After preceding ankle sprain external stabilization (i.e. taping or braces) is recommended to avoid further downtime for

the player and to decrease consequential damage [9]. Volleyball-specific coordination and jump exercises should be incorporated in daily training habits to help minimize the count of overuse injuries. Pastor et al. observed a significant decrease on injury levels for players within their first on to second season ( $p < 0004$ ). They traced this observation back to a constant and volleyball specific prevention program to which all athletes were included [5, 6, 13].

## Evidence

Table 70.1.

## Summary

- Volleyball, in comparison to other team (impact) sports like ice hockey, soccer or handball shows a relatively low count on injuries.
- Most occurring injuries in volleyball sports concern spine, shoulder, knee and ankle joint as well as fingers.
- Commonly ankle sprains during play or training are seen in vicinity to the net. Timing while jumping and landing phase and the stereotypical landing on a teammates foot is a usual pathomechanism.
- After concise and comprehensive inspection, palpation and careful functional testing the path of treatment should be followed regarding particular assessment parameter. Limited Range of motion, pain, instability, load capability and last but not least the self-estimation of the athlete are the ones that should be concerned before decision making of return to field.
- After ankle sprains external stabilization should be applied to reduce or even avoid down time for the athlete and. This can be achieved using various taping or bracing methods.

**Table 70.1** Evidence of injuries in volleyball

Author(s)	Journal, year	Target audience	Level of evidence
Agel et al.	<i>J Athl Train</i> , 2007	Women USA College	II
Augustsson et al.	<i>Scand J Med Sci Sports</i> , 2006	Both sex, Professional Sweden	II
Bahr & Bahr	<i>Scand J Med Sci Sports</i> , 1997	Professional Men Norway	II
Miltner et al.	<i>SportOrtho Trauma</i> , 2012	Professional Men Germany	II
Verhagen et al.	<i>Br J Sports Med</i> , 2004	Semi- Professional Men, Netherlands	II

## References

1. Agel J, Palmieri-Smith RM, Dick R, Wojtys EM, Marshall SW. Descriptive epidemiology of collegiate women's volleyball injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 through 2003–2004. *J Athl Train.* 2007;42(2):295–302.
2. Augustsson SR, Augustsson J, Thomee R, Svantesson U. Injuries and preventive actions in elite Swedish volleyball. *Scand J Med Sci Sports.* 2006;16(6):433–40.
3. Bahr R, Bahr IA. Incidence of acute volleyball injuries: a prospective cohort study of injury mechanisms and risk factors. *Scand J Med Sci Sports.* 1997;7(3):166–71.
4. Kerkhoffs GM, van den Bekerom M, Elders LA, van Beek PA, Hullegie WA, Bloemers GM, et al. Diagnosis, treatment and prevention of ankle sprains: an evidence-based clinical guideline. *Br J Sports Med.* 2012;46(12):854–60.
5. Verhagen EA, Van der Beek AJ, Bouter LM, Bahr RM, Van Mechelen W. A one season prospective cohort study of volleyball injuries. *Br J Sports Med.* 2004;38(4):477–81.
6. Pastor MF, Ezechieli M, Classen L, Kieffer O, Miltner O. Prospective study of injury in volleyball players: 6 year results. *Technol Health Care.* 2015;23(5):637–43.
7. Miltner O, Weihe W, Heinzinger A, Kieffer O. Injuries in male professional volleyball players – 4 year results. *Sports Orthopaedics Traumatol Sport Orthopädie Sport Traumatol.* 2012;28:163–9.
8. Bleakley C, McDonough S, MacAuley D. The use of ice in the treatment of acute soft-tissue injury: a systematic review of randomized controlled trials. *Am J Sports Med.* 2004;32(1):251–61.
9. Lamb SE, Marsh JL, Hutton JL, Nakash R, Cooke MW. Collaborative Ankle Support T. Mechanical supports for acute, severe ankle sprain: a pragmatic, multicentre, randomised controlled trial. *Lancet.* 2009;373(9663):575–81.
10. Clanton TO, Ford JJ. Turf toe injury. *Clin Sports Med.* 1994;13(4):731–41.
11. Valderrabano V PJ, Knupp M, Camathias C, Greitemann B, Furhamm R, Krüger Franke M, Engelhardt M. Akute und chronische OSG-instabilität. Sprunggelenksinstabilität GOTS Expertenmeeting. 2012;Burg Spreewald(31.5-3.6):43–55.
12. Miltner O. Stress reactions in bones of the foot in sport: diagnosis, assessment and therapy. *Unfallchirurg.* 2013;116(6):512–6.
13. Lian O, Refsnes PE, Engebretsen L, Bahr R. Performance characteristics of volleyball players with patellar tendinopathy. *Am J Sports Med.* 2003;31(3):408–13.
14. Reeser JC, Verhagen E, Briner WW, Askeland TI, Bahr R. Strategies for the prevention of volleyball related injuries. *Br J Sports Med.* 2006;40(7):594–600; discussion 599–600.
15. Fong DT, Hong Y, Chan LK, Yung PS, Chan KM. A systematic review on ankle injury and ankle sprain in sports. *Sports Med.* 2007;37(1):73–94.
16. Halasi T, Kynsburg A, Tallay A, Berkes I. Development of a new activity score for the evaluation of ankle instability. *Am J Sports Med.* 2004;32(4):899–908.
17. Willems TM, Witvrouw E, Delbaere K, Mahieu N, De Bourdeaudhuij I, De Clercq D. Intrinsic risk factors for inversion ankle sprains in male subjects: a prospective study. *Am J Sports Med.* 2005;33(3):415–23.

Mario Herrera-Perez and Anna Oller-Boix

**Abstract**

Several water sports have been connected to foot and ankle injuries. Most of these injuries are sprains, strains, contusions and skin lesions, with just a few of them being fractures. Ankle sprains are the most common injuries during water skiing followed by talus fractures, calcaneal tendon ruptures and ankle fractures. However, regarding the rest of the water sports reviewed in this chapter (rafting, canoeing, kayaking, sailing, rowing, snorkeling and diving), foot and ankle are not the most common areas involved in injuries during these activities. Otherwise, data reported in literature about dermatoses in aquatic sports (sailing, rowing, snorkeling and diving) is related to wet suits. In conclusion, there is little literature regarding foot and ankle injuries in water sports. Therefore, studies are needed to complete information and to get a higher level of evidence to lead sports practitioners to recognize, treat and prevent these injuries.

**Keywords**

Water skiing • Rafting • Canoeing • Kayaking • Sailing • Diving • Snorkeling • Rowing

Several water sports have been related to foot and ankle injuries but little literature has been written about it. The aim of this chapter is to expose an updated review of that topic focused on water skiing, rafting, canoeing and kayaking, sailing, rowing, snorkeling and diving.

**Water Skiing**

Ankle injuries are widespread in water skiing despite advances in boot technology [1, 2]. The following are the four most common injuries:

**Ankle Sprains**

They are the leading type of injury while water skiing. These typically occur with plantarflexion and inversion of the ankle. Syndesmotic sprains, however, imply an injury to the anterior tibiofibular ligament and the mechanism is typically a dorsiflexion with an external rotation. Sometimes, recurrent sprains can lead to chronic instability and joint damage [1]. Ankle sprains often require a period of 1–8 weeks of non-weight bearing (depending on the grade) and sometimes even a cast followed by a period of physiotherapy with a gradual return to sports. In these cases, surgery is rarely necessary [2].

**Lateral Process of the Talus Fractures (Snowboarder's Fracture)**

This fracture is often confused with an ankle sprain and the mechanism of injury is a forced eversion with a loaded hind-foot. A plain x-ray may diagnose the fracture but a CT scan

M. Herrera-Perez (✉) • A. Oller-Boix  
Department of Orthopaedic Surgery and Traumatology, University Hospital of Canary Islands, Ctra. Ofra, s/n, 38320 San Cristóbal de La Laguna, Santa Cruz de Tenerife, Canary Islands, Spain  
e-mail: [herrera42@gmail.com](mailto:herrera42@gmail.com); [annaoller@hotmail.com](mailto:annaoller@hotmail.com)

is often necessary for a complete definition of the injury [1]. Because this is an intraarticular fracture, it frequently requires anatomic reduction and stable fixation. If this is not possible, resection of the fragment should be considered. After surgery, early range of motion of the ankle and subtalar joint is advised. Weight bearing should be restricted for 6 weeks and once the fracture is healed, progressive weight bearing and gait rehabilitation is started.

### Calcaneal Tendon Ruptures

They can occur during a crash or when skiers violently kick off their skis [1]. To repair the injury, surgery is often needed to avoid loss of strength [2]. Weight bearing should be restricted for the first 6 weeks after surgery with progressive weight bearing for 3 months. Rehabilitation is started immediately after surgery. High impact sports such as water skiing are allowed 6 months after surgery.

### Ankle Fractures

Ankle fractures are uncommon, but they can occur. The most common mechanism is supination and external rotation [1]. Most ankle fractures related to water skiing can be treated conservatively in a cast, but unstable fractures need anatomic reduction and internal fixation. For a conservative treatment, weight bearing is forbidden for 4–6 weeks. In the case of surgery, early active exercises and light partial weight bearing followed by progressive weight bearing is recommended as tolerated.

### Rafting

The risk associated with whitewater rafting varies with the level of whitewater, weather, the health of the passenger and the experience of the guide. Although considered to be an extreme activity, rafting has a low morbidity rate. Attarian and Siderelis [3] in their examination of injuries in commercial rafting estimated that the majority of incidents were musculoskeletal injuries (sprains/strains 21.1%, dislocations 13.8%, fractures 12.2%), followed by soft tissue injuries (lacerations 29%, abrasions 13.3%, contusions 2.3%). Anatomically almost one half (44.3%) of injuries were to the head, neck and shoulders, followed by the lower extremities (33.9%). Fractures were most common in feet; however, sprains and strains were most common in knees. Dislocations occurred most often in shoulders. They also estimated that an 11% were ankle injuries and a 2.2% were foot injuries of the total number. Whisman and Hollenhorst [4] noted similar results and suggested that injuries were usually the result of

collisions with other passengers or being struck by a paddle. Data also revealed that injuries occurred when passengers were thrown into the river where they could hit rocks, floating debris, turbulent water or other hazards. To prevent, recognize and treat injury, all river guides should receive advanced training in wilderness medicine and carry with them the appropriate first aid equipment and supplies. Passengers should be trained before the rafting experience. In addition, it is recommended that the design of the raft be modified to prevent overturning and to determine the effectiveness of introducing new or modified safety equipment for costumers [3].

