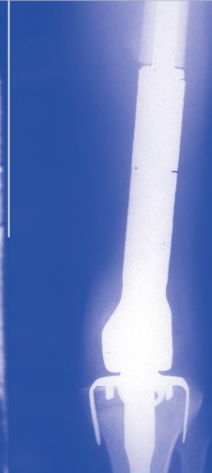
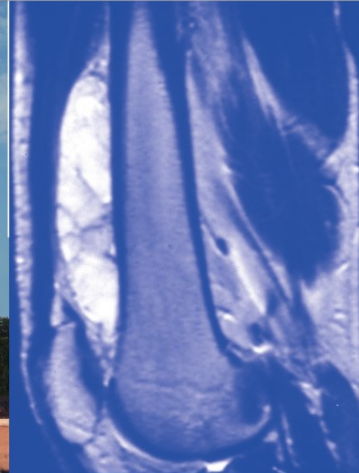


EFORT

IL Book  
Vol. 10 · 2010



# European Instructional Lectures



11th EFORT Congress, Madrid, Spain

*Edited by* George Bentley (UK)

European Federation of National Associations  
of Orthopaedics and Traumatology

# European Instructional Lectures

Volume 10, 2010

# European Federation of National Associations of Orthopaedics and Traumatology

## Committees and Task Forces

### EFORT Executive Committee

#### Executive Board

Prof. Dr. Miklós Szendrői, *President*  
Prof. Dr. Pierre Hoffmeyer, *Vice President*  
Dr. Manuel Cassiano Neves, *Secretary General*  
Prof. Dr. Karl-Göran Thorngren, *Immediate Past President*  
Mr. Stephen R. Cannon, *Treasurer*  
Prof. Dr. Enrique Caceres Palou, *Member at Large*  
Prof. Dr. Maurilio Marcacci, *Member at Large*  
Prof. Dr. Philippe Neyret, *Member at Large*

#### Co-Opted Members

Mr. John Albert  
Prof. Dr. Thierry Bégué  
Prof. Dr. George Bentley, *Past President*  
Prof. Dr. Nikolaus Böhler, *Past President*  
Prof. Dr. Karsten Dreinhöfer  
Prof. Dr. Klaus-Peter Günther  
Prof. Dr. Norbert Haas  
Ass. Prof. Dr. Per Kjaersgaard-Andersen  
Prof. Dr. Karl Knahr  
Prof. Dr. Wolfhart Puhl, *Past President*  
Prof. Dr. Nejat Hakki Sur  
Prof. Dr. Dieter C. Wirtz

### Scientific Coordination 11<sup>th</sup> EFORT Congress, Madrid 2010

#### Chairman

Prof. Dr. Pierre Hoffmeyer

#### Members

Prof. Dr. Enric Caceres Palou  
Dr. Manuel Cassiano Neves  
Dr. José Cordero  
Dr. Francisco Forriol  
Dr. Enrique Gil Garay  
Dr. José Antonio Hernández Hermoso  
Prof. Dr. Pierre Hoffmeyer  
Ass. Prof. Dr. Per Kjaersgaard-Andersen  
Prof. Dr. Philippe Kopylov

Prof. Dr. Maurilio Marcacci  
Prof. Dr. Philippe Neyret  
Mr. Dishan Singh  
Prof. Dr. Miklós Szendrői  
Prof. Dr. Wolfhart Puhl

### Standing Committees

#### EAR Committee

Prof. Dr. Nikolaus Böhler,  
Dr. Gerold Labek

#### Education Committee

Prof. Dr. Maurilio Marcacci

#### EA & L Committee

Prof. Dr. Wolfhart Puhl

#### Finance Committee

Mr. Stephen R. Cannon

#### Health Service Research Committee

Prof. Dr. Karsten Dreinhöfer

#### Portal Steering Committee

Prof. Dr. Klaus-Peter Günther

#### Publishing Committee

Prof. Dr. George Bentley (Books), Prof. Dr. Klaus-Peter  
Günther (Portal), Prof. Dr. Wolfhart Puhl (Journal)

#### Scientific Committee

Prof. Dr. Enrique Caceres Palou

### Task Forces and Ad Hoc Committees

#### Awards & Prizes Committee

Prof. Dr. George Bentley

#### Fora

Prof. Dr. Thierry Bégué

#### Speciality Societies Committee

Dr. George Macheras, Prof. Dr. Pierre Hoffmeyer

#### Travelling & Visiting Fellowships

Prof. Dr. Philippe Neyret

#### Musculoskeletal Trauma Task Force

Prof. Dr. Norbert Haas

European Federation of National Associations  
of Orthopaedics and Traumatology

# European Instructional Lectures

Volume 10, 2010  
11th EFORT Congress, Madrid, Spain

Edited by  
George Bentley



Springer



Prof. Dr. George Bentley  
Royal National Orthopaedic Hospital Trust  
Stanmore, Middlesex  
HA 7 4LP, UK  
george.bentley@efort.org

EFORT Central Office  
Technoparkstrasse 1  
8005 Zürich, Switzerland  
www.efort.org

ISBN: 978-3-642-11831-9            e-ISBN: 978-3-642-11832-6

DOI: 10.1007/978-3-642-11832-6

Springer Dordrecht Heidelberg London New York

Library of Congress Control Number: 2010925107

© EFORT 2010

This work is subject to copyright. All rights are reserved, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilm or in any other way, and storage in data banks. Duplication of this publication or parts thereof is permitted only under the provisions of the German Copyright Law of September 9, 1965, in its current version, and permission for use must always be obtained from Springer. Violations are liable to prosecution under the German Copyright Law.

The use of general descriptive names, registered names, trademarks, 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.

Product liability: The publishers cannot guarantee the accuracy of any information about dosage and application contained in this book. In every individual case the user must check such information by consulting the relevant literature.

*Cover design:* eStudio Calamar, Figueres/Berlin

Printed on acid-free paper

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

# Foreword

It is a privilege for me to introduce this Instructional Lecture book for the 11th EFORT Congress. The format of the Congress combines a National society, SECOT, and the European Federation, EFORT. For this lecture book, the topics selected are original and attractive and will predicate further lectures.

The main goal of EFORT is to convey the latest knowledge of diseases and trauma of the Musculoskeletal system to all European Surgeons. We also wish to welcome colleagues from all over the world, especially from Latin-America and Asia, who are joining us.

The Scientific programme combines multiple aspects of Orthopaedics and Traumatology, paying attention particularly to the latest treatments for infections, metastatic disease, Trauma care and arthroplasty registers, from a European perspective.

Our Instructional Lecturers are from all over Europe, and present topics from diverse areas of interest. These lectures give the opportunity to learn about various diseases but also our speakers' special experience based on their National philosophy - again a chance to widen our European horizons.

As the chairman of the Local Organising Committee. I thank all of our Lecturers for their excellent contributions for publication in this collection. My special thanks go to Professor George Bentley for organising this edition.

EFORT should be congratulated for all its efforts in providing training material for all Orthopaedic surgeons, and particularly for this selection of Instructional Lectures for the Congress in Madrid.

Madrid, Spain

Enric Caceres  
Chairman LOC Madrid 2010

---

## Preface

This 10th Volume of the EFORT European Instructional Lectures completes a series of 10 published since 1993 which cover 240 topics presented at the past 11 Congresses.

The topics reflect the current thinking and latest advances in Orthopaedics and Traumatology, written by specialists from across Europe, who bring a perspective unique to their own Country and personal practice.

All those involved in Orthopaedics and Traumatology can benefit from this book.

Especial thanks go to all the authors, not only for preparing and presenting the lectures, but for their other activities, such as reviewing and chairing Symposia and free paper sessions, without which the programme could not be completed.

As before, the preparation of the volume has been by the Internationally-recognised publishing Springer team, led by Gabriele Schroeder, to whom we are very grateful. My personal thanks go to Larissa Welti and the Central Office staff who have given unfailing and expert support.

This is the volume of the first decade of Instructional Lectures and is dedicated to all previous editors who have contributed their expertise, in particular Jacques Duparc, Pierre-Paul Castelyn, Philip Fulford, John Kenwright, Roli Jakob, Frank Horan, Karl-Goran Thorngren, Panyotis Soucacos, Jame Scott, Roger Lemaire, Richard Villar and Vitas Khanduja.

Stanmore, UK

George Bentley

# Contents

## General Orthopaedics

- Necrotising Fasciitis** ..... 3  
Nikolaos K. Kanakaris, Jas Tan, Mark I. Liddington,  
and Peter V. Giannoudis
- Trauma: Non-Union: New Trends** ..... 15  
Gerald Zimmermann and Arash Moghaddam

## Bone and Joint Tumours

- Bone Tumours: Work Up 2009** ..... 23  
I.M. Noebauer-Huhmann, J. Panotopoulos, and R.I. Kotz
- Diagnosis and Treatment of Soft Tissue Sarcomas** ..... 37  
Miklós Szendrői, Zoltan Sápi, Kinga Karlinger, and Zsuzsa Pápai

## Paediatrics

- Management of Lower Limb Deformities** ..... 53  
Franz Grill, Rudolf Ganger, and Christof Radler
- Total Hip Replacement in the Congenitally Dislocated  
Hip Using the Paavilainen Technique** ..... 61  
Bjørn Thorup, Inger Mechlenburg, and Kjeld Søballe

## Trauma

- Operative Strategy for Fracture-Dislocation of the Elbow** ..... 69  
Konrad Mader
- Fractures of the Scaphoid: Diagnosis and Management** ..... 79  
José Carlos Botelho



**Spine**

- Treatment of Thoraco-Lumbar Fractures** ..... 87  
Antonio A. Faundez
- The Rheumatoid Cervical Spine** ..... 97  
Zdenek Klezl, Girish N. Swamy, and Jan Stulik

**Upper Limb**

- Partial Rotator Cuff Ruptures** ..... 109  
Antonio Cartucho
- Massive Tears of the Rotator Cuff** ..... 121  
Fernando Marco and Carlos García-Fernández

**Hip**

- Hip Resurfacing** ..... 133  
Derek McMinn, Joseph Daniel, Hena Ziaee,  
and Chandra Pradhan

**Knee**

- Total Knee Arthroplasty in Extra-Articular Deformities** ..... 145  
José A. Hernández-Hermoso
- Unicompartmental Knee Arthroplasty** ..... 159  
Simon Thomas Donell
- Isolated Patellofemoral Osteoarthritis** ..... 167  
Ferran Montserrat

**Foot and Ankle**

- Achilles Tendinopathy** ..... 191  
Nicola Maffulli and Francesco Oliva
- The Painful Flatfoot** ..... 201  
Victor Valderrabano and Martin Wiewiorski

## Contributors

**José Carlos Botelho** Hand Surgery Unit, Hospital de Sant' Ana,  
Rua de Benguela, 2779-501 Parede, Portugal, jcbotelho@gmail.com

**Antonio Cartucho** Hospital Cuf Descobertas, R. Mário Botas, 1998-018 Lisbon,  
Portugal, antonio.carvalhopereira@jmellosaude.pt

**Joseph Daniel** The McMinn Centre, 25 Highfield Road, Edgbaston Birmingham,  
B15 3DP West Midlands, UK, mr.jdaniel@yahoo.co.uk

**Simon Donell** Norfolk and Norwich University Hospital, Colney Lane, Colney,  
Norwich NR4 7UY, UK, simon.donell@nnuh.nhs.uk

**Antonio Faundez** HUG - Hôpitaux Universitaires de Genève,  
24, Rue Micheli-du-Crest, 1211, Geneva, Switzerland, antonio.faundez@hcuge.ch

**Rudolf Ganger** Department for Paediatric Orthopaedics,  
Orthopädisches Spital Speising, Speisingerstrasse 109, 1130, Vienna, Austria,  
rganger@aon.at

**Carlos Garcia-Fernandez** Servicio de Traumatología,  
Hospital Clinico San Carlos, Martin Lagos s/n, 28040 Madrid, Spain

**Peter Giannoudis** Academic Department of Trauma and Orthopaedics,  
Leeds General Infirmary, Clarendon wing, Great George Street,  
LS13EX, Leeds, West Yorkshire, UK, pgiannoudi@aol.com

**Franz Grill** Department for Paediatric Orthopaedics, Orthopädisches Spital  
Speising, Speisingerstrasse 109, 1130 Vienna, Austria, franz.grill@oss.at

**Jose Hernandez-Hermoso** Orthopaedic Department, Terrassa Hospital,  
Carretera Torre Bonica s/n, 08227 Barcelona, Spain, jahernandez@cst.cat

**Nikolaos K. Kanakaris** Academic Department of Trauma and Orthopaedics,  
Leeds General Infirmary, Clarendon wing, Great George Street,  
LS13EX, Leeds, West Yorkshire, UK, nikolaskanakaris@yahoo.co.uk

**Kinga Karlinger** Department of Diagnostic Radiology and Oncotherapy,  
Semmelweis University Budapest, Karolina ut 27, 1113, Budapest, Hungary

**Zdenek Klezl** Department of Trauma and Orthopaedis, Derby Hospitals NHS  
Foundation Trust, Uttoxeter Road, Derby DE22 3NE, UK, zklezl@aospine.org

**Rainer Kotz** Department of Orthopaedics, Medizinische Universität Wien, Währinger Gürtel 18-20, 1090, Vienna, Austria, rainer.kotz@meduniwien.ac.at

**Mark I. Liddington** Department of Plastic Surgery, Leeds General Infirmary, Clarendon wing, Great George Street, LS13EX, Leeds, UK

**Konrad Mader** Ortopedisk Avdeling, Seksjon Traumekirurgi/ Håndkirurgi, Førde Sentralsjukehuset, 6807 Førde, Norway, konrad.mader@helse-forde.no

**Nicola Maffulli** Centre Lead and Professor of Sports and Exercise Medicine, Consultant Trauma and Orthopaedic Surgeon, Queen Mary University of London, Barts and The London School of Medicine and Dentistry, Institute of Health Sciences Education, Centre for Sports and Exercise Medicine, Mile End Hospital, 275 Bancroft Road, London E1 4DG, England, n.maffulli@qmul.ac.uk

**Fernando Marco** Professor of Orthopaedic Surgery, Chief Shoulder Surgery Service, Department of Orthopaedic Surgery, Clínico San Carlos Hospital, Complutense University of Madrid, Spain, fermarco@hotmail.com

**Derek McMinn** The McMinn Centre, 25 Highfield Road, Edgbaston Birmingham, B15 3DP West Midlands, UK, derekmcminn@mcminncentre.co.uk

**Inger Mechlenburg** Department of Orthopaedics, Aarhus University Hospital, Aarhus, Denmark

**Arash Moghaddam** BG Unfallklinik Ludwigshafen, Department of Traumatology and Orthopedics, Ludwig-Guttman Strasse-13, 67071 Ludwigshafen, Germany

**Ferran Montserrat** IMAS, Passeig Sant Gervasi 16-20, 08022, Barcelona, Spain, drfmontserrat@pulso.com

**Iris M. Noebauer-Huhmann** Department of Radiology, Medizinische Universität Wien, Währinger Gürtel 18-20, 1090 Vienna, Austria, iris.noebauer@akhwien.at

**Francesco Oliva** Centre for Sports and Exercise Medicine, Queen Mary University of London, Barts and The London School of Medicine and Dentistry, Mile End Hospital, 275 Bancroft Road, London E1 4DG, UK

**Joannis Panotopoulos** Department of Orthopaedics, Medizinische Universität Wien, Währinger Gürtel 18-20, 1090 Vienna, Austria

**Zsuzsa Papai** State Health Center, Semmelweis University Budapest, Karolina ut 27, 1113, Budapest, Hungary

**Chandra Pradhan** The McMinn Centre, 25 Highfield Road, Edgbaston Birmingham, B15 3DP West Midlands, UK

**Christof Radler** Department for Paediatric Orthopaedics, Orthopädisches Spital Speising, Speisingerstrasse 109, 1130, Vienna, Austria, cradler@chello.at

**Zoltan Sapi** Department of Pathology and Experimental Cancer Research, Semmelweis University Budapest, Karolina ut 27, 1113, Budapest, Hungary

**Kjeld Søballe** Department of orthopaedic surgery, University hospital of Aarhus, Denmark, kjeld@soballe.com

**Jan Stulik** Spine Surgery Department, Teaching Hospital Pragu Motol,  
Prague 5, Czech Republic

**Girish N. Swamy** Derby Hospitals NHS Foundation Trust, Uttoxeter Road,  
Derby DE22 3NE, UK, grishjam@yahoo.com

**Miklós Szendrői** Semmelweis University Budapest, Karolina ut 27,  
1113, Budapest, Hungary, szenmik@orto.sote.hu

**Jas Tan** Department of Plastic Surgery, Leeds General Infirmary, Clarendon wing,  
Great George Street, LS13EX, Leeds, UK

**Bjørn Thorup** Department of Orthopaedics, Aarhus University Hospital,  
Aarhus, Denmark

**Victor Valderrabano** Universitätsspital Basel, Spitalstrasse 21/ Petersgraben 4,  
4031 Basel, Switzerland, vvalderrabano@uhbs.ch

**Hena Ziaee** The McMinn Centre, 25 Highfield Road, Edgbaston Birmingham,  
B15 3DP West Midlands, UK

**Gerald Zimmermann** Theresienkrankenhaus Mannheim,  
Department of Trauma Surgery, Bassermannstrasse 1, 68165 Mannheim,  
Germany, g.zimmermann@theresienkrankenhaus.de

**Part I**

---

**General Orthopaedics**

# Necrotising Fasciitis

Nikolaos K. Kanakaris, Jas Tan, Mark I. Liddington, and Peter V. Giannoudis

## Abbreviations

ARDS	Acute respiratory distress syndrome
Atm	Atmospheres
CK	Creatine kinase
CRP	C-reactive protein
CT-scan	Computed tomography scan
ESR	Erythrocyte sedimentation rate
Hb	Haemoglobin
HBO	Hyperbaric oxygen therapy
IL-1/6	Interleukin 1/6
IV	Intravenous
IVIG	Intravenous immunoglobulin G
LFTs	Liver function tests
LRINEC	Laboratory risk indicator for necrotising fasciitis
MOF	Multiple organ failure
MRI	Magnetic resonance imaging
MRSA	Methicillin-resistant <i>Staphylococcus aureus</i>
NSTI	Necrotising soft tissue infection
SIRS	Systemic inflammatory response syndrome
TNF- $\alpha$	Tumour necrosis factor – alpha
WBC	White blood cells

## Definition

The term ‘Necrotising Fasciitis’ was initially used in 1952 by Wilson [64] to describe an inflammatory disease causing necrosis of the fascia planes and the subcutaneous fat with devastating results caused by several microbes and located at any anatomical site. Hippocrates described this

condition in the fifth century BC “many were attacked by the erysipelas all over the body when the exciting cause was a trivial accident... flesh, sinews, and bones fell away in large quantities... there were many deaths” [15].

Since then a number of authors used a variety of names; i.e. “hospital” gangrene, “Fournier” gangrene, phagedema, phagedema gangrenosum, progressive bacterial synergistic gangrene, non-clostridial gas gangrene, “flesh-eating bacterium”, necrotising soft-tissue infection (NSTI) [2, 58]; portraying this rare life-threatening infection.

## Aetiology

Typically an injury proximal to the affected anatomical site precedes the development of this infection. The severity of this local trauma may be minimal and there are reported cases associated with minor blunt, penetrating trauma or burns, surgical incisions, peri-rectal abscesses, childbirth or even chicken pox. In a number of cases no definite portal of entry is identified in the patients’ skin or positive traumatic event in their history.

The responsible micro-organisms vary significantly and may include aerobic/anaerobic, gram positive/negative, or even fungi (*Candida*, *Aspergillus*, *Rhizopus* species) [24]. Recently, community-acquired methicillin-resistant *Staphylococcus aureus* (MRSA)-related necrotising fasciitis has been described in relatively high proportions [28, 42]. It is reported that MRSA is currently cultured in 40% of necrotic wounds particularly in IV drug abusers, athletes and institutionalised groups of patients [36, 40, 42]. A classification related to the cultured bacteria and the Gram stain has been described [6] (Table 1).

## Epidemiology

According to recent reports there are almost 1,000 cases of necrotising fasciitis per year in the United States, or 0.4–0.53 cases per 100,000 population [52, 61, 62]. The prevalence of

P.V. Giannoudis (✉)

Academic Department of Trauma and Orthopaedics,  
Leeds General Infirmary, Clarendon wing,  
Great George Street, LS13EX, Leeds,  
West Yorkshire, UK  
e-mail: pgiannoudi@aol.com

**Table 1** Classification of Necrotising Fasciitis according to the microbial cause [6]

	Type 1 [24]	Type 2 [12, 24]	Type 3 [24, 38]
Aetiology	Polymicrobial (4–5 species) ( <i>Gram-positive cocci</i> , <i>Gram-negative rods</i> , <i>anaerobes</i> )	Monomicrobial ( <i>Streptococcus pyogenes</i> , <i>Staphylococcus</i> , <i>Clostridia</i> , <i>MRSA</i> , or <i>other species</i> )	Marine vibrios, Gram negative rods ( <i>Vibrio Vulnificus</i> %, <i>Klebsiella</i> , <i>Escherichia coli</i> , etc)
Incidence (%)	55–75	8–10	2–5
Anatomical site	Abdominal – perineal wounds	“Flesh eating” of extremities – toxic shock	Small wound from fish/cut/insect bite extremities
Epidemiology	Immunocompromised, diabetic patients	Healthy patients	Liver disease, chronic Hepatitis-B
Prognosis	High mortality	Moderate mortality	Highest mortality

this disease is such that the average clinician could not be anticipated to gain adequate experience in his usual practice.

For reasons yet undetermined the incidence has increased the last 2 decades. Possible explanations could be the increased microbial virulence and resistance due to the excessive universal use of antibiotics, the increased clinical awareness and the establishment of international reporting pathways [51].

In a recent systematic review [3] of the existing evidence male-to-female ratio was 2/1 with an age range from 5 to 88 years (average 45 years). The lower extremities are the most commonly affected anatomical site, with the perineum following the upper extremities and virtually any other body part in smaller numbers [6, 37].

## Risk Factors

Host conditions related to immune deficiency are considered as risk factors for necrotising fasciitis. Diabetes mellitus is present in 18–60% of the cases [44, 48], whilst other risk factors include obesity [59], peripheral vascular disease [12], intravenous drug/alcohol use [12, 34], malnutrition [16], smoking [12], chronic cardiac disease [12, 54], the continued or chronic use of non-steroidal anti-inflammatory drugs [4, 56], chronic corticosteroid therapy [22], cancer, and old age [10, 13].

Besides the numerous co-morbidities statistically related to an increased risk for necrotising fasciitis, it should be emphasised that 20–50% of all cases are previously healthy individuals [6, 16].

## Pathophysiology

Following mostly external trauma or more rarely direct spread from perforated visceral organs (lower gastro-intestinal or urogenital tract) microbes invade the

subcutaneous tissues. The secretion of endo- and exotoxins is followed by tissue ischaemia and necrosis. Thrombosis of the perforating vessels to the skin is the resulting key feature of necrotic fasciitis, which declares itself gradually by an increasing necrotic subcutaneous and skin lesion.

Infection can spread locally rapidly (1 cm/h) without major skin findings at the early stages. The speed of the pathologic sequel is depending on the microbes' characteristics, the local biology, and the defences of the host. The rare nowadays *Clostridium* species produce an  $\alpha$ -toxin that causes extensive local necrosis and systemic shock. *Staphylococcus aureus* and *Streptococci* species express surface proteins and toxins (M-1, M-3, exotoxins A-B-C, streptolysin O, and superantigen) that allow them to adhere to the host tissues, escape the defence mechanism of phagocytosis, cause damage to the endothelium, resulting in tissue oedema and impairment of the local blood flow. The stimulation of the defence mechanisms (CD4 cells and macrophages) and the production of cytokines of the acute phase in large quantities (TNF- $\alpha$ , IL-1, and IL-6) leads to a systemic inflammatory response (SIRS), and/or septic shock, and/or multisystem organ dysfunction, and in some cases eventually to death. The secretion of the cytokines (TNF- $\alpha$ ) interacts also with the vascular endothelium, stimulating the neutrophil degranulation, activating the coagulation cascade (complement, bradykinin/kallikrein system), promoting small vessel thrombosis (due to local hypercoagulable state, platelet-neutrophil plugging of small vessels, and the increased interstitial pressure). Thus, tissue perfusion, capillary blood flow, and subsequently local distribution of the antibiotics are all diminished, explaining the mechanism of ischaemic necrosis and the ineffectiveness of antibiotic therapy alone [9, 25, 51].

## Diagnosis

The diagnosis should be based principally to the clinical pre- and mostly intra-operative findings. Any delay caused in order to attain radiological or laboratory verification

may be detrimental, and surgical debridement should be performed early together with intravenous antibiotic therapy.

### Clinical Presentation

The typical presentation of necrotising fasciitis usually begins with a slightly inflamed area of soft tissue that rapidly advances to fasciitis combined with systemic toxæmia. In the first stages, the subtle clinical findings may be mistaken for simple cellulitis. In 20–40% of the cases there is a clear history of trauma or a break of skin within 48 h from the onset of symptoms. A high index of suspicion at these early stages may be critical for the subsequent outcome.

In this phase of acute inflammation, excessive and disproportionate to the local findings, pain and tenderness to palpation (in almost all cases), and rapid expansion (up to >1 cm/h) of non-specific skin findings (swelling, warmth, erythema, “wooden” skin) should alert the clinician to the seriousness of the patient’s condition [52, 56, 65].

As the disease progresses more classic signs develop including blisters and cysts with serosanguinous or hemorrhagic fluid. The skin passes stages from discoloration to black necrotic sloughing eschars, surrounded by rapidly-increasing oedema. In the presence of gas (depending on the type of the responsible bacteria) crepitus may develop. Analgesia over the necrotic skin areas is typical accompanying the destruction of cutaneous nerves. The subsequent necrosis of the fat and fascia produces the characteristic discharge of a greyish-watery foul-smelling purulent fluid [24].

Gradually the patient develops an escalating systemic state that leads to septic shock in over 30% of the cases. Fever-chills >38.5°C (59%), tachycardia, hypotension, decrease in the level of consciousness, accompanied by bacteraemia (50%), acute renal failure (35%), coagulopathy (29%), acute respiratory distress syndrome (14%) compose the cardinal features of a rapidly developing multiple organ failure and a rapidly deteriorating critically-ill patient [30, 32, 50].

The absence of any of the above symptoms may occur and should not mislead the clinician, who should follow the patient closely, and be alerted by the presence of disproportionate pain to the “cellulitic lesion”, and the rapid deterioration of the general condition [6, 68]. In fulminant cases (mostly type 3 – Table 1) it may be that cardiovascular collapse precedes the extensive soft tissue and skin changes. The reported average time from the first signs and symptoms to the diagnosis of the disease and the escalation of the clinical presentation varies from 2 to 4 days while in rare cases may take weeks [67].

### Laboratory Evaluation

The relevant laboratory evaluation includes a full blood count, biochemistry panel as well as liver function tests, and coagulation studies. Blood cultures should be always taken, positive in half of the cases, and following the sensitivity tests they guide the antibiotic therapy. In the presence of septic shock arterial blood gases are needed to monitor the acid-base balance and respiratory function [10]. Frequent other findings, dependent on the phase of the sepsis and the immune response of the host, are electrolyte deficits (Na, K), increase of creatine kinase, hypoalbuminaemia, hypertransaminasaemia, thrombocytopenia, anaemia, raised ESR and CRP. The progress of the observed laboratory parameters reflects the decline of the patient’s condition, or the successful response to the antibiotics and surgical interventions.

In 2000 Wall et al. [63] described a diagnostic model able to exclude in most cases the presence of necrotising fasciitis (sensitivity – 90%, specificity – 76%, poor predictive value – 26%). Simonart et al. [56] proposed that CRP levels >15 mg/dL have a sensitivity of 89% and specificity of 90%, while elevation of CK levels >600 U/L are highly specific (95%) for necrotising fasciitis in contrast to plain cellulitis.

In the same year (2004), the Laboratory Risk Indicator for Necrotising fasciitis (LRINEC) was developed (Table 2). It incorporates parameters such as the CRP, WBC, Hb, serum Sodium, Creatinine, and Glucose levels to distinguish the rare necrotising fasciitis cases from other

**Table 2** Laboratory risk indicator for necrotising fasciitis score [66]

Variables	Value	LRINEC score
CRP	<150	0
	≥150	4
WBC (cells/mm <sup>3</sup> )	<15	0
	15–25	1
	>25	2
Hemoglobin (g/dL)	>13.5	0
	11–13.5	1
	<11	2
Sodium (mmol/L)	≥135	0
	<135	2
Creatinine (µg/L)	≤141	0
	>141	2
Glucose (mmol/L)	≤10	0
	>10	1



more frequent soft tissue infections. The summation of the sub-scores varies between 0 and 13. Above 6 (cut-off point) the probability of necrotising fasciitis is >50%, while the overall positive and negative predictive values are 92% and 96% respectively [66].

### Radiologic Evaluation

The only related finding on plain X-rays is the presence of gas in the subcutaneous soft tissues after the necrosis has progressed significantly and only in a number of cases. Ultrasonography, a useful diagnostic tool for abscesses and cellulitis, is not considered sensitive or specific enough for differentiating necrotising fasciitis [39].

CT-scanning is more sensitive as it can identify the pathological signal of the affected subcutaneous fat and deep fascia in more than 80% of all necrotising fasciitis cases, as well as thickening and increased enhancement of the affected tissue planes, subcutaneous gas collection, and soft tissue oedema defining accurately the extent of the disease [5, 67]. Nevertheless, there are described cases with false negative CT-scan results reported [52, 68].

MRI scans are significantly more sensitive (>93%) even in the early stages of necrotising fasciitis, while specificity is quite low (50–85%). T2-weighted images detect the thickening of soft tissues followed by high signal of necrotic tissue and the fluid accumulation following the liquefaction of subcutaneous fat and fascia layers. Contrast-enhanced T1-weighted images detect the peripheral enhancement at the margins of the lesion as well as the oedema of the deep fascial planes [21, 68]. Nevertheless, an MRI scan may be impractical for critically-ill or unstable patients and should not delay the delivery of the necessary surgical treatment.

### Histologic Evaluation

Intra-operative biopsies provide the confirmatory diagnosis and are considered as the gold standard diagnostic modality. There is no role for culturing superficial skin lesions and blisters as the infection tracks at the subcutaneous level.

The “finger test” is a procedure that can be used for pre-operative tissue-based diagnosis under local anesthesia. Through a small skin incision a gloved finger is inserted down to the deep fascia under sterile conditions. Drainage of “dishwater” pus, absence of bleeding, and lack of tissue resistance to the insertion of the finger are considered as

positive findings. At the same time a frozen-section biopsy can be taken via a small elliptical section of skin-fat-fascia of the suspected area, as well from one at the periphery of the affected soft tissues.

Nevertheless, it should be emphasised that formal surgical debridement and an open biopsy is preferable whenever the patient is unstable, or the clinical suspicion strong. Attempts of gaining more proof regarding the establishment of a certain diagnosis and delaying surgical intervention should not be followed in the case of necrotising fasciitis due to its rapid escalation and detrimental effects locally and systemically.

A gram stain, microscopy analysis and a culture are able to provide early verification of the clinical suspicion as long as an experienced pathologist is available to review the samples [2]. Typical histopathological findings are the necrosis of the superficial fascia, subcutaneous fat and nerves with thrombosis and suppuration of the vessels, mixed inflammatory-cell infiltration and early fibroblast proliferation. At the early stages superficial epidermal hyaline necrosis, dermal oedema, polymorphonuclear infiltration and obliterative vasculitis is followed by thrombosis of penetrating fascial-to-skin vessels, and later by liquefactive necrosis of all tissue layers and the production of a dense predominantly neutrophilic infiltrate [12, 21, 57, 68].

---

### Differential Diagnosis

This disease or clinical syndrome (due to the array of its infectious aetiology), especially at its early stages, has mostly non-specific symptoms and findings. Due to the rapid escalation of events and the severity of its prognosis, the prompt differential diagnosis and subsequent immediate surgical debridement is of paramount importance.

In the early stages the most likely diagnosis is that of cellulitis, which is much more frequent, has identical skin findings (erythema, swelling), but in contrast only mild pain/tenderness and normal-looking subcutaneous fat and fascia.

Myonecrosis should be also considered in those cases that infection is excluded following the debridement and the microbiology cultures. Comparatively, it affects deeper layers of the soft tissues limited to the muscle groups. Eosinophilic fasciitis, an even more rare entity, has a more chronic course, affects also the fascia layer, is sterile and responds to steroids. Lymphoedema, or Myxoedema are easier to distinguish by the absence of systemic findings and the history of hypothyroidism respectively.

---

## Management

Successful management of this difficult clinical condition requires a multidisciplinary approach, close collaboration of the surgeon and the intensivist, and mostly prompt action and constant follow-up.

### Surgical Debridement

The effective minimisation of the bacterial load is achieved only by a thorough surgical debridement. This is essential in the attempt to stop the necrotic process and represents the cornerstone of necrotising fasciitis management. It has been proven to increase the survival rate of the affected patients and the timing and adequacy of this procedure have been identified as the most important clinical variable related with mortality [6, 10].

The consensus is that the first debridement should remove all necrotic tissues including muscle, fascia, fat and skin to the same extent as tumour-excision surgery [37]. Surgical approach is directed from the existing skin lesion, should be parallel to the local neurovascular bundles, or other vulnerable anatomical structures, and down to the level of the deep fascia. The margins of the resection should be to viable vascularised bleeding tissues, and all in-between necrotic or doubtful-looking elements should be removed [12, 68].

Perineal and scrotal infections pose particular surgical difficulties. In most cases at the time of the initial surgical debridement a diverting colostomy, as well as supra-pubic catheterization may be needed to allow wound hygiene and settling of the inflammation of the rectum/anus or the urethra. Surgical castration is not needed in most of the cases and the exposed testicles after scrotal resection are placed in the medial thighs [52].

The steps following the initial debridement include a re-evaluation of the wound on a daily basis, with repeated debridement mostly compulsory. It has been reported that optimally an average of three debridements within the first 2–3 days are needed to control gross infection [11, 17, 65]. In this process the wound should be protected against secondary infections, and also the formation of granulation tissue needs to be accelerated, as well as continuous drainage of the exudates. In the first stages the wound should be left open and treated with wet-to-dry dressings. It appears that topical negative pressure therapy (VAC-PAC) represents a viable option for wound management following initial infection control [26, 29, 43]. Although not well-studied in this particular clinical setting, other forms of

wound dressing (alginate and hydrogel, dilute sodium hypochlorite or iodine solution dressings, enzymatic debriding agents, or even skin the substitutes mainly used in burn patients) have been described with little benefit [1].

Once a healthy “bed” of granulation tissue is established, after the series of wound debridements, and the general condition of the patient is improved, the wound may be grafted (skin flap, split-thickness skin graft,) or left to complete its granulation by secondary intent with the help of the VAC-PAC. At a later stage a number of cases undergo reconstructive surgery with appropriate full-thickness free or rotational flaps [12].

In the cases of affected extremities an amputation represents a radical option which is often life-saving and mandatory. If the extent of the infection is rapidly spreading proximally, or includes a major joint, or has destroyed significant muscle groups of the extremity then an amputation should be considered and an informed consent should be obtained. Amputation has been reported to in 20% of all cases, particularly in IV drug users [2, 41].

---

### Antibiotic Therapy

Antibiotic therapy is an essential adjunct to surgical debridement. Except for one report [7] on a paediatric population of solely-conservative treatment for several days with antibiotics, the consensus is that they should not be used alone for the treatment of necrotising fasciitis due to the poor vascularity of fasciae, the poor blood supply of the necrotic lesion, and thus the poor delivery of the antibiotic agents locally. Nevertheless, they assist in the reduction of bacterial and toxin load, helping to prevent subsequent organ failure.

Intravenous broad-spectrum antibiotics should be administered on first presentation, optimally after microbial cultures are obtained. This initial empiric therapy should be efficient against gram-positive and negative organisms, as well as against anaerobes. In the past it included large doses of Penicillin with Clindamycin (Gram-positive and anaerobe coverage), and a third antibiotic for Gram-negative bacteria. Due to the major changes of microbial flora and the development of resistant species, currently the combination of clindamycin with vancomycin, imipenem, meropenem, ampicillin-sulbactam, piperacillin-tazobactam, daptomycin, quinupristin/dalfoprostin is preferred by most of authors as the initial empiric regime to cover Anaerobes and Gram-positive microbes [6, 52]. The addition of a Quinolone offers additional coverage for the Gram-negative bacteria, as they have excellent soft-tissue penetration [36, 40, 42].

Following the collection of samples from the wound and the gram stain of pus or of deep tissue, adjustments to the antibiotic regime should follow. The presence of Gram-positive cocci in clusters, as well as the increased prevalence of MRSA in many institutions dictates the use of Vancomycin in combination with Clindamycin [6, 19, 42, 69]. If the Gram-positive cocci are in pairs or chains then the combination of Clindamycin with a  $\beta$ -lactam antibiotic (ampicillin-sulbactam or piperacillin-tazobactam, or penicillin in high doses) suffices. Otherwise, if the gram stain identifies a polymicrobial flora or is inconclusive, the initial regime should continue until the final results of the cultures. Wound swabs should be sent at each debridement until final closure of the wound in order to identify early any secondary contamination, and adjust further the antibiotic therapy.

The duration of the antibiotics is still debatable and no proven time-frame can be recommended. It appears sensible to continue the antibiotics until no further debridement is needed, when healing healthy granulation tissue appears to cover the created defect and laboratory inflammatory and clinical markers are settled.

### Supportive Therapy

The general condition of these patients is usually grossly affected and a large proportion of them develop septic shock and multiple organ failure. Thus, adequate resuscitation and support of the vital functions is crucial. Analgesia is also essential as well as supplementation of the extensive fluid and electrolyte loss, and hypoalbuminaemia from the gross drainage of the large surgical wound. In the acute catabolic phase the calorie requirements of the patient are high and total parenteral nutrition may be needed for those patients where enteral feeding is not feasible.

### Adjunctive Therapies

Several authors have investigated the use of adjunctive therapies in the difficult clinical scenario of necrotising fasciitis in an attempt to optimise the outcome. Intravenous immunoglobulin-G (IVIG) is a concentrated product from a pool of immunoglobulin-G isotypes of human donors. It acts by inhibiting the activation of T-cells and the activity of streptococcal antigens. Theoretically, it can bind staphylococcal and streptococcal exotoxins limiting the systemic inflammatory response and its consequences specifically for necrotising fasciitis of this microbial aetiology [45, 60]. The reported results of its use, in the typical dosage of

1–2 g/kg of body weight for 1–5 days, are conflicting, underpowered, and non-randomised [31, 35, 53]. Some studies report decreased mortality in patients with streptococcal toxic shock where IVIG was administered [14, 20], while others report no clear advantage of its use [47].

Hyperbaric oxygen therapy (HBO) has been sporadically used in necrotising fasciitis cases, following its good results in cases of clostridial gangrene [47]. It is considered still an adjunct, with a probable beneficial effect that should never delay or hinder the primary treatment pillars of surgical debridement and intravenous antibiotics. In principle the increase of partial oxygen pressure (achieves arterial oxygen pressure of 2,000 mmHg, tissue oxygen pressure of 300 mmHg vs. 300 mmHg and 75 mmHg of arterial and tissue oxygen pressure respectively at normobaric conditions), reverses the effect of bacterial infection, and breaks the vicious triangle of infection-ischemia-reduced host defences. HBO also may limit the expansion of the necrosis and allow marking its boundaries and guiding the extent of the necessary debridement [55]. The typical HBO regime consists of 2–3 atmospheres of pressure for 0.5–2 h twice to four times daily until the progress of the infection is decreased or halted. The reported results vary significantly between a 3 [49] and 11-fold [18] decreased mortality rate and a significant decrease in amputation rates. Others report a non-effect of HBO treatment on survival rates and an increased risk of tympanic membrane rupture, seizures and central nervous system oxygen toxicity [8, 10]. It appears to offer advantages in tissue preservation and decreased mortality in the clostridial infections, which however, have a steadily decreased incidence on contemporary necrotising fasciitis cases, reflecting a decrease in the potential candidates for HBO treatment [27, 33].

Lately, the use of recombinant human-activated protein C has been described in a necrotising fasciitis case-report [47]. The authors proposed its evaluation in the future in a clinical trial focussed on necrotising fasciitis cases with involvement by group A streptococci.

---

### Outcome

The reported mortality rates have a wide range from 6 to 76% [6, 66]. The latest series report a somehow reduced mortality around 20%, reflecting the importance of early diagnosis and the advances in critical care [46].

Necrotising fasciitis of the perineum or abdominal wall have the highest mortality rates due to the inability for drastic surgical debridement or amputation in comparison to the cases where the extremities are affected.

In the series of Golger et al. [23] it was proven that the most important clinical variable related to mortality is the time from admission to surgical debridement, highlighting the importance of prompt diagnosis. Moreover they have found that for every year of life the risk of death is raised by 4%. In general the extremes of age (<1 and >60 years), streptococcal toxic shock and immunodeficiency syndromes are also associated with worse prognosis, as well as thrombocytopenia, hypoalbuminaemia, abnormal LFTs, acute renal failure, and elevated blood lactate levels [10, 12, 38].

In the study of Anaya et al. [2] multi-variate regression analysis of 166 patients identified as significant independent predictors of mortality the presence of clostridium as causative factor, the history of pre-existing coronary/heart disease, as well as high initial WBC (>30,000 cells/mm<sup>3</sup>) and serum Creatinine >2 mg/dL.

As expected, the survivors require prolonged hospitalisation and intensive care stay, undergo multiple surgical procedures and their in-hospital course is characterised by a number of serious complications. In these often are included nosocomial secondary infections (76%), ARDS and ventilator-dependent respiratory failure (29%), acute renal failure (32%), CNS episodes (seizures, stroke) (3–5%), heart episodes (arrest, heart failure) (2–3%) [17, 41, 52].

### Our Experience

Our unit encountered 29 cases of necrotizing fasciitis for the period of 2005 to 2009 (Table 3). Of 29 patients, 9 (31%) were female and 20 (79%) were male. The mean age of our patients was 54 years (range 4–87). Eight cases occurred in the lower limb (Fig. 1), 4 in the upper limb, 12 cases in the trunk and 5 cases in the head and neck. The most common identifiable cause for infection was intravenous drug use in 7 cases. Eight cases had no obvious cause and appeared to arise spontaneously. Five cases occurred after minor or trivial trauma. Other rarer causes for necrotizing fasciitis were associated with colonic malignancy, dental abscesses and pre-existing infections (Table 3). The mean length of hospital stay was 33 days with a mean of 3 days in intensive care and 2 days in a high dependency unit (HDU). Twenty cases were classified as Type 1 necrotising fasciitis due to their polymicrobial nature, seven cases with Type 2 (group A beta hemolytic streptococcus with or without *S. aureus*) whereas classification was not possible in two cases. The mean number of operations per patient was 4. Fourteen patients had VAC therapy to their wounds with skin grafts being the predominant method of reconstruction in 15 patients.



**Fig. 1** Case 3, spontaneous type-2 necrotising fasciitis, rapidly expanding cellulitis (6 h from initial marking), from groin to proximal tibia, just before initial surgical debridement

Five patients had either pedicled or free flap reconstruction. Two patients received hyperbaric oxygen as additional therapy; both these cases involved the head and neck. Of the 29 patients, five eventually died of overwhelming sepsis.