### Canoeing and Kayaking

Similar acute injuries, as found with whitewater rafting, were reported by canoeists and kayakers and involved sprains/strains, fractures and dislocations, followed by lacerations, contusions and abrasions. However, in these sports, the shoulder and upper extremities were the most common sites of injury instead of the head and lower extremities [3].

### Sailing

Sailing can be dangerous to the musculoskeletal system but the diversity in sailing makes a comparison of data complicated. The most important risk factors in sailing include a lack of general fitness, overuse and traumatic accidents. Many actions in sailing require muscles to perform with powerful and explosive movements, which can lead to injury. Furthermore, awkward postures such as rotation, hyperextension and twisting joints may cause musculoskeletal damage [5]. Regarding traumatic injuries in small boats, Allen and DeJong [6] reported that most injuries affect the shoulder (16%) and lumbar spine (16%), followed by the lower limbs (10%), the cervical spine (8%) and hands (7%). The most common injuries are burns (15.7%), followed by fractures (11%), lacerations (11%), strains, sprains and contusions (9%) from contact with boat hardware and head injuries (6.7%). On the contrary, in boardsailing the most common injured areas are the lower extremities (44.6%), followed by the upper extremities (18.5%), head and neck (17.8%) and trunk (16%). The most common types of injuries are sprains (26.3%), lacerations (21.2%), contusions (16.2%) and fractures (14.2%) [5]. No overuse foot and ankle injuries have been found in literature. To prevent injuries appropriate fitness training and proper care of previous injuries is the best method for all types of sailing because this sport requires aerobic endurance and muscular strength and flexibility. Furthermore, using very tight toe straps and pointed toes puts less strain on the ankle and foot. More

research is needed to develop the design of racing yachts, boats and hardware in order to better prevent injuries [5]. There is no literature regarding programs for returning to sailing after injury, however this should be done gradually.

---

## Rowing, Snorkeling and Diving

Although there is a higher risk of injury at an older age when participating in these water sports, there is a lack of evidence and more research is needed. However, data on the likelihood of injuries due to a lack of consistent training seems to be sufficient to warrant a general correlation between these water sports and injury [7].

No more literature has been found about these three water sports and foot and ankle injuries. However, data on contact dermatitis has been found in literature. Regarding this topic, one of the most frequent causes of dermatitis is the wet suit. In most cases, the diver reacts to some chemical used in the production of the rubber or neoprene material of the suit. Less commonly, other items such as footwear, gloves, snorkels and mouthpieces cause contact dermatitis in divers [8]. Preventive methods include cleaning diving suits with 0.45 % lactic acid after each use and showering immediately after diving [9]. Some other aquatic sports dermatoses are reported as rowing blisters, “pulling boat hands” and “the sailor’s marks” associated with friction in hands [9].

---

## Evidence

Level of evidence: IV, case series. Grade of recommendation: C

---

## Summary

Several water sports have been related to foot and ankle injuries.

Most of these injuries are sprains, strains, contusions and skin injuries and just a few of them are fractures.

Foot and ankle are not the most common areas involved in injuries during water sports except for water skiing.

Data about aquatic sports dermatoses is reported in literature.

There is little literature regarding foot and ankle injuries in water sports so more studies are needed to complete information and to get a higher level of evidence.

---

## References

1. Four Common Water Ski Ankle Injuries [Internet]. 2012 [Cited 4 June 2012]. Available from: <http://www.waterskimag.com/features/2012/06/04/four-common-water-ski-ankle-injuries/>.
2. Haro I. Ankle injuries from water skiing. Haro. Podiatry magazine [Internet]. 7 June 2013 [Cited 7 June 2013]. Available from: <http://haropodiatry.blogspot.com.es/2013/06/ankle-injuries-from-water-skiing.html>.
3. Attarian A, Siderelis C. Injuries in commercial whitewater rafting on the New and Gauley Rivers of West Virginia. *Wilderness Environ Med.* 2013;24(4):309–14.
4. Whisman SA, Hollenhorst SA. Injuries in commercial whitewater rafting. *Clin J Sport Med.* 1999;9(1):18–23.
5. Mitchell T. Smooth sailing. Overuse injuries in sailing and windsurfing [Internet]. 2013 [cited 21 Apr 2013]. Available from: <http://www.working-well.org/articles/pdf/Sailing.pdf>.
6. Allen JB, DeJong MR. Sailing and sports medicine: a literature review. *Br J Sports Med.* 2006;40(7):587–93.
7. Climstein M, Walsh J, Heazlewood I, DeBeliso M, Adams K, Sevene T, Kettunen J. Rowing, soccer and swimming: injury differences (location, type, consequence) in preparation or the world masters games. *J Sci Med Sport.* 2012;15:S127–87. doi:10.1016/j.jsams.2012.11.310.
8. Kockentiet B, Adams B. Contact dermatitis in athletes. *J Am Acad Dermatol.* 2007;56:1048–55.
9. Leventhal J, Tloughan B. Aquatic sports dermatoses: clinical presentation and treatment guidelines [Internet]. New York. Chapter 12. An international perspective on topics in sports medicine and sports injury. [Cited 17 Feb 2012]. Available from: <http://cdn.intechopen.com/pdfs-wm/28451.pdf>.

# Index

- A**
- Abbreviated injury scale (AIS), 508
- Achilles insertional tendinopathy (AIT), 188
- Achilles tendinitis, 528
- aerobics and fitness sports
    - epidemiology, 409–410
    - etiology, 408
    - evidence, 412
    - pathomechanism, 408
    - prevention, 411
    - rehabilitation and back-to-sports, 410
    - therapy, 410
  - ballet dance injuries, 448–449
- Achilles tendinopathy, 423, 525, 554
- back-to-sports, 194
  - chronic, rugby injuries, 517
  - diagnostics, 190
  - elite endurance runners, 188
  - enthesiopathies, 188
  - etiology, 188–189
  - insertional, 192
  - mid-portion tendinopathy, 188, 191–192
  - pathomechanism, 188–189
  - prevention measures, 195
  - rehabilitation, 194
  - symptoms, 189
  - therapy, 191–192
- Achilles tendon disorders
- diagnostics, 189–191
  - etiology, 188–189
  - gastrocnemius and soleus muscles, 187
  - pathomechanism, 188–189
  - prevalence, 187
  - prevention measures, 195
  - rehabilitation, 194–195
  - symptoms, 189
  - tendinopathy (*see* (Achilles tendinopathy))
  - tendon repair (*see* (Achilles tendinopathy))
- Achilles tendon ruptures, 528
- American football, 416–418
  - back-to-sports, 195
  - basketball-related injuries, 431–432
  - conservative treatment, 194
  - Copeland and O'Brien tests, 190
  - cycling, 441, 443–444
  - degenerative changes, 191
  - diagnosis, 190–191
  - etiology, 189
  - inspection and palpation, 190
  - Matles test, 190, 191
  - partial tear patients, 189
  - pathomechanism, 189
  - prevention measures, 195
  - racquet sports, 550, 551
  - rehabilitation, 195
  - rugby injuries, 513, 516–517
  - surgical treatment, 193–194
  - symptoms, 189
  - (Simmonds-) Thompson test, 190
- Achillodynia, 448, 487–488
- Acute ankle injury
- cartilage damage, 157
  - chondral lesions (*see* (Chondral lesions, ankle))
  - osteochondral lesions (*see* (Osteochondral lesions, ankle))
- Acute ankle instability (AAI), 510
- ankle arthroscopy, 253
  - classification, 251
  - diagnosis, 250–251
  - etiology, 249–250
  - evidence, 254–255
  - grades and treatment protocol, 257, 258
  - grading schemes, 251
  - modified Brostrom-Evans procedure, 253
  - modified Brostrom lateral ligament repair, 252–253
  - non-operative management, 252
  - pathomechanism, 249–250
  - post-operative care, 254
  - post-operative rehabilitation protocols, 254
  - prevention methods, 254
  - return to sport, 254
  - risks/complications, 254
  - surgical intervention
    - Brostrom-Gould procedure, 252
    - patient positioning, 252
    - preoperative planning, 252
    - symptoms, 250–251
    - treatment, 252
- Acute compartment syndrome, 171, 174
- Acute exertional compartment syndromes, foot, 172
- Acute fractures
- clinical investigation, 147–148
  - etiology, 147
  - in foot, cycling, 441
  - of forefoot, 153
  - hindfoot
    - calcaneal fractures, 150–151
    - Chopart/Lisfranc joints, 151–152
    - talar, 151
  - history, 147
  - imaging, 148

- Acute fractures (*cont.*)
  - level I and II evidence, 153–154
  - Lisfranc joint, 152–153
  - lower leg and ankle, 148–150
  - in lower limb, 437
  - pathomechanism, 147
  - symptoms, 147
- Acute posteromedial subtalar dislocation, 280
- Aerobics and fitness sports
  - Achilles tendinitis
    - epidemiology, 409–410
    - etiology, 408
    - evidence, 412
    - pathomechanism, 408
    - prevention, 411
    - rehabilitation and back-to-sports, 410
    - therapy, 410
  - ankle sprains
    - epidemiology, 409–410
    - etiology, 408, 409
    - evidence, 412
    - pathomechanism, 408, 409
    - prevention, 411
    - rehabilitation and back-to-sports, 411
    - therapy, 410
  - forefoot injuries
    - epidemiology, 409–410
    - etiology, 408
    - evidence, 412
    - pathomechanism, 408
    - prevention, 411
    - rehabilitation and back-to-sports, 411
    - therapy, 410
  - heel spurs
    - epidemiology, 409–410
    - etiology, 408
    - evidence, 412
    - pathomechanism, 408
    - prevention, 411
    - rehabilitation and back-to-sports, 411
    - therapy, 410
  - plantar fasciitis
    - etiology, 408
    - evidence, 412
    - pathomechanism, 408
    - prevention, 411
    - rehabilitation and back-to-sports, 411
    - therapy, 410
  - shin splints
    - epidemiology, 409–410
    - etiology, 408
    - evidence, 412
    - pathomechanism, 408
    - prevention, 411
    - rehabilitation and back-to-sports, 411
    - therapy, 410
  - stress fracture
    - epidemiology, 409–410
    - etiology, 408
    - evidence, 412
    - pathomechanism, 408
    - prevention, 411
    - rehabilitation and back-to-sports, 411
    - therapy, 410
- Alpine skiing injuries
  - back-to-sports, 529
  - elbow, wrist and hand, 528
  - epidemiology
    - competitive, 529
    - recreational, 529
  - etiology, 527
  - head and spine, 527
  - ISS, 529
  - knee, 528
  - lower extremity, 528
  - lower leg and ankle
    - distal fibula fractures, 528
    - heel injuries, 528
    - small breaches, 528
    - tibial pilon fractures, 528–529
  - pathomechanism, 527
  - prevention, 530
  - rehabilitation, 529
  - shoulder, 528
  - therapy, 529
  - upper extremity, 527
- American Academy of Orthopaedic Surgeons (AAOS) Clinical Practice Guidelines, 432
- American football
  - achilles tendon rupture, 416–418
  - Jones fracture, 414, 415
  - Lisfranc injuries, 414, 416, 417
  - syndesmotric ankle sprains, 417–419
  - turf toe, 413–414
- American Orthopaedic Foot and Ankle Society (AOFAS) hindfoot score
  - Ankle osteoarthritis, 311, 312, 313
  - OCLT, 293, 294, 295
  - posterior impingement syndrome, 448
  - TAR, 313
- Ankle Activity Scale, 312
- Ankle arthroscopy, OCLT, 292
- Ankle-foot orthoses (AFOs), 331
- Ankle osteoarthritis
  - ankle arthrodesis, 316–318
  - atraumatic, 307
  - corrective osteotomies
    - joint-sacrificing, 310
    - opening medial tibial wedge osteotomy, 313
    - realignment surgeries, 310, 311, 312
    - supramalleolar lateral closing wedge osteotomies, 310–311
    - valgus opening wedge supramalleolar osteotomies, 310–311
  - evidence, 322
  - malleolar fractures, 307
  - posttraumatic, 307
  - primary, 307, 308
  - risk factor
    - ankle instability, 309–310
    - sport, 308–309
  - secondary, 308
  - sports activities, in patients, 318–322
  - TAR (*see* (Total ankle replacement (TAR)))
  - treatment, 310
- Ankles
  - fractures, arthroscopy, 76–77
  - injuries, prevention, 143
  - instability, arthroscopy, 77
  - surface anatomy
    - anteroposterior view, 3, 4
    - lateral malleolus, 3, 4, 5



- medial malleolus, 3, 4, 5
  - posteroanterior view, 3, 4
  - superficial peroneal and saphenous nerve
    - branches, 3, 4
  - surgical safety, 3
- surgical approaches
  - anterior ankle approach, 10
  - anterolateral suprafibular approach, 4–6
  - medial approach, 6–9
  - posterolateral approach, 10–12
  - posteromedial approach, 12–13
- syndesmosis, 250, 259, 515–516
- Ankle sprains, 407
  - acute stage
    - cryotherapy, compression., 129, 130
    - orthotic protection, 128, 129
    - precaution, 129
    - PRICE, 128
  - aerobics and fitness sports
    - epidemiology, 409–410
    - etiology, 408, 409
    - evidence, 412
    - pathomechanism, 408, 409
    - prevention, 411
    - rehabilitation and back-to-sports, 411
    - therapy, 410
  - in athletes (*see* Acute ankle instability (AAI))
  - ballet dance injuries, 447–448
  - cross-country skiing, 531
  - cycling, 442–443, 444
  - equestrian sports, 455
  - floorball injuries, 466
  - functional treatment, 127
  - handball injuries, 479, 480
  - hiking, 486
  - ice hockey injuries, 491–492, 494
  - medial ligament injuries, 128
  - orienteering, 510
  - preventive ankle programme, 152
  - rehabilitation stage, 132–135
  - return to sports phase, 135
  - rock climbing, 437
  - running injuries, 524–525
  - snowboarding, 530
  - soccer injuries, 459–460, 463
  - sub-acute stage
    - criteria, 129, 130
    - duration, 129, 130
    - functional stability training, 130, 132–133
    - isometric exercises, 130, 131
    - neuromuscular stimulation, 130, 131
  - surfing, 535, 537, 538
  - syndesmosis injury, 128
  - syndesmotic ankle sprains, 418
  - tissue healing phases, timescale, 128, 129
  - water skiing injuries, 557
- Anterior ankle approach
  - capsulotomy, 10, 13
  - deep dissection, 10, 12, 13
  - hazards, 10
  - indications, 10
  - midline incision, 10, 11
  - position, 10
  - superficial peroneal nerve, 10, 11
- Anterior ankle impingement syndrome
  - ballet dance injuries, 447
  - basketball-related injuries
    - back-to-sports, 434
    - epidemiology, 433–434
    - etiology, 433
    - evidence, 434
    - pathomechanism, 433
    - prevention, 434
    - rehabilitation, 434
    - therapy, 434
  - soccer injuries, 462
- Anterior cruciate ligament (ACL), 528, 531
- Anterior inferior tibiofibular ligament (AITFL), 515
- Anterior talofibular ligament (ATFL), 6, 279, 282, 428, 429, 469, 481, 514, 524
- Anterior tarsal tunnel, 386–387
- Anterior tibial tendon lesion
  - anatomy, 239
  - back to sports, 242
  - conservative treatment, 240
  - diagnostics, 240
  - etiology, 239–240
  - evidence, 242
  - gracilis tendon graft, 241
  - pathomechanism, 239–240
  - rehabilitation, 242
  - surgical treatment, 240–241
  - symptoms, 240
  - tendon rupture, 240
  - therapy, 240
- Anterolateral suprafibular approach
  - deep dissection
    - anterolateral talar dome, 6, 7
    - calcaneocuboid joint, 6, 7
    - distal fibula, 6, 7
    - inferior extensor retinacle, 6, 7
    - subtalar joint, 6, 7
  - hazards, 5
  - indications, 5
  - position, 5
  - superficial dissection, 5–6
- Anteromedial impingement (AMI), 301
- Antibiotic therapy, 487
- AOFAS hindfoot score. *See* American Orthopaedic Foot and Ankle Society (AOFAS) hindfoot score
- Apophysitis
  - Ishelin's disease, 212
  - Sever's disease, 101, 212
- Aquatics. *See* Swimming injury
- Arthritis, 554
- Arthrodesis, subtalar joint degenerative joint
  - disease, 331
- Arthroscopic supplementation, imaging
  - anterior soft tissue, 75
  - bony impingement syndrome, 75
  - Haglund's deformity, 75–76
  - subtalar and posterior tibiotalar joint arthritis, 75–76
- Artistic gymnasts
  - back to sports, 474–477
  - epidemiology, 474
  - etiology, 474
  - injuries, 473
  - pathomechanism, 474
  - prevention, 477
  - rehabilitation, 474–477
  - therapy, 474
- ATFL. *See* Anterior talofibular ligament (ATFL)

- Athletic injuries  
 back-to-sports, 423–424  
 epidemiology, 422–423  
 etiology, 421–422  
 evidence, 424  
 initial approach, 38  
 jumping, 421  
 pathomechanism, 421–422  
 prevention, 424  
 rehabilitation, 423–424  
 running events, 421  
 therapy, 423  
 throwing, 421
- Autologous chondrocyte implantation (ACI), 120, 122, 294  
 Autologous matrix-induced chondrogenesis (AMIC), 293–294  
 Autologous osteochondral transplantation (AOT), 294
- B**
- Ballet dance injuries  
 Achilles tendinitis, 448–449  
 ankle sprain/joint instability, 447–448  
 anterior ankle impingement syndrome, 447  
 epidemiology, 450  
 etiology, 450  
 evidence, 450  
 hallux valgus, 449  
 metatarsal stress fractures, 449  
 osteoarthritis, 449–450  
 pathomechanism, 450  
 posterior impingement syndrome, 448  
 prevention, 450  
 therapy, 450
- Barouk-Osteotomy, 351
- Basketball-related injuries  
 achilles tendon ruptures, 431–432  
 anterior ankle impingement, 433–434  
 incidence, 427  
 Jones fractures, 430–431  
 lateral ankle sprain and instability, 428–429  
 OLTs, 432–433
- Bassett's ligament, 434  
 Baxter's nerve, 387–388  
 Bioadjuvants, 332
- Biomechanics  
 functional imaging  
 analysis of movements, 63  
 kinetic force-time-curves, 72  
 problem-oriented analysis of movement, 64  
 sports-specific demand profiles, 64  
 sports-specific profiles of strains, 64  
 unilateral analysis, 72  
 human walking  
 ankle joint, 28, 29  
 metatarsophalangeal break, 30, 31  
 plantar aponeurosis, 30, 31  
 subtalar joint, 28–29  
 transverse tarsal articulation, 30  
 of running, 31
- Blisters, 528  
 Body mass index (BMI), 163, 221, 428  
 Bone regeneration  
 arthrodesis, 119  
 BMP-2 and BMP-7 clinical trials, 120, 121  
 bone grafting, 119–120  
 bulk allografts, 120  
 recombinant growth factors and proteins, 120
- Bony injuries  
 accessory navicular, 523  
 os trigonum syndrome, 523–524  
 sesamoid injury  
 back-to-sports, 523  
 epidemiology, 522  
 etiology, 522  
 pathomechanism, 522  
 prevention, 523  
 rehabilitation, 523  
 therapy, 522  
 stress fractures  
 back-to-sports, 522  
 epidemiology, 521  
 etiology, 521–522  
 pathomechanism, 521–522  
 prevention, 522  
 rehabilitation, 522  
 therapy, 522
- “Boot-top”-injury, 493  
 Broden view technique, subtalar joints, 280, 331, 545  
 Bruises, 492–493  
 Bunionettes, 368, 369
- C**
- Calcaneal osteotomy  
 conservative treatment, 150  
 corrective and older age, 236  
 Essex Lopresti classification, 150  
 for Haglund's deformity, 216  
 Sanders classification, 150  
 stress fractures, 164  
 surgery, 151  
 tendon ruptures, 558  
 wedge osteotomy, 216
- Calcaneocuboid joint, 283, 284  
 Calcaneofibular ligament (CFL), 6  
 football injuries, 459–460  
 golf injuries, 469  
 handball injuries, 481  
 rugby injuries, 514  
 soccer injuries, 459–460  
 soft tissue injury, 524  
 subtalar instability, 282
- Calcaneonavicular coalition, 342, 343, 344  
 Callosities, 366  
 CAM. *See* Controlled ankle motion (CAM)  
 Canoeing, 558  
 Canyoning, 488  
 Cartilage repair  
 allograft technique, 120, 122  
 injections, 120  
 osteochondral autograft transplantations, 120, 122  
 OLTs, 120  
 pain and global functioning scores, 120
- Cerebral palsy (CP), children  
 diplegia, 115  
 evidence, 116  
 hemiplegia, 115  
 physical activity, 116  
 progressive defect/damage, 115  
 quadriplegia, 115  
 sport, therapy program, 115  
 therapy types