### Conclusions

Despite the expanded understanding of the pathophysiology of sepsis, the contemporary sophisticated diagnostic means and the modern advanced antibiotics, surgical debridement remains the cornerstone of treatment of necrotising fasciitis. The high mortality and morbidity of the disease remains a significant concern, while its incidence and microbiology appears to be evolving. Clinical research on this rare and lethal disease lacks high-level evidence, highlighting the necessity for multicentre collaboration of the scientific effort.

Table 3 Our centre's experience between the years July 2005 and January 2009

Patients	Sex	Age	Site	Cause	Comorbidities	LOS	ITU	HDU	Microbiology	Sensitivities	Treatment	No of ops	VAC	Recons	Mortality
1	F	34	Thigh	Inject	Type 2 diabetes, IVDU	79	9	6	<i>Strep milleri</i> / <i>Strep constellatus</i> , anaerobe, coag -ve Staph	Pen, Amox, Tazo, Metro	Pen, Metro, Clinda	5	Y	SSG	N
2	F	73	Abdo	Post op	None	22	1	2	<i>Strep milleri</i> , coliform, anaerobe	Tazo, Metro	Cef, Met, Merop, Clinda	1	Y	Secondary	N
3	M	54	Thigh	Spont	None	35	4	0	Group G Strep	Pen	Pen, metro, Clinda	4	Y	Secondary	N
4	M	76	Leg	Minor trauma	IHD, CVA	0	0	0	n/a	n/a	Merop, Metro	0	N	None	Y
5	M	4	Thigh	Spont	None	16	9	0	<i>S. aureus</i>	Flu	Flu, Cefotax, Metro, Pen, Clinda	3	N	SSG	N
6	M	70	Perineum	Perianal abscess	Type 2 diabetes	22	2	3	Coliform, Strep constellatus/ <i>milleri</i>	Amox/Amp	Merop	4	N	Secondary	N
7	M	44	Chest	Minor trauma	None	16	0	0	Coliform, enterococcus	n/a	Pen, Metro, Clinda, Gent	4	Y	SSG	N
8	M	71	Arm	Septic elbow	Rheumatoid, recurrent septic elbow	62	2	11	<i>S. aureus</i>	Flu	Flu, Tazo, Merop	6	Y	Pedicle brachio-radialis flap	N
9	M	25	Buttock	Inject	IVDU	26	0	1	Anaerobe, <i>Pseudomonas</i> , group C Strep	Metro, Tazo, Pen	Metro, Tazo, Merop	4	Y	SSG	N
10	M	35	Arm	Inject	IVDU	9	1	3	<i>S. aureus</i> , Group A Strep	Clinda, Flu, Pen	Pen, Flu, Clinda	2	Y	SSG	N
11	M	36	Arm	Inject	Steroid abuse	4	0	1	<i>Strep milleri</i> , anaerobe <i>S. aureus</i>	Pen, Flu	Pen, Flu, Clinda, Merop	1	N	Secondary	N

12	M	40	Head and neck	Dental abscess	None	7	1	0	<i>Strep milleri</i> , group F Strep, anaerobe, coag -ve staph	Pen	Pen, Flu, Metro, Clinda, Cipro	4	N	SSG, HBO	N
13	F	36	Leg	Minor trauma	None	15	1	7	Group A Strep, <i>S. aureus</i>	Flu, Vanc	Pen, Flu, Clinda	2	N	SSG	N
14	F	76	Head and neck	Spont	None	28	0	3	<i>S. aureus</i>	Flu	Pen, Flu, Metro; Clinda	3	N	Pedicle LD flap	N
15	M	37	Trunk	Inject	IVDU	87	8	0	Anaerobe, gram +ve cocci and bacilli	Flu, Metro, Gent	Pen, Metro, Gent	16	Y	Bipedicled abdo flap, SSG	N
16	M	39	Chest	Empyema	None	66	10	0	No growth	n/a	Cef, Met Tazo, Line, Metro	4	Y	SSG	N
17	M	40	Perineum	Inject	IVDU, DVT	13	0	0	<i>S. aureus</i> , Strep, anaerobe	Flu, Pen, Metro	Flu, Pen, Metro	3	N	SSG	N
18	F	50	Posterior maxilla	Dental abscess	None	19	0	0	<i>Strep milleri</i> , anaerobe	Pen, Metro	Pen, Cipro, Flu, Metro, Amox, Metro	3	N	Pharyngeal and buccal flap, HBO	N
19	F	52	Thigh	Spont	IHD	104	9	7	Anaer, <i>Strep constellatus/milleri</i>	Pen, Amox, Metro	Tazo, Clinda, Pen, metro	15	Y	SSG	N
20	F	42	Leg	Minor trauma	Alcoholic liver disease, drug use	1	1	0	Group A Strep, <i>S. aureus</i>	Pen, Flu	Flu, Clinda, Cipro, Gent	1	N	None	Y
21	F	68	Groin	Perforated colon	Colonic carcinoma	36	5	2	Coliform, anaerobe, <i>Enterococcus</i>	Amox, Metro, Pen	Clinda, Pen, Cipro, Metro	3	N	SSG	N

(continued)

Table 3 (continued)

Patients	Sex	Age	Site	Cause	Comorbidities	LOS	ITU	HDU	Microbiology	Sensitivities	Treatment	No of ops	VAC	Recons	Mortality
22	M	71	Parotid	Parotitis	None	32	11	7	<i>Strep milleri</i> , <i>Enterococcus</i>	Pen, Amox	Flu, Amox, Metro; Amox, Metro, Gent	5	N	SSG	N
23	F	74	Leg	Minor trauma	Psoriasis	16	2	3	Group A Strep, coag -ve staph	Pen	Pen, Clinda, Vanc	4	Y	SSG	N
24	M	83	Abdo	Spont	CRF, AF	2	2	0	Strep, anaerobe	Pen, Metro	Cef, Metro, Cipro, Vanc, Clinda	1	N	None	Y
25	M	87	Face	Spont	IHD, CRF, PVD	98	9	4	Beta haemolytic Strep	Pen	Cef, Pen, Metro	4	N	Forehead and radial forearm flap	N
26	M	75	Perineum	Anal SCC	Anal SCC	0	1	0	Coliform, beta haem Strep, <i>Enterococcus</i> , anaerobe	Amox, Cef	Cef, Metro, Gent	2	N	None	Y
27	M	43	Perineum	Spont	None	31	1	2	Gram -ve <i>Bacillus</i> , coag -ve Staph, <i>Proteus</i> , anaerobe	Amox, Metro	Merop, Metro, Flu	4	Y	None	Y
28	M	79	Perineum	Spont	CVA	83	6	15	Coliform, anaerobe	Cef, Met	Cef, Metro, Clinda, Gent, Merop	6	Y	Secondary	N
29	M	46	Arm	Inject	IVDU	30	0	2	Group G Strep, anaerobe	Pen, Metro	Pen, Metro, Flu, Pen, Metro, Clinda, Cipro, Amox, Metro	4	Y	SSG	N

Abdo abdominal; Amox amoxicillin; Anaer anaerobes; Cefcefturoxime; Cefotax cefotaxime; Cipro ciprofloxacin; Clinda clindamycin; F female; Gent gentamicin; HDU high dependency unit; ITU intensive care unit; Line linezolid; LOS length of stay; M male; Merop meropenem; Metro metronidazole; n/a not available/not applicable; Pen penicillin; Recons reconstruction; Spont spontaneous; SSG split skin graft; Secondary healing by secondary intention; Vanc vancomycin

## References

1. Akhtar S, Hasham S, Abela C, Phipps AR (2006) The use of Integra in necrotizing fasciitis. *Burns* 32:251–254
2. Anaya DA, Dellinger EP (2007) Necrotizing soft-tissue infection: diagnosis and management. *Clin Infect Dis* 44:705–710
3. Angoules AG, Kontakis G, Drakoulakis E et al (2007) Necrotising fasciitis of upper and lower limb: a systematic review. *Injury* 38(suppl 5):S19–S26
4. Aronoff DM, Bloch KC (2003) Assessing the relationship between the use of nonsteroidal antiinflammatory drugs and necrotizing fasciitis caused by group A streptococcus. *Medicine (Baltimore)* 82:225–235
5. Beauchamp NJ Jr, Scott WW Jr, Gottlieb LM, Fishman EK (1995) CT evaluation of soft tissue and muscle infection and inflammation: a systematic compartmental approach. *Skeletal Radiol* 24:317–324
6. Bellapianta JM, Ljungquist K, Tobin E, Uhl R (2009) Necrotizing fasciitis. *J Am Acad Orthop Surg* 17:174–182
7. Bingol-Kologlu M, Yildiz RV, Alper B et al (2007) Necrotizing fasciitis in children: diagnostic and therapeutic aspects. *J Pediatr Surg* 42:1892–1897
8. Brown DR, Davis NL, Lepawsky M et al (1994) A multicenter review of the treatment of major truncal necrotizing infections with and without hyperbaric oxygen therapy. *Am J Surg* 167:485–489
9. Cainzos M, Gonzalez-Rodriguez FJ (2007) Necrotizing soft tissue infections. *Curr Opin Crit Care* 13:433–439
10. Carter PS, Banwell PE (2004) Necrotising fasciitis: a new management algorithm based on clinical classification. *Int Wound J* 1:189–198
11. Chen JL, Fullerton KE, Flynn NM (2001) Necrotizing fasciitis associated with injection drug use. *Clin Infect Dis* 33:6–15
12. Childers BJ, Potyondy LD, Nachreiner R et al (2002) Necrotizing fasciitis: a fourteen-year retrospective study of 163 consecutive patients. *Am Surg* 68:109–116
13. Cox NH (1999) Streptococcal necrotizing fasciitis and the dermatologist. *Br J Dermatol* 141:613–614
14. Darabi K, Abdel-Wahab O, Dzik WH (2006) Current usage of intravenous immune globulin and the rationale behind it: the Massachusetts General Hospital data and a review of the literature. *Transfusion* 46:741–753
15. Descamps V, Aitken J, Lee MG (1994) Hippocrates on necrotising fasciitis. *Lancet* 344:556
16. Dufel S, Martino M (2006) Simple cellulitis or a more serious infection? *J Fam Pract* 55:396–400
17. Elliott DC, Kufera JA, Myers RA (1996) Necrotizing soft tissue infections. Risk factors for mortality and strategies for management. *Ann Surg* 224:672–683
18. Escobar SJ, Slade JB Jr, Hunt TK, Cianci P (2005) Adjuvant hyperbaric oxygen therapy (HBO2) for treatment of necrotizing fasciitis reduces mortality and amputation rate. *Undersea Hyperb Med* 32:437–443
19. Everest E (1999) Group A streptococcal fasciitis. *Crit Care Resusc* 1:63–68
20. Fontes RA Jr, Ogilvie CM, Miclau T (2000) Necrotizing soft-tissue infections. *J Am Acad Orthop Surg* 8:151–158
21. Fugitt JB, Puckett ML, Quigley MM, Kerr SM (2004) Necrotizing fasciitis. *Radiographics* 24:1472–1476
22. Geusens E, Pans S, Van Breuseghem I, Knockaert D (2004) Necrotizing fasciitis of the leg presenting with chest wall emphysema. *Eur J Emerg Med* 11:49–51
23. Golger A, Ching S, Goldsmith CH et al (2007) Mortality in patients with necrotizing fasciitis. *Plast Reconstr Surg* 119:1803–1807
24. Green RJ, Dafoe DC, Raffin TA (1996) Necrotizing fasciitis. *Chest* 110:219–229
25. Hsiao GH, Chang CH, Hsiao CW et al (1998) Necrotizing soft tissue infections. Surgical or conservative treatment? *Dermatol Surg* 24:243–247; discussion 247–248
26. Huang WS, Hsieh SC, Hsieh CS et al (2006) Use of vacuum-assisted wound closure to manage limb wounds in patients suffering from acute necrotizing fasciitis. *Asian J Surg* 29:135–139
27. Jallali N, Withey S, Butler PE (2005) Hyperbaric oxygen as adjuvant therapy in the management of necrotizing fasciitis. *Am J Surg* 189:462–466
28. Kalorin CM, Tobin EH (2007) Community associated methicillin resistant *Staphylococcus aureus* causing Fournier's gangrene and genital infections. *J Urol* 177:967–971
29. Kanakaris NK, Thanasis C, Keramaris N et al (2007) The efficacy of negative pressure wound therapy in the management of lower extremity trauma: review of clinical evidence. *Injury* 38 suppl 5:S9–S18
30. Kaul R, McGeer A, Low DE et al (1997) Population-based surveillance for group A streptococcal necrotizing fasciitis: clinical features, prognostic indicators, and microbiologic analysis of seventy-seven cases. Ontario Group A Streptococcal Study. *Am J Med* 103:18–24
31. Kaul R, McGeer A, Norrby-Teglund A et al (1999) Intravenous immunoglobulin therapy for streptococcal toxic shock syndrome—a comparative observational study. The Canadian Streptococcal Study Group. *Clin Infect Dis* 28:800–807
32. Kihiczak GG, Schwartz RA, Kapila R (2006) Necrotizing fasciitis: a deadly infection. *J Eur Acad Dermatol Venereol* 20:365–369
33. Korhonen K (2000) Hyperbaric oxygen therapy in acute necrotizing infections with a special reference to the effects on tissue gas tensions. *Ann Chir Gynaecol Suppl*:7–36
34. Lamagni TL, Neal S, Keshishian C et al (2008) Severe *Streptococcus pyogenes* infections, United Kingdom, 2003–2004. *Emerg Infect Dis* 14:202–209
35. Lamothe F, D'Amico P, Ghosn P et al (1995) Clinical usefulness of intravenous human immunoglobulins in invasive group A Streptococcal infections: case report and review. *Clin Infect Dis* 21:1469–1470
36. Lee TC, Carrick MM, Scott BG et al (2007) Incidence and clinical characteristics of methicillin-resistant *Staphylococcus aureus* necrotizing fasciitis in a large urban hospital. *Am J Surg* 194:809–812; discussion 812–813
37. Levine EG, Manders SM (2005) Life-threatening necrotizing fasciitis. *Clin Dermatol* 23:144–147
38. Liu YM, Chi CY, Ho MW et al (2005) Microbiology and factors affecting mortality in necrotizing fasciitis. *J Microbiol Immunol Infect* 38:430–435



39. Loyer EM, DuBrow RA, David CL et al (1996) Imaging of superficial soft-tissue infections: sonographic findings in cases of cellulitis and abscess. *AJR Am J Roentgenol* 166:149–152
40. Maltezou HC, Giamarellou H (2006) Community-acquired methicillin-resistant *Staphylococcus aureus* infections. *Int J Antimicrob Agents* 27:87–96
41. McHenry CR, Piotrowski JJ, Petrinic D, Malangoni MA (1995) Determinants of mortality for necrotizing soft-tissue infections. *Ann Surg* 221:558–563; discussion 563
42. Miller LG, Perdreau-Remington F, Rieg G et al (2005) Necrotizing fasciitis caused by community-associated methicillin-resistant *Staphylococcus aureus* in Los Angeles. *N Engl J Med* 352:1445–1453
43. Moues CM, Vos MC, van den Bemd GJ et al (2004) Bacterial load in relation to vacuum-assisted closure wound therapy: a prospective randomized trial. *Wound Repair Regen* 12:11–17
44. Nisbet AA, Thompson IM (2002) Impact of diabetes mellitus on the presentation and outcomes of Fournier's gangrene. *Urology* 60:775–779
45. Norrby-Teglund A, Kaul R, Low DE et al (1996) Plasma from patients with severe invasive group A streptococcal infections treated with normal polyspecific IgG inhibits streptococcal superantigen-induced T cell proliferation and cytokine production. *J Immunol* 156:3057–3064
46. Ogilvie CM, Miclau T (2006) Necrotizing soft tissue infections of the extremities and back. *Clin Orthop Relat Res* 447:179–186
47. Purnell D, Hazlett T, Alexander SL (2004) A new weapon against severe sepsis related to necrotizing fasciitis. *Dimens Crit Care Nurs* 23:18–23
48. Rajbhandari SM, Wilson RM (1998) Unusual infections in diabetes. *Diabetes Res Clin Pract* 39:123–128
49. Riseman JA, Zamboni WA, Curtis A et al (1990) Hyperbaric oxygen therapy for necrotizing fasciitis reduces mortality and the need for debridements. *Surgery* 108:847–850
50. Rodriguez RM, Abdullah R, Miller R et al (2006) A pilot study of cytokine levels and white blood cell counts in the diagnosis of necrotizing fasciitis. *Am J Emerg Med* 24:58–61
51. Salcido RS (2007) Necrotizing fasciitis: reviewing the causes and treatment strategies. *Adv Skin Wound Care* 20:288–293; quiz 294–295
52. Sarani B, Strong M, Pascual J, Schwab CW (2009) Necrotizing fasciitis: current concepts and review of the literature. *J Am Coll Surg* 208:279–288
53. Schrage B, Duan G, Yang LP et al (2006) Different preparations of intravenous immunoglobulin vary in their efficacy to neutralize streptococcal superantigens: implications for treatment of streptococcal toxic shock syndrome. *Clin Infect Dis* 43:743–746
54. Sharkawy A, Low DE, Saginur R et al (2002) Severe group A streptococcal soft-tissue infections in Ontario: 1992–1996. *Clin Infect Dis* 34:454–460
55. Shupak A, Shoshani O, Goldenberg I et al (1995) Necrotizing fasciitis: an indication for hyperbaric oxygenation therapy? *Surgery* 118:873–878
56. Simonart T (2004) Group A beta-haemolytic streptococcal necrotizing fasciitis: early diagnosis and clinical features. *Dermatology* 208:5–9
57. Stamenkovic I, Lew PD (1984) Early recognition of potentially fatal necrotizing fasciitis. The use of frozen-section biopsy. *N Engl J Med* 310:1689–1693
58. Stevens DL (1999) The flesh-eating bacterium: what's next? *J Infect Dis* 179(suppl 2):S366–S374
59. Sudarsky LA, Laschinger JC, Coppa GF, Spencer FC (1987) Improved results from a standardized approach in treating patients with necrotizing fasciitis. *Ann Surg* 206:661–665
60. Takei S, Arora YK, Walker SM (1993) Intravenous immunoglobulin contains specific antibodies inhibitory to activation of T cells by staphylococcal toxin superantigens [see comment]. *J Clin Invest* 91:602–607
61. Tang WM, Ho PL, Fung KK et al (2001) Necrotizing fasciitis of a limb. *J Bone Joint Surg Br* 83:709–714
62. Trent JT, Kirsner RS (2002) Diagnosing necrotizing fasciitis. *Adv Skin Wound Care* 15:135–138
63. Wall DB, Klein SR, Black S, de Virgilio C (2000) A simple model to help distinguish necrotizing fasciitis from nonnecrotizing soft tissue infection. *J Am Coll Surg* 191:227–231
64. Wilson B (1952) Necrotizing fasciitis. *Am Surg* 18:416–431
65. Wong CH, Chang HC, Pasupathy S et al (2003) Necrotizing fasciitis: clinical presentation, microbiology, and determinants of mortality. *J Bone Joint Surg Am* 85-A:1454–1460
66. Wong CH, Khin LW, Heng KS et al (2004) The LRINEC (Laboratory Risk Indicator for Necrotizing Fasciitis) score: a tool for distinguishing necrotizing fasciitis from other soft tissue infections. *Crit Care Med* 32:1535–1541
67. Wysoki MG, Santora TA, Shah RM, Friedman AC (1997) Necrotizing fasciitis: CT characteristics. *Radiology* 203:859–863
68. Young MH, Aronoff DM, Engleberg NC (2005) Necrotizing fasciitis: pathogenesis and treatment. *Expert Rev Anti Infect Ther* 3:279–294
69. Young LM, Price CS (2008) Community-acquired methicillin-resistant *Staphylococcus aureus* emerging as an important cause of necrotizing fasciitis. *Surg Infect (Larchmt)* 9:469–474

---

## Introduction

Fracture healing is a complex physiological process caused by the interaction of cellular elements that are activated and controlled by an array of cytokines and signalling proteins [11]. This process is both temporal and spatial in nature and usually results in the formation of new bone, which is structurally and mechanically similar to the pre-fracture state [10].

For a lot of reasons this process can fail and result in non-union of bone in 10% of all fractures and in up to 50% of open fractures of the tibia. These patients develop a non-union, which leads to long-lasting inability to work, loss of employment and high social costs. These costs are estimated in a paper of Sprague 2002 to be at approximately \$80,000 in case of 18 weeks delay of fracture healing [28]. The overall costs of delayed fracture healing are estimated to be at \$14.6 million in United States alone [6].

Many attempts have been made to reduce the rate of disturbed fracture healing but despite the correct osteosynthesis, which is mandatory, and new improved interlocking plates, biologic osteosynthesis and improved surgical techniques like subcutaneous plating, the overall rate has not been reduced. The standard procedure to induce or enhance bone healing in a delayed status is autologous bone grafting. This procedure provides osteogenic, osteoinductive and osteoconductive properties and has a success rate of 50–80% [7, 9, 29, 33]. But the success of this “gold standard” depends on the quality of the harvested bone and is naturally limited by the amount available from the donor.

Nowadays it could be stated, that correct technical debridement and knowledge about “correct osteosynthesis” is the basic treatment and the rules of stability have to be

implemented. But there are some new possibilities which can help as an adjunct to increase the healing rate of fractures and/or non-unions.

New trends are:

---

## Low Intensity Pulsatile Ultrasound (LIPUS)

Ultrasound treatment of a non-union or delayed union is dependent on specific pre-requisites. First the fracture or non-union has to be mechanically stable which is not often the case in non-union. Secondly, the non-union has to be viable. But most of the non-unions or fractures at-risk have exactly this problem and today the non-invasive diagnosis is very difficult to determine if a non-union is atrophic, oligotrophic or hypertrophic. Therefore the indication for conservative treatment with pulsatile ultrasound is also very limited.

But, if it is possible, it seems that it is sometimes successful.

In prospective, randomized, double-blind, placebo-controlled, multi-centre clinical studies, LIPUS has been proven to be effective in decreasing the time to heal in both fresh diaphyseal (tibia) and metaphyseal (distal radius) fractures. It also decreases the likelihood of a delayed union (>150 days to heal) in tibial fractures and loss of reduction in distal radius fractures. World-wide clinical studies of LIPUS for treatment of non-union, in a self-paired control study design, have demonstrated a healing rate of 88% with an average treatment time of 4.5 months in non-unions and an average fracture age of 23 months. The therapy is safe and non-invasive, and is used by the patient at home for a 20-min treatment session per day [8].

In a study of Jingushi, seventy-two cases of long bone fracture, including those of the femur, tibia, humerus, radius, and ulna, were analyzed. The mean time from the most recent operation to the beginning of LIPUS treatment was 11.5 (3–68) months. The relationship between the background factors and the union rate was analyzed

---

G. Zimmermann (✉)  
Theresienkrankenhaus Mannheim, Department of Trauma  
Surgery, Bassermannstrasse 1, 68165 Mannheim, Germany  
e-mail: g.zimmermann@theresienkrankenhaus.de

using a logistic regression method. In addition, long bone fractures in an upper extremity or in a lower extremity were analyzed separately. The union rate was 75% in all the cases of long bone fracture. There was a significant relationship between the union rate and the period from the most recent operation to the beginning of LIPUS treatment in all cases and in those that had long bone fracture of an upper extremity. There was also a significant relationship between the union rate and the time when a radiological improvement was first observed after the beginning of the treatment in all cases and in those with fractures in a lower extremity. When LIPUS treatment was started within 6 months of the most recent operation, 89.7% of all fractures healed. When an improvement in the radiological changes at the fracture site was observed after 4 months in those cases, then the sensitivity and specificity for union were more than 90%. Jingushi concluded that LIPUS treatment should be started within 6 months of the most recent operation. Because LIPUS has been shown to be effective without causing either serious invasiveness or any undue risk to the patient, it may be considered the treatment of first choice for cases of post-operative delayed union or non-union [17, 18].

LIPUS also appears as an effective and safe home treatment of aseptic and septic delayed-unions and non-unions, with a healing rate ranging from 70 to 93% in different, non-randomized, studies. Advantages of the use of this technology are: that it may avoid the need for additional complex operations for the treatment of non-unions and also efficacy, safety, ease of use and favourable cost/benefit ratio. Outcomes depend on the site of non-union, time elapsed from trauma, stability at the site of non-union and host type [19, 24].

The detailed biophysical process by which low-intensity pulsed ultrasound LIPUS stimulates bone regeneration still remains unknown, although various effects on bone cells in vitro and in vivo have been described.

LIPUS treatment has led to increased callus area and accelerated return of bone strength following fracture. Histological studies suggest that LIPUS influences all major cell types involved in bone healing, including osteoblasts, osteoclasts, chondrocytes and mesenchymal stem-cells. The affect of LIPUS seems to be limited to cells in soft tissues, whereas cells in calcified bone seem not to be effected. In vitro cell culture studies as well as tissue culture studies have shown some effects on cell differentiation and protein synthesis. Even though the energy used by LIPUS treatment is extremely low, the effects are evident. The most probable source of the therapeutic benefits observed with LIPUS treatment involves non-thermal mechanisms that influence cell membrane permeability and increase cellular activity. Despite clinical and experimental

studies demonstrating the enhancing effect of LIPUS on bone regeneration, the biophysical mechanisms involved in the complex fracture healing process remain unclear and require further research [4, 19].

---

## Magnetic Field Induction

Regarding the indications for the treatment of the magnetic field induction there is no difference to Lipus.

Electric and electromagnetic fields are, collectively, one form of biophysical technique which regulate extracellular matrix synthesis and may be useful in clinically stimulating repair of fractures and non-unions. Pre-clinical studies have shown that electric and electromagnetic fields regulate proteoglycan and collagen synthesis in models of endochondral ossification, and increase bone formation in vivo and in vitro. A substantial number of clinical studies have been done that suggest acceleration of bone formation and healing, particularly osteotomies and spine fusions, by electric and electromagnetic fields. Many of these studies have used randomized, placebo-controlled designs. In osteotomy trials, greater bone density, trabecular maturation, and radiographic healing were observed in actively-treated, compared with placebo-treated patients. In spine fusions, average union rates of 80–90% were observed in actively-treated patients across numerous studies compared with 65–75% in placebo-treated patients. Uncontrolled, longitudinal cohort studies of delayed and non-unions report mean union rates of approximately 75–85% in fractures previously refractory to healing. The few randomized controlled studies in delayed and non-unions suggest improved results with electric and electromagnetic fields compared with placebo treatment, and equivalent to bone grafts [13].

There is a consensus that electromagnetic stimulation is an effective adjunct to conventional therapy when used in the management of non-union of long bone fractures [5].

---

## Growth Factor Treatment

Bone Morphogenetic Protein 7 (BMP 7, Osigraft™) has been shown in a prospective randomised human trial to be a cytokine which can induce bone formation in tibial non-unions at the same rate autologous bone graft [9]. BMP's are members of the Transforming Growth Factor Beta (TGF  $\beta$ ) superfamily and are characterised by immense osteoinductive potential. They induce a sequential cascade

of events for chondro-osteogenesis during bone formation and ultimately fracture healing, including chemotaxis, proliferation of mesenchymal and osteoprogenitor cells and their differentiation into a chondrogenic or osteogenic lineage [10, 11, 26, 30, 31].

But not only BMP and TGF  $\beta$  are cytokines which trigger and are leading the normal process of bone healing. Today there are over 120 cytokines, growth factors hormones and other factors are known, which play a role in this process. Most of them are more or less involved but there are some key proteins like PDGF, IGF, VEGF and FGF which are known to have significant influence on the fracture healing process if they are supplemented.

Beside mechanical insufficiencies in treating fractures, it is obvious that also biological deficiencies can result in non-union.

Some clinically relevant studies can be found via Medline [4, 23, 25, 32, 34]. An overview is given in Table 1. In England, an overview of clinical BMP 7 application in all long hollow bones has recently been published. Of 653 documented cases, 60.5% had the application indicated for pseudoarthroses. In 74%, an autologous bone graft was also performed. In 23%, BMP 7 had been implanted without autologous bone transplantation. All cases that healed based upon radiological and clinical analyses without

requiring further treatment were considered successful, and the success rate was 82%. Details concerning location of the fracture, the number of previous therapies, other diseases of the patients or the duration of the healing process are not reported [12].

For BMP 7, one prospective, randomized and partly-blind clinical study exists, which describes 122 patients who had been treated over a period of 7 years in 7 different trauma centres in the USA. Criterion for inclusion in the study was tibial pseudoarthrosis of at least 9 months duration. The planned surgical techniques had to be intramedullary nailing and autologous bone grafting. The control group also received intramedullary nailing, but BMP 7, instead of autologous bone, was implanted. Nine months after treatment, the success was evaluated according to the clinical criteria of being full weight-bearing and radiological fusion. The success rate in the autologous bone grafting group was 84%, and that of the BMP 7 application group was 75%. This difference was not statistically significant ( $p=0.218$ ). In a sub-group of patients, which included those with nicotine abuse, BMP 7 showed a markedly higher success rate compared to autologous bone grafting. In addition, the frequency of infections was lower when BMP 7 was implanted. Since these were not the criteria the study was designed for, the results could not be interpreted as significant [9].

**Table 1** Clinical not randomised studies using BMP 7

Author	Study design	Indication	Patients	BMP	Union rate %
Giannoudis et al. [25] UK	Retrospective, observational, non randomized	Non-union, osteotomies etc. various sites	653	7	82
Dimitriou et al. (2005)	Retrospective, observational, non randomized	Non-union various sites	26	7	92
Ronga et al. [25] Bios study group Italy	Retrospective, observational, non randomized	Non-union various sites	105	7	89
Zimmermann et al. [32]	Retrospective, observational, non randomized	Non-union various sites	23	7	92
Zimmermann et al. [33, 34]	Prospective, matched pairs, controlled trial	Non-union tibia shaft	108	7	89 sign higher
Ristiniemi et al. [23]	Prospective, controlled trial	Pilon fractures	40	7	Sign faster
Giannoudis et al. [11]	Retrospective, observational, non randomized	Non-union pelvic girdle	9	7	89
Zimmermann Moghaddam et al. (2007)	Retrospective, observational, non randomized	Non-union various sites	172	7	79

## Stem-Cell Therapy

Several investigators have focussed their attention on a subset of autologous non-hematopoietic stem/progenitor cells contained in the adult bone marrow stroma, referred to as stromal stem-cells (SSC), as the appropriate cells to be transplanted [4, 20–22, 27]. The use of autologous cells is facilitated by less stringent ethical and regulatory issues and does not require the patient to be immunologically suppressed. In pre-clinical and clinical protocols of critical defects in which SSC are employed, two approaches are mainly used: in the first, SSC are derived from bone marrow and directly introduced at the lesion site, in the second, SSC are derived from several sites and are expanded *ex vivo* before being implanted. Both approaches, equally correct in principle, will have to demonstrate, with definitive evidence of their efficacy, their capability of solving a critical clinical problem such as non-union.

Overall there are only a few clinical studies where experience with the application of stem cells in the treatment of non-unions is reported. Most of these are from Hernigou. He investigated the relationship between number of injected cells to the healing rate [14–16].

He aspirated marrow from both anterior iliac crests, concentrated on a cell separator, and then injected into sixty non-infected atrophic non-unions of the tibia. Bone union was obtained in fifty-three patients, and the bone marrow that had been injected into the non-unions of those patients contained  $>1,500$  progenitors/cm<sup>3</sup> and an average total of  $54,962 \pm 17,431$  progenitors. There was a positive correlation between the volume of mineralized callus at 4 months and the number ( $p=0.04$ ) and concentration ( $p=0.01$ ) of fibroblast colony-forming units in the graft. There was a negative correlation between the time needed to obtain union and the concentration of fibroblast colony-forming units in the graft ( $p=0.04$ ).

Percutaneous autologous bone-marrow grafting seems to be an effective and safe method for the treatment of an atrophic tibial diaphyseal non-union. However, its efficacy appears to be related to the number of progenitors in the graft, and the number of progenitors available in bone marrow aspirated from the iliac crest appears to be less than optimal in the absence of concentration [1].

## References

1. Aaron RK, Ciombor DM, Simon BJ (2004) Treatment of nonunions with electric and electromagnetic fields. *Clin Orthop Relat Res* (419):21–29
2. Agata H et al (2009) Feasibility and efficacy of bone tissue engineering using human bone marrow stromal cells cultivated in serum-free conditions. *Biochem Biophys Res Commun* 382(2):353–358
3. Claes L, Willie B (2007) The enhancement of bone regeneration by ultrasound. *Prog Biophys Mol Biol* 93(1–3):384–398
4. Connolly JF (1995) Injectable bone marrow preparations to stimulate osteogenic repair. *Clin Orthop Relat Res* (313):8–18
5. Dimitriou R, Giannoudis PV (200) Application of rhOP-1 in non union. Report of 26 cases. *Injury* 375:524–528
6. Einhorn TA, Trippel SB (1997) Growth factor treatment of fractures. A.A.O.S.I.C. Lecture. American Academy of Orthopedic Surgeons Publications, Rosemont, IL, pp 483–494
7. Engelhardt P, Velasco R (1994) Prognosis of spongiosoplasty of the fractured tibial shaft. *Unfallchirurg* 97(10):525–529
8. Frankel VH, Mizuho K (2002) Management of non-union with pulsed low-intensity ultrasound therapy—international results. *Surg Technol Int* 10:195–200
9. Friedlaender GE et al (2001) Osteogenic protein-1 (bone morphogenetic protein-7) in the treatment of tibial nonunions. *J Bone Joint Surg Am* 83-A suppl 1(Pt 2):S151–S158
10. Gerstenfeld LC et al (2003) Fracture healing as a post-natal developmental process: molecular, spatial, and temporal aspects of its regulation. *J Cell Biochem* 88(5):873–884
11. Giannoudis PV, Kanakaris NK, Einhorn TA (2007) Interaction of bone morphogenetic proteins with cells of the osteoclast lineage: review of the existing evidence. *Osteoporos Int* 18(12):1565–1581
12. Giannoudis PV, Tzioupis C (2005) Clinical applications of BMP-7. The UK perspective. *Injury* 36S:47–50
13. Griffin XL, Warner F, Costa M (2008) The role of electromagnetic stimulation in the management of established non-union of long bone fractures: what is the evidence? *Injury* 39(4):419–429
14. Hernigou P et al (2005) The use of percutaneous autologous bone marrow transplantation in nonunion and avascular necrosis of bone. *J Bone Joint Surg Br* 87(7):896–902
15. Hernigou P et al (2005) Percutaneous autologous bone-marrow grafting for nonunions. Influence of the number and concentration of progenitor cells. *J Bone Joint Surg Am* 87(7):1430–1437
16. Hernigou P et al (2006) Percutaneous autologous bone-marrow grafting for nonunions. Surgical technique. *J Bone Joint Surg Am* 88 Suppl 1 (Pt 2):322–327
17. Jingushi S (2009) [Bone fracture and the healing mechanisms. Fracture treatment by low-intensity pulsed ultrasound]. *Clin Calcium* 19(5):704–708
18. Jingushi S et al (2007) Low-intensity pulsed ultrasound treatment for postoperative delayed union or nonunion of long bone fractures. *J Orthop Sci* 12(1):35–41
19. Malizos KN et al (2006) Low-intensity pulsed ultrasound for bone healing: an overview. *Injury* 37 suppl 1:S56–S62
20. Matsuda Y et al (1998) Percutaneous autologous bone marrow transplantation for nonunion of the femur. *Nippon Geka Hokan* 67(1):10–17

21. Meister K, Segal D, Whitelaw GP (1990) The role of bone grafting in the treatment of delayed unions and nonunions of the tibia. *Orthop Rev* 19(3):260–271
22. Muschler GF, Boehm C, Easley K (1997) Aspiration to obtain osteoblast progenitor cells from human bone marrow: the influence of aspiration volume. *J Bone Joint Surg Am* 79(11):1699–1709
23. Ristiniemi J (2007) External fixation of tibial pilon fractures and fracture healing. *Acta Orthop Suppl* 78(326):3, 5–34
24. Romano CL, Romano D, Logoluso N (2009) Low-intensity pulsed ultrasound for the treatment of bone delayed union or nonunion: a review. *Ultrasound Med Biol* 35(4):529–536
25. Ronga M et al (2006) Recombinant human bone morphogenetic protein 7 for treatment of long bone non-union. *Injury* 37(5):551–556
26. Sakou T (1998) Bone morphogenetic proteins: from basic studies to clinical approaches. *Bone* 22(6):591–603
27. Sen MK, Miclau T (2007) Autologous iliac crest bone graft: should it still be the gold standard for treating nonunions? *Injury* 38 suppl 1:S75–S80
28. Sprague S, Bhandari M (2002) An economic evaluation of early versus delayed operative treatment in patients with closed tibial shaft fractures. *Arch Orthop Trauma Surg* 122(6):315–323
29. Weise K, Winter E (1996) Role of intramedullary nailing in pseudarthrosis and malalignment. *Orthopade* 25(3):247–258
30. Wozney JM (1989) Bone morphogenetic proteins. *Prog Growth Factor Res* 1(4):267–280
31. Wozney JM et al (1988) Novel regulators of bone formation: molecular clones and activities. *Science* 242(4885):1528–1534
32. Zimmermann G et al (2006) Clinical experience with bone morphogenetic protein 7 (BMP 7) in nonunions of long bones. *Unfallchirurg* 109(7):528–537
33. Zimmermann G et al (2007) Therapeutic outcome in tibial pseudarthrosis: bone morphogenetic protein 7 (BMP-7) versus autologous bone grafting for tibial fractures. *Unfallchirurg* 110(11):931–938
34. Zimmermann G et al (2007) Ergebnisse der Anwendung von Bone Morphogenetic Protein 7 bei Pseudarthrosen langer Röhrenknochen in Deutschland. In Congress of the German Society of Trauma Surgery, Berlin

**Part II**

---

**Bone and Joint Tumours**

## Imaging Algorithm in the Diagnosis, Therapy, Control and Follow-Up of Primary Musculoskeletal Tumours and Metastases

I.M. Noebauer-Huhmann, J. Panotopoulos, and R.I. Kotz

### Introduction

Primary musculoskeletal (MSK) – tumours are rare. Most of the lesions seen in the daily routine work of Orthopaedic Surgeons are associated with trauma or infection [38]. It is crucial not to misinterpret a tumour as a haematoma or ganglion, and especially not to consecutively perform an accidental intra-lesional operation (which has thus been named “whoops” operation) [1]. Any case in doubt should be referred to a specialized centre. Here, the therapeutic strategy also depends on a thorough imaging work-up of the lesion.

The aim of all imaging algorithms is to detect the tumour early, to identify its character and extent, potentially to guide or perform a biopsy, to help in planning an operation or radiation, to assess further sites of polylocular disease and metastases, to detect potential complications (such as pathologic fractures or spinal cord compression), to monitor therapy and to perform follow-up for the detection of recurrence.

In the following article, the diagnostic algorithm of primary MSK-tumours and of metastases, which are far more common, are described.

### Primary Evaluation

#### X-Ray, CT

The first imaging modality, which should still be performed in all patients, is conventional projection radiography with

two projections. It will provide information on the status of the lesion in primary bone tumours and metastases, and of the tumour matrix [25, 33] (figure 1a, 3a). In the estimation of the status of a tumour, the well-established criteria like the destruction pattern (geographic, moth-eaten or permeated), the border and transition zone and the periosteal reaction are still valuable. The location of the lesion (long or flat bone, peripheral or in the axial skeleton, epi-/meta-/ or diaphyseal, central or peripheral), the extent and growth rate, monostotic or multilocular occurrence, the age and gender of the patient are still important criteria to be considered.

Some clearly benign lesions do not require further imaging for differential diagnosis, like small bone islands or single cartilaginous exostoses, or typical non-ossifying fibromas, the latter ones being followed-up with projection radiography.

To a limited extent plain radiography can also help in assessing the fracture risk in the long bones [32]. However, detection of osteolytic lesions requires bone mineral loss of at least 50% in the spine [18]. Overall, values of 30–75% have been reported [20].

In soft tissue tumours, calcifications of the lesion can be depicted (examples: ossifying fasciitis, myositis ossificans; liposarcoma, synovial sarcoma, extraosseous chondro- or osteosarcoma). In complex anatomical structures like the pelvic region, the spine, and the sternoclavicular region, cross-sectional imaging with CT is preferable. In osteoid osteoma, the nidus can also be visualized by the use of CT (Fig. 1).

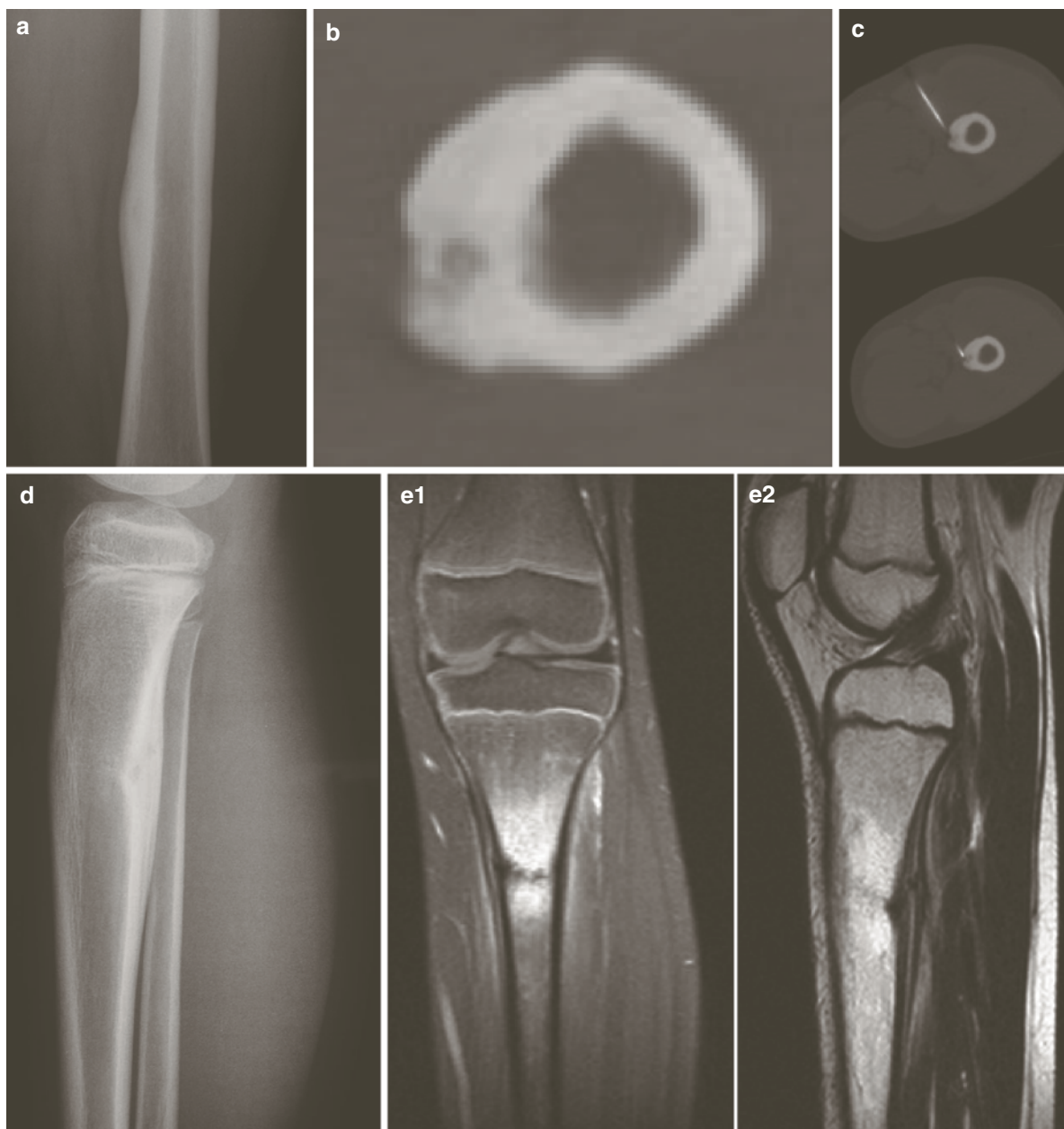
Modern Multi-detector CT systems (MDCT) allow multi-planar reconstructions.