- aquatic therapy, 116
  - hippotherapy, 115, 116
  - physical and occupational therapy, 115
  - strength and fitness training, 115
- CFL. *See* Calcaneo fibular ligament (CFL)
- Cheilectomy, 351, 353
- Chevron/short Scarf-osteotomy, 350, 351
- Children
  - with CP (*see* Cerebral palsy (CP), children)
  - growth and maturation, 97
  - overuse injuries, 97
  - physical exercise, 97
- Chondral lesions, ankle
  - augmented bone marrow stimulation, 159
  - back-to-sports, 159
  - bone marrow stimulation, 159
  - classification, 158
  - conservative treatment, 158–159
  - diagnostics, 158
  - etiology, 157–158
  - evidence, 159
  - fixation, 159
  - pathomechanism, 157–158
  - preventive measures, 159
  - reconstruction technique, 159
  - rehabilitation protocol, 159
  - surgery, 158–159
  - symptoms, 158
- Chopart instability
  - back-to-sports, 284
  - classification, 284
  - conservative treatment, 284
  - diagnosis, 284
  - etiology, 283
  - evidence, 285
  - pathomechanism, 283
  - prevention, 284–285
  - rehabilitation, 284
  - surgery, 284
  - symptoms, 283–284
- Chopart joint degenerative joint disease
  - anatomy, 333
  - back-to-sports, 335
  - classification, 333
  - diagnostics, 333
  - etiology and pathomechanism, 333, 334
  - nonoperative treatment, 333
  - operative treatment
    - approach, 334–335
    - arthrodesis position, 335
    - double arthrodesis, 334
    - fixation, 335
    - talonavicular joint arthrodesis, 334
    - triple arthrodesis, 334
  - prevention, 335
  - rehabilitation, 335
  - symptoms, 333
- Chopart joint dislocation fractures
  - back-to-sports, 152
  - conservative treatment, 151
  - evidence, 152
  - preventive training programs, 152
  - proprioceptive training, 152
  - rehabilitation, 152
  - surgery, 151–152
  - Zwipps classification, 151
- Chronic ankle instability (CAI), 143, 309, 510
  - anatomical reconstruction, 260
  - ankle arthroscopy, 260
  - ankle ligaments, 259
  - anterior pain, 299
  - back to sports, 261–262
  - classification and graduation, 258–259
  - conservative treatment and surgery, 259–260
  - diagnostics, 258–259
  - etiology, 258
  - evidence, 262
  - lateral, 481–482
  - midtarsal, 284
  - operative treatment, 260–261
  - osteochondral lesion, 259
  - pathomechanism, 258
  - rehabilitation, 261–262
- Chronic exertional compartment syndrome (CECS)
  - anterolateral fascial hernias, 173
  - diabetic patients, 171
  - diagnostic criteria, 174
  - elective fasciotomy, 178
  - intramuscular pressure, abnormal variations, 173
  - lower capillary density, 173
  - microtrauma, endurance activities, 171
  - non-operative management, 174–175
  - symptoms, 173
- Chronic overuse injuries
  - elderly athletes
    - osteoarthritis, 107
    - stress fractures, 107
  - in leg
    - CECS, 181
    - MTSS (*see* Shin splints syndrome)
    - “Spike Soreness” in runners, 181
    - tibial stress fracture, 181
- Compartment syndrome
  - acute, 171
  - anatomy, 172
  - anterior and lateral compartment fasciotomy
    - dual-incision lateral approach, 175
    - single-incision lateral approach, 175
  - back-to-sports, 177–178
  - chronic (*see* Chronic exertional compartment syndrome (CECS))
  - classification, 173–174
  - clinical diagnosis, 171, 173
  - complications, 177
  - diagnostics, 173–174
  - differential diagnosis, 174
  - dual-incision technique, 175
  - endoscopically-assisted compartment release, 176
  - evidence, 178
  - incidence, 171
  - non-operative management, 174–175
  - pathomechanism, 172–173
  - perifibular approach, four-compartment fasciotomy, 175–176
  - positioning, 175
  - posteromedial incision, fasciotomy, 176
  - post-operative care, 177
  - preoperative planning, 175
  - prevention, 178
  - rehabilitation protocol, 177–178
  - single incision medial approach, 175, 177
  - surgical treatment, 175
  - symptoms, 173
  - three incision approach, foot, 176–177

- Controlled ankle motion (CAM), 358, 363, 432, 470  
 Crescentic osteotomy, 351  
 Cross-country skiing, 531–532  
 Cycling injuries  
   Achilles tendon, 443–444  
   acute foot, 441  
   ankle, 442–443  
   back-to-sports, 444  
   contusions and skin, 441  
   evidence, 444  
   ingrowing nail, 444  
   metatarsalgia, 443  
   overuse injuries, 441, 442, 443  
   plantar neuropathy, 443  
   prevention, 444  
   rehabilitation, 444  
   skin lesions, 442
- D**  
 Daily adjustable progressive resistant exercises (DAPRE) program, 135  
 Deep peroneal nerve, 386–387  
 Disability sports  
   definition, 111  
   paralympic games, 112  
   prevention, 112  
 Distal tibial transitional fractures  
   Salter-Harris III fractures, 99, 100  
   tillaux fractures, 99, 100  
   triplane fracture  
     triplane I, 99  
     triplane II, 99, 100  
   two planes fracture, 99  
 Distal tibiofibular syndesmosis, 266  
 Dorsal capsulotomy, 368  
 Dorsal intermediate approach  
   deep dissection, 17, 18  
   hazards, 17  
   incision, 17  
   indications, 16  
   position, 17  
   superficial dissection, 17  
 Dorsal intermetatarsal space  
   common digital artery, 21, 22  
   deep dissection, 21, 23  
   extensor digitorum longus tendon, 21, 22  
   hazards, 20  
   incision, 21, 22  
   indications, 20  
   position, 20, 22  
   superficial dissection, 21, 22  
   transverse metatarsal ligament, 21, 23  
 Dorsolateral approach  
   deep dissection, 18, 19  
   hazard, 18  
   incision, 18  
   indications, 17–18  
   position, 18  
   superficial dissection, 18  
 Dorsomedial approach  
   anterior tibial tendon, 15, 16  
   deep dissection, 15, 16, 17  
   extensor hallucis longus tendon, 15, 17  
   hazards, 14  
   incision, 14, 16  
   indications, 13  
   position, 13  
   superficial dissection, 15, 16  
   tarsal-metatarsal joint, 15, 17  
 Double crush syndrome, 379  
 Drop foot syndrome, 67, 240  
 Dynamometry, 67
- E**  
 Elderly athletes. *See* Senior sports  
 Enthesiopathies, 188  
 Equestrian sports  
   back-to-sports, 456  
   disciplines, 453, 454  
   epidemiology, 455  
   etiology, 454, 455  
   evidence, 456–457  
   incidence, 453  
   overuse injuries, 453–454  
   pathomechanism, 454, 455  
   prevention, 456, 457  
   rehabilitation, 456  
   therapy, 456  
 Exostosis  
   apophysites, 212  
   Haglund's deformity (*see* (Haglund's deformity))  
   osteochondrosis, 209–212  
 Extraarticular hallux surgery, 350  
 Extracorporeal shock wave therapy (ESWT), 185, 204, 470
- F**  
 Fascio-cutaneous flaps, 403  
 FHL tendon disorders, athletes  
   anatomy, 243–244  
   biomechanics, 244  
   function, 244  
   history and physical examination, 244  
   imaging, 244–245  
   nonoperative management, 245  
   operative management, 245  
   pathology types, 244  
   prevention, 246  
   rehabilitation, 245–246  
 Fibular stress fracture, 167  
 Fifth metatarsal base fractures  
   avulsions fracture, 152, 153  
   back-to-sports, 153  
   Dameron-Quill classifications, 152–153  
   Jones fracture, 153  
   rehabilitation, 153  
   Torg Classification, 153  
 First metatarsophalangeal joint approach  
   deep dissection, 19, 20–21  
   extensor hallucis longus tendon, 19, 20  
   FHL tendon, 19, 21  
   hazards, 19  
   indications, 18–19  
   position, 19  
   ROM, 41  
   superficial dissection, 19, 20  
 Flatfoot  
   in sport, 220  
   surgery, 224  
 Flexor hallucis longus (FHL) tendon

- injury athlete, 245
- injury ballet dancers, 244
- management athlete, 245
- prevention athlete, 246
- rehabilitation athlete, 245–246
- tendinitis athlete, 244
- tenosynovitis athlete, 244, 245
- Floorball injuries
  - ankle sprains, 466
  - etiology, 466
  - evidence, 467
  - overuse injuries, 466
  - pathomechanism, 466
  - prevention, 465, 467
  - rehabilitation, 467
  - return-to-sports, 467
  - therapy, 466
  - traumatic ankle injuries, 466
- Foot and ankle ability measurement scores, 314, 315
- Foot compartment syndrome (FCS), 172, 173, 177, 178
- Foot Function Index scores, 314
- Forefoot injuries
  - aerobics and fitness sports
    - back-to-sports, 411
    - epidemiology, 409–410
    - etiology, 408
    - evidence, 412
    - pathomechanism, 408
    - prevention, 411
    - rehabilitation, 411
    - therapy, 410
  - classification, 153
  - conservative treatment, 153
  - metatarsal stress fracture
    - back-to-sports, 373
    - conservative treatment, 373
    - diagnostics, 373
    - etiology, 373
    - pathomechanism, 373
    - prevention, 373–374
    - rehabilitation, 373
    - surgery, 373
    - symptoms, 373
  - sesamoid disorders, 374
  - surgery, 153
  - traumatic hallux valgus
    - back to sports, 372
    - classification, 371
    - diagnostics, 371
    - etiology, 371
    - evidence, 372
    - pathomechanism, 371
    - prevention, 372
    - rehabilitation, 372
    - symptoms, 371
    - therapy, 372
  - turf toe
    - back-to-sports, 373
    - classification, 372
    - conservative treatment, 372–373
    - diagnostics, 372
    - etiology, 372
    - evidence, 373
    - pathomechanism, 372
    - prevention, 373
    - rehabilitation, 373
    - surgery treatment, 372–373
    - symptoms, 372
- Freiberg disease
  - back-to-sport, 369
  - back-to-sports, 211
  - classification, 210, 367
  - diagnostics, 210, 366
  - etiology, 210, 365, 366
  - evidence, 211, 370
  - non-operative management, 210–211
  - pathomechanism, 210, 365, 366
  - prevalence, 210
  - preventative measures, 211
  - rehabilitation, 211, 369
  - symptoms, 210, 366
  - therapy, 210–211, 369–370
- G**
- Gait analysis, 64–65
- Gait cycle, 26–27
- Geriatrics. *See also* Senior sports
  - musculoskeletal injury
    - articular cartilage, 106
    - bone structure, 106
    - osteoporosis, 106
    - skeletal muscle, 105–106
    - sports related, 106–108
    - tendons and ligaments, 106
  - physical performance, 106
- Golf injuries
  - back-to-sports, 470
  - epidemiology, 470
  - etiology, 469–470
  - evidence, 471
  - lateral ankle sprains, 469
  - musculoskeletal injury, 469
  - pathomechanism, 469–470
  - plantar fasciitis, 469
  - prevention, 470–471
  - rehabilitation, 470
  - therapy, 470
- Gross motor function classification system (GMFCS), 115, 116
- H**
- Haglund's deformity, 188
  - arthroscopic calcaneoplasty, 215, 216
  - back-to-sports, 216
  - calcaneal osteotomy, 216
  - calcaneoplasty, 215
  - classification
    - calcaneal pitch angle, 213, 214
    - Chaveaux-Liet angle, 213, 214
    - retrocalcaneal bursitis, 213, 214
  - conservative treatment, 214
  - definition, 212
  - diagnostics, 213
  - etiology, 213
  - operative treatment, 214
  - pathomechanism, 213
  - postoperative protocol, 215
  - prevention, 216–217
  - rehabilitation, 216
  - symptoms, 213
- Hallucal sesamoids, 211–212

- Hallux rigidus  
 back-to-sports, 353  
 classification, 349  
 conservative treatment, 351  
 diagnostics, 349  
 etiology, 347, 348  
 evidence, 354  
 minimally invasive surgery, 353  
 pathomechanism, 347, 348  
 prevention, 353–354  
 rehabilitation, 353  
 sports, 348  
 surgical treatment  
   arthrodesis, 352, 353  
   arthroscopic surgery, 351  
   cheilectomy, 351, 353  
   Keller-Brandes procedure, 352  
   OCD, 351  
   periarticular osteotomies, 351  
   silicone hemiarthroplasty (Swanson), 352  
 symptoms, 349
- Hallux valgus  
 back-to-sports, 353  
 ballet dance injuries, 449  
 classification, 349  
 conservative treatment, 350  
 diagnostics, 349  
 etiology, 347–348  
 evidence, 354  
 minimally invasive surgery, 353  
 pathomechanism, 347–348  
 prevention, 353–354  
 rehabilitation, 353  
 rock climbing, 437  
 sports, 348  
 stiffness, 350  
 surgical treatment, 350  
   Chevron/short Scarf-osteotomy, 350, 351  
   extraarticular hallux surgery, 350  
   lapidus-arthrodesis, 351  
   long/classical scarf-osteotomy, 350  
   proximal metatarsal osteotomies, 351, 352  
 symptoms, 349
- Handball injuries  
 ankle sprains, 479  
 back-to-sports, 481  
 epidemiology, 480  
 etiology, 479–480  
 pathomechanism, 479–480  
 prevention, 481–482  
 rehabilitation, 481  
 therapy, 480
- Heel injuries, 528  
 aerobics and fitness sports  
   epidemiology, 409–410  
   etiology, 408  
   evidence, 412  
   pathomechanism, 408  
   prevention, 411  
   rehabilitation and back-to-sports, 411  
   therapy, 410  
 in athletes (*see* (Plantar fasciitis (PF)))
- Hiking  
 acute pathologies  
   ankle sprain, 486  
   blisters, 485–486  
 chronic pathologies  
   achillodynia, 487–488  
   plantar fasciitis, 487–488  
 epidemiology, 485  
 etiology, 485  
 level of evidence, 488  
 paraesthesia, 488  
 pathomechanisms, 485  
 prevention, 488  
 rehabilitation, 488  
 return to sports, 488  
 risk factors, 485
- Hindfoot fractures  
 calcaneal fractures, 150–151  
 Chopart/Lisfranc joints, 151–153  
 talar fractures, 151
- Hippotherapy, 115, 116
- Human gait, 26–28
- Hyperbaric oxygen therapy, 487
- Hypothermia, 485, 488
- I**
- Ice hockey injuries  
 ankle sprains/syndesmosis, 491–492  
 back-to-sports, 494  
 bruises/fractures, 492–493  
 epidemiology, 493  
 etiology, 493  
 evidence, 494  
 pathomechanism, 493  
 prevention, 494  
 rehabilitation, 494  
 skin, 493  
 tendons, 493  
 therapy, 493–494  
 vessels and nerves, 493
- Impingement syndromes  
 anterior ankle  
   back to sports, 303  
   characterization, 299  
   epidemiology, 303  
   etiology, 299–301  
   evidence, 304, 305  
   nonoperative treatment, 303  
   operative treatment, 303  
   pathophysiology, 299–301  
   rehabilitation, 303  
 open surgery, 303  
 posterior ankle  
   aspects of, 301–302  
   back to sports, 303  
   epidemiology, 303  
   etiology, 301  
   evidence, 304, 305  
   nonoperative treatment, 303  
   operative treatment, 303–304  
   pathophysiology, 301  
   rehabilitation, 303
- Incidence of craniocerebral injuries (CCI), 527
- Injured athlete  
 epidemiology, 82  
 initial evaluation, 38  
 legal obligations, 85  
 medical coverage  
   functional examination, 83, 84

- initial assessment, 83
- inspection, 83
- Ottawa foot and ankle rules criteria, 83, 84
- palpation, 83
- patterns, young, 97–98
- primary treatment
  - bone fractures, 85–86
  - dislocated ankle joint, 85
- Injury severity score (ISS), 508, 529
- Injury Surveillance System, 363
- In-line skating injuries
  - back to sports, 498
  - etiology, 498
  - level of evidence, 499
  - pathomechanism, 498
  - prevention, 499
  - rehabilitation, 498
  - therapy, 498
- Insertional Achilles tendinopathy, 192
- Interosseous ligament (IOL), 515
- Interosseous talocalcaneal ligament (ITCL) tears, 78, 279, 282
- Ishelin's disease, 212
  
- J**
- Jones fractures
  - American football, 414
  - avulsions fracture, 153
  - basketball-related injuries, 430–431
  - forefoot adduction, 152
  
- K**
- Kayaking, 558
- Kinematic motion analysis, 26
  - analysis parameters, 66–67
  - 3D motion analysis, 66
  - image-based recording, 65
- Kinetic motion analysis, 26
  - analysis parameters, 68–70
  - dynamometry, 67
  - electromyography, 71
  - force-time-curves, 67, 68, 70
  - pedography, 67–68
  - treadmill analysis widespread, 68, 69
- Kitesurfing
  - back-to-sports, 538
  - epidemiology, 536
  - etiology, 535–536
  - evidence, 538
  - pathomechanism, 535–536
  - prevention, 538
  - rehabilitation, 538
  - therapy, 537
- Kohler's disease, 211
  
- L**
- Lapidus arthrodesis, 350, 351
- Lateral ankle sprain (LAS), 309
- Lateral calcaneal lengthening osteotomy (LCLO), 225
- Lateral ligamentous injuries, rugby, 514–515
- Lateral process of the talus (LPT), 544, 545
- Lateral squeeze test, 384
- Lesser toe deformities, 408–411
- Lisfranc degenerative joint disease
  - anatomy, 335
  - back-to-sports, 339
  - classification, 337–338
  - diagnostics, 337
  - etiology and pathomechanism, 335–337
  - nonoperative treatment, 338
  - operative treatment
    - approach, 338
    - arthrodesis position, 338
    - lateral column arthrodesis, 339
    - least invasive option, 338
    - medial and middle column arthrodesis, 338–339
    - pre-operative, 338
  - prevention, 339
  - rehabilitation, 339
  - symptoms, 337
- Lisfranc instability
  - American football, 414, 416, 417
  - back-to-sports, 286, 288
  - classification, 286, 287
  - conservative treatment, 286
  - diagnosis, 285–286
  - etiology, 285
  - evidence, 288
  - pathomechanism, 285
  - prevention, 288
  - rehabilitation, 286, 288
  - surgery, 286, 287
  - symptoms, 285
- Lisfranc joints, 17, 32, 56, 151, 222
  - dislocation
    - classification, 152
    - conservative treatment, 152
    - fifth metatarsal base fractures, 152–153
    - surgery, 152
  - sprain, 475
- Local plantar instep pedicled flaps, 403
- Long/classical scarf-osteotomy, 350
- Low-energy plantarflexion injury, 283
- Lower leg
  - and ankle fracture
    - back-to-sports, 149
    - classification, 148
    - conservative treatment, 148
    - level of evidence, 150
    - rehabilitation, 149
    - surgery, 148–149
    - trimalleolar, luxated fracture, 148, 149
  - compartment syndrome (*see* (Compartment syndrome))
  
- M**
- Malleolar fractures, 307, 546
- Martial arts injuries
  - back-to-sports, 503
  - epidemiology, 502, 503
  - etiology, 501–502
  - injury patterns, 501–502
  - level V evidence, 503
  - pathomechanism, 501–502
  - prevention, 502
  - rehabilitation, 503
  - therapy, 503
- Matles test, 43, 45
- Matrix-induced autologous chondrocyte implantation (MACI), 294
- Medial ankle ligament injuries, rugby, 517–518