MDCT as a cross-sectional modality also helps in planning open biopsy of osseous lesions. Often, open biopsy can even be replaced by CT-guided biopsy, which is a less invasive diagnostic modality (Fig. 2). In some cases of osteoid osteoma, pre-operative percutaneous marking of the lesion helps to find the nidus and minimize the accompanying tissue violation (Fig. 1).

R.I. Kotz (✉)

Department of Orthopaedics, Medizinische Universität Wien,  
Währinger Gürtel 18–20, 1090 Vienna, Austria  
e-mail: rainer.kotz@meduniwien.ac.at



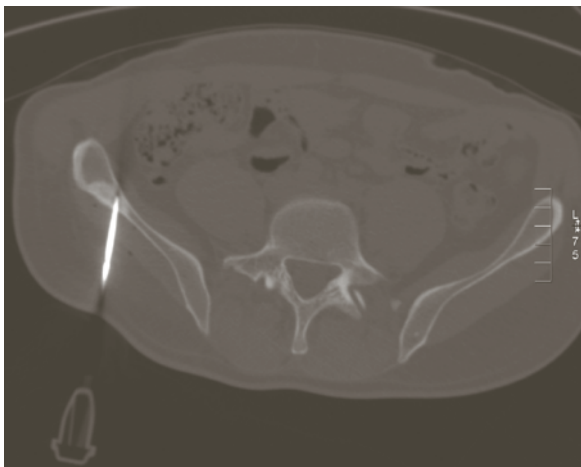


**Fig. 1** Osteoid osteoma of the distal diaphysis of the femur in a 29 year old male patient. He complained of night pain, and his symptoms typically declined after the application of aspirine. **(a)** projection radiograph, with extensive fusiform cortical thickening. **(b)** MSCT of the tumor, showing a small nidus with central calcification within the sclerosis **(c)** The lesion was preoperatively marked by CT guidance. Differential diagnosis:

Conventional staging CT (MSCT of the thorax and abdomen) initially performed is still considered state-of-the-art. However, with increasing availability of hybrid techniques, it can be expected that PET/CT will replace conventional MSCT

**(d)** Projection radiograph of the lower leg in a child with fatigue stress fracture, also leading to fusiform solid periosteal reaction. **(e)** The MRI reveals the fracture line and the edema in the coronal STIR sequence, as well as the periosteal thickening and signal alteration of the adjacent soft tissue (p.m. dorsally, as seen in the sagittal T2w sequence)

of the thorax and abdomen (see below). Multiple projection radiographs covering the whole skeleton for “whole-body” imaging as a screening method for osseous metastases have a low sensitivity (see above) and are considered obsolete.



**Fig. 2** 48 year old patient with a past medical history of bronchial carcinoma. The conventional FDG-PET had been negative, MSCT had revealed multiple sclerotic lesions in the spine and the pelvis. The CT guided biopsy of an iliacal lesion (Courtesy Prof. Dr. Czerny) confirmed a sclerotic lesion without vital tumour cells of an osteoblastic metastasis

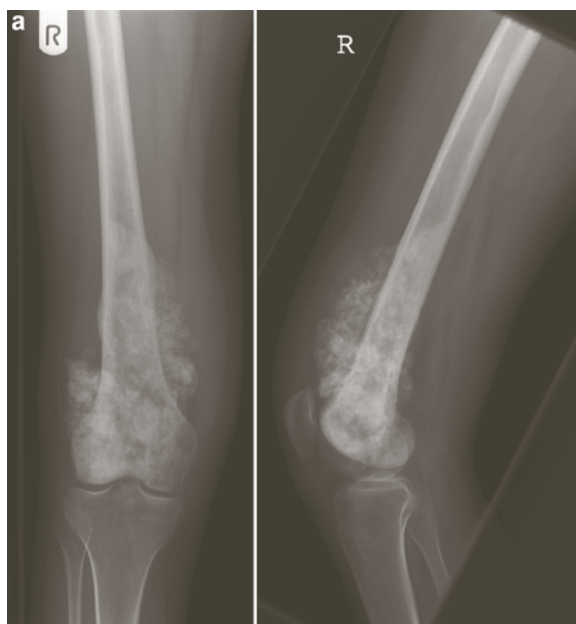
## MRI

MRI is the imaging method which provides the best tissue contrast.

Local MRI of the lesion is used to demonstrate the tumour extent with bone marrow replacement (Fig. 3), and the soft tissue component of osseous lesions (Figs. 4 and 5). It also helps in the assessment of compartmental infiltration, joint invasion, and the relation to neurovascular structures. It is also the modality of choice for soft tissue tumours (Fig. 6).

Routine protocols include fluid-sensitive, fat-saturated sequences. Here, the most sensitive sequence is the inversion recovery (IR) sequence. On modern MR scanners, proton density (PD) fat-saturated sequences are often used, and have replaced the T2w fat-saturated sequences. On both sequences, most tumours appear hyper-intense. Typically, tumours with a chondroid or myxoid matrix are especially hyper-intense. If present, surrounding oedema is diffusely hyper-intense. T1w sequences should also be performed. Here, most tumours show a signal similar to the muscle. The tissue types which are typically bright on T1w sequences are fat, methaemoglobin, melanin and some proteinaceous fluids [15]. T1w sequences serve as very sensitive sequence for the assessment of bone marrow involvement. Especially in the fatty marrow the replacement of the hyper-intense bone marrow by the hypo-intense tumour leads to an excellent contrast between those tissues. Oedema (as fluid) is also hypo-intense on T1. T1 is the

sequence used for the evaluation of contrast enhancement (it has proved useful to perform a T1w sequence in the same plane before and after the administration of contrast agent, to assess the extent of enhancement, followed by at least one fat-saturated sequence to image the areas of contrast enhancement more sensitively. Here, the axial plane is often most useful for the depiction of the relation to the adjacent tissue. On conventional T2-weighted sequences, fat and fluid are both hyper-intense. Therefore, the contrast between the tumour and surrounding fat of bone marrow or soft tissue is very low. The sequence is useful for the assessment of fibrotic or sclerotic areas (to differentiate calcified from fibrotic areas, to date, the images have to be correlated with radiographs/CT. The cortex of the bone, as well as tendons and ligaments are also hypo-intense on



**Fig. 3** G3 Osteosarcoma of the right distal femur in a 19 year old female patient, non-responder after preoperative Chemotherapy. **(a)** Projection radiograph (anterior/posterior and laterlal view) reveals signs of a malignant tumour with matrix calcifications typical of osteosarcoma. **(b)** MRI images with coronal STIR, coronal T1w and axial T2w sequence before contrast agent, and coronal T1w, followed by axial T1w images with fat saturation after the application of Gadolinium-containing contrast agent administration. The extent of bone marrow infiltration can be depicted, as well as the cortical destruction and the soft tissue involvement. **(c)** (Courtesy Prof. Dr. Susanna Lang) Macroscopic view of the resection specimen of another patient with an osteosarcoma of the distal femur shows the typical location in the metadiaphysis with heterogeneous appearance. The demarcation of the tumour against the normal fatty marrow is depicted

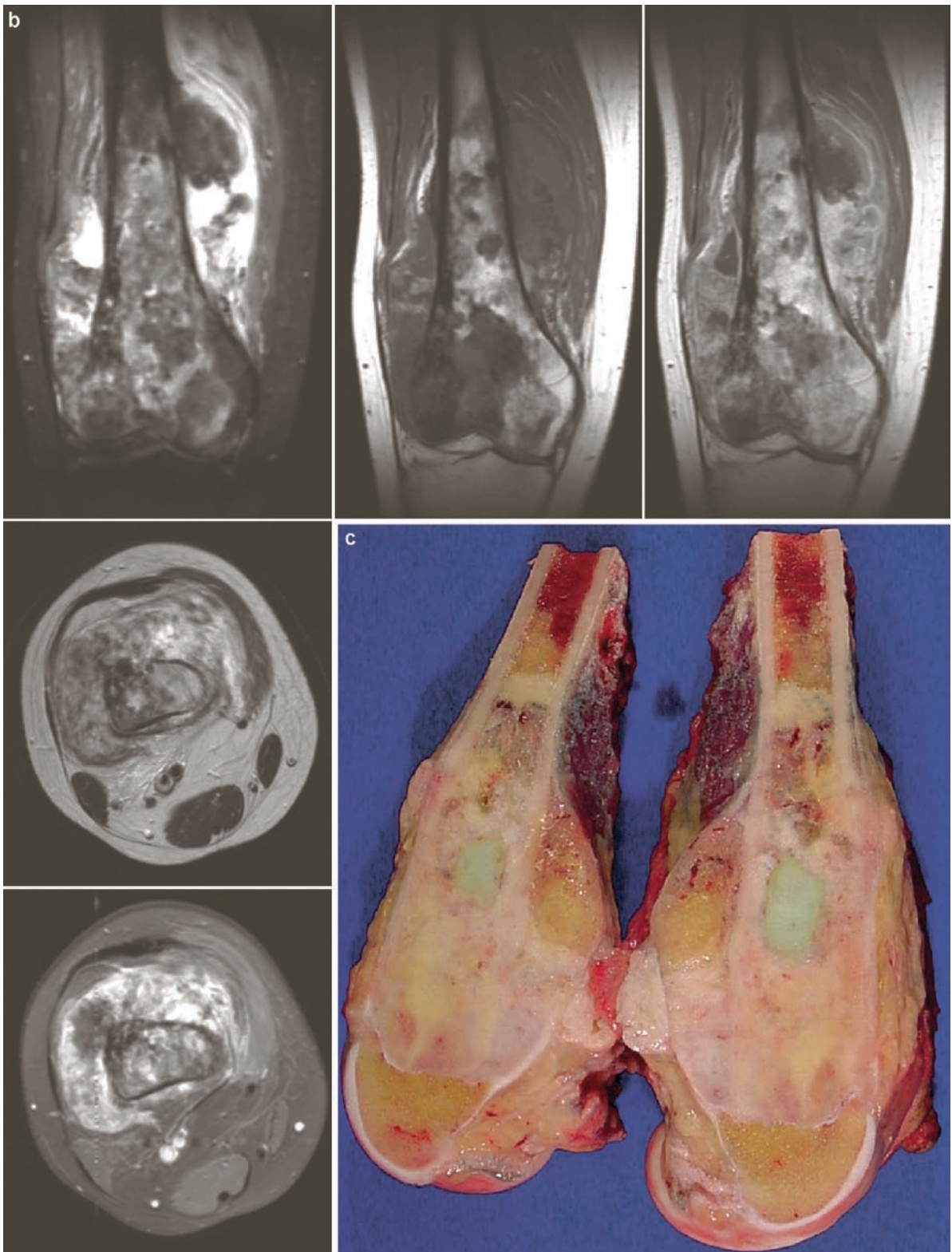
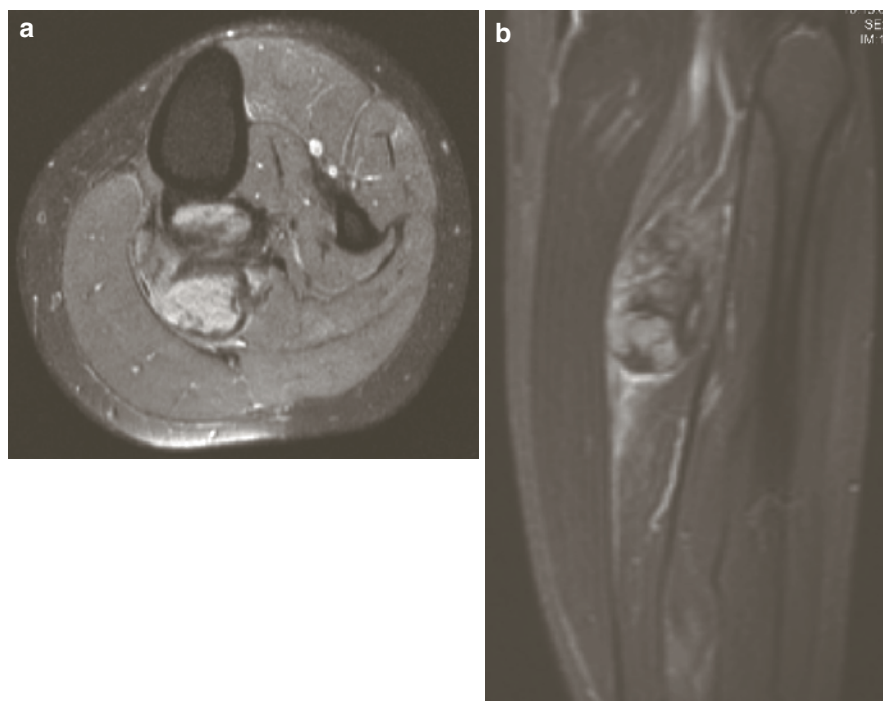


Fig. 3 (continued)



**Fig. 6** 25 year old patient with a past medical history of aggressive fibromatosis of the left upper leg and the left lower leg, which both had been resected. **(a)** Axial proton density fat saturate MR images of the left lower leg show the typically heterogeneous tumour with fibrous hypointense components. **(b)** The coronal T1w FS images after the application of contrast agent show an adjacent fascial enhancement. Recurrent fibromatosis was confirmed histologically



T2w. Alterations and infiltration of those structures can be depicted with T2. Apart from this basic protocol, additional sequences like the assessment of dynamic contrast enhancement (DEMRI) [47] sequences, diffusion weighted sequences (DWI), which show the diffusion capacity of tissue [26], or spectroscopy [22] are sometimes useful for the planning of the operation, MR-angiography sequences or CT-angiography have proved useful.

For staging in tumours like osteosarcoma, “skip lesion MRI”, which is MRI of the lower limbs, can be performed additionally. The area imaged is much bigger, and the spatial resolution is lower. This problem has become less pronounced by the use of modern MR systems with field strengths of 1.5 or 3 T, and modern coils. Still, this examination can not replace the local MRI of the tumour.

Whole-body MRI has become feasible by the use of scanners of 1.5 T and surface coil arrays, which allow accelerated image protocols (parallel imaging). To date, the examination time is about 40–50 min and is thus tolerable for the patient.

A typical protocol includes T1w and STIR images, which are performed in the coronal plane over the shoulder girdle and the thorax (using a breath-hold technique with respiratory gating), the pelvis and the legs, and in the axial plane over the head, and axial STIR images over the thorax, each with a slice thickness of 5.0 mm. 3.0 mm. thick sagittal T1w and STIR sequences over the whole spine (in two steps) complete the protocol (no contrast agent is applied) (Figs. 7 and 8) [11].

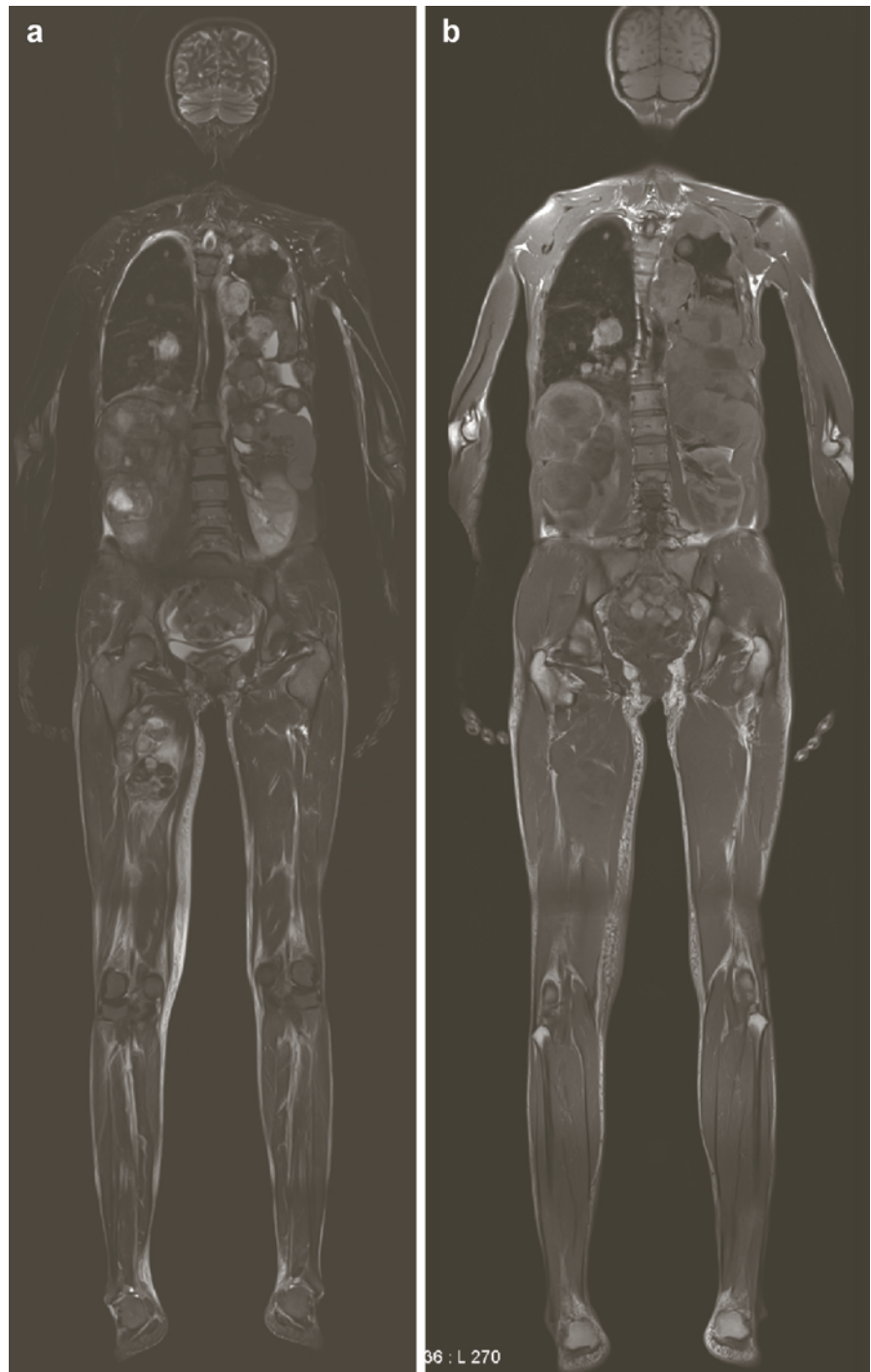
Recently, the fusion of MRI with CT has become feasible [48]. As fluoroscopy is available during the operation, the method might help the surgeon in theatre to better estimate the soft tissue borders of an invasive tumour.

MR-guided biopsy is a less invasive method than open biopsy, which is possible in soft tissue tumours or tumours with cortical destruction and adjacent soft tissue invasion. It enables selection of regions which appear most aggressive, or at least which enhance contrast agent and represent viable, non-necrotic tumour tissue (Fig. 9).

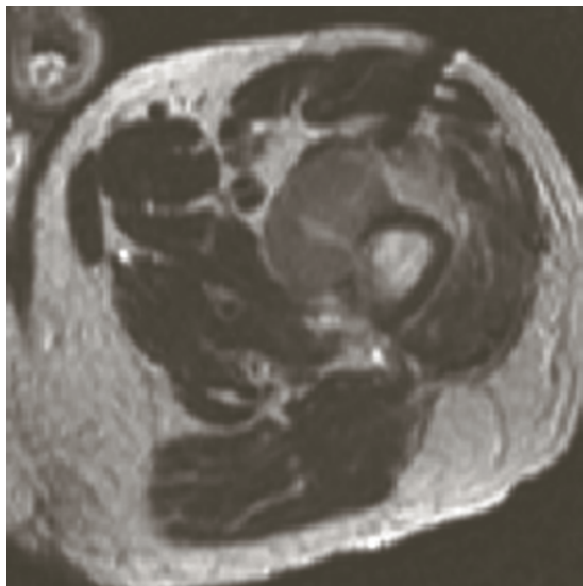
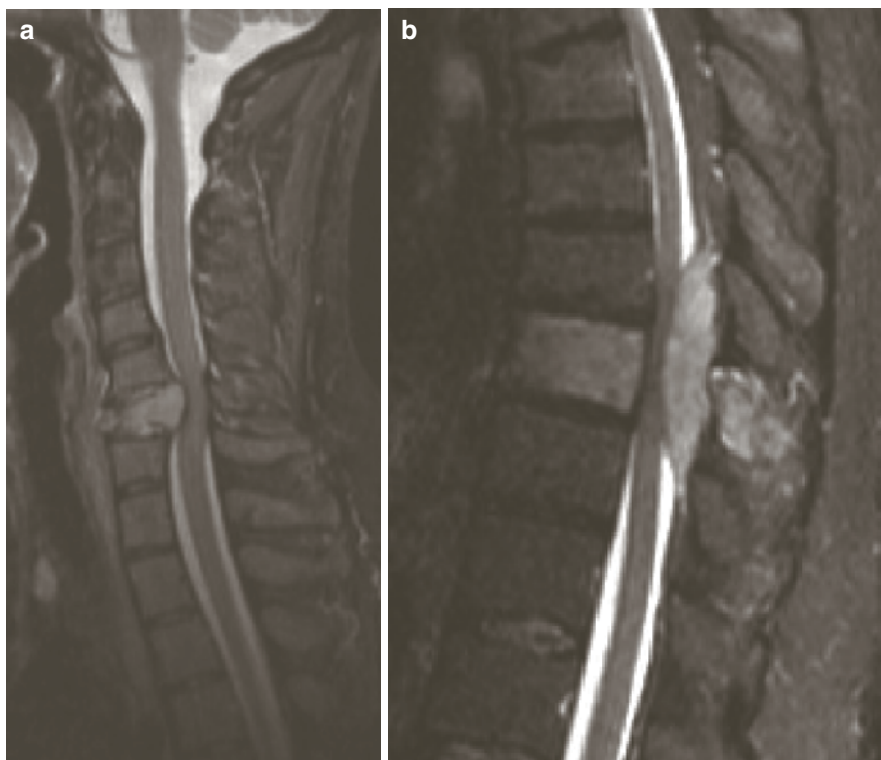
### Ultrasound

To date, ultrasound plays a minor role in the imaging of MSK tumours. Advantages of ultrasound are the easy access, wide availability, and the low costs. In recent years, ultrasound contrast agents have been developed, which help to gain more information on the vascularization of soft tissue tumours (contrast-enhanced ultrasound, CEUS) [31]. The major disadvantage of ultrasound in MSK tumours is the fact that bone and the main parts of the joints cannot be assessed. Moreover, the method is highly dependent on the experience of the investigator, leading to reproducibility limitations. It is an established method for the assessment of inguinal lymph node metastases. In some centres, soft issue tumour biopsy is regularly performed [16].

**Fig. 7** Whole body MRI in a 37 year old patient with a myxofibrosarcoma of the right upper leg. **(a)** The coronal STIR, and **(b)** coronal T1w images are able to show the primary tumour together with extensive metastases, especially in the liver and lung, and lymph nodes



**Fig. 8** MRI of the spine (which is performed in patients with neurological symptoms and sagittal STIR and T1w sequences also being part of the whole body MR protocol) **(a)** Sagittal T2w sequence of the cervical spine in a 49 year old patient with multiple myeloma, with a fracture of C6 and the tumour tissue protruding over the dorsal alignment (“butter sign”). **(b)** Sagittal STIR sequence over the thoracic spine, revealing extradural tumour growth in a 65 year old patient with known prostate cancer and scintigraphically positive osseous metastases. Both metastases lead to absolute central spinal canal stenosis



**Fig. 9** MR guided biopsy is regularly performed in soft tissue tumours, but is also possible in bone tumours with a soft tissue component. The biopsy approach was chosen together with the orthopaedic surgeons; a fast T2w sequence was used to control the needle. Histology revealed lymphoma

## Scintigraphy

Scintigraphy is a very well-established functional method. The widely available cost-effective technique allows whole body imaging within a reasonable examination time.

Both the most commonly used tracer, technetium-99m methylene diphosphonate (Tc-99m MDP), and also 18F-fluoride, a bone targeting PET tracer, very sensitively reflect osteoblastic activity [12, 20]. Only 5–10% change in the lesion, compared to normal bone, can be detected [12]. Scintigraphy may be negative in an early stage of the disease without osteoblastic response [19].

Purely lytic lesions may also be negative or only weakly positive in scintigraphy. Typically these include multiple myeloma, or aggressive metastatic lesions with rapid bone destruction without or with only minimal osteoblastic reaction.

Scintigraphy is widely used in the assessment of osseous metastases. The presence of multiple bone lesions narrows the differential diagnosis and has an impact on therapeutic options. Scintigraphy is also valuable for the detection of high stress area lesions (e.g. in the spine, or the hip).

It is still widely used to decide whether a biopsy is indicated in an osseous lesion or not. This is due to the fact that active

lesions can be differentiated from inactive lesions (the latter including entities like fibrous dysplasia, NOF, enchondroma). Minimal activity indicates that a non-malignant process is likely [24]. This is especially helpful in chondroid tumours [34]. Conversion from inactive to active lesions is indicative of malignant transformation (but also might represent lesions “burning out” or pathological fractures).

With the use of rotating detectors, it has become possible to acquire three-dimensional bone scan images, i.e. Single Photon Emission Computed Tomography (SPECT). SPECT was able to detect 20–50% more bone lesions in the spine than the planar scintigraphy [23]. For the detection of osseous metastases, the sensitivity, specificity, positive predictive value and negative predictive value of SPECT have been reported to be 91, 93, 78 and 98%, respectively [41].

## PET, PET/CT

### PET

Positron Emission Tomography (PET) represents a non-invasive functional nuclear medical imaging technique that allows the assessment of tissue blood flow and metabolism. Biologically relevant structures are labelled with short-lived positron-emitting radionuclides [2]. The positron ( $e^+$ , a positively-charged electron) interacts with a nearby electron. The masses of these particles are converted into two high-energy photons of 511 KeV each (that means, radiation energy, called annihilation radiation), moving in opposite directions. An electric circuit with two radiation detectors placed in opposite positions registers all events “simultaneously” (within 12 ns) occurring at both detectors (coincidence detection).

The radiolabelled tracer most commonly used for PET is [ $^{18}\text{F}$ ] 2-deoxy-2-fluoro-D-glucose (FDG). FDG uptake in biological tissue reflects the glucose metabolism and can be used as a marker for cell dysfunction.  $^{18}\text{F}$ FDG is usually directly taken up into the active tumour cells. Thus, tumours are  $^{18}\text{F}$ FDG positive before secondary reactive osteoblastic changes occur (which are necessary for a positive scintigraphy).

In modern scanners, the system resolution for clinical imaging lies around 4–5 mm [8].

The most common absolute measure to quantify the  $^{18}\text{F}$ FDG avidity is the Standard Uptake Value (SUV). It is dependent on the parameters used. Shin et al. tried to differentiate benign and malignant MSK-tumours and used a cut-off mean  $\text{SUV}_{\text{max}}$  3.7 in bone tumours and 3.8 in soft tissue tumours [43].

A meta-analysis on the detection of sarcomas revealed pooled values for sensitivity, specificity and accuracy of 0.91, 0.85 and 0.88, respectively. The mean SUV was

statistically significantly different between benign and malignant mixed and soft tissue sarcomas [10], and bone tumours [8]. It was also different between low and high grade mixed sarcomas [10].

The specificity is lower, as false positive findings include a variety of non-neoplastic and benign lesions, such as rheumatoid arthritis, sarcoidosis and Langerhans cell histiocytosis, osteomyelitis, Paget’s disease, fibrous dysplasia, NOF, juvenile bone cyst +fracture, aneurysmal bone cyst, giant cell tumour, chondroblastoma, chondromyxoid fibroma, desmoplastic fibroma [7, 17]. On the other hand, in grade I osteosarcoma, grade I Ewing sarcoma, and neuro-ectodermal tumour, FDG PET may give false negative results. Considering all evaluation parameters for  $^{18}\text{F}$ FDG PET, the specificity for ruling out malignant bone tumours is high, with about 97% [17].

### PET/CT

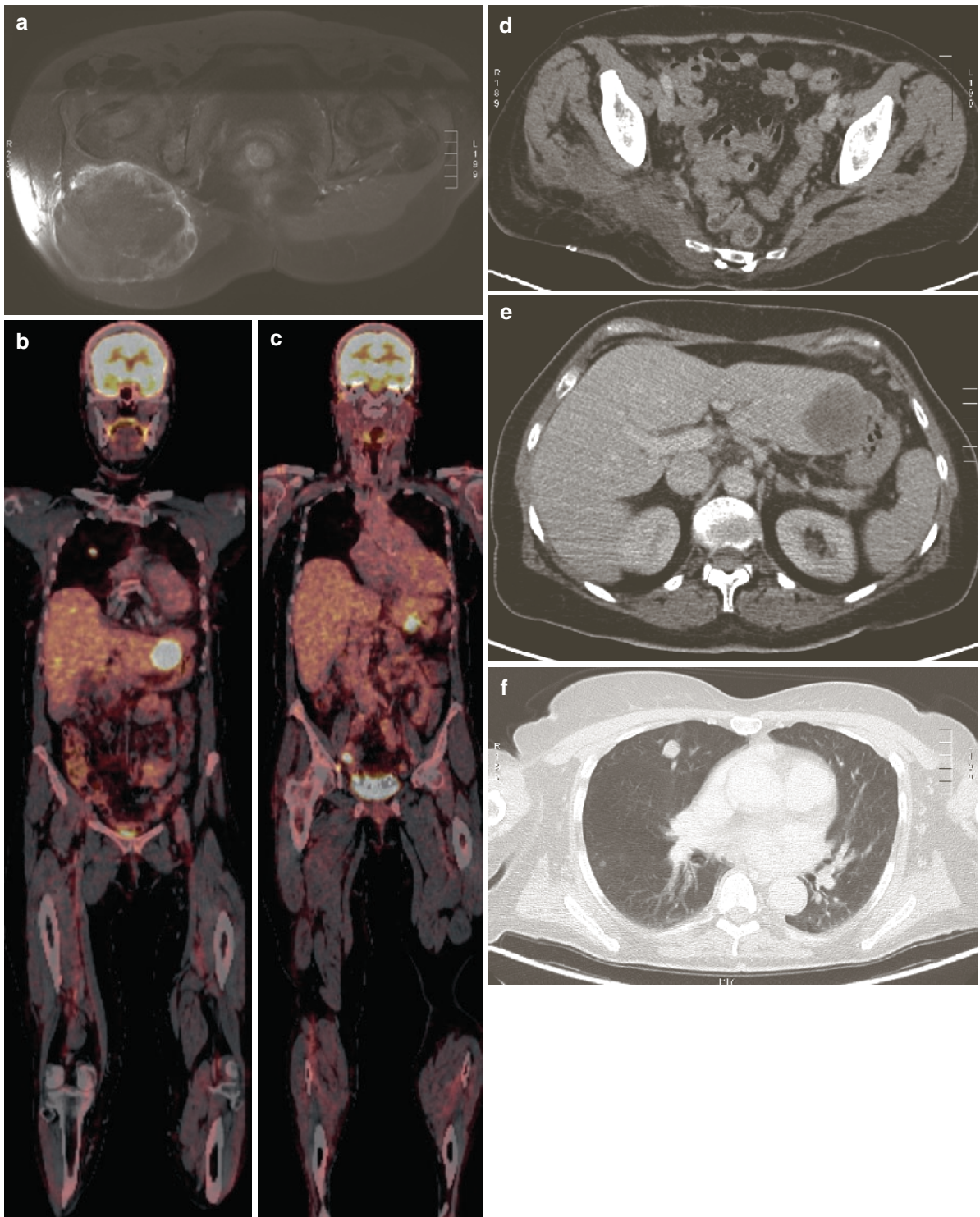
PET/CT is a hybrid modality that allows for a co-registration of the anatomical and functional information (Figs. 10 and 11), and thus enables an exact localization of increased tracer uptake [13, 46]. Modern systems use MSCT for diagnostic CT images. Contrast-enhanced CT images are acquired first. PET is acquired afterwards. The patient remains in the same position during the examination. The CT images are used to generate attenuation-correction factors to correct the effect of photon attenuation of the PET data. Finally, coregistered CT and PET images can be evaluated either separately or in a fused image mode [13].

Though FDG-PET is generally considered appropriate for all types of metastases, osteosclerotic metastases are less PET avid [42], what might be due to their relative hypocellularity, lesser aggressiveness and the lesser hypoxia. An increased  $^{18}\text{F}$ FDG uptake was found in 100% of the lytic and 88% of the osteosclerotic metastases detected on the CT part of PET/CT [37]. 13% of bone metastases are diagnosed by CT alone, whereas PET is negative [42]. Regarding primary bone tumours, a dedicated interpretation of the CT part improves the performance of FDG-PET/CT in the differentiation between benign and malignant bone tumours, increasing the sensitivity, specificity and diagnostic accuracy from 85, 35 and 68% with PET alone to 91, 77 and 86% with FDG-PET/CT [45].

## Comparison of the Different Methods and Summary

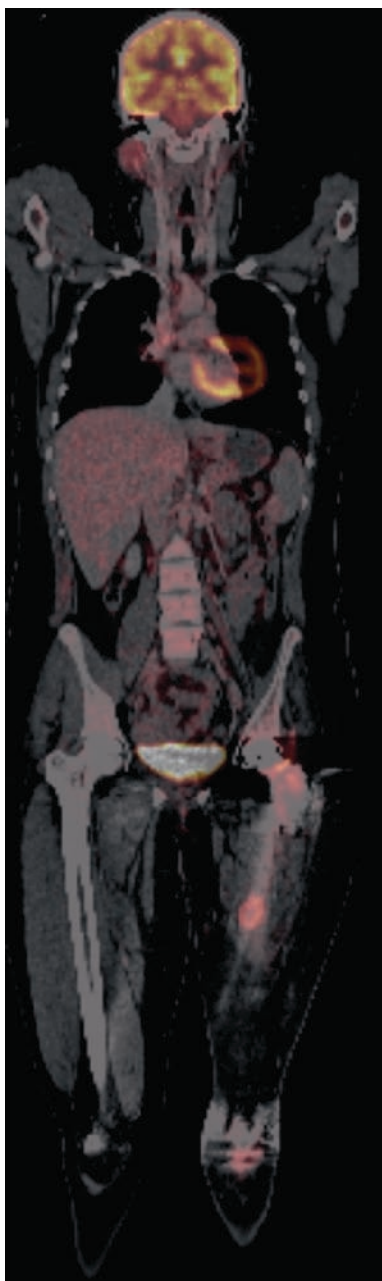
1. The local diagnosis should be performed with radiography (either projection radiography or MSCT) and MRI (the latter also provides functional information,





**Fig. 10** 64 year old female patient with a progressive swelling of the right gluteal region and pain radiating to the lower leg **(a)** In the pre-operative MRI, the axial fat saturated T1w sequence of the gluteal region shows a large soft tissue mass with peripheral contrast enhancement. Myxofibrosarcoma was confirmed by biopsy. **(b)** The FDG-PET/CT revealed several liver metastases. The largest one is shown

here, together with one of several lung metastases. **(c)** In another slice of the fused images a positive right parailiac lymph node metastasis is found. **(d)** CT evaluation of the FDG-PET/CT with diagnostic spatial resolution. The lymph node is surrounded by and might have been mistaken for the bowel. **(e)** Soft tissue window with the largest liver lesion, and **(f)** Lung window, with the largest lung metastasis



**Fig. 11** 17 year old female patient with a past medical history of osteosarcoma, and suspected recurrence in the projection radiograph. The FDG-PET/CT reveals local recurrence of an osteosarcoma of the left femur, adjacent to the tumour prosthesis. The glucose uptake of the FDG-PET is not influenced by metal artifacts

when sequences like DEMRI, diffusion or spectroscopy are performed). For functional imaging of the lesion only, scintigraphy is widely used to differentiate between active and inactive lesions and thus to help the orthopaedic surgeon in his therapeutic strategy.

2. The primary staging algorithm depends on the tumour and, in most facilities, on the availability of the imaging modalities.

If only CT, MRI and scintigraphy are available, in the assessment of skip lesions and metastases whole body MRI detects bone metastases before trabecular loss or cortical destruction is visible on CT [3]. MRI also detects spinal metastases before structural or metabolic alterations are detectable on scintigraphy [9]. It has to be kept in mind that osseous metastases, especially of renal cell carcinoma or thyroid carcinoma, may appear cold or normal on scintigraphy. Regarding the different regions, MRI proved superior to scintigraphy in bone metastases of the spine, pelvis, and the extremities; whereas in an older study with the limitations of that time, scintigraphy performed better in bone metastases of the skull and ribs [44].

If modern hybrid modalities such as PET/CT are available, there is an on-going debate about which modality should be favoured.

In the detection of osseous metastases, the overall diagnostic accuracy was found to be higher with MRI, with 91%, compared to 78% with PET/CT [42]. In several studies, the overall sensitivity was also better with MRI, compared to PET/CT, with values of 94% (MRI) vs. 78 % (PET/CT) [42], and of 85% (MRI) vs. 62% (PET/CT) [5]. The difference was even more pronounced in lesions which were smaller than 1 cm (88 vs. 56%) [42]. In contrast, the specificity of PET/CT was higher with 80%, compared to 76% in MRI [42]. Also PET/CT was superior to whole body MRI in the N-staging, and the detection of lung metastases, whereas MRI was superior in respect to liver metastases [6]. Thus, the diagnostic approach depends on the tumour type. Comparing “conventional” FDG-PET with <sup>99</sup>Tc-scintigraphy, in a study focussing on thyroid carcinoma metastases, PET/CT was superior (the sensitivity of <sup>18</sup>F FDG PET was 84.7%, compared to 78% with scintigraphy, the specificities were 99.6 vs. 91.4%, and the overall accuracy values were 97.8 vs. 89.8%, respectively) [28]. Though FDG-PET is generally considered appropriate for all types of metastases, osteosclerotic metastases may be negative with PET [42].

Recently, a new hybrid modality, PET/MRI, has been introduced. Whole-body scanners are to be expected in the near future. It is very likely that this modality will provide a more accurate TNM-stage than PET/CT or whole body MRI in musculoskeletal tumours [4].

## Monitoring Response to Therapy

After resection the definitive histological diagnosis and resection margins pave the way for adjuvant therapy. If wide resection is not possible a neo-adjuvant therapy is

applied. Re-staging follows and re-evaluation of the respectability is discussed.

The evaluation of the response to therapy for osteosarcoma in our department is evaluated by the method described by Salzer-Kuntshik [40]. Total body MRI combined with CT of the chest and abdomen are performed to complete the staging and tumour follow-up. Ideally, a PET-CT should be carried out, if available. The PET does not only reflect the internal metabolism of the tumour but allows monitoring the response to therapy [39].

Dynamic-enhanced MRI can be used for the assessment of responders in malignant musculoskeletal tumours, e.g. osteosarcoma [21, 29, 35].

As 18F-FDG is directly taken up by tumour cells, signs of response to therapy are the decrease or disappearance of uptake, in comparison to the baseline examination. This is an advantage over 99m Tc-MDP bone scintigraphy, where active disease cannot always be distinguished from repair processes [21]. Post-therapeutic lesions can remain present in CT, but not be 18F FDG avid. Those lesions may represent “burnt-out” lesions, but are also often sclerotic [27].

Post-therapeutic PET/CT results may be false negative shortly after chemotherapy (less than 2 weeks, due to a metabolic shutdown of the tumour cells). The uptake of bone exposed to a radiotherapy field may also be altered (either increased, or reduced) [14, 30]. GCSF therapy can lead to an increased tracer uptake.

In scintigraphy, healing may be indicated by a decrease in the tracer uptake intensity. However, the differentiation between ongoing disease and repair processes can be difficult, both leading to increased osteoblastic activity. Osteomyelitis, trauma, and metabolic bone disease and other non-malignant conditions also lead to positive bone scans.

Menendez et al. stated that *Thallium-201* Scanning appears to be useful in the prediction of the histological response of high-grade osteosarcomas and soft tissue sarcomas to chemotherapy.

In angiography the tumour blush or neo-vascularisation can disappear.

Though the mentioned methods proved to be useful to monitoring response to therapy, the use is limited by the high costs and lack of availability [36].