- Medial approach  
 deep dissection, 8, 9  
 hazards, 8  
 indications, 7–8  
 position, 8  
 superficial dissection, 8
- Medial tibial stress syndrome (MTSS), 531. *See also* Shin splints syndrome
- Metacarpophalangeal (MCP) joint  
 alpine skiing injuries, 528  
 Stener's lesion, 531
- Metatarsalgia, 408–411
- Metatarsalphalangeal (MTP) joint  
 back-to-sport, 369  
 classification, 366–367  
 diagnostics, 366–367  
 etiology, 365–366  
 evidence, 369–370  
 hallux rigidus, 348, 349, 351–354, 366  
 hallux valgus, 348–350, 353, 354, 366  
 hyperdorsiflexion of, 392  
 hyperplantarflexion of, 285  
 Morton's neuroma, 392  
 pathomechanism, 365–366  
 prevention, 369  
 rehabilitation, 369  
 symptoms, 366  
 therapy, 367–369  
 traumatic hallux valgus, 371
- Metatarsal (MT) stress fractures, 164–165  
 forefoot sports injuries  
 back-to-sports, 373  
 conservative treatment, 373  
 diagnostics, 373  
 etiology, 373  
 pathomechanism, 373  
 prevention, 373–374  
 rehabilitation, 373  
 surgery, 373  
 symptoms, 373  
 physeal fractures, 101
- Midfoot approaches  
 dermatofasciotomy, 17  
 dorsal intermediate approach, 16–18  
 dorsal intermetatarsal space, 20–23  
 dorsolateral approach, 17–18  
 dorsomedial approach, 13–17  
 first metatarsophalangeal joint approach, 18–20
- Midfoot sprains, 414, 456
- Mid-portion Achilles tendinopathy, 191–192
- Minimally invasive surgery  
 dorsal impingement syndromes, 351  
 hallux rigidus, 353  
 hallux valgus, 353
- Moberg osteotomy, 351
- Morton's neuroma, 47, 107  
 aetiology, 392  
 back-to-sports, 394  
 classification, 392  
 diagnosis, 392–393  
 evidence, 394  
 incidence, 392  
 location, 391  
 nerve entrapments  
 back-to-sports, 385  
 classification, 384  
 conservative treatment, 384–385  
 diagnostics, 384  
 etiology, 384  
 pathomechanism, 384  
 prevention, 385  
 rehabilitation, 385  
 surgery, 384–385  
 symptoms, 384  
 pathomechanism, 392  
 prevention, 394  
 rehabilitation, 394  
 surgical excision of, 443  
 symptoms and signs, 392  
 therapy, 393–394
- Motion analysis  
 kinematics (*see* (Kinematic motion analysis))  
 kinetics (*see* (Kinetic motion analysis))
- Motorsports injuries  
 back-to-sports, 506  
 epidemiology, 506  
 etiology, 505–506  
 foot and ankle fractures, 505  
 Level of evidence, 508  
 ligamentous injuries, 505  
 muscular strains, 505  
 ORIF, 507, 508  
 pathomechanism, 505–506  
 prevention, 506–507  
 rehabilitation, 506  
 therapy, 506
- Mountaineering  
 epidemiology, 485  
 etiology, 485  
 level of evidence, 488  
 paraesthesia, 488  
 pathomechanisms, 485  
 prevention, 488  
 rehabilitation, 488  
 return to sports, 488  
 risk factors, 485
- Mulder's Sign, 443
- N**
- National Electronic Injury Surveillance System (NEISS), 427, 497
- Navicular stress fractures, 165–166
- Negative-pressure wound therapy, 398
- Nerve conduction study (NCV), 174, 385, 470
- Nerve entrapments  
 conservative treatment, 383  
 deep peroneal nerve, 386–387  
 diagnosis, 383  
 first branch of the lateral plantar nerve, 387–388  
 medial plantar nerve, 387  
 Morton's neuroma, 384–385  
 neurogenic symptoms, 383  
 physical examination, 383  
 superficial peroneal nerve, 388–389  
 TTS, 385–386
- Non-life threatening injuries, 456
- Non-steroidal anti-inflammatory drugs (NSAIDs)  
 Baxter's nerve, 388  
 chopart joint DJD, 333



- golf injuries, 470
  - OLTs, 433
  - racquet sports injury, 551
  - subtalar joint DJD, 331
  - turf toe, 358
- O**
- On-field management, 81–82
  - Open reduction and internal fixation (ORIF), 507, 508, 545
  - Orienteering injuries
    - acute ankle instability, 510
    - chronic ankle instability, 510
    - epidemiology, 510
    - etiology, 509
    - evidence, 511
    - pathomechanism, 509
    - prevention, 510
    - therapy, 510
  - Orthobiologics
    - bone
      - arthrodesis, 119
      - BMP-2 and BMP-7 clinical trials, 120, 121
      - bone grafting, 119–120
      - bulk allografts, 120
      - recombinant growth factors and proteins, 120
    - cartilage
      - allograft technique, 120, 122
      - injections, 120
      - osteochondral autograft transplantations, 120, 122
      - OLTs, 120
      - pain and global functioning scores, 120
      - tendon & ligament, 122–124
  - Orthotics, Chopart instability, 285
  - Osteoarthritis (OA)
    - ballet dance injuries, 449–450
    - etiology, 90, 91
    - and muscles, 91–93
    - pathomechanism, 90–91
    - prevalence, 90
    - prophylaxis, 89
  - Osteochondral lesion of the talus (OCLT), 52, 53, 77–78, 99–100, 120, 432–433
    - back-to-sports, 295
    - classification, 292
    - conservative treatment, 292
    - diagnosis, 292
    - etiology, 291–292
    - evidence, 295, 296
    - pathomechanism, 291–292
    - prevention, 295
    - rehabilitation, 295
    - surgery
      - ACI/MACI, 294
      - allograft transplantation, 294
      - AMIC, 293–294
      - AOT, 294
      - autologous bone grafting, 293
      - bone marrow stimulation, 293
      - supplemental procedures, 294–295
    - symptoms, 292
  - Osteochondral lesions
    - ankle
      - augmented bone marrow stimulation, 159
      - back-to-sports, 159
      - bone marrow stimulation, 159
      - classification, 158
      - conservative treatment, 158–159
      - diagnostics, 158
      - etiology, 157–158
      - evidence, 159
      - fixation, 159
      - pathomechanism, 157–158
      - preventive measures, 159
      - rehabilitation protocol, 159
      - surgery, 158–159
      - surgical reconstruction technique, 159
      - symptoms, 158
      - soccer injuries, 460–461
    - Osteochondral talar lesions (OTLs). *See* Osteochondral lesion of the talus (OCLT)
    - Osteochondritis dissecans (OCD), 348, 351, 455
    - Osteochondrosis
      - classification system, 210
      - enchondral ossification, 209
      - etiology, 210
      - Freiberg's infraction, 210–211
      - hallucal sesamoids, 211–212
      - Kohler's disease, 211
      - pathomechanism, 210
    - Osteo-cutaneous composite flaps, 402
    - Osteonecrosis, 151, 210, 211
    - Osteophytes, 300, 448
    - Os trigonum syndrome, 523–524
    - Ottawa foot and ankle rules criteria, 83, 84
    - Overuse injuries, 37–38, 82, 188, 195, 212
      - apophysitis, calcaneus, 101
      - Sever's disease, 101
      - stress fractures, 101–102
      - tarsal coalition, 102
- P**
- Painful bone marrow edema syndrome (PBMES), 54–55
  - Painful os peroneum syndrome (POPS)
    - acute fracture, 233
    - chronic fracture, 233
    - conservative treatment
      - back to sports, 233
      - evidence, 233
      - surgery, 233
    - peroneus longus tendon, 233
  - Paralympics
    - classification, 112
    - limb prosthetics, 113–114
    - sports types, 112
    - wheelchairs
      - basketball, 113
      - racing, 112–113
  - Paratendinitis. *See* Paratenonitis
  - Paratenonitis
    - diagnostics, 190
    - infection, 188
    - long-distance runners, 188
    - rheumatoid arthritis, 188
    - seronegative arthropathies, 188
    - symptoms, 189
    - therapy measures, 192–193

- Pediatric sports injuries
  - ankle sprains, 98
  - distal tibial transitional fractures
    - Salter-Harris III fractures, 99, 100
    - tillaux fractures, 99, 100
    - two/triplane fracture, 99, 100
  - grade IV evidence, 102
  - growth-related problems, 101–102
  - health-promoting benefit, 97
  - intrinsic and extrinsic factors, 98
  - mechanism of trauma, 98–99
  - metatarsal physeal fractures, 101
  - OCD, 99–100
  - overuse injuries, 101–102
  - patterns, athlete, 97–98
  - phalangeal fractures, 101
  - prevention, 102
  - treatment, 97
- Pedicle instep island flap, 403
- Pedography, 67–68, 70
- Periarticular osteotomies, 351
- Permanent medical impairment injuries, 467
- Peroneal subluxation, 236
- Peroneal tendinitis and tenosynovitis
  - etiology, 232
  - pathomechanism, 232
  - symptoms and diagnosis, 232
- Peroneal tendinopathy, 554
  - conservative approach, 233
  - diagnosis, 232
  - extrinsic factors, 232
  - open procedure, 233
  - PRT, 233
  - symptoms, 232
  - tendoscopy, 233
- Peroneal tendonitis, 43–44, 46
- Peroneal tendon tears, 78–79
  - back to sports, 236
  - classifications, 234
  - conservative, 235
  - etiology, 234
  - evidence, 236
  - imaging, 236
  - open surgical procedure, 235
  - pathomechanism, 234
  - provocation tests
    - apprehension test, 236
    - compression test, 236
    - dorsiflexion standing test, 236
  - symptoms, 236
  - tendoscopy, 236
- Peroneus brevis (PB) tears
  - diagnosis, 234
  - etiology, 236
  - incidence, 234
  - pathomechanism, 236
- Peroneus longus (PL) tears
  - diagnosis, 235
  - etiology, 236
  - pathomechanism, 236
  - symptoms, 235
- Pes planovalgus deformity, 221, 222
- Pes planus development, 221
- Phalangeal fractures, 101, 455
- Physical and occupational therapy, 115
- Physical examination
  - ankle exam
    - Achilles tendonitis, 43, 45, 46
    - Achilles tendon rupture, 42–43, 45
    - Achilles tendon vs. gastrocnemius contracture, 43
    - ankle instability, 42
    - anterior ankle impingement, 42, 44, 45
    - anterior impingement, 48
    - external rotation test, 42, 44
    - forced dorsiflexion, 42, 44, 45
    - Matles test, 43, 45
    - neutral dorsiflexion, 42, 43
    - peroneal tendonitis, 43–44, 46
    - Silverskiold test, 43
    - squeeze test, 42, 44
    - Thompson test, 42, 45
  - foot exam
    - Morton's neuroma, 47
    - posterior tibialis tendon dysfunction, 47
    - stress fractures, 45–47
    - tarsal tunnel syndrome, 47
    - turf toe/plantar plate injury, 47
  - gait examination, 41
  - neurovascular exam, 41, 42
  - palpation, 41
  - patient's footwear inspection, 39–41
  - ROM assessment, 41
- Plantar approach, Morton's neuroma, 393
- Plantar fasciitis (PF), 58
  - aerobics and fitness sports
    - etiology, 408
    - evidence, 412
    - pathomechanism, 408
    - prevention, 411
    - rehabilitation and back-to-sports, 411
    - therapy, 410
  - back-to-sports, 205
  - classification, 202
  - diagnostics, 202–203
  - etiology, 201–202
  - evidence, 206
  - golf injuries, 469, 470, 471
  - heel pain, 58
  - hiking, 487
  - incidence, 201
  - morning plantar heel pain, 201
  - overuse injury, 101
  - pathomechanism, 201–202
  - prevention, 205–206
  - PTT dysfunction, 221
  - rehabilitation, 205
  - repetitive stress, 58
  - risk factors, 202
  - soft tissue injury, 525–526
  - strength and flexibility deficit athletes, 30
  - surgical treatment
    - minimally invasive techniques, 205
    - open technique, 205
  - symptoms, 202
  - therapy
    - ESWT, 204
    - foot insoles, 204
    - plantar heel pain management, 203
    - PRP injection, 205
    - treatment recommendation, 203, 204
- Plantar heel pain management, 202, 203
- Plantar neuropathy, cycling, 443
- Platelet-derived growth factor (PDGF), 120
- Platelet rich plasma (PRP), 122, 292, 461, 474