## Follow-Up Monitoring

Thorough regular follow-up is recommended, the intervals being dependent on the tumour type:

### Soft Tissue Sarcoma

	Year 1–3	Year 4–5	After year 5
Medical history	Every 4 months	Every 6 months	Once a year
CT chest	Every 4 months	Every 6 months	Once a year
MR, CT, Ultrasound or CXR of the initial TU region <sup>a</sup>	Every 4 months	Every 6 months	Once a year
Ultrasound abdomen	Every 4 months	Once a year	Once a year

### Adult Osteosarcoma

	Year 1–3	Year 4–5	After year 5
Medical history	Every 4 months	Every 6 months	Once a year
CT chest	Every 4 months	Every 6 months	Once a year
MR, CT, Ultrasound or CXR of the initial TU region <sup>a</sup>	Every 4 months	Every 6 months	Once a year
Ultrasound abdomen	Every 4 months	Every 6 months	Once a year
Bone scan	Every 6 months	Once a year	Once a year

### Adult Ewing-Sarcoma (incl. PNET)

	Year 1–3	Year 4–5	After year 5
Medical history	Every 4 months	Every 6 months	Once a year
CT chest	Every 4 months	Every 6 months	Once a year
MR, CT, Ultrasound or CXR of the initial TU region <sup>a</sup>	Every 4 months	Every 6 months	Once a year
Ultrasound abdomen	Every 4 months	Every 6 months	Once a year
Bone scan	Every 6 months	Once a year	Once a year

<sup>a</sup>Dependent on the local postoperative status of the site (e.g. metallic implants) and contraindications against certain modalities (e.g. pacemaker)

**Acknowledgement** We thank Teresa Zetl for the thorough administrative help

## References

1. "Der unerwartete Tumor aus interdisziplinärer Sicht" (The unexpected tumour from an interdisciplinary point of view). AMSOS – Austrian Musculoskeletal Oncology Society. Annual Meeting; Vienna, 21–22 November 2008
2. Homepage of Austin Hospital, Australia: <http://www.petnm.unimelb.edu.au/pet/detail/clinical.html>
3. Algra PR, Bloem L, Tissing H et al (1991) Detection of vertebral metastases: comparison between MR imaging and bone scintigraphy. *Radiographics* 11:219–232
4. Antoch G, Bockisch A (2009) Combined PET/MRI: a new dimension in whole-body oncology imaging? *Eur J Nucl Med Mol Imaging* 36:113–120
5. Antoch G, Saoudi N, Kuehl H et al (2004) Accuracy of whole-body dual-modality fluorine-18–2-fluoro-2-deoxy-D-glucose positron emission tomography and computed tomography (FDG-PET/CT) for tumour staging in solid tumours: comparison with CT and PET. *J Clin Oncol* 22:4357–4368
6. Antoch G, Vogt FM, Bockisch A et al (2004) Whole-body tumour staging: MRI or FDG-PET/CT? [Article in German]. *Radiologe* 44:882–888
7. Aoki J, Endo K, Watanabe H et al (2003) FDG-PET for evaluating musculoskeletal tumours: a review. *J Orthop Sci* 8:435–441
8. Aoki J, Watanabe H, Shinozaki T et al (2001) FDG-PET of primary benign and malignant bone tumours: standardized uptake value in 52 lesions. *Radiology* 219:774–777
9. Avrahami E, Tadmor R, Dally O et al (1989) Early MR demonstration of spinal metastases in patients with normal radiographs and CT and radionuclide bone scans. *J Comput Assist Tomogr* 13:598–602
10. Bastiaannet E, Groen H, Jager PL (2004) The value of FDG-PET in the detection, grading and response to therapy of soft tissue and bone sarcomas; a systematic review and meta-analysis. *Cancer Treat Rev* 30:83–101
11. Baur-Melnyk A, Buhmann S, Becker C et al (2008) Whole-body MRI versus whole-body MDCT for staging of multiple myeloma. *Am J Roentgenol* 190:1097–1104
12. Blake GM, Park-Holohan SJ, Cook GJ, Fogelman I (2001) Quantitative studies of bone with the use of 18F-fluoride and 99mTc-methylene diphosphonate. *Semin Nucl Med* 31:28–49
13. Blodgett TM, Meltzer CC, Townsend DW (2007) PET/CT: form and function. *Radiology* 242(2):360–385
14. Clamp A, Danson S, Nguyen H et al (2004) Assessment of therapeutic response in patients with metastatic bone disease. *Lancet Oncol* 5:607–616
15. de Kerviler E, Cuenod CA, Clement O et al (1998) What is bright on T1 MRI scans? *J Radiol* 79:117–126
16. De Schepper AM, Vanhoenacker F, Gielen J et al (eds) (2005) *Imaging of soft tissue tumours*, 3rd edn. Springer, Berlin, Germany
17. Dimitrakopoulou-Strauss A, Strauss LG, Heichel T et al (2002) The role of quantitative 18F-FDG PET studies for the differentiation of malignant and benign bone lesions. *J Nucl Med* 43:510–518
18. Edelstyn GA, Gillespie PJ, Grebbell FS (1967) The radiological demonstration of osseous metastases: experimental observations. *Clin Radiol* 18:158–162
19. Eustace S, Tello R, DeCarvalho V et al (1997) A comparison of whole-body turbo STIR MR imaging and planar 99mTc-methylene diphosphonate scintigraphy in the examination of patients with suspected skeletal metastases. *AJR* 169:1655–1661
20. Even-Sapir E (2005) Imaging of malignant bone involvement by morphologic, scintigraphic, and hybrid modalities. *J Nucl Med* 46:1356–1367
21. Even-Sapir E (2007) PET/CT in malignant bone disease. *Semin Musculoskelet Radiol* 11:312–321
22. Fayad LM, Barker PB, Bluemke DA (2007) Molecular characterization of musculoskeletal tumors by Proton MR spectroscopy. *Semin Musculoskelet Radiol* 11:240–245
23. Gates GF (1998) SPECT bone scanning of the spine. *Semin Nucl Med* 28:78–94
24. Gilday DL, Ash JM (1976) Benign bone tumours. *Semin Nucl Med* 6:33–46
25. Greenspan A, Jundt G, Remagen W (eds) (2007) *Differential diagnosis in orthopaedic oncology*, 2nd edn. Lippincott Williams & Wilkins, Philadelphia
26. Herneth AM, Guccione S, Bednarski M (2003) Apparent diffusion coefficient: a quantitative parameter for in vivo tumour characterization. *Eur J Radiol* 45:208–213
27. Israel O, Goldberg A, Nachtigal A et al (2006) FDG-PET and CT patterns of bone metastases and their relationship to previously administered anti-cancer therapy. *Eur J Nucl Med Mol Imaging* 33:1280–1284
28. Ito S, Kato K, Ikeda M et al (2007) Comparison of 18F-FDG PET and bone scintigraphy in detection of bone metastases of thyroid cancer. *J Nucl Med* 48:889–895
29. Kajihara M, Sugawara Y, Sakayama K et al (2007) Evaluation of tumour blood flow in musculoskeletal lesions: dynamic contrast-enhanced MR imaging and its possibility when monitoring the response to preoperative chemotherapy – work in progress. *Radiat Med* 25:94–105
30. Kazama T, Swanston N, Podoloff DA et al (2005) Effect of colony-stimulating factor and conventional- or high-dose chemotherapy on FDG uptake in bone marrow. *Eur J Nucl Med Mol Imaging* 32:1406–1411
31. Lassau N, Lamuraglia M, Vanel D et al (2005) Doppler us with perfusion software and contrast medium injection in the early evaluation of isolated limb perfusion of limb sarcomas: prospective study of 49 cases. *Ann Oncol* 16:1054–1060
32. Lee T (2007) Predicting failure load of the femur with simulated osteolytic defects using noninvasive imaging technique in a simplified load case. *Ann Biomed Eng* 35: 642–650
33. Lodwick GS (1966) Solitary malignant tumours of bone: the application of predictor variables in gnosis. *Semin Roentgenol* 1:293–313

34. Mankin HJ (2002) Chondrosarcoma of bone In: Mendez LR ed. Orthopedic knowledge update. Am Acad of Orthop Surgeons 187–194
35. McCarville MB (2008) New frontiers in pediatric oncologic imaging. *Cancer Imaging* 8:87–92
36. Menendez LR, Fideler BM, Mirra J (1993) Thallium-201 scanning for the evaluation of osteosarcoma and soft-tissue sarcoma. A study of the evaluation and predictability of the histological response to chemotherapy. *J Bone Joint Surg Am* 75(4):526–531
37. Metser U, Lerman H, Blank A et al (2004) Malignant involvement of the spine: assessment by 18F-Fluorodeoxyglucose PET/CT. *J Nucl Med* 45:279–284
38. Parsons TW III, Filzen TW (2004) Evaluation and staging of musculoskeletal neoplasia. *Hand Clin* 20:137–145
39. Peterson JJ (2007) F-18 FDG-PET for detection of osseous metastatic disease and staging, restaging, and monitoring response to therapy of musculoskeletal tumours. *Semin Musculoskelet Radiol* 11(3):246–260
40. Salzer-Kuntschik M, Delling G, Beron G, Sigmund R (1983) Morphological grades of regression in osteosarcoma after polychemotherapy – study COSS 80. *J Cancer Res Clin Oncol* 106 Suppl:21–24
41. Savelli G, Maffioli L, Maccauro M et al (2001) Bone-scintigraphy and the added value of SPECT (single photon emission tomography) in detecting skeletal lesions. *Q J Nucl Med* 45:27–37
42. Schmidt GP, Schoenberg SO, Schmid R et al (2007) Screening for bone metastases: whole-body MRI using a 32-channel system versus dual-modality PET-CT. *Eur Radiol* 17:939–949
43. Shin D-S, Shon O-J, Han D-S (2008) The clinical efficacy of 18F-FDG-PET/CT in benign and malignant musculoskeletal tumours. *Ann Nucl Med* 22:603–609
44. Steinborn MM, Heuck AF, Tiling R et al (1999) Whole-body bone marrow MRI in patients with metastatic disease to the skeletal system. *J Comput Assist Tomogr* 23: 123–129
45. Strobel K, Exner UE, Stumpe KDM et al (2008) The additional value of CT images interpretation in the differential diagnosis of benign vs. malignant primary bone lesions with 18F-FDG-PET/CT. *Eur J Nucl Med Mol Imaging* 35:2000–2008
46. Townsend DW, Beyer T, Blodgett TM et al (2003) PET/CT scanners: a hardware approach to image fusion. *Semin Nucl Med* 33:193–204
47. Van Rijswijk CS, Geirnaerd MJ, Hogendoorn PC et al (2004) Soft-tissue tumours: value of static and dynamic gadopentetate dimeglumine-enhanced MR imaging in prediction of malignancy. *Radiology* 233:493–502
48. Wong KC, Kumta SM, Antonio GE et al (2008) Image fusion for computer-assisted bone tumor surgery. *Clin Orthop Relat Res* 466:2533–2541

# Diagnosis and Treatment of Soft Tissue Sarcomas

Miklós Szendrői, Zoltan Sági, Kinga Karlinger, and Zsuzsa Pápai

## Introduction

Soft tissue tumours form a very heterogeneous group containing more than 200 clinico-pathological sub-types [15]. According to the WHO classification, all tumours originating from non-epithelial tissues, all extra-skeletal bone and cartilage-forming tumours, tumours of the peripheral nerves and neuro-ectodermal tissues are discussed here except those that develop from the reticuloendothelial system, the glia and parenchymal connective tissue.

Whilst the benign soft tissue tumours are relatively common, the soft tissue sarcomas (STS) are rare, their distribution being 100:1. The annual incidence of STS is around 30/1 million inhabitants. They comprise less than 1% of all malignant tumours in adults but 15% of all paediatric malignancies [32]. The consequence of this is that the general pathologist and orthopaedic surgeon may not meet these types of tumours for years. The rarity of the STS underlines that these patients should be sent to tumour centres where enough experience is accumulated and specialized pathologists, surgeons, oncologists and radiotherapists give interdisciplinary treatment according to multi-modal protocols.

## Clinical Features

Up to 95–99% of benign connective tissue tumours are superficially located and are less than 5 cm in size, 70% of the STS are deep-seated with a median diameter of 9 cm [17]. About 75% are located in the extremities (two-thirds in the lower, one-third in the upper limb), 10% in the head

and neck and 15% in the retroperitoneum, abdomen or chest wall. STS becomes more common with increasing age (median age: 60–65 years). The age-related incidences vary and may be characteristic for some of the STS: the embryonal sub-type of rhabdomyosarcoma occurs almost exclusively in children, synovial sarcoma in young adults and liposarcoma in elderly patients. Ten to fifteen percent of the patients present with distant metastases at the first diagnosis.

Although there are as many as 50 sub-types of STS, liposarcoma, malignant fibrous histiocytoma, synovial sarcoma, fibromyxosarcoma, leiomyosarcoma and malignant peripheral nerve sheath tumour are the most common types comprising about 70% of all STS occurring in adult patients. In children and adolescents, however, the distribution of STS is rather different. According to the data [25] of the Paediatric Tumour Registry in Kiel, STS in children comprise about 14% of all malignancies and the most frequent tumours are: rhabdomyosarcoma (42%), pPNET (16%), extra-osseous Ewing's sarcoma (9%) and synovial sarcoma (5%).

Most STS in the extremity present as a painless, firm mass which in its early stages does not influence the general health or any vital function of the patient. The laboratory values are usually within the normal range. The patient often connects the appearance of the tumour with a trauma event and observes it for months before visiting his doctor. Additionally, some kind of tumour-like synovial sarcoma can be present for months or years like a nodule without changing apparently his size. The seemingly innocent presentation of STS can lead to misinterpretation as benign condition and in a significant number of cases these lumps are marginally excised without previous biopsy.

Disturbance of the blood supply of the extremity or contracture in a joint are late symptoms of a deep seated STS. Superficially-located STS are recognized at an earlier stage which explains their smaller size (median 5 cm).

---

M. Szendrői (✉)  
Semmelweis University Budapest,  
Karolina ut 27  
1113 Budapest, Hungary  
e-mail: szenmik@orto.sote.hu

## Diagnostic Algorithm and Imaging of the STS

It is important that the patient presenting with a firm mass in the soft tissues goes through a standard procedure to obtain a correct diagnosis and as many relevant investigations as possible to optimise planning of further treatment (Fig. 1). Evaluating the case history is still of outstanding importance; the onset and duration of the symptoms and the occurrence of a traumatic event which can help to differentiate between haemorrhage or rupture of muscle from true tumours. STS with necrosis is often treated for a long time as an abscess first or can be mis-diagnosed as thrombophlebitis with limb threatening anticoagulation especially in an elderly patient when the patient has varicosities and the deep-seated tumour in the lower extremity cannot be palpated. The simple and cost-effective *ultrasound* examination is still under-utilized in clinical practice, but should be used as first-line examination in many situations. In proper clinical context it can help to differentiate between tumours and tumour stimulating lesions of the soft tissues, such as trauma-related haemorrhages, infections (abscess formation), fluid filled cystic lesions (ganglia, ruptured or intact Baker cysts) or a muscular tear. It is very useful as guidance for core-needle or aspiration biopsy for deep-seated STS.

The *plain radiograph* is still useful for detection of cortical erosion, periosteal reaction or impending pathological fracture but often soft-tissue calcification, phleboliths or a soft tissue mass can also be demonstrated near to the adjacent bone especially when the tumour is of lipomatous origin.

*Angiography* is seldom use in the diagnostic procedure having lost its importance in the epoch of MRI- and

CT-angiography. Pre-operative embolization can be useful in highly vascularized large STS.

The role of *computer tomography (CT)* is limited in the diagnosis of STS, though its additional use to MRI can be helpful in complex anatomical situations i.e. paraspinal-, midfoot-, carpal- and shoulder-girdle locations. CT is however superior to MR in detection of distant lung metastases.

*Magnetic resonance (MR)* as a multi-planar imaging method with excellent soft tissue contrast is superior to CT and should follow conventional radiography of a suspected soft-tissue mass [34]. The use of both ionising radiation sources and iodized contrast media can be avoided during this procedure. It can show the peri-tumoural oedema, the extent of soft tissue tumours and their relationship to neurovascular structures and joints. The standard gadolinium-enhanced “static” *MR images* give a tissue but not a histological differentiation. At present MRI rarely allows a specific diagnosis unless there are typical morphological features like central tissue-necrosis and infarcts with heterogeneous signal intensity on T2-weighted images in malignant lesions or smooth, well-defined margins with homogenous signal intensities in the usually small (95% less than 5 cm in size) benign tumours. The growth pattern of STS is more invasive than infiltrative and many subtypes respect their anatomical barrier for a while pushing the adjacent tissues before them.

The recently developed *dynamic contrast-enhanced MR* imaging provides information about tissue vascularity and perfusion, capillary permeability and composition of interstitial compartments. Most malignant lesions exhibit an early and peripheral enhancement because of their high vascularity, however, there is a significant overlapping between certain benign and malignant tumours, e.g. haemangioma and myositis ossificans may also show high contrast enhancement.

A new and promising diagnostic tool could be the still experimentally-used in vivo *1H MR spectroscopy* [47]. Based on the cholin content of different lesions a differentiation between benign and malignant tumours may be possible.

The use of *positron imaging with (F-18) fluorodeoxyglucose (FDG-PET)* is based on the detection of enhanced glucose metabolism of the tumours. This increases with increasing grade of malignancy or with de-differentiation of tumour cells. A further assumption is that the recurrent tumour and the metastasis will show the same increased metabolic rate (isotope uptake) than the primary lesion. New hybrid techniques i.e. *PET-CT*, which combine information about the metabolism and morphology of the STS, are promising in the detection of primary (sensitivity: 93.7%) and residual or recurrent tumours and in evaluation of chemotherapy-response of STS [7, 30, 35]. FDG PET/CT is an efficient tool in detection of the STS sub-types

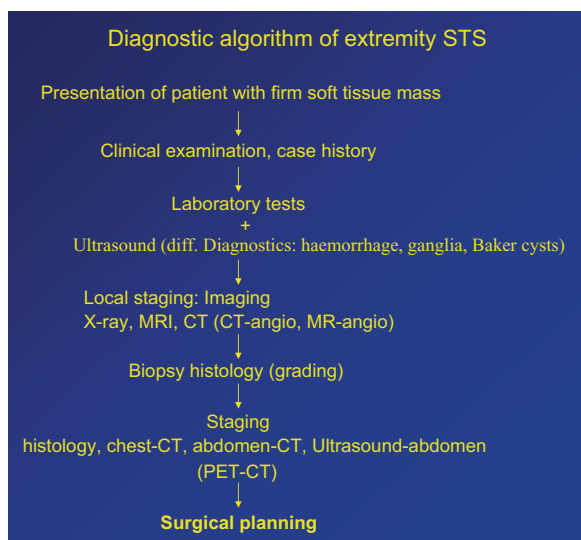


Fig. 1 Diagnostic algorithm of soft tissue sarcomas

leiomyosarcoma, Ewing's sarcoma/PNET family, fibroblastic and myoblastic sarcomas, malignant fibrohistiocyte tumours, and also in liposarcomas and synovial sarcomas.

PET-CT has limited significance in the first round of diagnostics: its use being mainly in detection of distant metastases or local recurrences.

*Biopsy* should always be performed after imaging of the STS. Core needle to a less extent, but incisional biopsy always, produce reactive changes like haemorrhage and oedema which modifies the MR and CT pictures and will interfere with later staging of the tumour.

Apart from the imaging of the STS locally, searching for distant metastases by chest and abdominal CT, ultrasound examination of the lymph nodules, bone scan and PET-CT are important for the staging of the tumour. The stage of STS predicts the survival of the patient and helps in planning the therapy.

---

## Biopsy and Histological Examination

Even in the era of modern imaging techniques where the newest dynamic contrast-enhanced MR imaging or MR-spectroscopy allow the determination of the tumour's morphological attributes (size, location, vascular relations) and may give information about the malignancy and tissue origin of soft tissue tumours, the basic procedure for establishing the malignancy, assessing the histological grade and determination of the specific histological sub-type is still the histo (cyto) pathological examination of the tumour tissue taken by biopsy. This information is fundamental for the staging of the sarcoma and important additional data for the surgical planning.

The widely-used standard technique for harvesting tissue samples is the *incisional biopsy*. The advantage of this procedure is that the surgeon can remove, under direct visual control, a large enough sample from a viable area of the tumour. It is important especially in the STS family where there are so many sub-types with different outcomes and many of the tumours consist of different cell types and can overlap each other in their histological appearance. In addition, in many cases the tumour contains a significant per cent of necrotic area which is useless for the histology. The surgeon's routine and a careful evaluation of the previous MRI pictures help to find viable areas. An additional advantage is that the tissue sample can usually be divided in different parts and saved for special examinations (electronmicroscopy, etc.) or for the tumour bank. The disadvantage of the procedure is that it is an operative intervention with bleeding, post-operative haemorrhage and infection as possible complications and the dissemination of tumour cells is more likely into the

surrounding tissues. A much wider excision of the normal tissue (in most cases the entire compartment) is necessary to obtain a sufficiently radical margin at the definitive surgery.

There are some intra-operative technical factors which must be taken into consideration to minimize the contamination of normal tissues. Incisions should be longitudinal so that the entire biopsy tract can be completely excised en bloc with the tumour at the time of definitive surgery. The surgeon should directly reach the tumour through the muscle, contamination of other compartments, especially neurovascular bundles must be avoided. Viable tissue is found usually near the pseudocapsule at the periphery. The excised chunk should contain normal tissue, reactive zone, pseudocapsule and peripheral tumour tissue as one block. Adequate haemostasis is essential and if necessary a drain is placed in the line of incision to avoid haematoma formation.

*Excisional biopsy* is only justified if the tumour is less than 2 cm. diameter and located superficially. The shelling-out operations of larger deep-seated tumours have a high risk of leaving residual tumour tissue and contamination of the normal tissues. The surgical plane is usually through the pseudocapsule. It is done usually if a benign lesion like ganglion or lipoma is assumed by its clinical appearance, however, in a significant number of cases the malignant nature of the tumour is revealed only by histology. In this case a re-excision of the tumour bed is necessary with the removal of at least 1 cm normal tissue followed by local radiotherapy.

*Core-needle biopsy* is a safer, more accurate, economical and less-invasive procedure for diagnosing soft tissue sarcomas than the incisional biopsy. Enough tissue is usually obtained to differentiate between benign and malignant tumours, to make a correct grading but it fails to predict the precise histological sub-types in one third of the cases. Heslin et al. [28] reported in their series a 95%, 88% and 75% accuracy of the core-needle biopsy correlated with the final resection diagnosis for malignancy, grade and histologic sub-type, respectively. Complications occur in less than 1% of patients. Most frequently 14–18-gauge side-cutting or tip-cutting needles are used and the procedure is performed under local anaesthesia (except in children). Careful analysis of the previous MR images, eventual use of CT or US guidance and a consultation between the surgeons, radiologist and pathologist is mandatory to avoid necrotic and cystic areas. If possible, multiple samples are taken in different parts of the lesion since STS's. are often composed of more than one histologic tissue. In the absence of adequate tissue, open biopsy is required.

*Intra-operative frozen section biopsy* is not the tool of the diagnostic procedure. Its accuracy concerning the malignancy, grade or histologic sub-type of STS does not reach that of the core-needle or incisional biopsy.



The value of frozen section histology is mostly limited to the intra-operative evaluation of the surgical margins in questionable situations of contamination.

The role of *fine-needle aspiration biopsy (FNAB)* in the diagnostic procedure of STS still provokes a controversial discussion among the experts. It is widely accepted as a tool for confirming the clinico-radiographic diagnosis of a tumour with classic presentation, for detecting recurrences or metastatic lesions of known primary tumours and for identifying abscesses, ganglions, etc. However, the frequent histopathological heterogeneity with overlapping features occurring together in the STS have discouraged many authors from recommendation FNAB in the diagnostic procedure. The reported diagnostic accuracy of FNAB ranges from 55% to 90–95% in the literature depending on the centre [1]. This high accuracy is achieved only in those special centers where experienced cytopathologists and ancillary techniques such as immunocytochemistry, fluorescence in situ hybridization (FISH) and PCR techniques are available.

The Scandinavian countries introduced FNAB decades ago. Its use is increasing as experience is gained with this procedure. The advantage of the FNAB is that it is quick, inexpensive, and minimally-invasive. It can be performed usually without any anesthesia using a 23- or 25-gauge needle and it is well tolerated by the patients. The procedure is atraumatic with minimal potential for tumours spill. Therefore a much less extensive excision of the biopsy site is radical enough at the definitive surgery.

However, if tumour grading or determination of the exact subtype of STS is essential for treatment planning (i.g. justification of pre-operative neo-adjuvant chemotherapy) FNAB has limitations. In general, the amount of material obtained by FNAB is small, and the diagnostic accuracy depends on the experience and skill of the Cytopathologist. Even if the procedure of FNAB is guided by US or CT it can be that there are no identifiable malignant cells on the glass slide. However, a negative FNAB does not confirm the absence of malignant tumour, it is only one component of an overall diagnostic procedure.

Regardless of the cytological diagnosis surgeons usually take an excisional biopsy for histology and, what is sometimes more important, the oncology team usually do not apply pre-operative chemo- or radiotherapy based on cytological diagnosis. Beyond this, it is well known that the first surgical treatment is crucial to the fate of the patient. To solve this problem (and to avoid open biopsy), it is recommended to have a much more accurate clinicopathological approach with the help of ancillary techniques.

Among the ancillary techniques, FISH has become a revolutionary technique because it makes possible the examination of interphase cells in large numbers. One of

the most suitable materials for this purpose is the fresh material gained by FNAB. If necessary, the Pathologist is able to get samples from different parts of a large STS to avoid (or just to prove) intra-tumoural heterogeneity. With the help of FISH (sensitive break-apart probes), the specific translocation, which is, in many cases very specific for the different types of soft tissue tumour, can be demonstrated [10].

They include the well-known synovial sarcoma t(X;18)(p11;q11), SYT probe (18q11.2); Ewing/PNET tumour t(11;22)(q23;q22), Ewing probe (22q12); myxoid/round cell liposarcoma t(12;16)(q13;p11), CHOP (12q13) and FUS (16p11) probes; alveolar rhabdomyosarcoma t(2;13)(q35;q14), FKHR (13q14) probe and rhabdoid tumour INI1 deletion BCR probe (22q11.2). The Ewing probe is especially useful, because the Ewing gene is also involved in many other diseases and in this way, together with the proper cytomorphology and characteristic immunocytochemistry, all these tumours can be accurately diagnosed. Such involvement can be found in clear-cell sarcoma t(12;22)(q13;q12), extra-skeletal myxoid chondrosarcoma t(9;22)(q22;q11), desmoplastic small round cell tumour t(11;22)(p13;q12), angiomatoid fibrous histiocytoma t(12;22)(q13;q12) and in round-cell liposarcoma t(12;22)(q13;q12). FISH is also useful to identify numerical alteration i.e. amplification of MDM2 and CDK4 genes which is a great help to assess whether a lipomatous tumour is a simple lipoma or it should be considered as an atypical lipomatous tumour /well-differentiated liposarcoma.

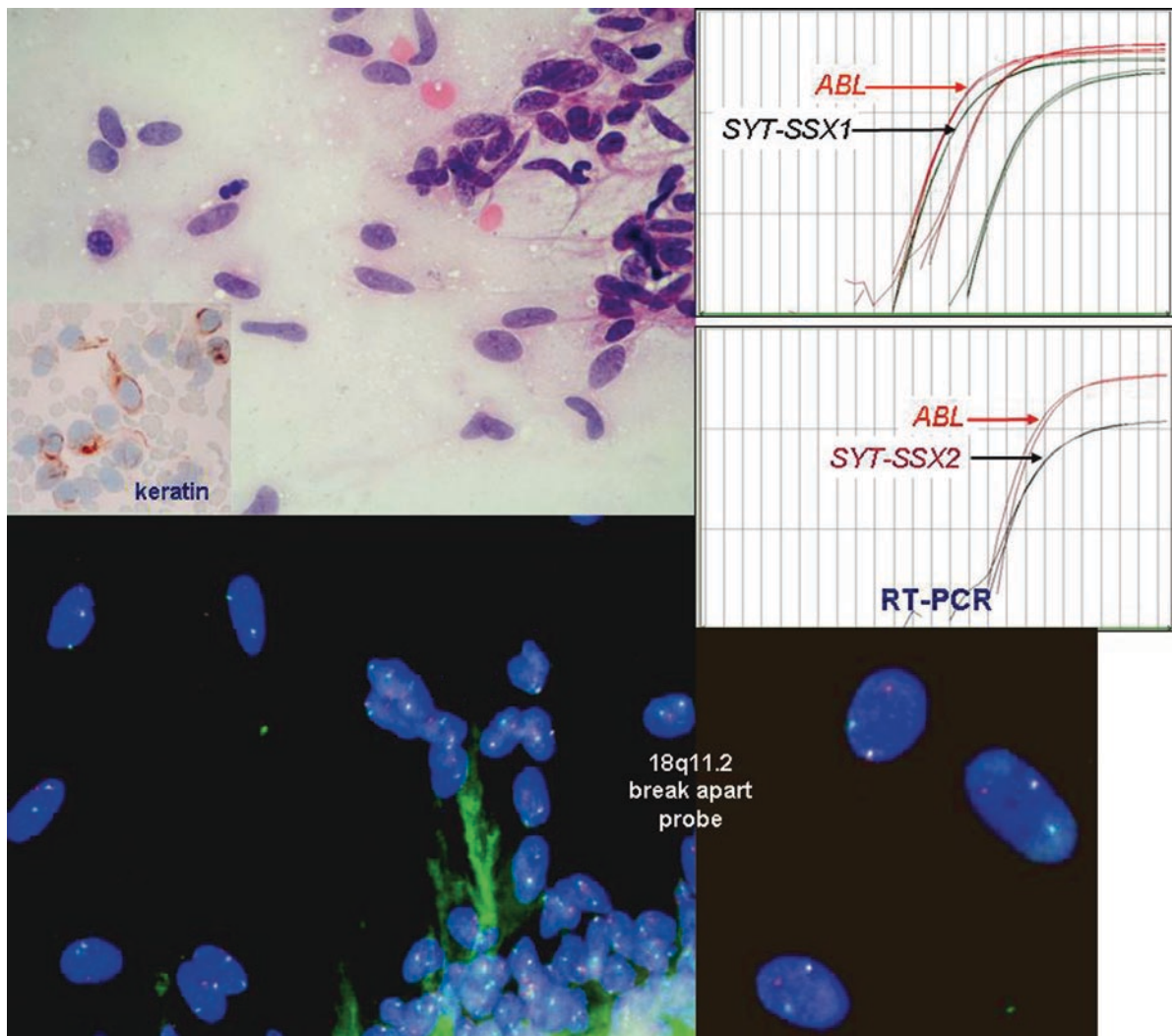
Beside FISH there is possibility to perform PCR or RT-PCR using aspiration material, and practically any kind of chimeric fusion mRNA product can be detected allowing even more specific detection of the above mentioned translocations (Fig. 2).

According to the literature [42, 48] FNAB in conjunction with ancillary techniques can correctly identify malignancy in 93–95% of the cases and histogenetic sub-typing of the STS can be achieved in approximately 54–83%.

If the FNAB findings are not consistent with the clinical and imaging findings and a malignancy is suspected a core biopsy or at least an open biopsy should be performed.

*Immunocytochemistry* also plays a crucial role in the differential diagnosis, to specify the lesions, and in cases of metastatic carcinomas and primary soft tissue lymphomas, to show if they appear as primary STS. Based on our experience we want to emphasize the use of fresh (not stored) material to avoid false negativity. This is especially true if we want standardise the circumstances for counting the Ki-67 index, which may be important to decide whether the tumour is highly malignant or not.

For practical reasons we divided the soft tissue tumours into four groups to guide the surgeon and oncologist



**Fig. 2** *Left top*: typical cytological picture of monomorph but atypical elongated tumour cells with cyokeratin positivity (insert). *Bottom*: fluorescent in situ hybridization (FISH) with 18q11.2 break apart probe, specific for the translocation of syn-

ovial sarcoma. *Right top*: more than 99% of synovial sarcomas show either SYT-SSX1 or SYT-SSX2 chimeric RNA product. Abelson (ABL) is the housekeeping gene that is used for internal control

for the best choice of operation and/or pre-operative treatment:

- High-grade malignant neoplasms (high-grade sarcomas, metastatic carcinomas, lymphoma).
- Tumours with precise histogenetic origin by cytogenetics.
- Benign tumours.
- Tumours of questionable nature.

Regarding groups a and b, pre-operative chemotherapy – for certain indications (see later)-can be useful. In this way diagnosis given by aspiration cytology provides great freedom for pre-operative and surgical treatment, thus providing the best chance for cure.

## Staging and Prognostic Factors

Different staging systems have been developed [14, 20, 23] for soft tissue sarcomas based on the most important prognostic factors which influence the survival and help the oncologic team to decide the appropriate treatment strategy for the patient. The most widely used American Joint Committee on Cancer (AJCC) system is an extension of the tumour-node-metastasis (TNM) system and is based on tumour size, depth (superficial or deep tumours), histologic malignancy, grade and distant spread (presence or absence of lymph node or other distant metastases). It accommodates

2, 3, 4-tiered histopathological grading systems (Table 1). This system applies to all STS with the exception of angiosarcoma, dermatofibrosarcoma, infantile fibrosarcoma and malignant mesenchymoma [29].

The importance of *histologic grade* in the staging system is unique to STS since it is the most important factor in predicting the risk for distant metastasis and tumour-related death [16, 18, 33]. The 10-year disease-specific survival for high grade STS in the extremity is only 55% compared with 90% for low grade lesions. High grade STS develop distant metastases in approximately 50–60%, the intermediate malignancy lesions in 25–30% and the low grade lesions in 5–10%, respectively [8].

The *size* of the tumour is defined by whether the sarcoma is smaller (T1) or greater (T2) than 5 cm in its greater dimension. Large tumours have a worse outcome concerning the overall survival of the patient [8, 23, 43, 49]. Gadgeel et al. [18] found that further stratification of the tumours led to more accurate prognostic information. Three or four groups were formed on basis of tumour size: <5 cm, 5 to <10 cm, 10 to <15 cm and >15 cm and each group was found to have a different prognosis.

The tumour size is further sub-categorized by the *depth*, as a further important prognostic factor. Superficial tumour is located exclusively above the superficial fascia, while the deep tumour is located either exclusively beneath the superficial fascia or superficial to the fascia with invasion through the fascia. Especially high grade sarcomas located

deep to the investing fascia have been shown to have a worse prognosis (Stage III) [26].

Approximately 10% of patients have already evidence of distant *metastatic disease* at the first presentation. The most common site of metastases for extremity STS is the lung (34%), bone (24%) and liver (16%) and less frequently the lymph nodes (3%) [18]. Visceral STS metastasize mostly to the liver (80%), extremity STS in approximately equal percentages to the lung. These stage IV STS are usually considered as incurable. The 5-year survival in this group varies between 5 and 20% [31]. Long-term survival associated with lymph node metastases is similar to that associated with metastasis to any other site.

The most important factor influencing *local recurrence* is the status of the surgical margin [21]. Marked increase in local recurrence have been found in patients whose surgical margins were positive for tumour cells, even when surgery was completed by postoperative chemo- and radiotherapy. Therefore a surgical re-revision is advisable in these cases whenever it is possible to perform. Further negative factors for local recurrence are the initial inadequate excision [45] patient's age greater than 50 years, high histological grade, deep location, and sub-types such as malignant peripheral nerve sheath tumour and fibrosarcoma.

Factors that predict *distant metastases* and *survival* which have been consistently found in different studies are as follows: high grade, large size, deep depth, recurrent disease at presentation, positive resection margins (?), sub-types such as leiomyosarcoma and visceral, retroperitoneal, mediastinal and distal extremity-location of the lesion. Vascular invasion, growth pattern and tumour necrosis have also been found to be adverse prognostic factors [12, 23].

**Table 1** TNM Classification of soft tissue sarcomas

Stage IA	T1a	N0,Nx	M0	Low grade
	T1b	N0,Nx	M0	Low grade
Stage IB	T2a	N0,Nx	M0	Low grade
	T2b	N0,Nx	M0	Low grade
Stage IIA	T1a	N0,Nx	M0	High grade
	T1b	N0,Nx	M0	High grade
Stage IIB	T2a	N0,Nx	M0	High grade
Stage III	T2b	N0,Nx	M0	High grade
Stage IV	Any T	N1	M0	Any grade
	Any T	Any N	M1	Any grade

T1 tumour less or equal 5 cm in greatest dimension (a superficial, b deep)

T2 tumour larger than 5 cm in greatest dimension (a superficial, b deep)

N0 no regional lymph node metastases, N1 regional lymph node metastases

M0 no distant metastasis, M1 distant metastasis

Histopathological grading: Low grade: grade 1 in three grade system, grade 1 and 2 in four grade system. High grade: grade 2 and 3 in three grade system and grade 3 and 4 in four grade system

## Local Control Rate and Survival

The effect of local recurrence on overall survival remains highly controversial according to the literature. Many authors suggested that other independent prognostic factors such as grade, size and depth of the tumour have a greater influence on the overall prognosis than local recurrence [8, 24, 38, 43, 46], while others found that local recurrence is an adverse significant risk factor for STS-specific mortality [18, 33]. This contradiction could be solved by the hypothesis that there are two types of local recurrences: one is an innocent event following an inadequate surgery of the tumour and represents a local persistence, while the other type of recurrence appear at negative margin and is a marker for a high grade aggressive lesion

[32]. This latter is supported by the result that improved local control did not translate into survival benefit.

---

### Other Factors Influencing Survival

Radiation therapy is associated with reduced local recurrence and improved survival in extremity sarcomas, whereas pre-operative chemotherapy reduces the size of the lesion and post-operative chemotherapy the rate of local recurrences, however, it does not influence the overall survival of patients [18].

*Sub-types.* Although histologic grading and extent of tumour necrosis, vascular invasion and growth pattern were proved as very strong prognostic factors [12], Hashimoto et al. [27] showed after evaluation of 1,116 patients with STS that sub-types like malignant schwannoma, liposarcoma, leiomyosarcoma and synovial sarcoma each also showed determinable factors of prognostic significance.

---

### Surgery of STS

Historically, until the late Seventies successful local control of extremity STS has been obtained mainly by ablative surgery. Resection of the entire compartment or amputation of the involved extremity resulted in local tumour control with a recurrence rate of 5–30%. Randomized trials from different tumour centres, i.g. the National Cancer Institute (NCI) of USA (1982) found, however, that the local recurrence rate after limb-saving surgery and radiotherapy was reduced to 15% only, with no difference in survival compared with ablative surgery [40]. With the introduction of multi-modality therapy (surgery plus pre- or post-operative radiotherapy, brachytherapy, chemotherapy, intra-arterial perfusion, etc.) the rates of limb-saving surgery have reached 85–95%. Despite improvements in local control rates, metastasis and tumour-related death remain still a significant problem in patients with high risk STS (sarcomas larger than 5 cm, with high histologic grade, localized at sites other than the extremities and presented already with metastasis at first recognition).

Surgical resection with negative margins remains the principal therapeutic modality in STS. The tumours should be resected en bloc with the biopsy scar within the normal tissue. STS are surrounded by a zone of compressed reactive tissue that forms a pseudocapsule around the tumour. Microscopic extension of tumour cell colonies in and beyond the pseudocapsule, however, must always be considered

at planning of the surgical excision. Contaminated margins have a consequence of high rate of local recurrence because of tumour infiltration of the pseudocapsule and reactive zone. Enneking [13] classified the different surgical procedures according their margins and this classification is now widely accepted.

---

### Surgical Margins

*Intra-capsular or intra-lesional excision* is the procedure if the tumour is curetted or excised within the lesion leaving micro- or macroscopic tumour tissue behind. It is only justified in case of benign, non-aggressive tumours, hence the local recurrence of a malignant tumour is almost certain. In cases of *marginal local excision* the surgical margin is between the reactive zone and the pseudocapsule of the tumour which contains viable tumour cells and foci. This “shell-out” procedure is performed at excisional biopsy and has a local recurrence rate of 60–80% in cases of malignant lesions. *Wide excision* or resection means that the tumour is excised en bloc with a wide surgical margin in the normal tissue but within the muscular compartment. In cases of high grade large STS, skip metastases may be present occasionally in the remaining muscle tissue but using multi-modality therapy e.g. post-operative radiotherapy, the rate of local recurrence is low, varying from 10 to 30%. Wide excision is mostly used and acceptable radical procedure in most cases of STS. *Radical resection* means by definition that the entire anatomical compartment with the tumour inside must be resected beyond the fascia together with the biopsy track.

Amputation is thought to be a radical intervention, although it is only a type of surgical procedure and should also be classified by its radicality as intra-capsular or de-bulking amputation, marginal-, wide or radical amputation/disarticulation.

These definitions mentioned above, especially the term “wide” is rather subjective. Most authors use the term “wide” if the resected tumour is surrounded by 1.5–2.5 cm of normal surrounding tissue [29]. Gronchi et al. [21] categorized the surgical margin as negative by: absence of tumour cells within 1 mm. from the linked surface. Obviously it is not a wide resection and it is also questionable how precisely can the Pathologist examine the whole surface of the tumour mm by mm.

It is characteristic for STS that its extension follows the path of least resistance, principally along the muscle fibres and neurovascular bundles. On the other hand, fascial planes, periosteum, joint capsules or even tendon sheets,

perivascular or perineural sheets all form natural barriers to local extension of malignant tumours. The tumour tends to grow proximally and distally rather than crossing fascial septal planes or invading the cortical bone and joint. Therefore it is obvious that whenever possible a 1–2-cm thick muscle layer should surround the resected sarcoma. However, when the tumour reaches the fascia or periosteum of the adjacent bone, a “wide” resection is performed if the 1–2 mm thick fascial plane or periosteum is also excised together with the tumour. Furthermore, the definition of the term “wide” is also influenced by the biological nature, grade and aggressiveness of the tumour. The term “wide” also has different meaning for the surgical planning whether a low grade liposarcoma or a large high grade synovial sarcoma must be excised.

Grossly positive post-operative margins result in local failure rates of up to 80%, whilst marginal resection gives 20%, [6] which decreases when wide or radical resections are performed to 20% and 5%, respectively. Local recurrences appear mostly in the first two post-operative years.

A small percentage of *STS*'s arise *superficial* to the fascia in the subcutaneous tissues. Most of these tumours are less than 5 cm in size and predominantly low grade. Therefore they can be treated by surgery alone and additional radiotherapy is seldom necessary [39]. The 5-year overall survival reported in the literature is between 85 and 90% [19] which is also superior to that of the remaining *STS*'s. A wide en bloc excision with a skin margin of 2–3 cms. around the protruding tumour and with a deep margin (resection line beyond the underlying fascia) should be performed. When the tumour is attached to the fascia, 1 cm of muscle cuff just deep to the fascia should be excised. If the primary closure of the skin is not possible, the usually excellent vascular bed should be covered by a split-thickness skin graft. A plastic surgeon is rarely needed.

The majority of *STS*'s grow *deep*, they invade or are beneath the superficial fascia and often exceed their anatomical barriers, ie. they are extra-compartmental. These tumours are typically large, high-grade lesions in close relationship with the adjacent bone, joint capsule or neurovascular structures. Wide resection is often only partly achieved, the histological examination of the margins reveals at least in some parts marginal margin, therefore post-operative radiotherapy and chemotherapy is mandatory to reduce the rate of local recurrence [6]. When the careful pre-operative MRI examination reveals a close attachment of the tumour to the neurovascular structures, for given indications (high-grade chemotherapy-sensitive tumours, etc.) multi-modality therapy should be strongly considered to achieve the conditions of limb-sparing surgery [29].

*Re-resection of the tumour bed* is necessary if the surgical margins are contaminated i.e. obtaining a negative margin.

Several studies [44, 51] have also documented that in about one third to one half of the cases the margins are microscopically positive when *STS*'s. were treated with simple excision by a primary physician or subsequent “unplanned” excision of a sarcoma occurred. Although the re-excision of the tumour bed is unfortunately inadequate in about 15% of the cases (still microscopical tumour cells remaining on the cut surface), the local control was significantly improved (up to 82% recurrence-free survival at 10 years), as were the disease-free survivals. The results were inferior in those cases when instead of re-resection with additional radiotherapy only post-operative radiotherapy was performed [51].

---

## Reconstructive Surgery in Deep *STS*

Involvement of the *vessels* should be carefully examined before the definitive surgery by using echo-Doppler, CT-angio. or MRI-angio. If the vessels are compressed by the tumour, but a “blunt dissection” is feasible retracting the artery is possible and the major vein can be sacrificed (if the superficial veins are preserved). However, if the adventitia of the vessels has become part of the tumoural pseudocapsule, the vessels must be resected en bloc with the tumour to achieve adequate margins and the artery (less often also the vein) reconstructed either by using autograft (contralateral saphenous vein) or artificial prosthesis. Although there is a significant rate of complications (deep vein thrombosis, embolism, wound complications, haematoma and infection), reports suggest a comparable local tumour control and distant metastases rate [41].

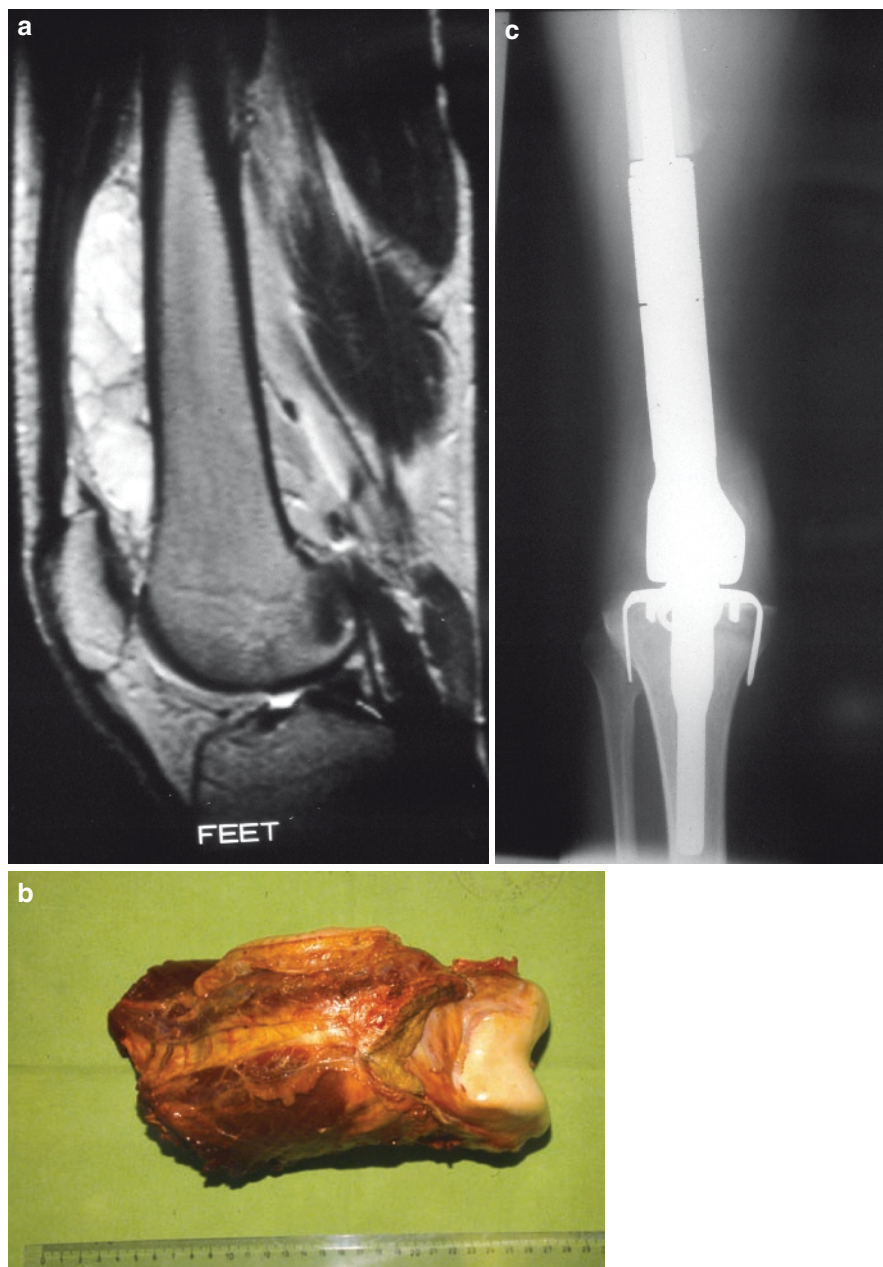
*Nerves* can also show strong adherences to the pseudocapsule of the *STS*. If the nerve is pushed by the tumour only, the epineurium (strong anatomical barrier) should be longitudinally split and the nerve peeled off and extracted. (the epineurium remains attached to the pseudocapsule). If the nerve is surrounded by the tumour, it should be resected and either replaced (nerve autograft) or not depending on the circumstances. Sometimes, even the sciatic nerve can be resected when the femoral nerves are saved. However, if the quality of life decreases significantly after resection of the nerve, an amputation should be considered. A below-knee amputation and prosthesis may provide better function (and radicality) than a preserved limb with sacrificed nerves damaged by additional radiation.

*Tissue defects* after tumour resection are often extensive, and microvascular reconstruction using free-flaps or transfer of vascularized musculocutaneous flaps are frequently required. These methods are performed by experienced plastic surgeons to achieve best results [2].

If the STS is adjacent to a *bone*, the surgeon must make the decision as to whether the bone or part of it should be removed en bloc with the tumour or not.

Clinically if the tumour reaches the periosteum it is usually not freely moveable. A very careful examination of the MRI images is necessary before the operation. If there is a close association between the pseudocapsule and periosteum (the pseudocapsule involves the periosteum), but the cortical bone and intra-medullary cavity do not show any changes in their signal and bone scan is negative, an en bloc

resection of the tumour with the adjacent periosteum – which acts as a strong barrier is recommended. At rare indication, when an aggressive benign or low grade STS is attached to the periosteum and the cortical bone shows some reaction, is that the bony segment should be cut in half and the affected part should be removed together with the periosteum and tumour. If the tumour is highly malignant and surrounds the bone (Fig. 3a–c) or the intramedullary cavity is already invaded, a segmental resection of the bone is necessary [5]. Depending on the size and site of the bone



**Fig. 3** MRI image (T1-weighted, sagittal plane) reveals a high grade T2b liposarcoma in the extensor compartment of the thigh surrounding the distal femoral metaphysis (a). Wide resection of the tumour means in that case en bloc excision of the distal part of the femur together with the tumour and 1 cm. muscle cuff (b). Modular tumour endoprosthesis was used for reconstruction of the defect (c). The patient died 3 years later from pulmonary metastases

defect either bone graft or tumour endoprosthesis can be used by the surgeon for reconstruction. Extensive stripping of the periosteum and post-operative radiotherapy increases the risk of a pathological fracture in the operative field.

---

### Treatment of Local Recurrent Tumour

The rate of local recurrence after initial surgical treatment for STS is high: 90–100% after intra-lesional and marginal excision and 30% after en bloc resection. Wide surgical margin and the introduction of radiotherapy and chemotherapy reduce this rate significantly to 8–20% [36, 50]. If the recurrent tumour is detected in its early phase, limb-sparing surgery is still possible in most patients, although the probability of further recurrences are higher after surgical re-resection. Wide surgical margins are important to achieve and additional radiation therapy might be used unless it was a part of the initial treatment. A regular follow-up of the patient is necessary for early detection of local and distant recurrences. Depending on the grade and stage of the primary STS, guidelines recommend chest imaging every 6–12 months for stage I tumours, 3–6 months for stage II and III tumours in the first 2–3 years and then annually. Post-operative scar tissue and the deep localization makes the detection of local recurrences often difficult. Contrast medium enhanced MRI is superior to CT for establishing the right diagnosis, but simple ultrasound examination and PET-CT in certain type of STS are also useful tools in the follow-up. Absence of high signal intensity excludes recurrent tumour in most cases.

---

### Surgery of Lymph Node and Other Distant Metastases

The involvement of regional lymph nodes by STS is very unlikely with most sub-types. Even tumours with a predilection for such behaviour, such as synovial sarcomas, angiosarcomas, embryonal rhabdomyosarcomas and epithelioid sarcomas produce regional lymph node metastases infrequently, ranging from 10 to 16% [4]. No studies have demonstrated any benefit of additional lymph node dissection associated with the routine surgical procedure. Given the morbidity of lymph node dissections, the rarity of its involvement and the lack of demonstrated efficacy, their excision is only recommended if the regional lymph nodes are suspicious, enlarged and the lymphoscintigraphy performed by labeled nanocolloid or the aspiration cytology are positive for metastasis.

Distant metastatic disease remains the major cause of death among patients with STS, hence the mortality rate is between 20 and 40%. In the past, lung metastasis was regarded as a fatal condition and palliative treatment was recommended only. Complete pulmonary resection can, however, achieve long term survival in approximately 15–40% of patients with a pre-requisite that the primary tumour is oncologically controlled, there are no more than four resectable nodules in the lung without endobronchial invasion, and the disease-free interval is long [9].

Patients with STS, however, who present with synchronous lung metastases have a very poor prognosis. Kane et al. [31] reported a 15 months' median overall survival and 21% 2-year survival in this group and found no improved survival for patients treated with metastasectomy vs. those treated by chemotherapy alone.

---

### Radiotherapy

Based on the evidence and positive results of different prospective studies [50], the combination of conservative (function-preserving) surgery and adjuvant radiation therapy to eliminate the microscopical tumour residuum has become standard practice in the treatment of STS. The reported 5-year and 10-year local control rate (but not the survival) increased significantly and is now between 70 and 91% [3, 36, 39]. It should be considered after resection of high-grade malignant tumours, following resection of any grade STS when the surgical margins are inadequate and a re-resection of the tumour bed is not possible, and after resection of a recurrent tumour. Low grade STS tumours with less mitotic rate have a worse response and the results of local recurrence rate do not differ significantly from that of surgery alone with wide surgical margins. Small high grade tumours ( $\leq 5$  cm) removed radical or wide in normal tissue are less frequently associated with local recurrence, therefore radiotherapy may not be necessary in these patients.

The delivery of radiotherapy (RT) in patients with STS can be performed by pre-operative RT, post-operative RT, intra-operative RT and brachytherapy. Highest doses are delivered intra-operatively as a boost (12–20 Gy), lower doses are given fractionated during post-operative RT (6,000–7,000 cGy), pre-operative RT (5,000 cGy) and brachytherapy (4,500 cGy over a 4–5-day period) [11]. Intra-operatively delivered single fraction dose is biologically more effective than would be the same dose that is fractionated. The disadvantage of this procedure includes transporting the patient during the operation to another room or may necessitate special equipment in the

operating room; in addition radiation neuritis and neuropathy can occur frequently.

The optimal timing, pre-operative or intra-operative, of radiation therapy (RT) have yet to be defined. The decision is usually made according to the preference of the treating centre, although for some indications one treatment sequence may be preferable. *Pre-operative RT* has the advantage that the treatment volume is smaller, the delivered doses are also smaller and the resectability of the tumour may increase. However, wound complications (generally reversible) are seen more commonly, surgery delays for up to 8 weeks and also the pathological examination of the surgical margins is more difficult. In cases of large high grade tumours which are closely associated with neurovascular structures, an effective pre-operative RT and chemotherapy only can enable limb-sparing surgery.

In the case of *post-operative RT* immediate surgery is possible, the risk of wound healing complication decreases. However, the late effects of radiation, in particular, tissue oedema, fibrosis, contractures around the joints are more commonly seen in patients treated with post-operative RT. It is mandatory, however, if a re-resection of the tumour bed is performed or the margins are inadequate and a re-resection is not possible.

*Interstitial brachytherapy*, which involves the placement of multiple catheters in the tumour resection bed, can be used alone or combined with post-operative high-energy external beam irradiation. The primary benefit of brachytherapy is the smaller dose, shortened treatment time (4–6 days) and irradiation of critical anatomical regions (lower arm, wrist, etc.) is minimized. Disadvantages of this procedure are the increased rate of complications (neuritis, neuropathy, infection, wound-healing problems) and the treatment volume is limited to tumour bed.

Finally, it is important to remark that neither the radiation therapy nor chemotherapy can replace the efficacy of surgery. To achieve local control after surgical resection of the tumour is mostly dependent on the negative margin.

---

## Chemotherapy

The use of chemotherapy in the treatment of STS remains controversial. There is general agreement that adjuvant chemotherapy is not indicated for patients with low-grade STS and those with small (T1) intermediate- or high-grade soft tissue sarcomas. However, in case of high grade, large, deep located, stage IIIB and stage IV soft tissue sarcomas as well as recurrent, irresectable and metastatic tumours, the patient needs to undergo multimodality therapy, i.e. the

surgery should be completed with neo-adjuvant or adjuvant chemotherapy and radiotherapy.

Treatment of locally advanced STS includes a variety of therapeutic approaches like radiotherapy alone, post-operative chemotherapy alone or in combination with radiotherapy, pre-operative chemotherapy and/or radiotherapy as well as isolated limb-perfusion.

The assessment of the *neo-adjuvant chemotherapy* is still controversial, although some authors report on encouraging results. Pre-operative systemic chemotherapy may shrink the primary tumour, resulting in improved resectability of locally advanced and otherwise irresectable tumours. It treats immediately the micrometastases and it may yield prognostic information about the chemotherapy-response of the tumour. A possible negative aspect can be the delay in surgery especially when the treatment is ineffective.

Different studies reported an improved local recurrence-free survival when a combined pre-operative chemotherapy regimen consisting of mesna, Adriamycin, ifosfamide and dacarbazine (MAID) was used. However, no benefit in overall survival could be demonstrated [26].

Several phase I and phase II studies have been conducted to present the benefit of *combined pre-operative chemo and radiotherapy*. One of them demonstrated a 3-year-survival of 75% in patients with large high grade STS when neo-adjuvant chemotherapy (three cycles MAID) was combined with 44 Gy radiation therapy. In addition, when adequate surgical margins could be achieved the rate of local recurrence was only 5.7% after 3 years. Combined neo-adjuvant chemo and radiotherapy can be recommended in patients with deep T2 high grade locally advanced STS, hence an effective treatment can result in a down-staging of the tumour enough to permit a limb-sparing surgery.

Recently, a highly successful treatment modality, the isolated limb perfusion (ILP+ hyperthermia) was implemented in patients with locally advanced STS. Conventional chemotherapeutic drugs (mainly melphalan) are used in combination with other biologic response-modifying agents i.e. tumour necrosis factor-alpha and interferon. The overall response rate was 75% and limb salvage was achieved in 87% of the perfused limbs [22]. Beside its high efficacy, severe local reactions i.e. extensive epidermolysis, local toxicity, motor neuropraxia was observed in up to 8%. Further randomized prospective studies are necessary to prove the benefit of this method on the overall survival of patients.

The role of *adjuvant* chemotherapy in the treatment protocols of STS is still an issue for debate. In different studies, the neo-adjuvant chemotherapy has been completed with the use of adjuvant treatment approaches. According to the results based on large meta-analysis studies the risk for local relapse, distant relapse and disease-free survival



showed a significant reduction following the use of doxorubicin and ifosfamide-containing chemotherapy. However, there was no difference in the overall survival compared with that of the control group. Systemic adjuvant chemotherapy (doxorubicin/ifosfamide) combined with post-operative radiotherapy can be recommended in patients with high grade (G3-4), large T2 tumours especially after inadequate surgery. Further randomized prospective studies are necessary to evaluate the optimal duration time of treatment and doses of the effective chemotherapeutic drugs in the adjuvant and neo-adjuvant chemotherapy of STS.

The palliative chemo and radiotherapy of patients with metastatic STS should prolong their overall survival and result in an improvement of the quality of life.

### The Effectivity of Different Drugs in the Treatment of STS

There are only a few drugs until now which show consistent effectivity in the treatment of STS. Among the drugs used in single-agent treatment, etoposid had a response rate of 13–15%, dacarbazine 15–17%, and cisplatin 23–25%. The two most extensively examined and most efficient drugs, doxorubicin and ifosfamide showed in mono-drug treatment a remission rate of 25–30% and 28–30%, respectively.

The effectivity of many drug-combinations have been studied in the last decades. In the Eighties, with the delivery of cyclophosphamide-, vincristin-, doxorubicin and dacarbazine-based chemotherapy (CYVADIC) a 30% remission rate could be achieved. This was followed by the combination of doxorubicin and ifosfamide in different doses and application protocols. In the last decade, the most popular combination was the MAID (methotrexate, Adriamycin, ifosfamide and dacarbazine) with an response rate of 38–40%. The effectivity of the combination of vepesid, ifosfamide and cisplatin reached 45% [37]. There is extensive research currently to work out new and more effective chemotherapeutic drug-combinations for the treatment of STS.

### Conclusions

Soft tissue sarcomas with their more than 50 sub-types belong to the most challenging group of tumours in oncology amounting to only 0.5–1% of all malignancies.

Using multi-modality therapies in dedicated centres, limb salvage and function-preserving surgery is the choice of treatment for most extremity-STS. Although the overall 5-year survival rate in patients with STS of all stages

remains only 50–60%, the majority of patients with localized lesions can be cured. The size, grade, depth and histology of the tumours strongly influence the development of metastatic disease. However, local recurrence has a minor adverse effect on the overall survival only. Pre- or post-operative radiotherapy and chemotherapy reduce the rate of local recurrence, but have little influence on overall survival. Patients with STS are best treated in specialized centres with multi-disciplinary oncology-teams.

### References

1. Akerman M, Rydholm A, Persson BM (1985) Aspiration cytology of soft tissue tumours. *Acta Orthop Scand* 56:407–412
2. Barner-Rasmussen I, Popov P, Böbling T et al (2009) Microvascular reconstruction after resection of soft tissue sarcoma of the leg. *Brit J Surg* 96:482–489
3. Beltrami G, Rüdiger HA, Mela MM et al (2008) Limb salvage surgery in combination with brachytherapy and external beam radiation for high-grade soft tissue sarcomas. *EJSO* 34:811–816
4. Brien EW, Terek RM, Geer RJ et al (1995) Treatment of soft-tissue sarcomas of the hand. *J Bone Joint Surg* 77-A:564–571
5. Capanna R, De Biase P (2003) Excision of bone tumours. In: Duparc J (ed) *Surgical techniques in orthopaedics and traumatology*. Elsevier, Philadelphia, pp 55-050-B-20
6. Chansky HA (2002) Surgical management of malignant soft-tissue tumours. In: Menendez LR (ed) *Orthopaedic knowledge update. Musculoskeletal tumours*. AAOS, Rosemont, pp 231–241
7. Charest M, Hickeys M, Lisbona R et al (2009) PET/CT imaging in primary osseous and soft tissue sarcomas: a retrospective review of 212 cases. *Eur J Nucl Med Mol Imaging*. Epub. 2009 Jul 11
8. Coindre JM, Terrier Ph, Bui NB et al (1996) Prognostic factors in adult patients with locally controlled soft tissue sarcoma: a study of 546 patients from the French Federation of Cancer Centers Sarcoma Group. *J Clin Oncol* 14:869–877
9. Cormier JN, Pollock RE (2004) Soft tissue sarcomas. *CA Cancer J Clin* 54:94–109
10. Dei Tos AP, Dal Cin P (1997) The role of cytogenetics in the classification of soft tissue tumours. *Virchows Arch* 431: 83–94
11. Einck JP, Schwartz D (2002) Radiation therapy for malignant soft-tissue tumours. In: Menendez LR (ed) *Orthopaedic knowledge update. Musculoskeletal tumours*. AAOS, Rosemont, pp 289–301
12. Engellau J, Bendahl P-O, Persson A et al (2005) Improved prognostication in soft tissue sarcoma: independent information from vascular invasion, necrosis, growth pattern, and immunostaining using whole-tumour sections and tissue microarrays. *Hum Pathol* 36:994–1002

13. Enneking WF (1983) *Musculoskeletal tumour surgery*. Churchill Livingstone, New York, Edinburgh, London
14. Enneking WF, Spanier SS, Goodman MA (1980) System for the surgical staging of musculoskeletal sarcoma. *Clin Orthop* 153:106–120
15. Enzinger FM, Weiss SW (1983) *Soft tissue tumours*. Mosby, St. Louis, Toronto, London
16. Ferrari A, Casanova M, Collini P et al (2005) Adult-type soft tissue sarcomas in pediatric-age patients: experience at the Istituto Nazionale Tumori in Milan. *J Clin Oncol* 23:4021–4030
17. Fletcher CDM, Rydholm A, Singer S (2002) Soft tissue tumours: epidemiology, clinical features, histopathological typing and grading. In: Fletcher CDM, Unni K, Mertens F (eds): *WHO classification, tumours of soft tissue and bone*. IARC Press, Lyon, pp 12–18
18. Gadgeel SM, Harlan LC, Zeruto CA et al (2009) Patterns of care in a population-based sample of soft tissue sarcoma patients in the United States. *Cancer* 115:2744–2754
19. Gibbs CP, Peabody TD, Mundt AJ et al (1997) Oncological outcomes of operative treatment of subcutaneous soft-tissue sarcomas of the extremities. *J Bone Joint Surg* 79-A: 888–897
20. Green FL, Page DL, Fleming ID et al (2002) *AJCC cancer staging manual*, 6th edn. Springer, New York
21. Gronchi A, Casali PG, Mariani L et al (2005) Status of surgical margins and prognosis in adult soft tissue sarcomas of the extremities: a series of patients treated at a single institution. *J Clin Oncol* 23:96–104
22. Grunhagen DJ, de Wilt JHW, Graveland WJ et al (2006) Outcome and prognostic factor analysis of 217 consecutive isolated limb perfusions with tumour necrosis factor- $\alpha$  and melphalan for limb-threatening soft tissue sarcoma. *Cancer* 106:1776–1784
23. Gustafson P, Akerman M, Alvegard TA et al (2003) Prognostic information in soft tissue sarcoma using tumour size, vascular invasion and microscopic tumour necrosis – the SIN-system. *Eur J Cancer* 39:1568–1576
24. Ham SJ, van der Graf WTA, Pras E, Molenaar WM et al (1998) Soft tissue sarcoma of the extremities. A multimodality diagnostic and therapeutic approach. *Cancer Treat Rev* 24:373–391
25. Harms D (1997) The pediatric tumour registry in Kiel. *Acta Orthop Scand (suppl 274)*:65
26. Hartman JT, Bauer S (2006) Soft tissue sarcoma. *Update Cancer Ther* 1:385–402
27. Hashimoto H, Daimaru Y, Takeshita S et al (1992) Prognostic significance of histologic parameters of soft tissue sarcomas. *Cancer* 70:2816–2822
28. Heslin MJ, Lewis JJ, Woodruff JM et al (1997) Core needle biopsy for diagnosis of extremity soft tissue sarcoma. *Ann Surg Oncol* 4:425–431
29. Huetan MT, Thornton K, Herman JM et al (2008) Management of extremity soft tissue sarcomas. *Surg Clin N Am* 88:539–557
30. Jagaru A, Quon A, McDougall RI et al (2006) F-18 FDG PET/CT evaluation of osseous and soft tissue sarcomas. *Clin Nucl Med* 31:754–760
31. Kane JM, Finley JW, Driscoll D et al (2002) The treatment and outcome of patients with soft tissue sarcomas and synchronous metastases. *Sarcoma* 6:69–73
32. Khatri VP, Goodnight JE (2005) Extremity soft tissue sarcoma: controversial management issues. *Surg Oncol* 14:1–9
33. Lahat G, Tuvin D, Wei C et al (2008) New perspectives for staging in soft tissue sarcoma. *Ann Surg Oncol* 15: 2739–2748
34. Maeda M, Matsumine A, Kato H et al (2007) Soft-tissue tumours evaluated by line-scan diffusion-weighted imaging: influence of myxoid matrix on the apparent diffusion coefficient. *J Magn Reson Imaging* 25:1199–1204
35. Mintzer DM, King JJ, Alavi A et al (2007) Utility of PET scan in predicting chemotherapeutic response in soft tissue sarcoma patients. *ASCO Annual Meeting Proceedings Part I. J Clin Oncol* 25(18S) (June 20 supplement):10021
36. Muhic A, Hovgaard D, Petersen MM et al (2008) Local control and survival in patients with soft tissue sarcomas treated with limb sparing surgery in combination with interstitial brachytherapy and external radiation. *Radiother Oncol* 88:382–387
37. Pápai Z, Bodoky G, Szántó J et al (2000) The efficacy of a combination of Etoposide, Ifosfamide and Cisplatin in the treatment of patients with soft tissue sarcoma. *Cancer* 89:177–200
38. Pisters PWT, Leung DHY, Woodruff J et al (1996) Analysis of prognostic factors in 1041 patients with localized soft tissue sarcomas of the extremities. *J Clin Oncol* 14: 1679–1689
39. Pisters PWT, O’Sullivan B, Maki RG (2007) Evidence-based recommendations for local therapy for soft tissue sarcomas. *J Clin Oncol* 25:1003–1008
40. Rosenberg SA, Tepper J, Glatstein E et al (1982) The treatment of soft tissue sarcomas of the extremities: prospective randomized evaluations of (1) limb-sparing surgery plus radiation therapy compared with amputation and (2) the role of adjuvant chemotherapy. *Ann Surg* 196: 305–315
41. Schwarzbach MH, Hormann Y, Hinz U et al (2005) Results of limb-sparing surgery with vascular replacement for soft tissue sarcoma in the lower extremity. *J Vasc Surg* 42: 88–97
42. Singh HK, Kilpatrick SE, Silverman JF (2004) Fine needle aspiration biopsy of soft tissue sarcomas: utility and diagnostic challenges. *Adv Anat Pathol* 11:24–37
43. Stefanovski PD, Bidoli E, De Paoli A et al (2002) Prognostic factors in soft tissue sarcomas: a study of 395 patients. *Eur J Surg Oncol* 28:153–164
44. Sugiura H, Takahashi M, Katagiri H et al (2002) Additional wide resection of malignant soft tissue tumours. *Clin Orthop* 394:201–210
45. Van Geel AN, Egermont MM, Hanssens PEJ et al (2003) Factors influencing prognosis after initial inadequate excision (IIE) for soft tissue sarcoma. *Sarcoma* 7:159–165
46. Vraa S, Keller J, Nielsen OS et al (2001) Soft tissue sarcoma of the thigh. Surgical margin influences local recurrence but not survival in 152 patients. *Acta Orthop Scand* 72:72–77

47. Wang CK, Li CW, Hsieh TJ et al (2004) Characterization of bone and soft-tissue tumours with in vivo  $^1\text{H}$  MR spectroscopy: initial results. *Radiology* 232:599–605
48. Ward WG, Savage P, Boles CA et al (2001) Fine-needle aspiration biopsy of sarcomas and related tumours. *Cancer Control* 8:232–238
49. Wunder JS, Healy JH, Davis AM et al (2000) A comparison of staging systems for localized extremity soft tissue sarcoma. *Cancer* 88:2721–2730
50. Yang JC, Chang AE, Baker AR et al (1998) Randomized prospective study of the benefit of adjuvant radiation therapy in the treatment of soft tissue sarcomas of the extremity. *J Clin Oncol* 16:197–203
51. Zagara GK, Ballo MT, Pisters PW et al (2003) Surgical margins and resection in the management of patients with soft tissue sarcoma using conservative surgery and radiation therapy. *Cancer* 97:2544–2553

**Part III**

---

**Paediatrics**

---

## Introduction

Most congenital lower limb deficiencies present with shortening of the leg and deformities of the bones and joints. Regardless of severity, a limb deformity is always a handicap for the child with a negative functional and psychological impact. The goal of treatment is to achieve a well-functioning leg with normal alignment and stability, and a normal range of motion of the joints.

We face a wide spectrum of congenital conditions, like congenital femoral deficiency (CFD), Fibula Deficiency, tibial hemimelia, congenital pseudarthrosis of the tibia, and hemi-hyper- or hypotrophy, all with different pathologies. Despite great advances in the understanding of the genetics associated with embryological limb development and of genetic signalling errors in the animal model the aetiologies of these deficiencies are still unknown [4].

However, limb length inequality or axial mal-alignment can occur any time during infancy, childhood and adolescence following infection of bones and joints or fractures with or without damage to the growth-plate. Additionally, tumors, skeletal dysplasias, Ollier's disease, Blount's disease, hypophosphataemic rickets, CDH and Perthes' disease can cause leg-length discrepancy.

During the last decade, based on the revolutionary ideas of callus distraction and osteoneogenesis of Ilizarov [15], lengthening and deformity correction has become a predictable and safe surgical procedure.

Today, limb-lengthening is usually recommended for discrepancies greater than 2–3 cm. The biological principle of limb lengthening developed by Ilizarov is based on a

percutaneous osteotomy [13]. If possible the osteotomy should be performed in the metaphysis as this is an area of the bone that has the highest metabolic turn-over and therefore yields the best formation of bone regenerate [3]. The distraction osteogenesis is ideally performed with a daily rate of lengthening of about 1 mm/day. A lower rate increases the risk of premature bone consolidation while a higher rate decreases intra-membranous ossification [14]. The mechanical lengthening device must insure stability and protect the distraction area against undesirable angular or shear movements [13]. The period of distraction is followed by a period of consolidation until corticallization of the bone regenerate has been achieved and the fixator can be removed.

Usually the healing index for full bone consolidation per cm. of lengthening is around 30 days depending on the severity of the pathology and the patient's age. Especially in congenital deformity the healing index can grow up to 50 days/cm. The removal of the frame is indicated when the regenerated bone is capable of full independent weight-bearing.

---

## Advances in Deformity Correction

The biggest advances in deformity correction have been achieved in deformity analysis and correction planning, understanding of patho-morphology, development of new technology and surgical techniques, prevention of complications and improvement of rehabilitation.

The basic requirement for treatment planning is the deformity analysis including prediction of the remaining growth in children. In cases of congenital defects leg-length discrepancy can be calculated and predicted easily because growth remains proportional [24]. For calculation of remaining growth the straight line graph introduced by Mosely [19], the arithmetic method by Menelaus and the Paley multiplier method [20] can be used.

For deformity analysis digital solutions like TraumaCad (Orthocrat, Petach-Tikva, Israel) integrated into the

---

F. Grill (✉)  
Department for Paediatric Orthopaedics,  
Orthopädisches Spital Speising,  
Speisingerstrasse 109, 1130 Vienna, Austria  
e-mail: franz.grill@oss.at

PACS-system allow measurement of the standardized angles [22] and definition of the centre of rotation of angulation (CORA) (Fig. 1a). The surgical correction can be simulated after choosing the osteotomy level and the location of the angulation correction axis (ACA) (Fig. 1b).

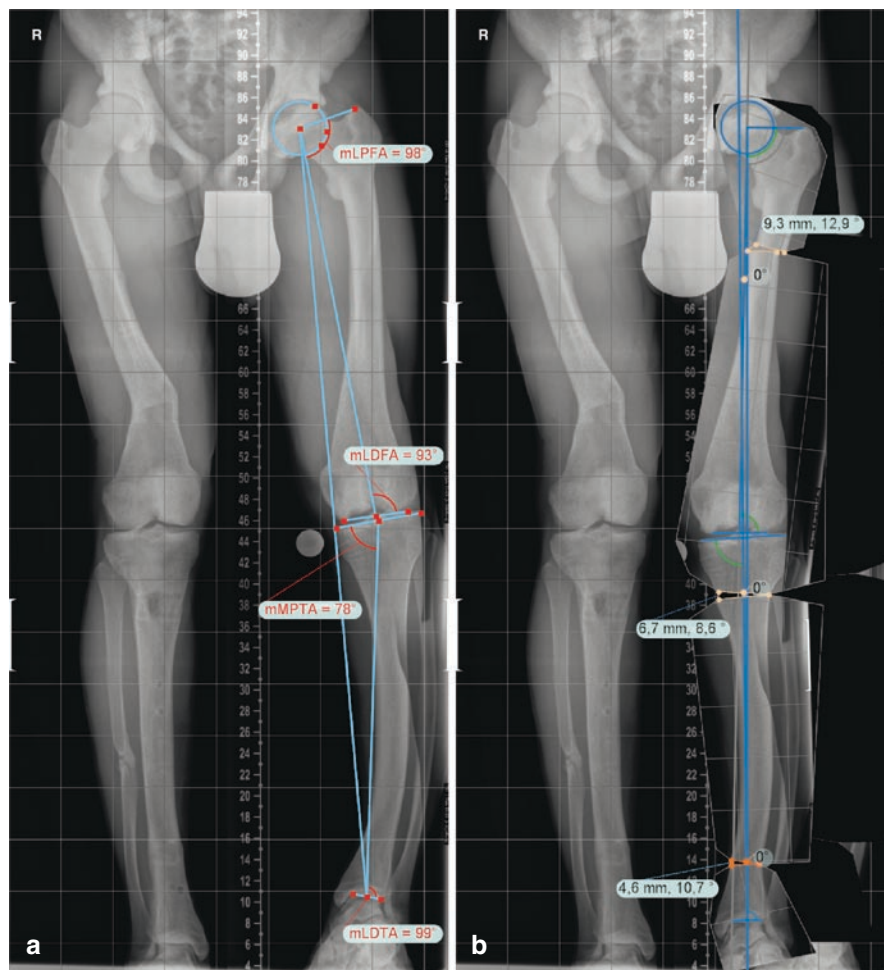
However, advances were also achieved in the development of new high-tech devices for lengthening and deformity correction. For years the use of the Ilizarov circular frame was the gold standard at most centres performing limb-lengthening. Today there is still an indication for the Ilizarov system in certain cases but in the majority of cases the Taylor Spatial Frame (Smith and Nephew, Memphis, Tennessee, USA) has replaced the conventional Ilizarov ring fixator.

The Taylor Spatial Frame is a hexapod system consisting of two rings, connected with six telescopic struts at universal joints. By adjusting only strut length one ring can be re-positioned in relation to the other. Simple and complex

deformities are treated using the same frame (Fig. 2a). The Taylor Spatial Frame fixator is capable of correcting six axis deformities guided by a web-based software program. Additionally foot deformities can be corrected with the same hexapod system using special foot programs and foot plates. The Taylor Spatial Frame is easier to handle than the conventional Ilizarov system, especially for correction of rotational and translation deformities (Fig. 2b–e). Patients find the system easy to use because a daily adjustment schedule is provided. The struts exchanges are listed and explained in chronological sequence.

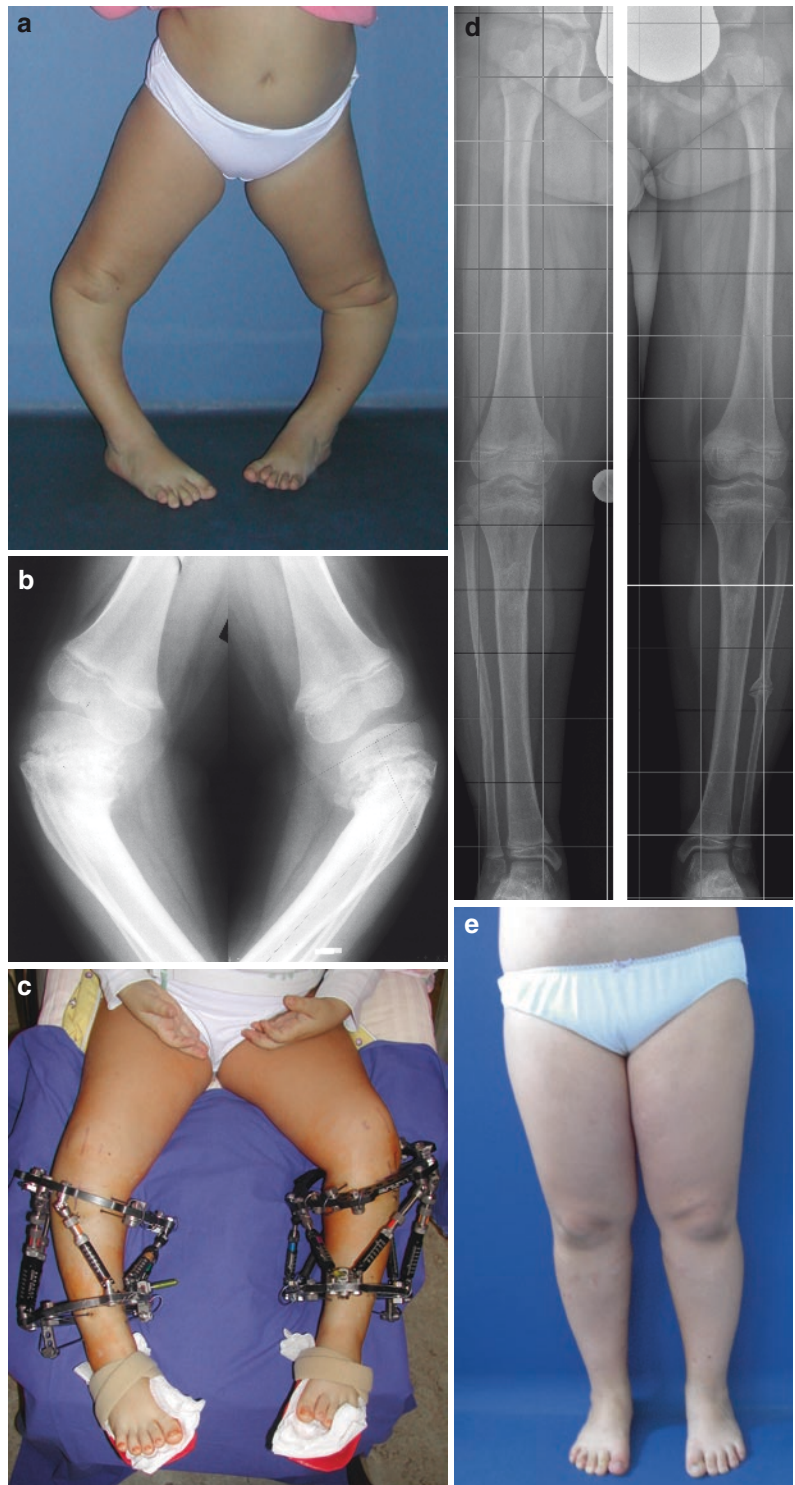
Since 1981 a total of 806 patients were treated using the Ilizarov circular frame at the department for Paediatric Orthopaedics at the Hospital Speising in Vienna. The TSF was introduced at our department in 1999 and another 502 patients have been treated using this new device since then.

Comparing the final outcome of corrections using the TSF vs. the Ilizarov system it was evident that the Taylor



**Fig. 1** (a) New planning software allows for fast and easy analysis of the deformity using the same principles and terminology drawn from conventional planning. (b) The correction can be simulated and the final alignment and joint orientation angles, as well as changes in the length of the mechanical axis, can be analyzed

**Fig. 2** (a) For this 5-year-old female presented with severe Blount's disease with varus and rotational mal-alignment. (b) Radiographs showed the mal-alignment and the collapse of the medial plateau. (c) A bilateral TSF was implanted and simultaneous gradual correction of the multi-directional deformity was performed. (d, e) Remodelling of the epiphysis (d) and a good clinical correction (e) was observed at follow-up 4 years after the initial surgery



Spatial Frame allowed for much higher accuracy in all deformity corrections [16]. The Ilizarov ring fixator showed major deficiencies in the treatment of multi-dimensional and especially rotational deformities.

Additionally, new mono-lateral devices were developed such as the MAC (Multi-Axial Correction) Fixation System (Biomet, Warsaw, Indiana, USA), which allows deformity correction in three dimensions. Mono-lateral fixators have the advantage of increased patient comfort due to the smaller fixator size and the lateral implantation allowing full hip and knee extension when lying down. However the amount of correction is limited, especially at the secondary hinge.

Despite all these improvements the striking disadvantages of external fixators is that the patient's activities of daily life are severely limited and painful pin-site infections occur in nearly all cases. The answer to this problem is the use of telescopic nails for intra-medullary lengthening which were developed in Russia [6], France (Albizzia Nail [11], United States (ISKD) [9] and Germany (Fitbone) [5].

The ISKD nail (Orthofix Int. Curaçao Netherlands Antilles) is elongated mechanically by rotational movements which the patient has to perform. The voluntary rotational movements which are necessary to lengthen are sometimes very painful and may lead to an increased lengthening rate due to uncontrolled rotational movements of the patients. Another disadvantage is that in lengthening of the tibia the callus formation is rather poor and therefore we stopped using these nails. Our current preference is the Fitbone nail (Fitbone, Wittgenstein, Igersheim, Germany), a motor-driven intra-medullary distraction nail with a power supply through a transmitter to the receiver which is embedded in the subcutaneous tissue. Using this nail an additional single level axial deformity correction can be performed (Fig. 3a–d). The advantages are a low infection risk, an excellent range of motion during the treatment period, a short rehabilitation time, an excellent cosmetic result and high comfort for the patient. Up to now we have treated 20 patients using the Fitbone nail with excellent results. The disadvantage is the high cost of the implant and the limited indications.

Regarding the timing of the lengthening procedures the calculated leg length difference at the end of growth is an important factor. Today, with a staged programme of lengthening, cases with predicted final discrepancies of up to 25 cm can be corrected. To reach this goal usually three lengthening steps and an epiphyseodesis of the contra-lateral side have to be performed. The first step of lengthening should be performed before school age and the final lengthening step at an age of 14–15 years. For the last lengthening step – if the bone diameter is wide enough – an intra-medullary lengthening device can be used. During growth,

valgus or varus mal-alignment can be treated with temporary hemi-epiphyseodesis using 8-hole plates (Orthofix Int. Curaçao Netherlands Antilles).

For patients with a discrepancy from 2 to 5 cm an epiphyseodesis may be appropriate as a single procedure depending on the predicted final total height. This procedure closes the growth of the opposite leg by altering the activity of the growth plate thereby allowing the affected limb to “catch up” by the end of growth. There is a choice of two different techniques, temporary epiphyseodesis or definitive epiphyseodesis. If the predicted increase in discrepancy over time equals, or is more than the remaining growth of the longer leg a definitive epiphyseodesis can be performed safely. The method of choice is the mini-invasive Canale technique [7].

---

## Guidelines for the Treatment of Congenital Deformities of the Lower Extremity

### Congenital Femoral Deficiency (CFD)

There is a wide range of deformities from mild hypoplasia to complete absence of the femur [21, 23]. The crucial indication for treatment is the patho-morphology and function of the hip and knee joint.

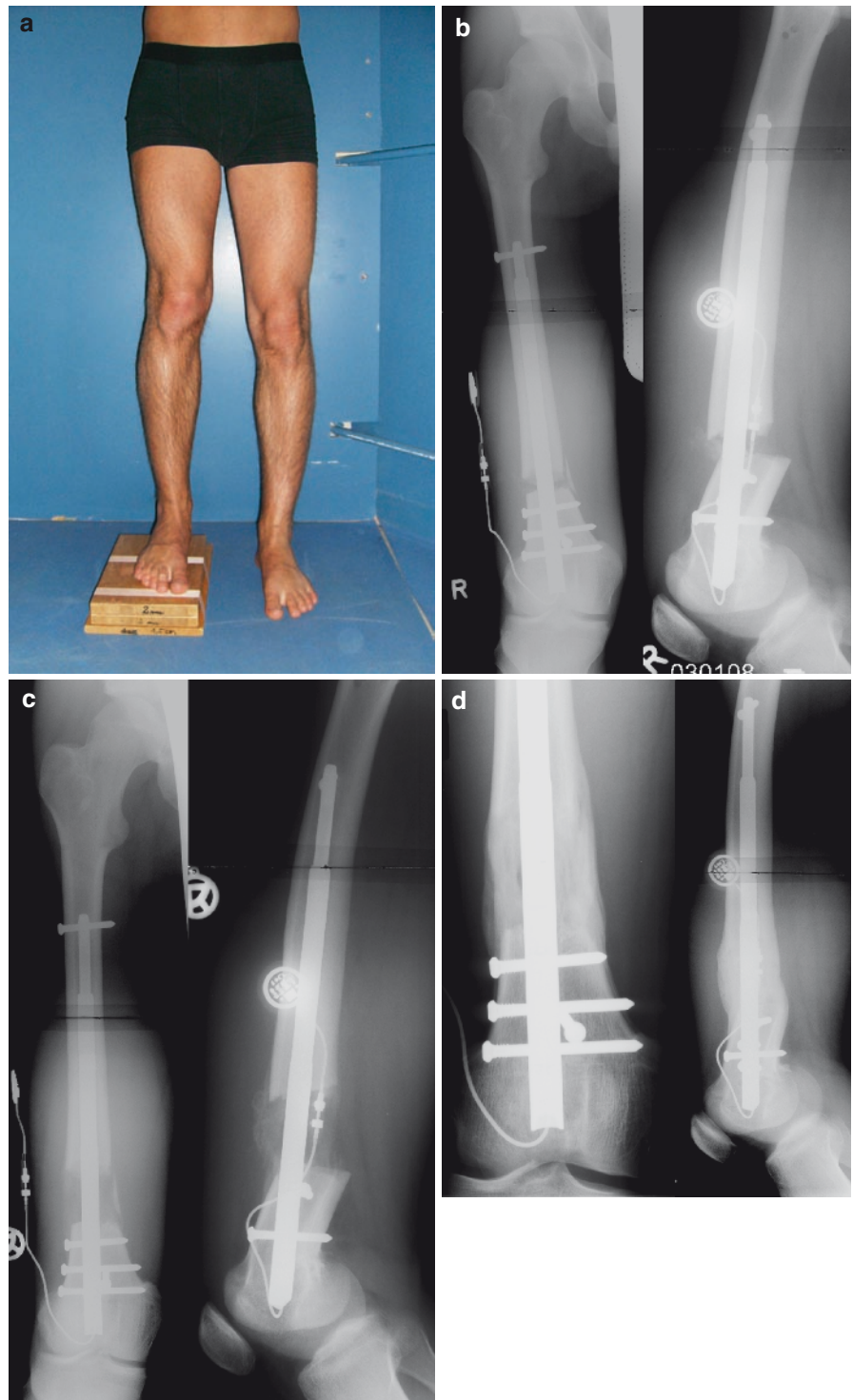
Lengthening should only be performed in the presence of a stable hip joint or when creating a stable hip joint with a pelvic osteotomy. Surgery must address the varus and retroversion of the femoral neck and the dysplasia of the acetabulum. If there is a mobile pseudarthrosis of the femoral neck or the subtrochanteric region, which is very often accompanied by a severe bowing of the proximal femur, a stable and anatomically normal situation has to be restored by an extensive combined soft tissue and bone reconstruction (Paley “Superhip” procedure) [20].