- Posterior impingement syndrome, ballet dance, 448
- Posterior inferior tibiofibular ligament (PITFL), 428
- Posterior talofibular ligament (PTFL), 428
- Posterior tibial tendon insufficiency (PTTI)
- aetiology, 221
  - back-to-sports, 227–228
  - classification, 222, 223, 224
  - degenerative changes, 221
  - diagnostics, 222–223
  - evidence, 228
  - incidence, 221
  - inflammatory process, 221
  - pathomechanism, 221–222
  - physiotherapy protocol, 223
  - plantar calcaneonavicular ligament lesion, 221
  - prevention, 228
  - rehabilitation period, 228
  - spring ligament lesion, 221
  - structured physical therapy program, 227
  - surgical treatment
    - in flatfoot, 224, 225
    - flatfoot deformity correction, 225, 226
    - grade II-IV PTTI, 225, 226
    - spring ligament integrity, 225
    - syndesmotic insufficiency, 227
    - synovectomy, 224
  - symptoms, 222
  - therapy, 223–224
- Posterior tibial tendon (PTT) lesions, 219–221
- aetiology, 219–220
  - chronic insufficiency (*see* (Posterior tibial tendon insufficiency (PTTI)))
  - diagnostics, 220
  - direct/indirect trauma, 219
  - dislocation, 220, 221
  - incidence, 219
  - pathomechanism, 219–220
  - symptoms, 220
  - therapy, 220–221
- Posterolateral approach
- deep dissection, 11–12
  - flexor halux longus muscle, 11, 14
  - hazards, 11
  - indications, 10
  - peroneus brevis tendon, 11, 14
  - position, 11
  - posterior talofibular ligament, 12, 14
  - superficial dissection, 11, 13, 14
  - superior peroneal retinacle, 11, 14
- Posteromedial approach
- Achilles tendon and plantaris tendon, 12, 15
  - deep dissection, 13, 15, 16
  - flexor halluc longus tendon, 13, 16
  - Haglund exostosis, 13, 15
  - hazards, 12
  - incision, 12
  - indications, 12
  - Kager fat pad, 13, 15
  - prone position, 12
  - superficial dissection, 12, 15
- Post-traumatic subtalar arthritis, 329
- Prophylactic ankle support, 135, 143
- Proprioceptive exercises, 129, 132, 135, 136, 185
- Protection, rest, ice, compression and elevation (PRICE) protocol, 85
- Proximal closing wedge osteotomy, 351, 352
- Proximal metatarsal osteotomies
- closing wedge, 351
  - crenscentic osteotomy, 351
  - open wedge, 351
- Proximal open wedge osteotomy, 351, 352
- PTT dysfunction (PTTD). *See* Posterior tibial tendon insufficiency (PTTI)
- ## R
- Racquet sports injury
- achilles tendon ruptures, 550
  - acute extremity injuries, 550
  - chronic injuries, 550
  - epidemiology, 550–551
  - etiology, 550
  - lower extremity injuries, 550
  - pathomechanisms, 550
  - prevention, 551–552
  - rehabilitation, 551–552
  - serious overuse stress injuries., 550
  - therapy, 551
- Radiofrequency thermoneurolysis therapy (RTT), 393
- Rafting, 558
- Range of motion (ROM), 348, 349
- ankle sprains, 41, 448
  - anterior ankle impingement, 448
  - hallux valgus, 449
  - ice hockey injuries, 492
  - MTPJ, 41, 368
  - posterior impingement syndrome, 448
  - subtalar motion, 41
  - turf toe, 362
- Recombinant human bone morphogenetic proteins (rhBMPs), 120
- Regional propeller flaps, 400, 401
- Rest, ice, compression and elevation (RICE) principle, 84, 85
- ankle syndesmotic injuries, 516
  - chopart instability, 284
  - equestrian sports, 456
  - football injuries, 466
  - foot and ankle fractures, 518
  - golf injuries, 470
  - lateral ligamentous injuries, 514
  - lisfranc injuries, 518
  - medial ankle ligament injuries, 517
  - orienteering, 510
  - soccer injuries, 460
  - subtalar instability, 281
  - turf toe, 358
- Retrocalcaneal bursitis, 188
- Return to sports
- agility training stages sports-specific, 135, 141
  - ankle fracture, 135, 140
  - duration, 135, 141
  - exercises, 135, 141–143
  - functional progression, 135
  - phase duration, 135
  - progression criteria, 135, 143
- Rock climbing
- acute injuries, 438
  - chronic injuries, 437, 438, 439
  - epidemiology, 437–438
  - etiology, 437, 438
  - evidence, 439
  - pathomechanism, 437, 438
  - pressure marks, 437, 438
  - prevention, 438–439
  - typical feet injuries, 437
  - typical overstrain injuries, 437

- Roller skating injuries
  - back to sports, 498
  - etiology, 497–498
  - level of evidence, 499
  - pathomechanism, 497–498
  - prevention, 499
  - rehabilitation, 498
- Rugby injuries
  - achilles tendon, 516–517
  - ankle syndesmotric, 513, 515–516
  - chronic achilles tendinopathy, 517
  - foot and ankle fractures, 518
  - lateral ligamentous injuries, 514–515
  - lisfranc injuries, 518
  - medial ankle ligament injuries, 517–518
- Running injuries
  - bone (*see* (bony injuries))
  - soft tissue
    - Achilles tendinopathy, 525
    - ankle sprains, 524–525
    - plantar fasciitis, 525–526
  
- S**
- Sailing, 558–559
- Senior sports. *See also* Geriatrics
  - diagnosis and treatment, 108
  - evidence, 108
  - foot and ankle problems
    - Achilles tendon problems, 107
    - Mortons neuroma, 107
    - PTTD, 107
    - stress fracture, 107–108
  - health care, 105
  - life expectancy, 105
  - medical prevention strategies, 108
  - musculoskeletal changes
    - acute traumatic injuries, 106–107
    - chronic overuse injuries, 107
  - physical performance, 106
  - structural and functional alterations, 105
  - training schedule, 108
- Sesamoiditis, 408–411
  - back-to-sports, 523
  - epidemiology, 522
  - etiology, 522
  - hallucal, 211–212
  - pathomechanism, 522
  - prevention, 523
  - rehabilitation, 523
  - stress fractures, 166–167
  - therapy, 522
- Sever's disease, 212
- Shin splints syndrome
  - aerobics and fitness sports
    - epidemiology, 409–410
    - etiology, 408
    - evidence, 412
    - pathomechanism, 408
    - prevention, 411
    - rehabilitation and back-to-sports, 411
    - therapy, 410
  - back-to-sports, 185
  - bone scintigraphy, 182, 183
  - classification, 182
  - diagnostics, 182
  - etiology, 181–182
  - level of evidence, 186
  - MRI scan, 182, 184
  - pathomechanism, 181–182
  - prevention, 185–186
  - rehabilitation program, 185
  - surgical treatment, 185
  - symptoms, 182
  - treatment modalities
    - cast immobilization, 185
    - exercise program, 185
    - fascial distortion, 185
    - ice fomentation, 185
    - orthotics, 185
    - physiotherapy, 185
    - rest, 184–185
- Silverskiold test, 43
- Skin lesions, cycling, 442
- Slip-catch-mechanism, 528
- Snowboarding injuries, 557–558
  - back-to-sports, 531
  - clinical signs, 545
  - concomitant calcaneus fracture, 546
  - diagnosis, 545
  - disciplines, 542
  - epidemiology, 530–531, 543–545
  - etiology, 530
  - foot, fractures of, 530
  - hard boots and plate bindings, 542
  - LPT, 545
  - malleolar fractures, 546
  - pathomechanism, 530
  - peroneal tendon injuries, 530
  - plate bindings, 543, 544
  - processus lateralis tali, fractures of, 530
  - rehabilitation, 531
  - Slip-in bindings, 542
  - soft strap binding, 543, 544
  - surgery, 546
  - therapy, 530
  - windsurfing straps, 542
- Soccer injuries
  - ankle sprains, 459–460
  - anterior ankle impingement syndrome, 462
  - bruises, 460
  - chondral/osteochondral lesions, 460–461
  - contusions, 460
  - epidemiology, 462–463
  - etiology, 462
  - evidence, 463
  - fractures, 460
  - long-term effects, 459
  - pathomechanism, 462
  - prevention, 463
  - tendon lesions, 461–462
  - therapy, 463
- Soft tissue injury, 454
  - management
    - achilles tendon reconstruction, 400, 402
    - anatomical subunits, 398
    - ankle reconstruction, 400, 401
    - back-to-sports, 403