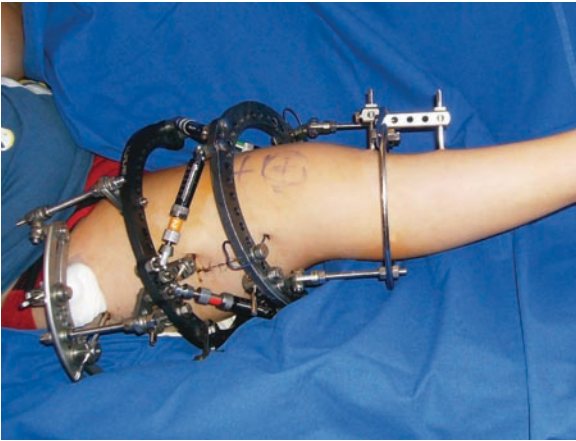
Manner et al. reported in 90% of CFD cases an aplasia of the anterior cruciate ligament and in 40% also an aplasia of the posterior cruciate ligament [17]. There was a clear correlation between severity of the deformity and aplasia of the cruciate. He also described a distinct hypoplasia within the distal lateral femoral epiphysis which is the cause for recurrent valgus deformity of the knee joint in this disease [18]. The knee therefore has to be protected against subluxation or dislocation by bridging the knee joint with an Ilizarov frame with hinges in the centre of rotation of the knee joint to allow physiotherapy during the lengthening process with the device in place (Fig. 4).

In cases presenting a severe or multi-directional instability of the knee joint a stabilization of the knee joint has to be



**Fig. 3** (a) This 20-year-old patient presented with a post-traumatic valgus and procurvatum deformity and a leg-length discrepancy of 5.5 cm. (b) Intra-operatively an acute correction of the axial mal-alignment, with special consideration to anatomic lengthening axis, was performed. (c) A good distraction gap and early callus formation was seen 8 weeks after the initial surgery. (d) At 6 months follow-up leg-length equalization with a full correction of mal-alignment was achieved and full bone healing and beginning of remodeling was observed





**Fig. 4** The hinge is placed at a point where a line running at the posterior cortex of the femur intersects with the growth-plate. The hinges are locked in full extension at night time and are opened during the day to allow knee flexion and physical therapy

created by soft tissue surgery before performing a lengthening procedure (Paley “Superknee” procedure) [21].

The amount of lengthening in CFD should depend on age and the severity of the deformity but not exceed 5–7 cm. for the first stage to avoid unnecessary complications.

### Fibula Deficiency

As in congenital short femur there is a range of deformity from hypoplasia to complete absence of the fibula [1]. The only remnant of the fibula may be a fibrous fibular anlage. Together with a shortening of the gastro-soleus complex this can cause a severe bow-stringing with anterior bowing and valgus mal-alignment of the tibia.

The tibia is shorter depending on the severity of the deformity and commonly a foot deformity can be found. The foot on the affected site is shorter with absence of lateral rays. The hindfoot often presents with a talo-calcaneal coalition or a clubfoot type-deformity [1]. Fibular hemimelia is commonly combined with congenital femoral deficiency and a shortening of the femur with hypoplasia of the lateral femoral condyle and instability of the knee joint are common findings.

If shortening is excessive and a single ray foot is present, knee stabilization, alignment correction and chondrodesis of the ankle joint should be performed followed by an adequate orthoprosthetic management.

In most of the cases even with a 2-ray foot, correction of the deformity and lengthening with a staged treatment can be performed. For reconstruction the first stage of surgery within the first year of life consists of resection of the fibular

anlage, and lengthening of the Achilles tendon. At an age of 2–3 years the foot deformity and ankle varus have to be corrected. The calcaneus has to be aligned to the mechanical axis of the tibia by an inclined osteotomy through the coalition of the talus and the calcaneus. To stabilise the ankle joint a chondrodesis can be performed and fixed with two K-wires. During the same procedure the first lengthening step can be performed, including the correction of the valgus and procurvatum deformity. In case of knee instability to prevent knee dislocation and knee flexion contracture the frame is bridged to the femur with flexible hinges. As an alternative the knee can be braced by an orthosis which is connected to the Taylor Spatial Frame and includes pneumatic struts that push the knee into extension but allow active flexion against an individually set resistance (Fig. 5).

According to the calculated leg-length discrepancy at the end of growth a treatment plan has to be developed with



**Fig. 5** The orthosis with the pneumatic struts is fixed directly to the Taylor Spatial Frame

several steps of lengthening and eventually an epiphysiodesis of the contra-lateral tibia.

### **Tibial Hemimelia**

Tibial hemimelia is another congenital hypoplasia of the lower limb. Especially in bilateral cases a genetic aetiology is common. The best classification was published by Weber [26]. Weber distinguished between seven different types of deformity. According to the different types a treatment plan has to be made. If for example a proximal tibial anlage is present and the knee can be actively stretched, the centralisation of the fibula with a fusion of the fibula with the tibial anlage is recommended.

If there is no tibial anlage but presence of the patella, Weber described a technique using the patella as a tibial plateau and reconstructing the lower leg by a centralisation of the fibula underneath the patella [25, 26]. If there is no extensor mechanism, no tibial anlage, and no patella, a centralisation of the fibula should not be performed. In these cases, a better function can be achieved with new orthoprosthetic management.

In all cases of tibial hemimelia a chondrodesis between fibula and talus has to be performed with lengthening of the Achilles tendon and correction of the foot deformity. Lengthening in tibial hemimelia patients should be performed only if the fibula remodels, gets wider and takes the form of a tibia.

### **Lengthening of Short Stature Patients**

The indication for lengthening of short stature patients is under constant debate [2, 8, 10]. A height at the end of growth below 150 cm for a female and below 160 cm for a male is below the limit of normal and most likely caused by a genetic disorder like achondroplasia or hypochondroplasia [12]. This group of patients has functional, anatomic, ergonomic and psychosocial problems. Therefore, for this group of patients lengthening is indicated if they feel their short stature as a handicap and if activities of daily life can be improved. The advantage of lengthening may that they can drive a normal car, look proportional, are not overlooked at the desk, can use a normal toilet, can cook in a normal kitchen and can buy ready-made regular clothes. For this group of patients the first step of lengthening should be performed at an age between 5 and 8 years. Usually achondroplastic patients develop a bi-level deformity of the tibia which has to be corrected and this correction can be combined with a first lengthening step of about 5–7 cm.

Preferably the lengthening procedure should be performed simultaneously at the same segment bilaterally. This offers the advantage to the patient that in case of psychological problems the treatment can be stopped at any time.

The next step of treatment is lengthening of both femora again with the goal to gain a length increase of about 7 cm. At this stage an acceptable proportion can be achieved. For further steps of lengthening the patient should be informed that also the upper extremities have to be lengthened not only because of cosmetic reasons but also because of ergonomic reasons.

---

### **Prevention of Complications**

In lengthening of congenital deformities the fracture rate after frame removal is about 26%.

To avoid these complications we now remove the fixator and stabilize the limb by inserting an intra-medullary rush rod or an interlocking nail in the same session.

Another complication is joint instability or stiffness. In case of a dysplastic hip joint before lengthening of the femur a surgical correction of the dysplasia has to be performed. To protect the knee joint against dislocation in congenital deformities with absence of the cruciate ligaments the knee joint has to be bridged by an Ilizarov ring. A perfect hinge placement in the centre of rotation of the knee joint is mandatory for pain free movement of the joint as long as the lengthening device is in situ. To keep stability and enhance motion we developed an orthosis which is fixed to the Taylor Spatial Frame with hinges at knee level and hydrostatic tethers, available in different strengths, which keep the knee in a stretched position and allows active flexion movement altered by using different strengths of tethers. To avoid equinus through lengthening the lower leg bridging of the ankle joint is advisable especially in congenital deformities such as fibular hemimelia. Protection of the adjacent joints should be the primary concern during all lengthening procedures.

---

### **In Conclusion**

Advances in deformity analysis, planning of surgery, and understanding of pathology, implant design and prevention and management of complications have made lengthening procedures much safer with a predictable outcome concerning function and an increased quality of life. For the future new biological approaches to limb lengthening using

adequate treatment to improve and speed up bone healing to reduce the time in the fixator and also the further development of new devices will make lengthening more and more comfortable for the patient and also the surgeon.

## References

- Achterman C, Kalamchi A (1979) Congenital deficiency of the fibula. *J Bone Joint Surg Br* 61-B(2):133–137
- Aldegheri R, Dall'Oca C (2001) Limb lengthening in short stature patients. *J Pediatr Orthop B* 10(3):238–247
- Aronson J, Shen X (1994) Experimental healing of distraction osteogenesis comparing metaphyseal with diaphyseal sites. *Clin Orthop Relat Res* (301):25–30
- Barham G, Clarke NM (2008) Genetic regulation of embryological limb development with relation to congenital limb deformity in humans. *J Child Orthop* 2(1):1–9
- Baumgart R, Betz A, Schweiberer L (1997) A fully implantable motorized intramedullary nail for limb lengthening and bone transport. *Clin Orthop Relat Res* (343):135–143
- Bliskunov AI (1983) [Intramedullary distraction of the femur (preliminary report)] *Ortop Travmatol Protez* (10): 59–62
- Canale ST, Christian CA (1990) Techniques for epiphysiodesis about the knee. *Clin Orthop Relat Res* (255):81–85
- Catagni MA, Loviseti L, Guerreschi F, Combi A, Ottaviani G (2005) Cosmetic bilateral leg lengthening: experience of 54 cases. *J Bone Joint Surg Br* 87(10):1402–1405
- Cole JD, Justin D, Kasparis T, DeVlught D, Knobloch C (2001) The intramedullary skeletal kinetic distractor (ISKD): first clinical results of a new intramedullary nail for lengthening of the femur and tibia. *Injury* 32 suppl 4:SD129–SD139
- Daniel E, Kent G, Binney V, Pagdin J (2005) Trying to do my best as a mother: decision-making in families of children undergoing elective surgical treatment for short stature. *Br J Health Psychol* 10(Pt 1):101–114
- Guichet JM, Casar RS (1997) Mechanical characterization of a totally intramedullary gradual elongation nail. *Clin Orthop Relat Res* (337):281–290
- Herzenberg JE, Paley D (2007) Stature lengthening: skeletal dysplasia. In: Rozbruch RS, Ilizarov S (eds) *Limb lengthening and reconstruction surgery*. Informa Healthcare USA, New York, pp 575–596
- Ilizarov GA (1989) The tension-stress effect on the genesis and growth of tissues. Part I. The influence of stability of fixation and soft-tissue preservation. *Clin Orthop Relat Res* (238):249–281
- Ilizarov GA (1989) The tension-stress effect on the genesis and growth of tissues: Part II. The influence of the rate and frequency of distraction. *Clin Orthop Relat Res* (239): 263–285
- Ilizarov GA (1990) Clinical application of the tension-stress effect for limb lengthening. *Clin Orthop Relat Res* (250):8–26
- Manner HM, Huebl M, Radler C, Ganger R, Petje G, Grill F (2007) Accuracy of complex lower-limb deformity correction with external fixation: a comparison of the Taylor Spatial Frame with the Ilizarov ring fixator. *J Child Orthop* 1(1):55–61
- Manner HM, Radler C, Ganger R, Grill F (2006) Dysplasia of the cruciate ligaments: radiographic assessment and classification. *J Bone Joint Surg Am* 88(1):130–137
- Manner HM, Radler C, Ganger R, Grill F (2006) Knee deformity in congenital longitudinal deficiencies of the lower extremity. *Clin Orthop Relat Res* 448:185–192
- Moseley CF (1977) A straight-line graph for leg-length discrepancies. *J Bone Joint Surg Am* 59:174–179
- Paley D, Bhave A, Herzenberg JE, Bowen JR (2000) Multiplier method for predicting limb-length discrepancy. *J Bone Joint Surg Am* 82:1432–1446
- Paley D, Standard SC. Lengthening reconstruction surgery for congenital femoral deficiency. In: Rozbruch RS, Ilizarov S (eds) *Limb lengthening and reconstruction surgery*. Informa Healthcare USA, New York, pp 393–412
- Paley D, Tetsworth K (1992) Mechanical axis deviation of the lower limbs. Preoperative planning of uniapical angular deformities of the tibia or femur. *Clin Orthop Relat Res* (280):48–64
- Pappas AM (1983) Congenital abnormalities of the femur and related lower extremity malformations. *J Pediatr Orthop* 3:45–60
- Shapiro F (1982) Developmental patterns in lower-extremity length discrepancies. *J Bone Joint Surg* 64-A: 639–651
- Weber M (2002) A new knee arthroplasty versus Brown procedure in congenital total absence of the tibia: a preliminary report. *J Pediatr Orthop B* 11(1):53–59
- Weber M (2007) Tibial hemimelia. In: Rozbruch RS, Ilizarov S (eds) *Limb lengthening and reconstruction surgery*. Informa Healthcare USA, New York, pp 429–448

# Total Hip Replacement in the Congenitally Dislocated Hip Using the Paavilainen Technique<sup>1</sup>

19 Hips Followed for 1.5–10 Years

Bjørn Thorup, Inger Mechlenburg, and Kjeld Søballe

Total hip replacement (THR) for correction of the congenitally dislocated hip (CDH) is a technical challenge and different approaches have been used [7, 8, 12]. At our department we use the cementless THR procedure described by Paavilainen et al. [10], whereby the cup is placed at the level of the true acetabulum and a shortening osteotomy of the proximal part of the femur is carried out. There have only been a few reports on the results achieved with this technique [1, 6, 10, 11]. We evaluated the clinical and radiographic results in a series of Paavilainen procedures performed at our hospital over a 10-year period.

## Patients and Methods

From 1996 to 2006, 19 CDHs (15 patients, 10 women) were operated on by the senior author (KS) using the Paavilainen technique (Table 1). The indications for surgery were CDH with walking difficulties and hip pain. The average age at the time of surgery was 38 (16–73) years. Seven hips had been operated on previously with a Schanz osteotomy. The mean follow-up time was 4.8 (1.5–10) years.

All 15 patients were clinically evaluated preoperatively and at the most recent follow-up with the Harris hip score (HHS). The radiographs were classified as either Eftekhar type C (4 hips) or D (15 hips) [3, 4] and they were assessed for aseptic loosening using the Gruen and Charnley classification.

Pre-operatively, templating was performed on radiographs. With the patient standing, the leg-length inequality was estimated by placing wooden blocks of different height (i.e. 0.5, 1, 1.5, 2, 3, 4, and 5 cm) until the position of the pelvis was balanced. In young adults, almost full correction of the leg-length

discrepancy was planned whereas in older patients with a more rigid spine, we tried to find the most comfortable lengthening. The operation was performed as described by Paavilainen [9] using a posterolateral approach (Figs. 1 and 2). All acetabular components were seated in the original acetabulum. In most cases, autologous cancellous bone material was placed under the acetabular component before inserting it. We did not need to re-inforce the acetabular wall with bone strut grafts in any of the cases. Cementless Mallory-Head cups with hydroxyapatite (HA) coating, two or three screws, and ArCom polyethylene liner were used in all but one hip (case no. 15). This patient had a Duraloc cup (metal-metal articulation).

The femoral shaft was reamed and a trial implant was inserted and trial reduction was performed before insertion of a straight cementless stem. In the first three hips, we used Biomet's dysplasia stem (Biomet, Warsaw, IN) and in the following 16 hips we used S-ROM femoral stems of the dysplastic type (DePuy, Warsaw, IN). After reduction, the greater trochanter was trimmed to fit the lateral part of the proximal diaphysis. With the hip in abduction, the greater trochanter was advanced distally until the gluteus medius muscle was tight, and then fixed with two or three cortical screws with washers. A cable around the femur was used frequently to avoid fracture of the bone during insertion of the stem. Finally, the vastus lateralis muscle was reinserted at the greater trochanter.

Post-operatively, the patients were mobilized using two crutches and allowed 30 kg of weight-bearing on the operated leg for the first 6 months. Active abduction of the leg was not allowed in the same period. Full weight-bearing was allowed when healing of the osteotomy had been verified radiographically.

K. Søballe (✉)  
Department of orthopaedic surgery, University hospital of  
Aarhus, Denmark  
e-mail: kjeld@soballe.com

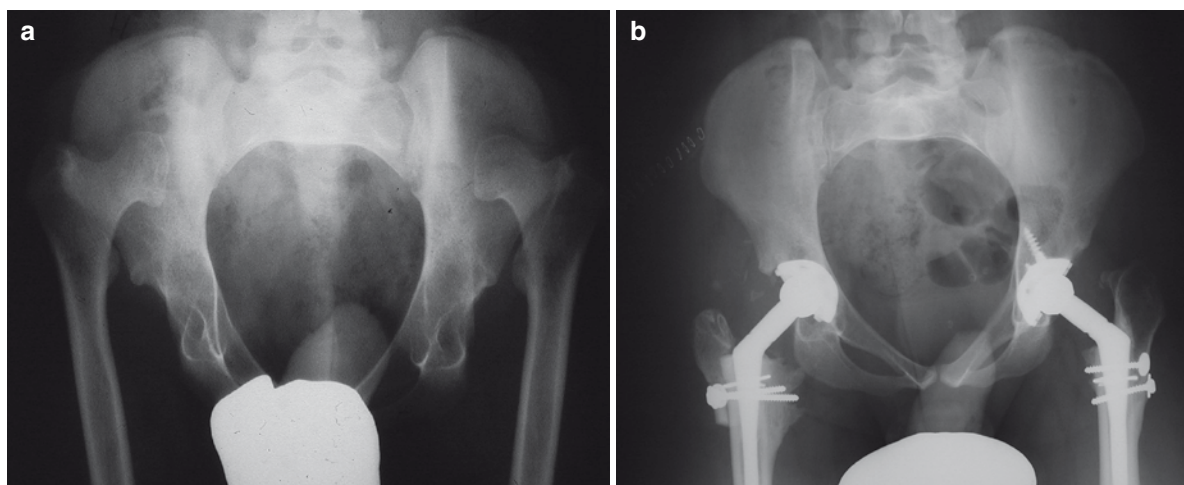
<sup>1</sup>Copyright Informa Healthcare Ltd 2009. ISSN 1745-3674.  
Printed in Sweden – all rights reserved. DOI  
10.3109/17453670902876789

Publication details, including instructions for authors and subscription information: <http://www.informaworld.com/smpp/title~content=t113400243>

**Table 1** Demographic data and results of clinical and radiographic examinations of 19 CDHs operated on with the Paavilainen technique

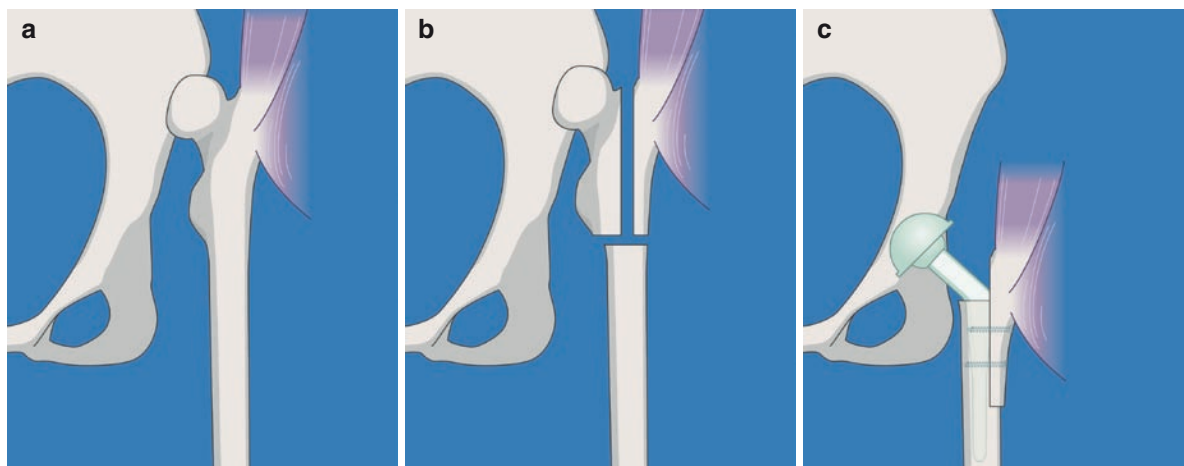
Case no	Follow-up (months)	Sex	Age at surgery	Eftkhar class	Formerly operated	Trendelenburg preop./postop.	HHS preop./postop.	Gruen zones	Dislocation postop.	Liner replacement
1-sin	120	F	38	C	+	+/-	35/92	1(4F)	-	-
2a-dx	120	M	16	C	+	+/-	10/72	0	+	+
2b-sin	108	M	17	D	-	+/-	36/99	1(1F)	-	+
3-dx	120	F	17	D	-	+/-	43/88	0	-	+
4-sin	120	F	19	D	+	-/-	63/96	0	-	-
5-dx	48	M	26	D	+	+/+	50/83	0	-	-
6a-dx	30	F	16	D	+	+/-	45/93	0	-	-
6b-sin	24	F	17	D	+	+/-	42/89	0	-	-
7-dx	48	M	73	D	-	+/-	38/99	0	-	-
8-sin	48	F	36	C	-	+/-	63/91	0	-	-
9-sin	48	M	42	D	-	+/-	42/95	2(1,4F)	-	-
10-sin	120	F	43	C	-	+/-	63/49	1(2A)	-	-
11a-sin	24	F	46	D	-	+/-	46/89	0	-	-
11b-dx	18	F	47	D	-	+/-	46/50	0	-	-
12a-dx	24	M	45	D	-	+/-	22/95	0	-	-
12b-sin	18	M	45	D	-	+/-	51/88	0	+	-
13-dx	24	F	58	D	-	+/-	55/93	0	+	-
14-sin	24	F	64	D	-	+/-	42/91	0	-	-
15-sin	18	F	16	D	+	+/-	43/82	0	-	-
Total	1,104				7	18/1		5	3	3
Median	58		38				44/86			

Four patients were operated on bilaterally and are thus specified by letters a and b in the case column



**Fig. 1** Case no. 2. Congenital dislocated hips bilaterally; grade D according to Efthkhar. **(a)** Preoperatively. **(b)** Postoperatively, the acetabular components (Mallory head) were seated in the true acetabulum. A straight cementless dysplasia stem (Biomet) with a

22-mm head was used. The greater trochanter was fixed with two screws. A prophylactic cable was placed around the femur before insertion of the stem



**Fig. 2** **(a)** CDH grade D. **(b)** Consistent with the preoperative templating, an extended trochanteric osteotomy is performed followed by a transverse osteotomy of the femur. The medial segment is removed. **(c)** A straight stem is inserted and the

acetabular component is placed at the level of the true acetabulum. The greater trochanter is advanced distally until the gluteus medius muscle is tight, and then it is fixed with two screws

## Results (Table 1)

Pre-operatively, the patients had a median HHS of 44; post-operatively, it was 86. The Trendelenburg test was positive in 18 hips pre-operatively and in only one post-operatively. One case of incomplete peroneal nerve palsy (drop foot) occurred and the patient recovered completely after 2 weeks. In 14 of the 15 unilateral hips, the leg length was

equalled within 1 cm. In one case, the operated leg was 1–2 cm too short. There were four cases of intra-operatively fissures of the proximal femur. They were all secured with cables and none of these patients experienced post-operatively complications. No deep vein thrombosis or early infections were observed.

The radiographic evaluation showed no aseptic loosening of the components. In three patients, radiolucency was observed in one Gruen zone. In one patient, radiolucency

was observed in two Gruen zones at the time of final follow-up (case no. 9). None of these four patients had clinical symptoms of loosening. Three hips dislocated in the follow-up period; two were reduced openly and one had a closed reduction. One of the dislocations was due to malposition of the acetabular component. The acetabular component was repositioned and no further dislocation occurred. Three patients needed the liner replaced due to wear of the polyethylene. In one of these, deep infection was diagnosed 6 months post-operatively. This patient underwent a 2-stage revision surgery, which cured the infection.

---

## Discussion

Our good results are in agreement with those of the other three follow-up studies on patients with C or D dislocation operated with the same technique [1, 6, 11].

In our series, most of the well-known complications were present but at an acceptable rate. The major problem was the need for re-operation due to wear of the polyethylene liner. Eskelinen et al. [6] concluded that wear was secondary to suboptimal design of the acetabular components used. We have started using hard bearings in our younger patients: ceramic-ceramic for females and metal-metal for males, which will hopefully be a better solution than polyethylene. Metal bearings may be of concern in women in childbearing age; metal ions pass the placenta [14]. Highly cross-linked polyethylene has shown very low wear rates [2], and the use of this instead of conventional polyethylene is also a possible solution in young, active patients.

We experienced two dislocations within 2 days of surgery. The first hip was reduced closed without any further complications. The second dislocation was due to malposition of the cup, and an open reduction was performed. In addition, we performed an open reduction on a chronic dislocation discovered at a routine check 3 months after it had occurred. The patient had noted pain and shortening of the leg, but had not found it necessary to contact a doctor. Both Paavilainen et al. [11] and Carlsson et al. [1] reported late dislocations in their series, and a plausible reason for the relatively high dislocation rate was the use of small 22-mm heads.

We had one case of temporary incomplete peroneal palsy in a patient whose leg had been lengthened 3 cm. The nerve monitoring with electric-evoked potentials during operation did not indicate problems and the palsy disappeared 2 weeks post-operatively. Egli et al. [5] found no correlation between lengthening of the leg and nerve palsy

and concluded that nerve contusion was responsible in most cases. Eskelinen et al. [6] reported peroneal palsy in 3 of 68 patients in their series.

One problem that occurred during operation in all series – our own included – was a fissure in the proximal femur. We stabilized the four fractures we experienced with Dall-Miles cable, with good results, and used a prophylactic cable in several other patients. A potential complication is nonunion between the greater trochanter and the proximal diaphysis [13] but we did not encounter this complication.

In conclusion, THR using the Paavilainen technique has provided our series of patients with a stable and functional hip, with a limited rate of complications. We are mainly concerned about the wear of the polyethylene, which is excessive in these mostly younger patients. This is thus a problem that must be considered.

BT and KS designed the study. KS performed the operations. BT gathered the data and both BT and IM analyzed it. IM revised the first draft of the manuscript and BT wrote the final draft.

No competing interests declared.

---

## References

1. Carlsson A, Bjorkman A, Ringsberg K, von Schewelov T (2003) Untreated congenital and posttraumatic high dislocation of the hip treated by replacement in adult age: 22 hips in 16 patients followed for 1–8 years. *Acta Orthop Scand* 74:389–396
2. Digas G, Karrholm J, Thanner J, Herberts P (2007) 5-year experience of highly cross-linked polyethylene in cemented and uncemented sockets: two randomized studies using radiostereometric analysis. *Acta Orthop* 78:746–754
3. Eftekhar N (1971) Charnley “low friction torque” arthroplasty. A study of long-term results. *Clin Orthop* 81:93–104
4. Eftekhar NS, Stinchfield FE (1973) Total replacement of the hip joint by low friction arthroplasty. *Orthop Clin North Am* 4:483–501
5. Egli S, Hankemayer S, Muller ME (1999) Nerve palsy after leg lengthening in total replacement arthroplasty for developmental dysplasia of the hip. *J Bone Joint Surg (Br)* 81:843–845
6. Eskelinen A, Helenius I, Remes V, Ylinen P, Tallroth K, Paavilainen T (2006) Cementless total hip arthroplasty in patients with high congenital hip dislocation. *J Bone Joint Surg (Am)* 88:80–91
7. Gul R, Masterson E (2005) Cementless total hip arthroplasty in the treatment of severe hip dysplasia or dislocated hips. *Eur J Orthop Surg Traumatol* 15:101–104
8. Masonis JL, Patel JV, Miu A, Bourne RB, McCalden R, Macdonald SJ, Rorabeck CH (2003) Subtrochanteric shortening and derotational osteotomy in primary total hip



- arthroplasty for patients with severe hip dysplasia: 5-year follow-up. *J Arthroplasty* 18:68–73
9. Paavilainen T (1997) Total hip replacement for developmental dysplasia of the hip. *Acta Orthop Scand* 68:77–84
  10. Paavilainen T, Hoikka V, Solonen KA (1990) Cementless total replacement for severely dysplastic or dislocated hips. *J Bone Joint Surg (Br)* 72:205–211
  11. Paavilainen T, Hoikka V, Paavolainen P (1993) Cementless total hip arthroplasty for congenitally dislocated or dysplastic hips. Technique for replacement with a straight femoral component. *Clin Orthop* [297]:71–81
  12. Park MS, Kim KH, Jeong WC (2007) Transverse subtrochanteric shortening osteotomy in primary total hip arthroplasty for patients with severe hip developmental dysplasia. *J Arthroplasty* 22:1031–1036
  13. Sanchez-Sotelo J, Berry DJ, Trousdale RT, Cabanela ME (2002) Surgical treatment of developmental dysplasia of the hip in adults: II. Arthroplasty options. *J Am Acad Orthop Surg* 10:334–344
  14. Ziaee H, Daniel J, Datta A K, Blunt S, McMinn DJ (2007) Transplacental transfer of cobalt and chromium in patients with metal-on-metal hip arthroplasty: a controlled study. *J Bone Joint Surg (Br)* 89:301–305

## Part IV

---

# Trauma

# Operative Strategy for Fracture-Dislocation of the Elbow

Konrad Mader

## Introduction

The elbow joint is one of the most inherently stable articulations of the skeleton [49]. When in addition to the dislocation of the joint at least one of the osseous or articular component structures that contribute to the stability of the elbow is disrupted, the injury is termed “fracture-dislocation of the elbow” and the risk of recurrent or chronic instability and the development of post-traumatic arthritis of the elbow are increased [2, 11–13, 17, 18]. Treatment of these injuries is challenging due to difficulties in applying accurate definitions of the patterns of the injury, in evaluating the specific roles of the component structures that contribute to the instability of the elbow and the lack of a standardized rationale for the operative treatment. Soft tissue injury including open wounds and vascular or nerve compromise add to the complexity of the injury. In recent years investigative efforts were put into the definition of the components that contribute to the stability of the elbow and the concept of operative fixation of the osseous and ligamentous lesions (i.e., articular fragments of the distal humerus [7, 20, 31, 32, 35, 36], the radial head [33, 41], the coronoid process [3, 14, 15, 21, 34, 59, 61–64, 72, 75], the olecranon [16, 42, 50, 51, 65, 70] the collateral ligaments [18, 54, 76] and combinations of these lesions [19, 28, 29, 37, 38, 43–45, 58, 60, 67, 68, 78] in order to reconstitute elbow stability. In addition to this more recently the development and use of the trans-articular external fixator with motion capacity of the elbow has extensively broadened the therapeutic spectrum of treating complex elbow trauma and partially changed therapeutic pathways [6, 10, 22–27, 39, 40, 46–48, 52, 55, 57, 66, 69,

71, 77]. The purpose of this lecture is to analyse the current literature and own prospective data in order to establish and discuss a strategy in the treatment fracture-dislocations of the elbow (Fig. 1).

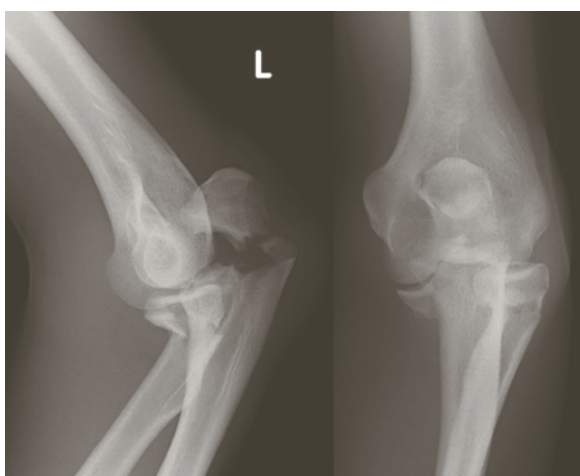
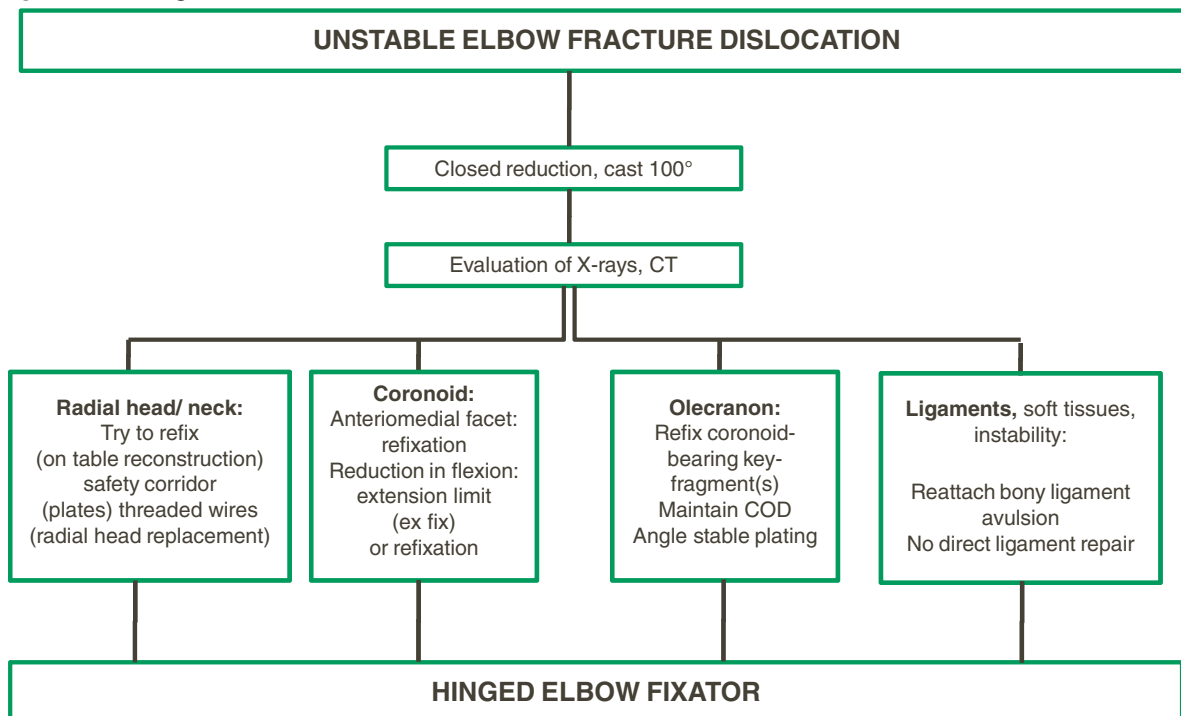
## Diagnostic Tools

### Conventional Imaging

Any elbow trauma is assessed initially using standard antero-posterior (AP) and lateral radiographs, which gives an idea of the type and personality of injury involved (Fig. 2). The AP is taken with the elbow extended and the forearm supinated (when possible) with the patient seated or supine, the beam is directed perpendicular to the midpoint of the elbow joint. The features demonstrated on this view should include the distal humerus, epicondyles, trochlea, capitellum, radial head including proximal radius and olecranon. The lateral view is taken with the patient seated if possible, the shoulder abducted to 90°, so that the shoulder is level with the film and the elbow is flexed at 90°. In this position the forearm is placed in supination and the beam is directed perpendicular to the humeral epicondyles centered on the elbow joint. On this view the epicondyles should be superimposed, the olecranon should be seen in profile, and the radial head superimposed on the coronoid process. Relationships that should be checked include the radiocapitellar line, which should bisect the radial head and the capitellum, and the anterior humeral line, which should run through the middle third of the capitellum. A thorough assessment of the initial radiographs at this stage should include the soft tissue envelope, anterior and posterior fat pads, cortical continuity, joint congruency, and bony alignment. Coonrad et al. proposed the “drop sign,” which is an increase in ulno-humeral distance on the lateral X-ray-film. This objective and static radiographically-measurable increase in ulno-humeral distance can be

K. Mader  
Ortopedisk Avdeling,  
Seksjon Traumekirurgi/ Håndkirurgi,  
Førde Sentralsjukehuset, 6807 Førde, Norway  
e-mail: konrad.mader@helse-forde.no

Fig. 1 Treatment algorithm of unstable elbow dislocation



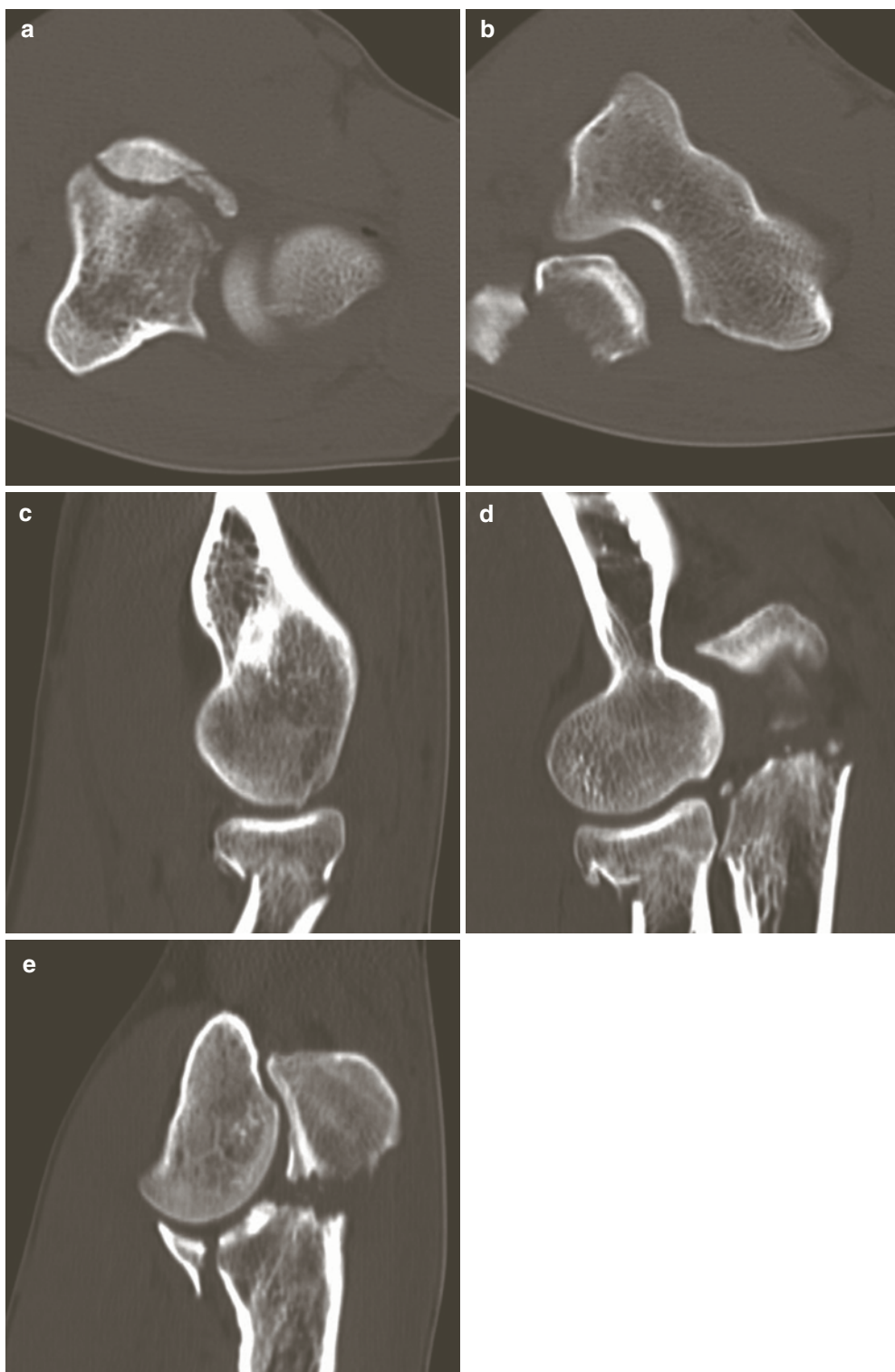
**Fig. 2** Antero-posterior and lateral X-ray of a fracture-dislocation of the left elbow in a 30-year-old male patient: note the compound fracture of the proximal ulna, the ventral subluxation of the distal humerus, the involvement of the radial head/neck and the coronoid process

viewed as warning sign for the presence of ligamentous instability [9]. A radial head-capitellum view can be obtained by having the patient positioned for a lateral elbow view and then angling the beam 45° medial to lateral and centred 2.5 cm. inferior to the epicondylar axis. This view gives an

unrestricted view of the radial head. Careful analysis of these three conventional X-rays will give information about the bony lesions involved and any existing dislocation or subluxation of the elbow joint. Repeat AP and lateral X-rays after reduction and casting in a well-padded upper arm cast (in 100° of flexion) are required and again cautiously analysed for any persisting subluxation of the joint. Joint incongruity or asymmetry (i.e., rotatory subluxation) or a “drop sign” should be cautiously evaluated.

### Computed Tomography

Computed tomography is the tool of choice to evaluate the type and extent of bony fragmentation and presence/absence and direction of any persistent dislocation/subluxation of the elbow joint (Fig. 3a–e). Helical scanning with two-dimensional reconstruction can be done quickly with the arm rested in the cast. The “Superman” position (with the arms overhead) produces the best image quality, but scans can be done with the elbow at the side (giving some more volume artefacts). It is useful to start with the original transverse sections, followed by the analysis of the frontal and sagittal reconstruction images and finally the three-dimensional reconstruction. The transverse plane is used to evaluate the proximal radio-ulnar joint and the distal



**Fig. 3** CT scans of the same patient; **(a)** transverse scan depicting the involvement of the coronoid process; **(b)** transverse scan showing the ventral dislocation of the distal humerus and the compound destruction of the proximal ulna; **(c)** sagittal reconstruction

showing involvement of the radial neck; **(d)** sagittal reconstruction showing the extent of the ulnar destruction; **(e)** sagittal reconstruction with subluxation of the humerus

humerus, olecranon, coronoid and radial head including the pattern of articular fragmentation. The coronal or frontal plane provides an image similar to the standard PA radiograph and will provide better bone detail and intra-articular fragmentation. The sagittal plane will give a clear view of ventral or dorsal dislocation of the humero-ulnar joint and the dislodgement of fragments of the coronoid process and the radial head.

### Evidence-Based Medicine?

For all readers with the interest in evidence-based orthopedics medicine, randomized trials and the meta-analysis of the medical literature there is sad news: randomized studies and a meta-analysis of treatment concepts at the elbow do only exist in the treatment of chronic epicondylitis. The digestion of 250 scientific papers on fracture-dislocation of the elbow was a sour tour through Level III (case-control studies), IV (case studies) and predominantly Level V studies (expert opinion). First prospective protocols and a randomized controlled trial comparing the outcome of patients treated with reduction and early physiotherapy vs. hinged-fixation in unstable elbow dislocations are underway.

### Injury Mechanism

Fracture-dislocation of the elbow mainly occurs in the joints between the humerus and ulna and primarily is caused by compression, shearing, and avulsion forces. Radial head and neck fractures are produced by compression or shearing force. Olecranon and coronoid process fractures, accompanied by dislocation, are also caused by compression or shearing force. The most common type of fracture of the olecranon is made by traction force or pulling of the triceps muscle. Concurrent fractures of both condyles are produced mainly by avulsion or shearing forces. Almost all dislocations of the elbow are accompanied by rupture of collateral ligaments or avulsion fractures of the medial or lateral epicondyle. Because the incidence of coronoid, olecranon, and condyle fractures seems to increase to the amount of compression force, the current biomechanical studies focussing on fracture-dislocations resulting from experimental compression force [1, 4, 5]. Amis and Miller, in a cadaveric study, demonstrated a flexion-extension "arc of injury," which relates fracture types to elbow position at the moment of impact [1]. Radial head and coronoid fractures followed impact along the forearm up to 80° flexion. Olecranon fractures occurred by direct impact around 90° flexion. Distal humeral fractures mostly occurred above 110° flexion.

### Fracture Fixation

During the last decade, in America and Europe standard surgical protocols to treat complex elbow fracture-dislocations have evolved [16, 26, 28, 29, 34, 38, 39, 44, 45]. These provide guidance in reconstructing both ligamentous and bony components. Recent advances in fracture fixation of the radial head [41], olecranon [42], the introduction of angle-stable plating systems in fractures of the distal humerus and olecranon [73], and the strategic use of hinged external fixation have further broadened the therapeutic spectrum.

### Surgical Approaches

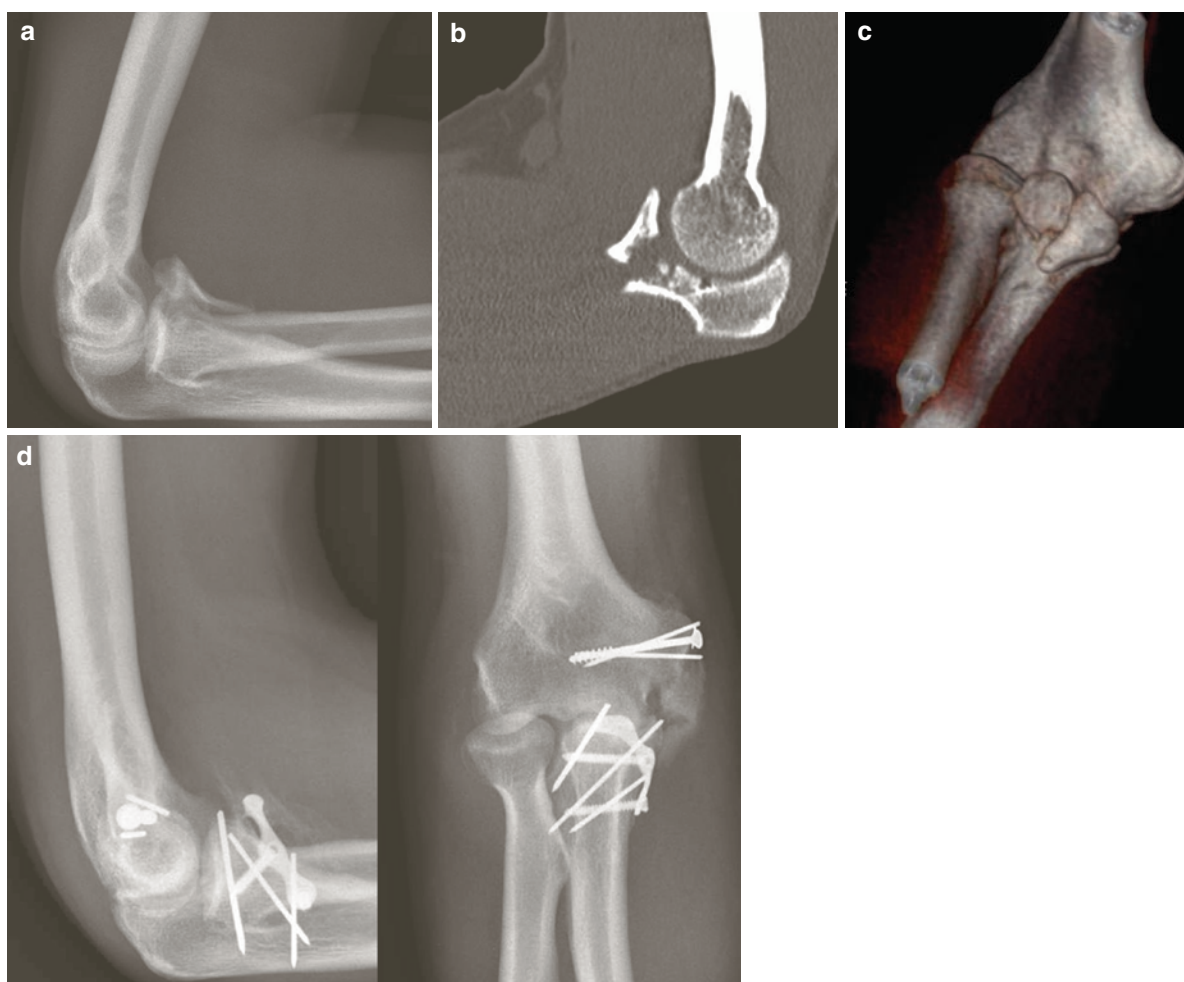
Surgical approaches to the elbow can be classified according to the aspect of the joint exposed, as anterior, lateral, medial, posterior and global [56]. The aim of a surgical approach to the elbow is to provide an adequate extensile exposure with preservation of the neurovascular structures. Modern surgical approaches to the elbow began with the lateral approach to the elbow described as early as 1911 by Kocher. Subsequently, many authors have described approaches to the elbow with the intention of providing improved visualization, primarily of the anterior elbow, without compromising the surgical outcome [38, 56]. Henry described the generally followed principle of "extensile exposure." The majority of these exposure use internervous or intermuscular intervals. Therefore, it is a pre-requisite to be familiar with the surgical anatomy of the major nerves and their cutaneous branches, the muscles and the medial and lateral ligament complex [25, 56, 74]. When a dorsal approach is mandatory, olecranon osteotomy should be avoided according to McKee et al. [8, 37].

### Coronoid Process

Coronoid fractures of the ulna are relatively uncommon yet critical injuries to recognize [59]. They definitely play an important role in elbow instability. Historic recommendations are to fix all large coronoid fracture fragments, as well as small fracture fragments associated with instability. The coronoid process acts as a bony buttress to prevent posterior dislocation and has three soft tissue insertions which lend stability as well: the anterior joint capsule of the elbow, the brachialis muscle and the medial ulnar collateral ligament. If the elbow is unstable, management usually consists of a combination of bony and soft-tissue repairs often including coronoid process repair [3, 15, 59]. Loss of motion is the most common complication of these

injuries. The current recommendation is to repair virtually all coronoid fractures associated with instability [72]. Recent biomechanical and clinical investigations have emphasized the importance of the coronoid process in the stability of the ulno-humeral articulation. Recently, Doornberg and Ring indicated the importance of the antero-medial facet of the coronoid as a distinct type of coronoid fracture resulting from a varus postero-medial rotational injury force resulting in complex instability [14]. They concluded that antero-medial fractures of the coronoid are associated with either subluxation or complete dislocation of the elbow in most patients. In order to

detect this very unstable sub-set of coronoid fractures again a CT scan is mandatory [14–16]. Usually there are two or more fragments, which make stable re-fixation a demanding procedure with possible complications such as heterotopic ossification and secondary dislocation (Fig. 4a–d). Although most experts recommend open reduction and direct or indirect fixation of coronoid fragments [14, 19, 21, 34, 61, 69, 75] there is a new concept, when using hinged external fixation, to limit extension to 30° for a period of 2–3 weeks and thereby indirectly to create stability. The personal experience with 85 consecutive cases will be discussed in the lecture.



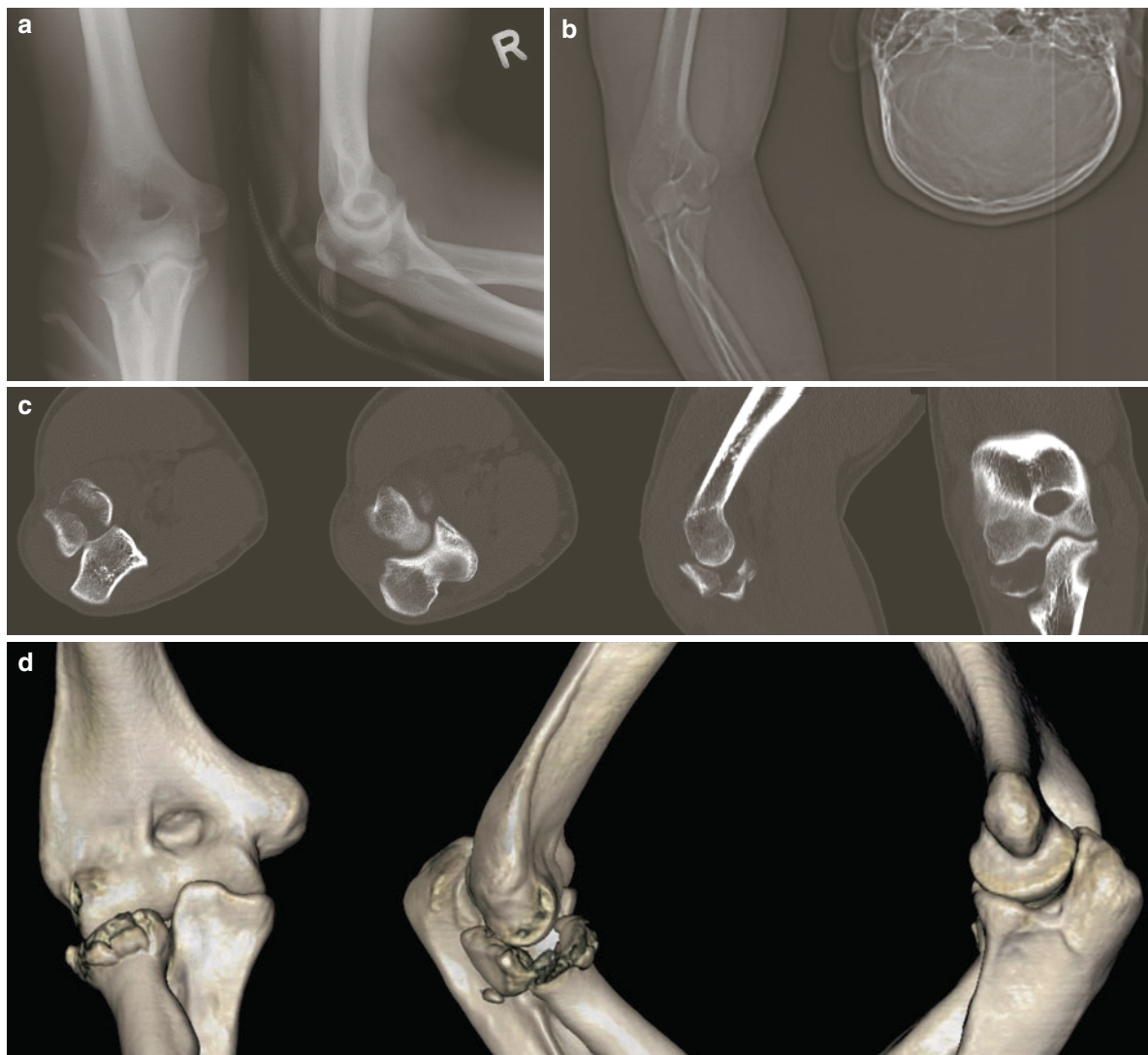
**Fig. 4** Fracture dislocation of the right elbow in a 35-year-old man: **(a)** lateral X-ray demonstrating a complex coronoid fracture; **(b)** sagittal CT reconstruction showing basal multi-fragmented coronoid fracture pattern. **(c)** 3-D CT reconstruction depicting two distinctive fragments, of which one is an antero-medial facet

fragment; **(d)** X-ray control 3 months post-operatively after open reduction and internal fixation of the coronoid with help of an ulnar osteotomy showing severe heterotopic ossification (Ilahi IV) on the ulnar side leading to complete ankylosis of the joint [30]

## Radial Head

“If in doubt, resect” was the old dogma regarding the treatment of fractures of the radial head. Patients with comminuted radial head fractures and those with associated soft-tissue injuries of the elbow have a poor outcome if the radial head is simply resected. Complications include subsequent distal radio-ulnar pain, weakness and instability of the elbow, cubitus valgus and ulnar neuritis [33, 41]. The role of the radial head as an important stabiliser of the forearm and

elbow is now better understood [58, 62]. It should not be resected without careful consideration [62, 63]. The treatment of Mason type-III fractures and those with associated ligamentous damage or dislocation (Mason type IV) is challenging. Several methods of reconstruction have been described and prosthetic replacement of the radial head is recommended for comminuted fractures, especially if the medial collateral ligament is disrupted [58, 60, 62]. There are some concerns about the non-anatomical shape of the prostheses which may cause loosening, subsequent degenerative changes

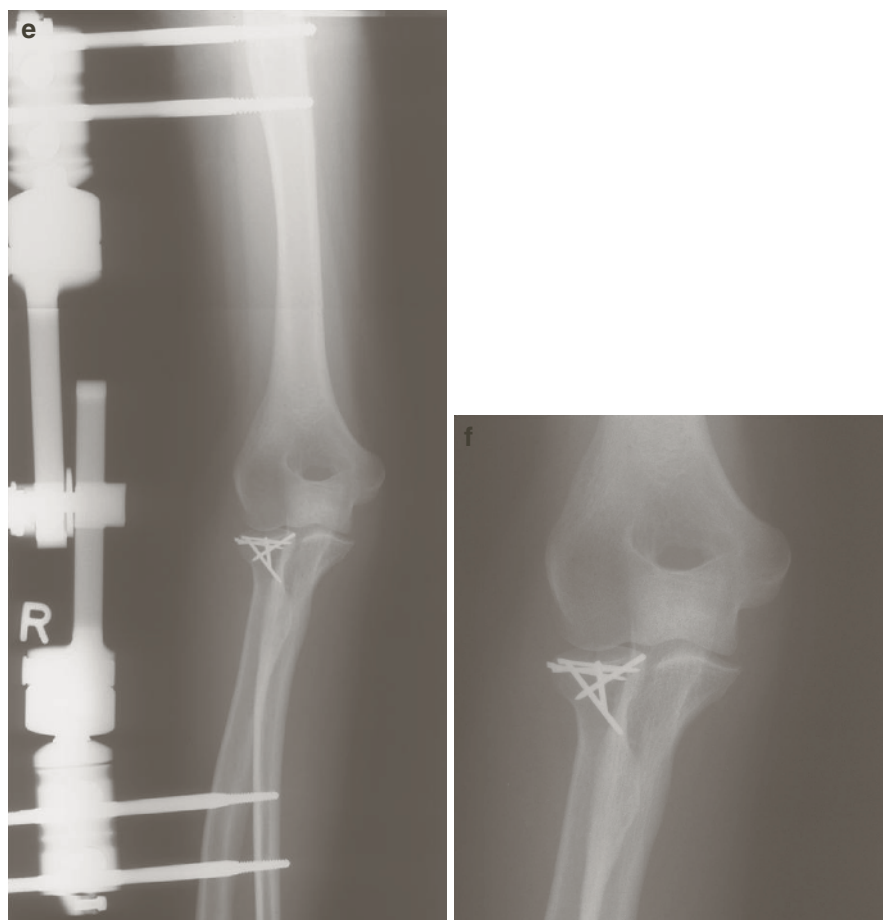


**Fig. 5** Twenty-three-year-old patient with complex fracture-dislocation of the right elbow with a comminuted fracture of the proximal radius; (a) X-rays in a cast after reduction; (b) Scout CT scan showing the “superman position.” (c) exemplary CT scans showing the extent of the radial head destruction; (d) 3-D

reconstruction of the bony injury. (e) conventional anteroposterior X-ray 5 weeks after the injury after ORIF of the radial head and application of an hinged external fixator; (f) higher resolution demonstrating reconstruction of the radial head with fine threaded screws



Fig. 5 (continued)



and instability of the elbow. We always try to reconstruct the radial head, even as an “on-table” reconstruction, as it will be the major radial stabilizer after bony healing. If there is severe comminution of the radial head and neck, after reconstruction a hinged fixator is mandatory in order to unload the radial head. Next to pre-formed plates, in the so-called “safe-zone” headless mini-fragment screws with fine threads are useful in reconstructing the radial head (Fig. 5a–f) [40, 41].

### The “Terrible Triad”

The “terrible triad” injury (radial head fracture, coronoid fracture and ulnar ligament disruption) has a history of complicated outcomes as the surgeon attempts to maximize functional range-of-motion goals while maintaining stability [78]. Several investigators have displayed standardized surgical protocols relying on stable reconstruction of the bony elements and extensive ligament repair [19, 34, 38, 39, 62, 63]. While most American colleagues rely on the protocol of

reconstruction/replacement of the radial head with coronoid and ligament repair, the integrative use of hinged external fixation is challenging this [23, 24, 26, 57, 69].

### Trans-Olecranon Dislocation

Trans-olecranon fracture-dislocation of the elbow occurs when a high-energy direct blow is applied to the dorsal aspect of the forearm with the elbow in mid-flexion and causes an olecranon fracture associated with an anterior dislocation of the forearm with respect to the distal humerus [50, 51]. The trochlea appears to have fractured through the olecranon process as the forearm is displaced anteriorly. The trans-olecranon fracture-dislocation is different from the anterior Monteggia, Bado one lesion, because in the former, there is a loss of stability in the ulno-humeral joint but the radio-ulnar relationship is preserved. The capsule-ligamentous restraints, in particular the annular ligament, remain intact. Most of the failures of the ulno-humeral joint

are a result of the bony disruption rather than the ligamentous component. The osseous injury can be a simple, non-comminuted, transverse, or oblique fracture of the olecranon, but is more commonly a complex and comminuted fracture involving the trochlear notch and, sometimes, the coronoid process as well. This lesion, often caused by high-energy trauma, can result in a complex skeletal disruption including an avulsion or shear fracture. It became quite clear that this injury represents an intrinsically-unstable bony lesion that requires good pre-operative planning and stable internal fixation with complete restoration of articular congruence and the coronoid buttress. This allows early active mobilization, which is critical to obtain good long-term results. If there is additional soft tissue injury or severe bone loss, maintenance of the coronoid-olecranon tip distance (COD) is of utmost importance in order to avoid post-traumatic stiffness. Again a hinged mono-lateral fixator is a suitable tool to control the soft tissues and allow early mobilization [50, 51, 55, 65, 66].

### The Role of Hinged External Fixation

It has become very clear in recent years, that hinged external fixation with motion capacity is an important tool in the treatment of severe fracture-dislocation of the elbow [6, 10, 22–27, 39, 40, 46–48, 52, 55, 57, 66, 69, 71, 77]. Several biomechanical and clinical studies have demonstrated, that stable movement in a nearly physiologic range of motion is possible, that an external fixator with adequate stability minimizes varus or valgus load after bony stabilization and with severed ligaments, and that it should be used in a situation of persistent instability/subluxation in complex elbow fracture-dislocations [24, 53, 55]. In recent reviews the role of hinged external fixation has expanded from using it as a “last help” to use early in the treatment protocol. Now hinged fixation has become such an integrated part in the treatment protocol in some centres in Europe, that it is *the* advance in the treatment of these injuries. As shown in the treatment algorithm (Fig. 1), a structured use of hinged fixation will actually change common protocols of ligament reconstruction and the treatment of “terrible triad” injuries. A suitable hinged external fixator (with appropriate biomechanical features) as an integral part in the treatment protocol challenges actual treatment algorithms [24, 55, 69].

### References

- Amis AA, Miller JH (1995) The mechanisms of elbow fractures: an investigation using impact tests in vitro. *Injury* 26:163–168
- Broberg MA, Morrey BF (1987) The results of the treatment of fracture-dislocations of the elbow. *Clin Orthop* 216:109–119
- Chung CH, Wang SJ, Chang YC, Wu SS (2007) Reconstruction of the coronoid process with iliac crest bone graft in complex fracture-dislocation of elbow. *Arch Orthop Trauma Surg* 127:33–37
- Chou PH, Chou YL, Lin CJ, Su FC, Lou SZ, Lin CF, Huang GF (2001) Effect of elbow flexion on upper extremity impact forces during a fall. *Clin Biomech* 16:888–894
- Closkey RF, Goode JR, Kirschenbaum D, Cody RP (2000) The role of the coronoid process in elbow stability. A biomechanical analysis of axial loading. *J Bone Joint Surg [Am]* 82-A:1749–1753
- Cobb TK, Morrey BF (1995) Use of distraction arthroplasty in unstable fracture dislocations of the elbow. *Clin Orthop* 312:201–210
- Cobb TK, Morrey BF (1997) Total elbow arthroplasty as primary treatment for distal humeral fractures in elderly patients. *J Bone Joint Surg [Am]* 79-A:826–832
- Coles CP, Barei DP, Nork SE, Taitsman LA, Hanel DP, Bradford Henley M (2006) The olecranon osteotomy: a six-year experience in the treatment of intraarticular fractures of the distal humerus. *J Orthop Trauma* 20:164–171
- Coonrad RW, Roush TF, Major NM, Basamania CJ (2005) The drop sign, a radiographic warning sign of elbow instability. *J Shoulder Elbow Surg* 14:312–317
- Deuel CR, Wolinsky P, Shepherd E, Hazelwood SJ (2007) The use of hinged external fixation to provide additional stabilization for fractures of the distal humerus. *J Orthop Trauma* 21:323–329
- O’Driscoll SW (2000) Classification and evaluation of recurrent instability of the elbow. *Clin Orthop* 370:34–43
- O’Driscoll SW, Jupiter JB, King GJ, Hotchkiss RN, Morrey BF (2001) The unstable elbow. *Instr Cours Lect AAOS* 50:89–102
- O’Driscoll SW, Bell DF, Morrey BF (1991) Posterolateral rotatory instability of the Elbow. *J Bone Joint Surg [Am]* 73-A:440–446
- Doornberg JN, Ring D (2006) Coronoid fracture patterns. *J Hand Surg Am* 31:45–52
- Doornberg JN, Ring DC (2006) Fracture of the anteromedial facet of the coronoid process. *J Bone Joint Surg [Am]* 88-A:2216–2224
- Doornberg J, Ring D, Jupiter JB (2004) Effective treatment of fracture-dislocations of the olecranon requires a stable trochlear notch. *Clin Orthop Relat Res* 429:292–300
- Duckworth AD, Ring D, Kulijdian A, McKee MD (2008) Unstable elbow dislocations. *J Shoulder Elbow Surg* 17: 281–286
- Eyngendaal D, Verdegaal SHM, Obermann WR, Van Vugt AB, Pöll RG, Rozing PM (2000) Posterolateral dislocation of the elbow. *J Bone Joint Surg [Am]* 82-A:555–560
- Fern SE, Owen JR, Ordyna NJ, Wayne JS, Boardman ND (2009) Complex varus instability: a terrible triad model. *J Shoulder Elbow Surg* 18:269–274
- Garcia JA, Mykula R, Stanley D (2002) Complex fractures of the distal humerus in the elderly. The role of total elbow

- replacement as primary treatment. *J Bone Joint Surg [Br]* 84-B:812–816
21. Garofalo R, Bollmann C, Kombot C, Moretti B, Borens O, Mouhsine E (2005) Minimal invasive surgery for coronoid fracture: technical note. *Knee Surg Sports Traumatol Arthrosc* 13:608–611
  22. Gausepohl T, Mader K, Pennig D (2006) Mechanical distraction in post-traumatic stiffness of the elbow in children. *J Bone Joint Surgery [Am]* 88-A:221–223
  23. Gausepohl T, Pennig D, Mader K (1997) Der transartikuläre Bewegungsfixateur bei Luxationen und Luxationsfrakturen des Ellenbogengelenkes. *Osteosynthese Int* 5:102–110
  24. Gausepohl T, Mader K, Pennig D (2005) Early functional treatment of unstable elbow dislocation with the aid of a hinged fixator. *Aktuelle Traumatol* 35:65–71
  25. Gausepohl T, Koebke J, Pennig D, Hobrecker S, Mader K (2000) The anatomical base of unilateral external fixation in the upper limb. *Injury Suppl* 1:11–20
  26. Gausepohl T, Pennig D, Mader K (1999) The early motion fixator concept for treatment of acute unstable fracture dislocations of the elbow. *J Bone Joint Surg [Br]* 81-B(Suppl II):191
  27. Hall J, Schemitsch EH, McKee MD (2000) Use of a hinged external fixator for elbow instability after severe distal humeral fracture. *J Orthop Trauma* 14:442–445
  28. Hastings H II, Engles DR (1997) Fixation of complex elbow fractures, part I. *Hand Clin* 13:703–717
  29. Hastings H II, Engles DR (1997) Fixation of complex elbow fractures, part II. *Hand Clin* 13:721–735
  30. Ilahi OA, Strausser DW, Gabel GT (1998) Post-traumatic heterotopic ossification about the elbow. *Orthopedics* 21:265–268
  31. Johansson H, Olerud S (1971) Operative treatment of intercondylar fractures of the humerus. *J Trauma* 11:836–842
  32. Josefsson PO, Gentz CF, Johnell O, Wendeberg B (1989) Dislocations of the elbow and intraarticular fractures. *Clin Orthop* 246:126–130
  33. Judet T (2001) Results of acute excision of the radial head in elbow radial head fracture-dislocations. *J Orthop Trauma* 15:308–309
  34. Kalicke T, Westhoff J, Wingenfeld C, Muhr G, Arens S (2003) Fracture dislocation of the elbow involving the coronoid process. *Unfallchirurg* 106:300–305
  35. Kamineni S, Morrey BF (2005) Distal humeral fractures treated with noncustom total elbow replacement. Surgical technique. *J Bone Joint Surg [Am]* 87-A(Suppl 1):41–50
  36. Kamineni S, Morrey BF (2004) Distal humeral fractures treated with noncustom total elbow replacement. *J Bone Joint Surg [Am]* 86-A:940–947
  37. McKee MD, Kim J, Kebaish K, Stephen DJG, Kreder HJ, Schemitsch EH (2000) Functional outcome after open supracondylar fractures of the humerus-effect of the surgical approach. *J Bone Joint Surg [Br]* 82-B:646–651
  38. McKee MD, Pugh DM, Wild LM, Schemitsch EH, King GJ (2005) Standard surgical protocol to treat elbow dislocations with radial head and coronoid fractures. Surgical technique. *J Bone Joint Surg [Am]* 87-A(Suppl 1):22–32
  39. McKee MD, Bowden SH, King GJ, Patterson SD, Jupiter JB, Bamberger HB, Paksima N (1999) Management of recurrent, complex instability of the elbow with a hinged external fixator. *J Bone Joint Surg [Br]* 80:1031–1036
  40. Koslowsky TC, Mader K, Dargel J, Koebke J, Hellmich M, Pennig D (2007) Reconstruction of a Mason type – III fracture of the radial head using four different fixation techniques: an experimental study. *J Bone Joint Surg [Br]* 89-B:1545–1550
  41. Koslowsky TC, Mader K, Dargel J, Pennig D, Koebke D (2007) Communitated radial head fractures – can they all be fixed? *Acta Orthopaed* 78:151–156
  42. Koslowsky TC, Mader K, Dargel J, Schadt R, Koebke J, Pennig D (2009) Olecranon fracture fixation with a new implant: biomechanical and clinical considerations. *Injury* 40:618–624
  43. Lashjian RZ, Katarincic JA (2006) Complex elbow instability. *J Am Acad Orthop Surg* 14:278–286
  44. Lee DH (2001) Treatment options for complex elbow fracture dislocations. *Injury* 32-S:41–69
  45. Lill H, Korner J, Hepp P, Verheyden P, Josten C (2001) Fracture dislocations of the elbow joint-strategy for treatment and results. *Arch Orthop Trauma Surg* 121:31–37
  46. Mader K, Koslowsky TC, Gausepohl T, Pennig D (2007) Mechanical distraction in post-traumatic stiffness of the elbow in children: operative technique. *J Bone Joint Surg [Am]* 89-A:26–35
  47. Mader K, Pennig D, Gausepohl T, Wulke AP (2004) Arthrolysis of the elbow joint. *Unfallchirurg* 107:403–414
  48. Mader K, Gausepohl T, Pennig D (2005) The operative technique of distraction arthrolysis (arthrodiatasis) for elbow stiffness. *Unfallchirurg* 107:115–119
  49. Morrey BF, An KN, Chao EYS (1993) Functional evaluation of the elbow. In: Morrey BF (ed) *The elbow and its disorders*, 2nd edn. W.B. Sanders, Philadelphia, pp 73–91
  50. Mouhsine E, Akiki A, Castagna A, Cikes A, Wettstein M, Borens O, Garofalo R (2007) Transolecranon anterior fracture dislocation. *J Shoulder Elbow Surg* 16:352–357
  51. Mortazavi SM, Asadollahi S, Tahririan MA (2006) Functional outcome following treatment of transolecranon fracture-dislocation of the elbow. *Injury* 37:284–288
  52. Mueller LP, Kamineni S, Rommens PM, Morrey BF (2005) Primary total elbow replacement for fractures of the distal humerus. *Oper Orthop Traumatol* 17:119–142
  53. Nielsen D, Nowinski RJ, Bamberger HB (2002) Indications, alternatives, and complications of external fixation about the elbow. *Hand Clin* 18:87–97
  54. Papandrea RF, Morrey BF, O' Driscoll SW (2007) Reconstruction for persistent instability of the elbow after coronoid fracture-dislocation. *J Shoulder Elbow Surg* 16:68–77
  55. Paksima N, Panchal A (2004) Elbow fracture-dislocations: the role of hinged external fixation. *Bull Hosp Jt Dis* 62:33–39
  56. Patterson SD, Bain GI, Mehta JA (2000) Surgical approaches to the elbow. *Clin Orthop* 370:19–33
  57. Pennig D, Gausepohl T, Mader K (2000) Transarticular fixator with motion capacity in fracture dislocations of the elbow. *Injury Suppl* 1:35–44

58. Perry CR, Tessier JE (1987) Open reduction and internal fixation of radial head fractures associated with olecranon fracture or dislocation. *J Orthop Trauma* 1:36–42
59. Regan W, Morrey BF (1989) Fractures of the coronoid process of the ulna. *J Bone Joint Surg [Am]* 71-A:1348–1354
60. Ring D, Jupiter JB (2002) Fracture-dislocation of the elbow. *Hand Clin* 18:55–63
61. Ring D, Jupiter JB (2000) Operative fixation and reconstruction of the coronoid process. *Tech Orthop* 15:147–154
62. Ring D, Jupiter JB (1998) Current concepts review: fracture-dislocations of the elbow. *J Bone Joint Surg [Am]* 80-A:566–580
63. Ring D, Jupiter JB, Zilberfarb F (2002) Posterior dislocation of the elbow with fractures of the radial head and coronoid. *J Bone Joint Surg [Am]* 84-A:547–551
64. Ring D, Hannouche D, Jupiter JB (2004) Surgical treatment of persistent dislocation or subluxation of the ulnohumeral joint after fracture-dislocation of the elbow. *J Hand Surg [Am]* 29:470–480
65. Ring D, Jupiter JB, Sanders RW, Mast J, Simpson NS (1997) Transolecranon fracture-dislocation of the elbow. *J Orthop Trauma* 11:545–550
66. Ruch DS, Triepel CR (2001) Hinged elbow fixation for recurrent instability following fracture dislocation. *Injury* 32-S:70–78
67. Saati AZ, McKee MD (2004) Fracture-dislocation of the elbow: diagnosis, treatment, and prognosis. *Hand Clin* 20: 405–414
68. Schneeberger AG, Sadowski MM, Jacob HA (2004) Coronoid process and radial head as posterolateral rotatory stabilizers of the elbow. *J Bone Joint Surg [Am]* 86-A: 975–982
69. Stavlas P, Gliatis J, Polyzois V, Polyzois D (2004) Unilateral hinged external fixator of the elbow in complex elbow injuries. *Injury* 35:1158–1166
70. Strauss EJ, Tejwani NC, Preston CF, Egol KA (2006) The posterior Monteggia lesion with associated ulnohumeral instability. *J Bone Joint Surg [Br]* 88-B:84–89
71. Tan V, Daluiski A, Capo J, Hotchkiss R (2005) Hinged elbow external fixators: indications and uses. *J Am Acad Orthop Surg* 13:503–514
72. Terada N, Yamada H, Seki T, Urabe T, Takayama S (2000) The importance of reducing small fractures of the coronoid process in the treatment of unstable elbow dislocation. *J Shoulder Elbow Surg* 9:344–346
73. Stoffel K, Cunneen S, Morgan R, Nicholls R, Stachowiak G (2008) Comparative stability of perpendicular versus parallel double – locking plating systems in osteoporotic comminuted distal humerus fractures. *J Orthop Res* 26:778–784
74. Wilkinson JM, Stanley D (2001) Posterior surgical approaches to the elbow: a comparative anatomic study. *J Shoulder Elbow Surg* 10:380–382
75. Whitcomb Pollock J, Brownhill J, Ferreira L, McDonald CP, James J, King G (2009) The effect of anteriomedial facet fractures of the coronoid and lateral ligament injury on elbow instability and kinematics. *J Bone Joint Surg [Am]* 91:1448–1458
76. Yadao MA, Savoie FH 3rd, Field LD (2004) Posterolateral rotatory instability of the elbow. *Instr Cours Lect AAOS* 53:607–614
77. Yu JR, Throckmorton TW, Bauer RM, Watson JT, Weikert DR (2007) Management of acute complex instability of the elbow with hinged external fixation. *J Shoulder Elbow Surg* 16:60–67
78. Zeiders GJ, Patel MK (2008) Management of unstable elbows following complex fracture-dislocations – the “terrible triad” injury. *J Bone Joint Surg [Am]* 90-A:75–84

# Fractures of the Scaphoid: Diagnosis and Management

José Carlos Botelho

---

## Foreword

In this written lecture we will not present the basic science fundamentals of scaphoid pathology – anatomy, physiological movements and vascular supply – that may be read in a good textbook – or all the possibilities and the most recent advances in diagnosis and treatment of scaphoid fractures, but rather concentrate on what is considered to be good clinical practice, based on the literature and our own experience.

Being a mobile link between the proximal and distal rows of carpal bones [21], the scaphoid is by far the most commonly fractured bone of the carpus – more than 80% [15]. Scaphoid fractures are specially frequent among young men and in the upper extremity are second only in incidence to fractures of the distal radius, that, by contrast, occur more commonly in older females [17].

Many scaphoid fractures are undisplaced, difficult to see in plain X-rays of the wrist and are commonly missed [21]. Being a poorly vascularised bone, especially its proximal pole, scaphoid fractures are slow to heal and non-union is frequent [17].

These are the main reasons why scaphoid fractures have been one of the most common topics of the Orthopaedic literature in the last 50 years.

---

## Diagnosis

Scaphoid fractures are commonly caused by a fall on the hand, but can also occur by a direct blow [16, 22]. Stress fractures have been described in young athletes [8].

---

J.C. Botelho  
Hand Surgery Unit, Hospital de Sant'Ana,  
Rua de Benguela,  
2779-501 Parede, Portugal  
e-mail: jcbotelho@gmail.com

Generally, the patient will easily locate the pain on the radial side of the wrist, distal to the radial styloid, and the area between the long and short extensors of the thumb – the “anatomic snuffbox” – will frequently be swollen. Typically, that area and the palmar scaphoid tuberosity will be most painful on palpation, but the patient’s complaints and the clinical examination are sometimes misleading and some patients with a scaphoid pseudarthrosis don’t even remember the initial accident.

Some clinical tests have been described – painful axial compression of the thumb and others – but are of little practical use [22].

The initial X-rays often show the fracture if carefully looked for after a suspicious clinical examination – they should include at least the normal PA and lateral projections of the wrist and the so called “scaphoid projection”. This is a view of the scaphoid bone in all its length and is taken PA with some extension and cubital deviation of the wrist [14]. Many other special projections have been described but they are of little practical use, especially nowadays with easily available bone scans [22].

If the initial X-rays do not show a suspected scaphoid fracture, it is time-honoured good practice to splint the wrist for 2 weeks and then repeat the radiological examination – a slight bone resorption at the fracture site will then show it [16, 21], but not always [22]. This is why other ways of dealing with the suspected scaphoid fracture have been described, with the use of bone scans, MRI, scintigraphy and ecography.

These methods have in common being expensive and not always readily available. On the other hand, splinting a wrist sprain for a fortnight is a good treatment, especially with a removable splint that allows washing of the hand. But they can be useful in special situations – high-level athletes for example – when ruling out a fracture is paramount. For this purpose, the MRI is especially useful [22] – a normal MRI will rule out a scaphoid fracture and even the image of a scaphoid “bone bruise” (marrow oedema – increased signal on T2) does not indicate a scaphoid

fracture, although in one published study the wrist was immobilised for 6 weeks [15].

So, as a rule of thumb, a suspected fractured scaphoid should have at least a repeat 3-projection X-ray at 2 weeks. If pain persists, repeat it after another fortnight or ask for a bone scan, MRI or scintigraphy (sensitive but not specific). A suspected scaphoid fracture is a fracture until proved otherwise!

### Differential Diagnosis

Differential diagnosis should include fractures of other carpal bones, although rare, that can be revealed by a bone scan, and serious wrist sprains, especially a rupture of the scapho-lunate ligament. This should be searched for if a scaphoid fracture is ruled out and pain persists.

### Classification

A diagnosed scaphoid fracture must be classified, not only for academic reasons but especially for correct treatment.

Many classifications have been proposed [14]. They are useful for clinical studies, but to decide treatment the important points to consider are:

- The fracture location – proximal, middle or distal third, tuberosity.
- Undisplaced or displaced.
- The presence of comminution?
- The age of the fracture – fresh, or weeks old...
- Is the fracture part of a trans-scapho-perilunate dislocation of the wrist? (a grossly displaced scaphoid fracture could mean a self-reduced one).

To classify and decide the treatment of a scaphoid fracture a bone scan is sometimes needed, especially in doubtful cases [22] (Fig. 1).

### Treatment

By and large, an undisplaced fracture of the scaphoid can still be treated by immobilisation but a displaced one should be fixed.

Classically, immobilisation should be with an antebrachial-palmar plaster including the base of the thumb but with its interphalangeal joint free, for up to 3 months, but including the elbow or leaving the thumb free have been advocated on anatomical and clinical grounds [22].



**Fig. 1** Undisplaced middle-third scaphoid fracture

If the surgeon decides to treat a scaphoid fracture conservatively the immobilisation should last at least 8–10 weeks [16, 21]. Then, if correct X-rays without the cast show bone union, the patient can start to mobilise his wrist but it is a sensible practice to repeat X-rays 15 days later to confirm bone union – if the fracture line re-appears it was once advised to re-apply the cast [21] but nowadays a surgical procedure seems advisable. Immobilisation for more than 3 months is considered worthless [20].

Repeating X-rays at 6 months is a useful practice that will disclose some painless non-unions [12].

However, even in undisplaced fractures of the proximal pole of the scaphoid bone, the surgeon and the patient should be aware of the fact that, due to the well-known poor vascularisation of the proximal small fragment, bone union can take more than 3 months with a cast and even never occur in up to 30% of cases [14]. For these reasons, a correct osteosynthesis may be a better alternative, to shorten the immobilisation time and to enhance the chances of bone union (Fig. 2).

Undisplaced fractures of the middle and distal thirds of the scaphoid do not have the same tendency to pseudarthrosis [14], but surgical treatment, with effective osteosynthesis, can be proposed confidently to the patients to shorten the immobilisation and off-work times, and many of them accept it gladly.

Distal tuberosity fractures, generally undisplaced, are said to unite in 6 weeks with an antebrachial palmar cast [22], although I have needed to operate on two delayed unions.

The acceptable displacement of a scaphoid fracture is questionable, but most surgeons now consider that more than 1 mm or any degree of angulation mean a displaced fracture that should be fixed surgically to shorten the immobilisation time and the possibility of non-union [22].



**Fig. 2** The same fracture after osteosynthesis with a double-thread cannulated screw inserted by a small palmar approach

Many distal third scaphoid fractures have a typical displacement with a dorsal angulation or hump. Grossly displaced scaphoid fractures, of course, must be reduced and fixed, to avoid pseudarthrosis and mal-union, and the same applies to those which are part of a transscaphoid-perilunar dislocation or associated with distal radius fractures [22].

Many types of osteosynthesis have been proposed to treat scaphoid fractures in the last 40 years [11], from simple K-wires to dozens of screws and even staples or plates, but the following are the most used screws nowadays. These are the double-thread cannulated ones, much easier to use than the original Herbert screw, and plain mini screws, used in the proximal third fractures.

The headless Herbert screw and others like it compress the fracture by the effect of the different screw threads, proximal and distal, and can and should be completely introduced in the bone to maximize this effect [12] – an oversized screw that protrudes at its insertion point should be avoided.

In undisplaced or little-displaced fractures of the middle and distal thirds of the scaphoid, those cannulated screws can be inserted through a short palmar approach to the scaphoid tuberosity or even percutaneously, with the control of an image intensifier. The K-wire and the cannulated drill and screw must not be inserted directly into the tuberosity but just deep to it, to reach the proximal pole and give a better compression [3]. This is not, in fact, a straightforward procedure for untrained hands and the mentioned short incision makes it easier than a percutaneous procedure, with no practical inconvenience. A 24-mm screw is most commonly used, after a correct measurement.

After surgery, immobilisation has ranged from none [11] to 6 weeks [3] with a plaster splint, but a plaster splint for 2 weeks – until stitch removal – and protection with a removable

wrist splint for another 2 or 4 weeks is better, depending on the X-ray appearances and the patient compliance.

Frankly-displaced middle and distal third scaphoid fractures should have reduction and osteosynthesis. This means for most surgeons an open reduction by a classical palmar Russe approach (radial to the FCR tendon) to the entire scaphoid [22], although some advocate an indirect closed reduction, even with the help of arthroscopy [9]. A K-wire, and then the cannulated drill and the double-thread cannulated screw are then inserted in the usual way, with the control of an image-intensifier. In very unstable fractures a K-wire can be added to control rotation during insertion of the screw which is then removed. As important wrist ligaments are severed in this approach, a plaster splint is generally used for 4 weeks at least [22].

Failure to reduce these fractures, just fixing them percutaneously, leads to scaphoid mal-union, with limitation of wrist extension, although often asymptomatic [2].

We generally do not remove these screws after bone union unless they protrude proximally or distally – and they shouldn't! Distally-protruding screws can even cause rupture of the FCR tendon [5].