- classification, 397–398
- composite ankle, 402
- diagnosis, 397–398
- dorsum of foot, 402
- etiology, 397
- evidence level IV, 403
- flap, algorithm for, 400, 401
- fracture and vessel management, 398
- osteocutaneous composite flaps, 402
- pathomechanism, 397
- pressure zones of foot, 399
- principles of, 399
- reconstruction principle, 398
- reconstructive options and evaluation, 399–400
- regional propeller flaps, 400, 401
- rehabilitation, 403
- sole reconstruction, 402–403
- surgical debridement, 398
- timing of reconstruction, 398
- wound conditioning, 398
- running injuries
  - Achilles tendinopathy, 525
  - ankle sprains, 524–525
  - plantar fasciitis, 525–526
- Split skin graft (SSG), 402
- Sport injuries. *See also* Paralympics
  - acute, 37
  - ankle instability, 90
  - in disabled and handicapped people
    - ankle, 115
    - biomechanics, lower extremity, 114
    - children with CP, 115–116
    - lower leg, 115
    - prevalence, 114
    - rehabilitation period, 114
    - risk factors, 114
  - in geriatrics (*see* (Geriatrics))
  - high-risk stress fractures, 37
  - legal obligations, 85
  - OA
    - prevention, 93
    - risk factor, 92
    - treatment, 93
  - overuse injuries, 37–38
  - of pediatric foot (*see* (Pediatric sports injuries))
  - prognostic evidence Level II studies, 93
- Sports biomechanics, 33
- Sports medicine
  - acute athletic injuries, 35
  - biomechanical abnormalities, 36–37
  - epidemiology, 35–36
  - intrinsic and extrinsic risk factors, 36
  - natural grass, 36
  - osseous abnormalities
    - fracture healing, 52, 54
    - osteochondral injuries, 52, 53
    - PBMES, 54–55
    - sesamoid pathology, 51–52
    - stress fractures, 50–51
  - patient care
    - MR imaging, 50
    - Spect and Spect CT, 50
    - ultrasound imaging, 50
    - weight bearing CT scanners, 49
  - shoewear types, 37
  - soft tissue abnormalities
    - ankle ligament injuries, 55
    - Lisfranc injuries, 55–57
    - plantar fasciitis, 58
    - plantar plate injuries, 57–58
    - tendon tears, 55
    - turf toe injuries, 57–58, 59
  - structural abnormalities, 35
  - Squeeze test, 42, 44
  - Strength and fitness training, 115
  - Stress fractures
    - aerobics and fitness sports, 407
    - epidemiology, 409–410
    - etiology, 408
    - evidence, 412
    - pathomechanism, 408
    - prevention, 411
    - rehabilitation and back-to-sports, 411
    - therapy, 410
  - ballet dancers, 162, 165
  - bony injuries
    - back-to-sports, 522
    - epidemiology, 521
    - etiology, 521–522
    - pathomechanism, 521–522
    - prevention, 522
    - rehabilitation, 522
    - therapy, 522
  - calcaneal stress fractures, 164
  - classification, 162–163
  - diagnostics, 162–163
  - etiology, 161–162
  - fibular stress fracture, 167
  - military recruits, 162, 164, 165, 167
  - MT stress fractures, 164–165
  - navicular stress fractures, 165–166
  - pathomechanism, 161–162
  - prevention, 163–164
  - rehabilitation
    - duration, 132, 134
    - early mobilization, 132
    - elastic band exercises, 134, 135
    - exercises, 135, 136–139
    - gait pattern, 132
    - orthosis, 135, 140
    - precautions, 135
    - progression criteria, 132
    - structured physical therapy program, 227
  - runners, 162, 163, 167
  - sesamoid stress fractures, 166–167
  - symptoms, 162
  - treatment, 163
- Subtalar instability
  - back-to-sports, 282
  - conservative treatment, 281–282
  - diagnosis, 280–281
  - etiology, 279–280
  - evidence, 282
  - pathomechanism, 279–280
  - prevention, 282
  - rehabilitation, 282
  - surgery, 281
  - symptoms, 280

- Subtalar joint degenerative joint disease  
 back-to-sports, 332  
 classification, 331  
 diagnostics, 330–331  
 etiology, 329, 330  
 nonoperative treatment, 331  
 operative treatment  
   arthrodesis position, 332  
   arthrodesis site preparation, 332  
   fixation, 332  
   supplemental adjuncts, 332  
   surgical approach, 331–332  
 pathomechanism, 329, 330  
 postoperative care, 332  
 prevention, 332  
 rehabilitation, 332  
 symptoms, 329–330
- Subtalar synovitis, 78
- Superficial peroneal nerve, 388–389
- Superior peroneal retinaculum (SPR)  
 back to sports, 237–238  
 classification, 237  
 dislocation, 236  
 evidence, 238  
 subluxation, 236  
 therapy, 237
- Supination external rotation injuries, 498
- Supramalleolar medial closing wedge osteotomy, 227
- Supramalleolar osteotomy (SMOT), 295
- Surfing  
 back-to-sports, 538  
 epidemiology, 536, 537  
 etiology, 535–536  
 evidence, 538  
 pathomechanism, 535–536  
 prevention, 538  
 rehabilitation, 538  
 therapy, 537
- Surgical biomechanics  
 of ankle fusion, 32  
 ankle ligaments, 33  
 of ankle osteoarthritis, 31  
 of chopart joint fusion, 32  
 chronic ankle instability, 33  
 of forefoot corrections, 32  
 of subtalar joint fusion, 32  
 tendon transfer, 33  
 of total ankle replacement, 31–32
- Swimming injury  
 aquatic therapy, 116  
 back-to-sports, 540  
 epidemiology, 540  
 etiology, 539  
 evidence, 540  
 pathomechanism, 539  
 prevention, 540  
 rehabilitation, 540  
 therapy, 540
- Syndesmosis, 417, 491–492, 508, 515, 516, 532  
 ankle sprains, American football, 417–419  
 athletic injuries, 266  
 back-to-sports, 274  
 classification, 268  
 conservative treatment, 268–269  
 diagnostic arthroscopy, 269–270  
 etiology, 266  
 evidence, 274  
 imaging and arthroscopy, 265, 267–268  
 pathomechanism, 266  
 physical examination, 266–267  
 radiographic assessment, 269  
 reduction technique, 269  
 rehabilitation, 274  
 treatment  
   dynamic suture-button fixation, 272–273  
   screw fixation and removal, 271, 272, 274  
   surgical reduction and stabilization, 270
- Synovitis, 366, 368
- T**
- Talar fractures  
 conservative treatment, 151  
 Hawkins classification, 151  
 hindfoot fractures, 151  
 surgery, 151
- Talocalcaneal coalition, 342, 343, 344
- Talonavicular joint arthritis, 333
- Tarsal coalitions  
 back-to-sports, 345  
 classification, 342–343, 3454  
 diagnosis, 342–343, 344  
 etiology, 341  
 evidence, 345  
 pathomechanism, 341  
 prevention, 345  
 rehabilitation, 345  
 symptoms, 341–342  
 therapy, 343–345  
 TTS, 379
- Tarsal tunnel syndrome (TTS), 47  
 aetiology, 378–379  
 anatomy, 377–378  
 back-to-sports, 381  
 diagnosis, 380  
 nerve entrapments  
   back-to-sports, 386  
   classification, 385  
   conservative treatment, 386  
   diagnostics, 385  
   etiology, 385  
   pathomechanism, 385  
   prevention, 386  
   rehabilitation, 386  
   surgery, 386  
   symptoms, 385  
 presentation, 379–380  
 rehabilitation, 381  
 symptoms, 379–380  
 Tinel's sign, 377  
 treatment, 381
- Tarsometatarsal (TMT) joint. *See* Lisfranc degenerative joint disease
- Tendinosis, 55, 188, 235
- Tendon/ligament regeneration  
 acellular human dermal tissue matrix, 123–124  
 amniotic tissue membranes, 124  
 autografts and allografts, 122–123  
 PRP, 122  
 soccer injuries, 461–462
- Thompson test, 42, 45
- Tinel's sign, 392
- Total ankle replacement (TAR)

- Agility prosthesis, 315
  - ankle arthrodesis, 315, 316
  - AOFAS hindfoot score, 313
  - contraindication, 313
  - failure rate, 313
  - Foot and Ankle Ability Measurement scores, 314, 315
  - Foot and Ankle Outcome Score, 315
  - Foot Function Index scores, 314
  - haemophilia patient, 315
  - ideal candidate for, 313
  - obese patients, 315
  - 3rd-generation prosthesis designs, 313
  - sports activities, in patients, 318, 321, 322
  - survivorship, 313, 318
  - Valderrabano's sports frequency score, 313, 314, 315
  - "Transitional zone" injuries, 333, 335
  - Transverse tarsal injuries, chopart joint degenerative joint disease, 333
  - Traumatic boot-top injuries, 494
  - Traumatic hallux valgus
    - forefoot sports injuries
      - back to sports, 372
      - classification, 371
      - diagnostics, 371
      - etiology, 371
      - evidence, 372
      - pathomechanism, 371
      - prevention, 372
      - rehabilitation, 372
      - symptoms, 371
      - therapy, 372
    - turf toe, 359
  - Treadmill analysis
    - advantages, 64–65
    - EMG, 71
    - with force sensors, 66, 67
    - gait analysis, 64–65
    - in hyper-supination, 68, 69
    - medical diagnostics, 72
    - orthopedic and therapeutic appraisal, 64
  - Turf toe
    - American football, 413–414
    - anatomy, 356
    - classification, 358
    - etiology, 356–357
    - evidence, 363
    - forefoot sports injuries
      - back-to-sports, 373
      - classification, 372
      - conservative treatment, 372–373
      - diagnostics, 372
      - etiology, 372
      - evidence, 373
      - pathomechanism, 372
      - prevention, 373
      - rehabilitation, 373
      - surgery treatment, 372–373
      - symptoms, 372
    - non-operative treatment, 358
    - rehabilitation, 363
    - surgical treatment
      - complications, 363
      - for Grade III injuries, 359
      - medial approach, 359, 360
      - plantar approach, 359–361
      - soft-tissue fixation, 362
      - sutures placement, 359–361
      - traumatic hallux valgus, 359
      - work-up, 357–358
- U**
- Unhealed medial ankle sprain, 228
- V**
- Valderrabano's sports frequency score, 313, 314, 315
  - VISA-A score, 423
  - Visual analog scale (VAS), 312
  - Volleyball specific injuries
    - acute injuries, 553, 554
    - epidemiology, 554
    - etiology, 553–554
    - evidence, 555
    - overuse injuries, therapy for, 554–555
    - pathomechanism, 553–554
    - prevention, 555
    - rehabilitation, 555
    - return to sports, 555
    - stress-injuries, 553
- W**
- Waterman-Green Osteotomy, 351
  - Water skiing injuries
    - ankle fractures, 558
    - ankle sprains, 557
    - calcaneal tendon ruptures, 558
    - talus fractures, lateral process of, 557–558
  - Water sports
    - canoeing, 558
    - diving, 559
    - evidence, 559
    - kayaking, 558
    - rafting, 558
    - rowing, 559
    - sailing, 558–559
    - skiing injuries
      - ankle fractures, 558
      - ankle sprains, 557
      - calcaneal tendon ruptures, 558
      - talus fractures, lateral process of, 557–558
    - snorkeling, 559
  - Weil osteotomy, 367, 368
  - Windsurfing
    - back-to-sports, 538
    - epidemiology, 536
    - etiology, 535–536
    - evidence, 538
    - pathomechanism, 535–536
    - prevention, 538
    - rehabilitation, 538
    - therapy, 537–538