Seldom, a palmar comminution of a distal fracture of the scaphoid will be found, leaving a gap after reduction and needing a bone graft [22], generally taken in these case from the palmar surface of the distal radius by a proximal extension of the surgical approach. One can either choose a cortico-cancellous graft and use a compression screw or just cancellous bone – in this case with a non-compressive fixation (2 K-wires) and a longer post-operative immobilisation.

If osteosynthesis is used to treat a proximal third scaphoid fracture, either because of its displacement, supposed instability or tendency to non-union, many surgeons prefer to place a mini-screw, 1.5 or 2.0 mm. in diameter, by a dorsal oblique articular approach, between the EDC and the EPL tendons [1] – this should be searched for and its sheath opened proximally – although percutaneous osteosynthesis with arthroscopy has been advised by some very experienced surgeons [9]. The articular capsule is opened cautiously to avoid damaging the scapho-lunate ligament, generally on the cubital side of the ECRB tendon, until the fracture line is seen and not beyond it to avoid disturbing the important dorsal vascularisation of the scaphoid. The fracture is then reduced if displaced, with a small bone lever, and a K-wire introduced into the pole of the scaphoid, pointing to the thumb. This must be done with the wrist in complete flexion and indeed the surgery must progress in this rather awkward position. If the image-intensifier confirms the correct position of the K-wire, this is withdrawn and replaced by a motor-driven drill corresponding to the desired screw. After the image-intensifier confirms the



**Fig. 3** Proximal scaphoid fracture, 6 weeks old, not seen in repeated X-rays, disclosed by a CT bone scan



**Fig. 4** The same fracture, after osteosynthesis with a 1.5-mm screw by inserted by a proximal articular approach with radius cancellous bone grafting

correct position of the drill, this one is replaced by a screw, almost always of a 22-mm length so that we no longer measure it any more (Fig. 3).

The capsule and skin are then sutured and a plaster splint is put in place until the stitches are removed at 2 weeks. It is then replaced by a removable wrist splint for 2 or 4 weeks more, depending on stability of the fracture and the reliability of the patient.

Although we introduce the screw head under the cartilage surface, we routinely remove it in a second surgery, some 4–5 months later, when X-rays (or scan) show a sound bone union – this is not generally easy, due to the fibrosis, but has in our experience the advantage of increasing the wrist mobility, apart from removing a possible cause of damage to the articular surface of the radius.

In some cases with a bigger proximal scaphoid fragment, two screws can be inserted [16].

We do not use double-thread screws for proximal fractures because even the mini-Herbert one seems to cause more harm to the proximal pole cartilage than a 1.5-mm normal screw (Fig. 4).

### Transcaphoid-Perilunar Dislocations

In these cases, the issue is whether or not the dislocation was reduced in the emergency department. The fracture often lies in the middle third or waist of the scaphoid.

If the dislocation was not reduced it is probably better to operate on the patient by a dorsal mid-line approach to the wrist, place a retrograde k-wire in the triquetrum, reduce the dislocation, reintroduce this k-wire to the lunate – Voche's trick [20] – and then fix the scaphoid

fracture by the dorsal approach with K-wires or a screw [13]. Radio-lunate or luno-capitate K-wires are sometimes needed as well.

If the dislocation is already reduced, the scaphoid fracture can be reduced and fixed with a double-thread screw by a palmar approach and the luno-triquetral joint fixed percutaneously or with a dorsal approach by a K-wire.

In some rare cases, a scaphoid fracture – normally of its middle third – co-exists with a ruptured scapho-lunate ligament, apparent on plain X-rays by comparing it to the other wrist. This should also be addressed surgically [14].

The rare Fenton syndrome – a scaphoid fracture with an associated displaced fracture of the capitate – should not be forgotten [14].

I once removed a proximal pole of the scaphoid, in a widely-displaced transcapho-perilunar dislocation, that was located just palmar to the distal radius and most probably devascularised, and replaced it with a rolled tendon, but this is a debatable [14] and exceptional procedure.

Surgical reduction and fixation of late-diagnosed transcapho-perilunar dislocations is easier after a week-long distraction with an external fixator [18].

### Scaphoid Fractures in Children

These are very rare, occurring mostly in the distal third before the age of 10 years and the middle third after that age, proximal ones being exceptional. Conservative treatment is the rule and pseudarthrosis is a rarity [6, 22]. Congenital pseudarthrosis of the scaphoid should only be cautiously considered in bilateral cases [10].



## “Old” Fractures

The borderline between a late diagnosed fracture and a pseudarthrosis is hard to define, but perhaps a 3-month delay is acceptable [22]. The patient’s information on the age of the fracture is sometimes unreliable and plain X-rays can be misleading – a bone scan can be useful.

Although success has been claimed in treating late-diagnosed undisplaced fractures conservatively [15] or with a percutaneous screw [9], I think it is wiser to approach most of these fractures surgically and treat them according to what is found.

Late-diagnosed proximal pole fractures are generally treated, with success, with a single screw placed by the dorsal approach, as described, with no bone grafting [1] – although this can be needed in some rare cases.

Middle and distal third fractures should be treated by a palmar approach – if they appear to be uniting, a double-thread headless screw is then simply applied. If they appear to be developing pseudarthrosis, they should be treated accordingly. We use either the Diego-Fernandez technique, with an intercalary cortico-cancellous graft, for angulated distal pseudarthrosis [7]; or a Starck-modified Russe technique for less displaced middle-third pseudarthroses [19]. Some in-between cases can be managed with a screw and some cancellous bone chips in the fracture line.

## Bone Union

This is notoriously difficult to evaluate in scaphoid fractures, specially in the operated ones, but the absence of a visible gap in all X-rays projections at 3 months can be accepted as a good sign, but X-rays should be repeated at 6 and probably 12 months. Before 3 months it is difficult to say that a scaphoid fracture is united and the so-often-used “time to union” is prone to error [4].

CT scans can be useful in doubtful cases. In operated cases, progressive lucency around part or the entire screw normally means non-union of the fracture.

## Rehabilitation

Although formal rehabilitation of scaphoid fractures has been considered unnecessary [19], most surgeons begin it after bone union is achieved. In fact, with solid osteosynthesis and experienced therapists, it can begin well before that. Some limitation of wrist mobility is common at least in the short-term [3] and the patients should be warned in advance.

**Acknowledgements** I want to thank Dr. Silvia Silverio, from Hospital de Sant’Ana, for her help in most of these surgical procedures and in writing this manuscript and also Mr. Henrique Marques for his information and help.

## References

1. Alnot JY (1988) Les fractures et pseudarthroses polaires proximales du scaphoïde carpien. *Rev Chir Orthop* 74:740–743
2. Amadio et al (1989) Scaphoid malunion. *J Hand Surg* 14A:679–687
3. Cheveigne C (2004) Vissage conventionnel des fractures fraiches du scaphoïde carpien. In: Lussiez B, Rizzo C, Lebreton E (eds) *Le Scaphoïde*. Sauramps Medical, Montpellier
4. Dias JJ (2004) Criteria for the diagnosis of healing of a scaphoid fracture or nonunion. In: Lussiez B, Rizzo C, Lebreton E (eds) *Le Scaphoïde*. Sauramps Médical, Montpellier
5. Ducharme G, Frick L, Schoofs M (2009) Rupture du tendon du fléchisseur radial du carpe après ostéosynthèse percutanée d’une fracture de scaphoïde. À propos d’un cas. *Chir de la main* 28:50–52
6. Duteille F, Dautel G (2004) Les fractures du scaphoïde avant maturité osseuse. In: Lussiez B, Rizzo C, Lebreton E (eds) *Le Scaphoïde*. Sauramps Médical, Montpellier
7. Fernandez DL (1984) A technique for anterior wedge-shaped grafts for 3 scaphoid nonunions with carpal instability. *J Hand Surg* 9A:733–737
8. Fichiez O (2004) Les fractures de fatigue du scaphoïde carpien. In: Lussiez B, Rizzo C, Lebreton E (eds) *Le Scaphoïde*. Sauramps Medical, Montpellier
9. Fontes D, Roure P (2004) Vissage percutané des fractures du scaphoïde carpien sous contrôle arthroscopique. In: Lussiez B, Rizzo C, Lebreton E (eds) *Le Scaphoïde*. Sauramps Medical, Montpellier
10. Genestet M et al (2004) Scaphoïde bipartite: mythe ou réalité? In: Lussiez B, Rizzo C, Lebreton E (eds) *Le Scaphoïde*. Sauramps Médical, Montpellier
11. Herbert TJ (2004) Internal fixation of the scaphoid – history. In: Lussiez B, Rizzo C, Lebreton E (eds) *Le Scaphoïde*. Sauramps Médical, Montpellier
12. Herbert TJ, Fisher WE (1984) Management of the fractured scaphoid bone using a new bone screw. *J Bone Joint Surg* 66B:114–123
13. Herzberg G (2004) Fractures du scaphoïde dans les luxations périlunaires du carpe fermées récentes. In: Lussiez B, Rizzo C, Lebreton E (eds) *Le Scaphoïde*. Sauramps Medical, Montpellier
14. Irisarri C (2002) *Patologia del escafoïdes carpiano*. Norgrafica, Vigo
15. La Hei N, Mc Fayden I, Brock M et al (2007) Scaphoid bone bruising – probably not the precursor of asymptomatic non-union of the scaphoid. *J Hand Surg* 32E:337–340
16. Leslie IJ, Dickson RA (1981) The fractured carpal scaphoid, natural history and factors influencing outcome. *J Bone Joint Surg* 63B:225–230

17. Romano S, Couturier C, Sokolow C (2004) Fracture du sca-  
phoïde: double visage proximal. In: Lussiez B, Rizzo C,  
Lebreton E (eds) *Le Scaphoïde*. Sauramps Medical,  
Montpellier
18. Sousa HP, Fernandes H, Botelho JC (1995) Pre-operative  
progressive distraction in old transcapho-*peri-lunate* dislo-  
cations. *J Hand Surg* 20B:603–605
19. Starck HH et al (1988) Treatment of ununited fractures of  
the scaphoid by iliac bone grafts and Kircher wire fixation.  
*J Bone Joint Surg* 70A:982–991
20. Voche P (1995) Luxations perilunaires du carpe. In: Merle  
M, Dautel G (eds) *La main traumatique 2*. Masson, Paris
21. Wilson JN (1976) *Watson-jones fractures and joint injuries*.  
Churchill Livingstone, Edinburgh, London, New York
22. Wolfe SW (2004) Fractures of the carpus: scaphoid frac-  
tures. In: Berger RA, Weis AP (eds) *Hand surgery*. Lippincott  
Williams & Wilkins, Philadelphia

**Part V**

---

**Spine**

# Treatment of Thoraco-Lumbar Fractures

Antonio A. Faundez

---

## Introduction

Thoraco-lumbar fractures (Th10-L2) in young adults are common and often associated with profound socio-economic consequences [20]. Most of these result from motor vehicle accidents and falls from heights, which involve high kinetic energy and affect mainly males. Very often, patients are polytraumatized and present with associated thoracic and/or abdominal injuries. Initial in-hospital management is carried out following the Advanced Trauma Life Support (ATLS) guidelines, where priority is given to stabilization of vital functions and only then to neurologic functions. The traumatized spine is assessed using standard radiologic imaging, as well as CT scan. MRI can provide valuable information about neural tissue and disco-ligamentous injuries. Specific treatment decisions will then rely on both intrinsic (e.g., fracture morphology, neurologic status, mechanical instability) and extrinsic factors (e.g., age, occupation, level of physical activity). The main goal of surgical treatment is to protect the neural tissue by mechanically stabilizing the spine and additionally decompressing the spinal canal if necessary. We present here an overview of current treatment options available to surgeons for the treatment of thoraco-lumbar fractures.

---

## Epidemiology of Spinal Injuries

Thoraco-lumbar fractures affect mostly males between 20 and 30 years old and are due to high energy trauma, mostly motor vehicle accidents (40–50%) and falls (around 20%)

---

A.A. Faundez  
HUG - Hôpitaux Universitaires de Genève,  
24 Rue Micheli-du-Crest,  
1211 Geneva, Switzerland  
e-mail: antonio.faundez@hcuge.ch

[4]. It is difficult to present exact numbers for the incidence of spine fractures because of inconsistent data collection amongst Trauma centers. In a recent epidemiological review, it was estimated that the incidence of adult thoraco-lumbar fractures in the United Kingdom is around 117/10<sup>5</sup> inhabitants/year [10]. The incidence of spinal cord injuries is better documented and is reported to range between 27 and 47 per million population in North America, with an acute mortality rate that has dramatically decreased from 38 to 15.8% over the past 30 years [20]. Major improvements have been made in pre- and in-hospital spinal cord injury management, as well as in surgical implants and techniques, thus allowing provision of better trauma care today.

---

## Initial Management of Polytrauma Patients with an Associated Spine Injury

Patients with spine injuries are often polytraumatized. Strict adherence to ATLS guidelines is required before and upon arrival to the trauma center [16]. Taking pictures of the scene of the accident can be very useful to determine the mechanism of trauma and is a current practice now in several paramedic teams. The patient should be adequately ventilated and oxygenated and the cervical spine immediately immobilized in a rigid collar. In the emergency room, after vital functions have been stabilized, a detailed physical examination and a thorough neurologic clinical assessment is performed in the conscious patient. As polytrauma patients frequently present with altered consciousness, they are usually immediately screened with a total body CT scan that also allows detection of occult fractures of the spine, which are frequently overlooked in this category of patient [2]. If a spinal cord injury is diagnosed, neurologic impairment is evaluated according to the American Spinal Injury Association (ASIA) classification (Fig. 1). Mean blood pressure should be maintained above 90 mmHg

**STANDARD NEUROLOGICAL CLASSIFICATION OF SPINAL CORD INJURY**

**MOTOR KEY MUSCLES**

	R	L
C2	<input type="checkbox"/>	<input type="checkbox"/>
C3	<input type="checkbox"/>	<input type="checkbox"/>
C4	<input type="checkbox"/>	<input type="checkbox"/>
C5	<input type="checkbox"/>	<input type="checkbox"/>
C6	<input type="checkbox"/>	<input type="checkbox"/>
C7	<input type="checkbox"/>	<input type="checkbox"/>
C8	<input type="checkbox"/>	<input type="checkbox"/>
T1	<input type="checkbox"/>	<input type="checkbox"/>
T2	<input type="checkbox"/>	<input type="checkbox"/>
T3	<input type="checkbox"/>	<input type="checkbox"/>
T4	<input type="checkbox"/>	<input type="checkbox"/>
T5	<input type="checkbox"/>	<input type="checkbox"/>
T6	<input type="checkbox"/>	<input type="checkbox"/>
T7	<input type="checkbox"/>	<input type="checkbox"/>
T8	<input type="checkbox"/>	<input type="checkbox"/>
T9	<input type="checkbox"/>	<input type="checkbox"/>
T10	<input type="checkbox"/>	<input type="checkbox"/>
T11	<input type="checkbox"/>	<input type="checkbox"/>
T12	<input type="checkbox"/>	<input type="checkbox"/>
L1	<input type="checkbox"/>	<input type="checkbox"/>
L2	<input type="checkbox"/>	<input type="checkbox"/>
L3	<input type="checkbox"/>	<input type="checkbox"/>
L4	<input type="checkbox"/>	<input type="checkbox"/>
L5	<input type="checkbox"/>	<input type="checkbox"/>
S1	<input type="checkbox"/>	<input type="checkbox"/>
S2	<input type="checkbox"/>	<input type="checkbox"/>
S3	<input type="checkbox"/>	<input type="checkbox"/>
S4-5	<input type="checkbox"/>	<input type="checkbox"/>

Elbow flexors  
Wrist extensors  
Elbow flexors  
Finger flexors (distal phalanx of middle finger)  
Finger abductors (little finger)

Hip flexors  
Knee extensors  
Ankle dorsiflexors  
Long toe extensors  
Ankle plantar flexors

Voluntary anal contraction (Yes/No)

**TOTALS**  +  =  **MOTOR SCORE**  
(MAXIMUM) (50) (50) (100)

**SENSORY KEY SENSORY POINTS**

	R	L
C2	<input type="checkbox"/>	<input type="checkbox"/>
C3	<input type="checkbox"/>	<input type="checkbox"/>
C4	<input type="checkbox"/>	<input type="checkbox"/>
C5	<input type="checkbox"/>	<input type="checkbox"/>
C6	<input type="checkbox"/>	<input type="checkbox"/>
C7	<input type="checkbox"/>	<input type="checkbox"/>
C8	<input type="checkbox"/>	<input type="checkbox"/>
T1	<input type="checkbox"/>	<input type="checkbox"/>
T2	<input type="checkbox"/>	<input type="checkbox"/>
T3	<input type="checkbox"/>	<input type="checkbox"/>
T4	<input type="checkbox"/>	<input type="checkbox"/>
T5	<input type="checkbox"/>	<input type="checkbox"/>
T6	<input type="checkbox"/>	<input type="checkbox"/>
T7	<input type="checkbox"/>	<input type="checkbox"/>
T8	<input type="checkbox"/>	<input type="checkbox"/>
T9	<input type="checkbox"/>	<input type="checkbox"/>
T10	<input type="checkbox"/>	<input type="checkbox"/>
T11	<input type="checkbox"/>	<input type="checkbox"/>
T12	<input type="checkbox"/>	<input type="checkbox"/>
L1	<input type="checkbox"/>	<input type="checkbox"/>
L2	<input type="checkbox"/>	<input type="checkbox"/>
L3	<input type="checkbox"/>	<input type="checkbox"/>
L4	<input type="checkbox"/>	<input type="checkbox"/>
L5	<input type="checkbox"/>	<input type="checkbox"/>
S1	<input type="checkbox"/>	<input type="checkbox"/>
S2	<input type="checkbox"/>	<input type="checkbox"/>
S3	<input type="checkbox"/>	<input type="checkbox"/>
S4-5	<input type="checkbox"/>	<input type="checkbox"/>

0 = absent  
1 = impaired  
2 = normal  
NT = not testable

Any anal sensation (Yes/No)

**TOTALS**  +  =  **PIN PRICK SCORE** (max: 112)  
(MAXIMUM) (56) (56) (56) (56)

+  =  **LIGHT TOUCH SCORE** (max: 112)  
(MAXIMUM) (56) (56) (56) (56)

**NEUROLOGICAL LEVEL**  
The most caudal segment with normal function

	R	L
SENSORY	<input type="checkbox"/>	<input type="checkbox"/>
MOTOR	<input type="checkbox"/>	<input type="checkbox"/>

**COMPLETE OR INCOMPLETE?**

Incomplete = presence of any sensory or motor function in lowest sacral segment

**ZONE OF PARTIAL PRESENTATION**  
Partially innervated segments

	R	L
SENSORY	<input type="checkbox"/>	<input type="checkbox"/>
MOTOR	<input type="checkbox"/>	<input type="checkbox"/>

**Fig. 1** The ASIA scale of neurologic impairment. The motor and sensory deficits are recorded on the data sheet *left*. The scale of impairment (A, B, C, D, E) is detailed on the *right*

to protect the cord from secondary ischaemic injury [4]. Until relatively recently, the administration of steroids was also considered as a standard of care [6]. However, various methodological flaws of the clinical trials conducted by the National Acute Spinal Cord Injury Study Group (NASCIS) have seriously questioned the validity of their conclusions, and because of possible serious adverse effects, steroids should no longer be administered without further clinical research [18, 33]. A more detailed description of the medical management of spinal cord injury is provided in the article by Bernhard et al. published in 2005 [4]. Once urgent care has been delivered, treatment strategy decisions for the spinal injury need to be developed, also including management of possible fractures of the appendicular skeleton.

## Thoraco-Lumbar Trauma Imaging

Polytraumatized patients are often immediately taken to the radiology department for a total body CT scan once vital functions have been stabilized. However, standard radiologic imaging must still be part of the initial assessment as it provides an aerial view of the osseous lesions of the spine that

cannot be completely replaced yet by the CT scan. Several basic pathomorphologic signs can already be identified on plain radiographs, such as the amount of height loss of the vertebral body, the interpedicular distance on antero-posterior views, interspinous distance and interruption of the posterior wall on lateral views, and the amount of kyphotic deformity. CT scan is useful to precisely analyze the bony contour of the spinal canal, but also the amount of vertebral body destruction, comminution and spread of fragments.

MRI is another important tool for the analysis of spinal cord and ligamentous injuries that cannot readily be detected on plain radiographs or CT scan. It often shows the real extent of vertebral injury by detecting changes of bone marrow signal intensity. It has been evaluated for the characterization of spine injuries since 1989 [17] and proposed for inclusion in future spine trauma classification schemes as early as 1995 [38]. Only more recent studies have investigated its clinical validity in the management of thoraco-lumbar fractures [11, 25, 26]. However, in an article by Dai et al. published in 2009, the practical role of MRI in clinical decision-making was questioned, in particular for burst fractures [11]. The authors argued that although it may be a reliable instrument for the assessment of ligamentous injuries, it did not correlate with

neurologic status or fracture severity, and as such should not be used routinely. Further studies are awaited to better define indications for MRI investigation of non-osteoporotic spinal fractures.

## Classification Systems

A variety of thoraco-lumbar fracture classifications have been described in the literature, but none has reached a consensus amongst Spine and Trauma surgeons. The most frequently cited classifications systems are the Denis classification (three-column theory), the load-sharing classification, and the AO classification (named after the founding Swiss group “Arbeitsgemeinschaft für Osteosynthesefragen”).

### Evolution of Classification Systems

Lorenz Boehler (1885–1973) was one of the first Trauma surgeons in Europe, and head of the first hospital for labourers, based in Vienna. His seminal work on the treatment of fractures was first published in 1929 [5]. Despite difficulties to achieve publication, the book encountered an important success and was soon translated into English and later into other languages. Boehler’s book is richly illustrated with drawings and pictures of various thoracic and lumbar fracture types and their long-term deformity if not treated appropriately. He described five categories of thoracolumbar injuries which served later as a basis for the Watson-Jones’ classification in 1938 [49]. Holdsworth was the first to use the term “burst fracture” [22]. He also introduced the concept of “column”, dividing the spine into an anterior (vertebral body and disc) and a posterior column (posterior facet joints and posterior ligamentous complex [PLC]). Some of the aspects of Holdsworth’s classification were later redefined by Kelly and Whitesides [23] and served as the basis of the more recent AO classification published in 1994 by Magerl et al. [30].

### Denis Classification (1983)

A major stage was reached in the management of spine trauma with the advent of CT scan imaging in the eighties. Using this new radiologic tool, Denis reviewed 412 patients with thoraco-lumbar fractures and published in 1983 one of the most frequently-cited thoraco-lumbar fracture classification systems today [13]. The results of this study and the concept of “middle column” originated from the observation

that during scoliosis surgery, where he would release both anterior and posterior columns, he did not observe any major mechanical instability as defined in Holdsworth’s classification. Denis concluded that the middle column had to be disrupted to result in a clinically significant instability. Four major types were defined: compression fracture, burst fracture, seat belt fracture and fracture-dislocation (flexion injury). It is often claimed that Denis’ classification is incomplete, and does not describe other pathomorphologic fracture types, e.g., the “lumberjack” fracture type, which was however described by himself later in 1992 [7, 14].

### The Load-Sharing Classification (1994)

In 1994, McCormack and Gaines described their load-sharing classification in an attempt to help the surgeon to predict the risk of implant failure in short-posterior segment constructs, such as the ones obtained using the AO internal fixation device [31] (Fig. 2). They proposed a decisional algorithm to decide whether an additional reconstruction of the anterior column was necessary in burst fractures based on three criteria: comminution of the vertebral body; apposition of fragments of the vertebral body; and reducibility of sagittal deformation. Although a few studies have reported its validity in clinical decision-making [12, 28, 35], the disadvantage of this algorithm is that it does not take into account the neurologic status of the patient, which is a major drawback in clinical care.

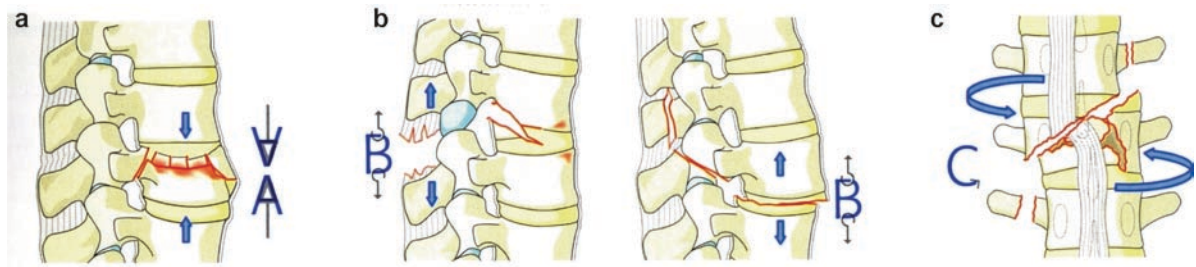
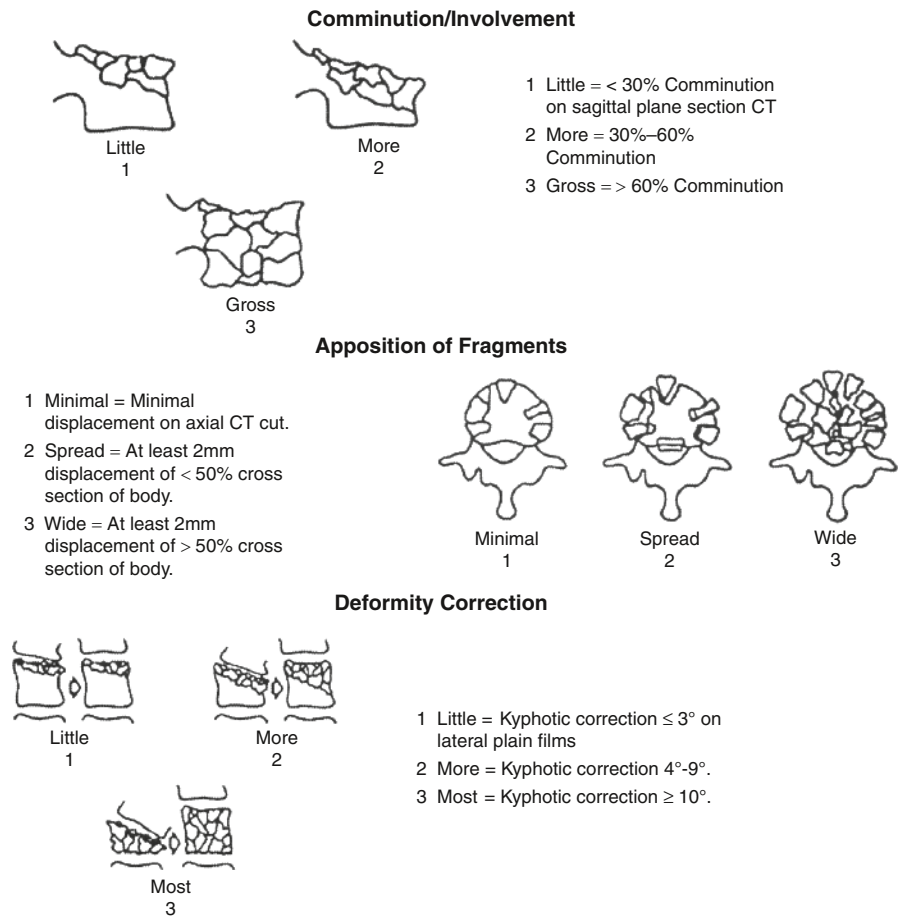
### The AO Classification (1994)

In 1994, Magerl et al. proposed the AO classification of thoraco-lumbar fractures following a review of 1,445 cases [30]. Fractures are classified according to three pathomorphologic types: type A (flexion-compression fractures); type B (distraction); and type C (rotational-shearing) (Fig. 3). In an attempt to design a system describing every possible fracture, the authors further divided each type into sub-groups, sub-types and sub-divisions, resulting in a total of 53 patterns.

### The Thoraco-Lumbar Injury Classification and Severity System (TLICS, 2005)

At present, none of these classifications has been adopted as a universal reference, mainly because of their poor intra- and inter-observer reliability [52]. The latest classification system described in the literature is the Thoraco-lumbar Injury Classification and Severity System (TLICS) [36, 43]

**Fig. 2** The load-sharing classification of Burst fractures. The classification was proposed in an attempt to predict the risk of implant failure in short-posterior segment constructs. Three items are considered: 1 comminution; 2 apposition of fragments; 3 reducibility of the deformity. Each item is given a numerical value. Originally, the authors concluded that a score equal or greater than seven represented a high risk of failure of short segment fixations (adapted from [31, 42])



**Fig. 3** The AO classification of fractures. Three major traumatic mechanisms were described: (a) Flexion-compression fractures; (b) distraction injuries, either in hyperflexion or hyperextension; (c) rotational-shear fractures. There is a progressive scale of

severity of the injury from type A to type C with a reported frequency of neurologic deficit of 14% in type A, 32% in type B and 55% in type C (with permission from [1])

that results from another classification system initially called the Thoraco-lumbar Injury Severity Score (TLISS). TLICS is a spine trauma evaluation score that considers three parameters: (1) the fracture morphology, based on the main mechanisms described in the AO classification; (2) the neurologic status; (3) the integrity of the PLC, inferred

by clinical and radiologic examination, including MRI (Table 1). A numerical value is assigned for each injury subcategory, depending on the severity of injury. The sum of each numerical value is used to guide the treatment decision: a score of 5 or more suggests a surgical treatment; a score of 3 or less, a conservative treatment. For a score

Category	Points
<b>injury morphology</b>	
compression	1
burst	+1
translational/rotational	3
distraction	4
<b>neurological status</b>	
intact	0
nerve root	2
cord, conus medullaris	
incomplete	3
complete	2
cauda equina	3
<b>PLC</b>	
intact	0
injury suspected/indeterminate	2
injured	3

**Table 1** The Thoraco-Lumbar Injury Classification and Severity System (TLICS adapted from [36]). The classification is based on three items: 1 Injury morphology; 2 Neurological status; 3 Integrity of PLC. A numerical value is attributed to each item and a total score is calculated: if  $\leq 3$ , non-surgical treatment is advocated; if  $\geq 5$ , surgical treatment is recommended; for a score of 4, either non-surgical or surgical treatment can be decided based on other confounding factors

of 4, either surgical or conservative treatment can be recommended, also based on other confounding factors, such as the age of the patient, amount of kyphosis, quality of bone, etc [36]. TLICS has shown improved intra- and inter-rater reliability in recent studies, but only within the group of physicians who developed the system [37, 43], and further studies are needed to more widely validate this promising decisional algorithm.

## Non-Surgical Treatment of Thoraco-Lumbar Fractures

### A0 Types A1 and A2

With the advent of less-invasive surgical techniques, there will probably be a future shift towards surgical treatment for lesions that would have been classically treated conservatively. Nevertheless, conservative treatment of thoraco-lumbar fractures still has a role to play at present. It can be divided into functional treatment or bracing with or without external reduction manoeuvres. In our institution, functional treatment (isometric muscular exercises) is

applied for AO fractures of type A1 (impaction and wedge fractures,  $5^\circ$  vertebral kyphotic angulation). For most of type A2 (split fractures), we recommend bracing, usually a three-point thoraco-lumbar orthosis, for 6–12 weeks, depending on the radiologic follow-up. As already pointed out by Boehler at the beginning of the twentieth century, intensive and immediate physical therapy with the brace in place should be an integral part of the treatment plan [5]. In the particular case of the pincer-type fracture (A2.3), surgical treatment is recommended because of a high risk of non-union [30].

### Burst Fractures (AO Type A3)

There is no consensus in the literature on the treatment of burst fractures [42, 51]. In our institution, mainly complete burst fractures (AO A3.3) are treated surgically. Incomplete burst fractures with acceptable sagittal deformity, up to  $15^\circ$  in the thoraco-lumbar junction, can be handled with a custom-made brace. However, despite an ongoing debate for years over the amount of tolerable kyphotic deformity of the thoraco-lumbar junction, it is interesting to note that only recent studies have started to focus on global sagittal balance and thoraco-lumbar fractures. Koller et al. published a retrospective study analyzing the long-term radiologic and clinical outcome for regional post-traumatic kyphosis of conservatively-treated thoraco-lumbar and lumbar burst fractures according to the global spino-pelvic alignment of each patient [24]. They concluded that the patient's global spine compensates for the post-traumatic regional kyphosis within the limits dictated by their pelvic geometry, in particular the pelvic incidence. They also found that clinical outcome correlated with regional kyphosis. Finally, the authors recommended that fractures with a load-sharing classification score of more than 6 should be treated by aggressive surgical reconstruction.

### A0 Fractures Type B and C

Except for type B2.1, also known as “Chance fracture” [9], which can be successfully treated by bracing, surgical treatment is recommended for most type B as well as type C injuries. Type B and C fractures result from very high energy trauma and usually include ligamentous disruptions that have a very poor healing potential. These fracture types are also associated with neurologic symptoms in 32% and 55% of patients, respectively [30]. As for the strategy of stabilization and reconstruction, the same principles of



amount of kyphosis and vertebral body destruction apply (see Chap. 7). It is important to recall, however, that not all neurologic symptoms imply surgical treatment. In a well-done Instructional Lecture course, Rechtine discusses other similar unfounded myths around treatment indications for thoraco-lumbar fractures [39, 40].

---

## Surgical Treatment

### Surgical Treatment for Fractures with Neurologic Deficit

As mentioned above, type B and C injuries usually require surgical treatment and are characterized by serious biomechanical instability and deformity, frequently accompanied by neural tissue damage. A treatment strategy is quite simple to define for patients with immediate and complete spinal cord damage. If any intervention is to be planned, it should be carried out only once vital functions have been stabilized, keeping in mind that the primary goal of surgery is to enhance nursing care and rehabilitation [8, 21]. For incomplete or progressive lesions, it is accepted that surgical decompression and stabilization should be performed within 6 to a maximum of 24 h from injury [19]. Posterior decompression and stabilization can be recommended as a first emergency procedure. However, it is not mandatory that any residual anterior compression be relieved as an emergency. Even if it is now suggested that the amount of canal narrowing is strongly associated with severity of the neurologic deficit [32, 34], it does not correlate with the prognosis of functional recovery. In other words, the removal of a large intra-canal bone fragment will not necessarily improve the chances of neurologic recovery.

### Surgical Treatment for AO Type A and B Fractures Without Neurologic Deficit

The treatment decision for thoraco-lumbar fractures of AO types A and B (predominantly osseous injury) without neurologic deficit remains very controversial and it must be recalled that there is a very real risk to end up with irreversible iatrogenic nerve tissue damage. However, as described by Boehler and others [5, 24, 42], there is a rationale to treat these patients surgically given the risk of mid to late onset deformity and its possible progressive cord compression and/or chronic disabling pain. Indications for surgical treatment of fractures without neurologic deficit should be based on the amount of deformity and weighed against its anatomical location, and on the amount of mechanical instability inferred by the analysis of radiologic documentation, including MRI

(fracture classification). As an example, for burst fractures (AO type A3) of the thoraco-lumbar junction, a maximum of 15° of regional kyphosis (measured between the upper end-plate of the vertebra above and the lower end-plate of the vertebra below) is usually tolerated for non-surgical treatment. However, it will also depend on the amount of destruction of the vertebral body. For example, an incomplete burst fracture (AO 3.1) with 10–15° of regional kyphosis will be treated by external reduction and bracing in our institution. But if the burst is complete (AO 3.3), reaching a high level of instability in compression, we will treat it surgically [24, 42].

A further decision, even more controversial, has to be made for complete burst fractures, i.e., whether a short posterior fusion will be enough or if an anterior approach and vertebral body reconstruction is needed, according to the load-sharing classification [31]. We continue to use the AO “fixateur interne” as a posterior stabilization implant. The technique is based on the principles of posterior short segment stabilization and ligamentotaxis: the indirect reduction of sagittal deformity and intra-canal bone fragments of the posterior wall through posterior longitudinal ligament (PLL) retensioning [15, 27]. The success of the technique obviously relies on the integrity of the PLL. The “reverse cortical sign” is a radiologic sign corresponding to a 180° flip of the postero-superior wall fragment and a consequent rupture of the PLL [3]. If present, this sign normally precludes any efficiency of ligamentotaxis alone for reduction of the fragment and an additional anterior approach for direct decompression should then be considered. Of note, despite previous recommendations to remove intra-canal bone fragments [48], the compromise of the spinal canal is not in itself an indication for surgical treatment in the absence of neurologic symptoms [39]. In a minimum 5-year follow-up study, Wessberg et al. have confirmed that intra-canal fragments stemming from the posterior wall are subject to remodelling and that the cross-sectional area of the spinal canal recovers up to 87% of its normal value without surgery [50]. If an anterior vertebral reconstruction is deemed necessary, it can be done by a classic open approach, for example the extra-pleural approach of the thoraco-lumbar junction, or by a video-assisted, less invasive approach [45]. More recently, vertebral body augmentation with calcium-phosphate cement has gained popularity and might be an alternative to more aggressive surgery in fractures without neurologic injury, but the risk of intra-canal cement extravasation has to be assessed. In addition, improvements in cement resistance are required before it can be recommended as a routine procedure.

Some AO type B fractures can also be treated either surgically or non-surgically. Figure 4 presents a typical case of a young male patient who suffered a bi-column fracture (AO type B2.1 or Chance type). The accident occurred in an old car with only two-point seat belts and he suffered from

**Fig. 4** Chance fracture of L2 (AO type B2.1) in a young male patient involved in a car accident, without neurologic deficit. Decision was taken with the patient to surgically treat the fracture using a minimally-invasive technique to avoid external reduction and bracing. At 2 year follow-up, the patient was symptom-free and the fracture radiologically healed. **(a)** Pre-operative X-ray. **(b)** Post-operative X-ray. **(c)** Skin incisions at 2 years follow-up

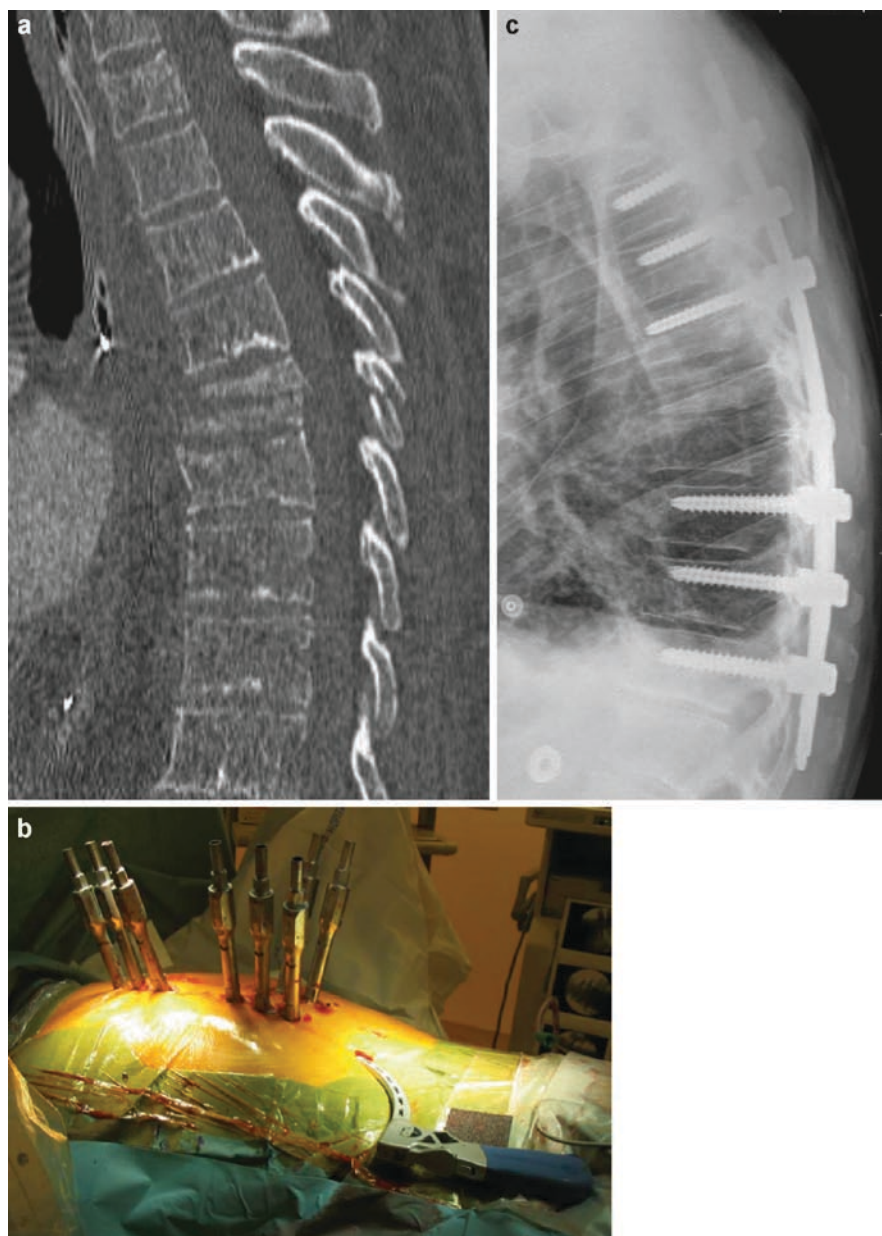


splenic and hepatic contusions, in addition to a hyperflexion fracture. It is known that these types of fractures with a predominant osseous instability respond very well to external reduction and bracing [30]. For various reasons, bracing can be very impractical (hot weather, very active patient, overweight patient, etc.) and surgery can reasonably be proposed. However, it is of the utmost importance that the decision is taken together with the patient, and not by the surgeon alone. As for any other treatment, risks and benefits have to be discussed and weighed against each other.

### Less-Invasive and Recent Surgical Techniques

Major improvements have been made during the past decade in spine surgical techniques and new instruments have been developed to insert implants through small incisions. For instance, surgeons have acquired an expertise in endoscopic treatment of vertebral fractures and extensive exposure of

the thoraco-lumbar junction is no longer necessary to perform corpectomies and vertebral reconstructions with structural allografts or cages [45]. Pedicle screws can also be inserted percutaneously, with the consequence of lowering blood loss and operative time. An example of a fracture treated by percutaneous pedicle screws and rod placement is shown in Fig. 5. A technique that has recently gained popularity is cement augmentation of the vertebral body. It has been used for treatment of osteoporotic fractures for a long time, but only more recently for high energy spine fractures [47]. A few prospective non-randomized and non-controlled studies have been published and suggest that stand-alone vertebral body augmentation with calcium phosphate cements might become an alternative to bracing in non-osteoporotic AO type A1 up to A3.1 fractures [29, 41]. Bone resorption around the calcium phosphate cement has been reported in type A3.2 and A3.3 fractures and, for this reason, it probably should not be recommended in these types as a “stand-alone” technique. An additional posterior short segment construct could be added percutaneously to



**Fig. 5** A two-level fracture (Chance type of Th7 with also some amount of height loss and compression fracture of Th8) in a 57-year-old male patient involved in a motorcycle accident. He also sustained multiple rib fractures and a haemo-pneumothorax precluding any bracing technique. To stabilize the fractures while easing nursing care, we performed a multiple-level percutaneous pedicle screw fixation. Blood loss was minimal and the patient recovered from surgery uneventfully. Initially, we had planned to also provide a cement augmentation to the vertebral bodies, but for technical reasons it could not be done simultaneously. Once

discharged from the intensive care unit, the patient eventually declined to undergo the cementoplasty and at 3-month follow-up we did not observe further vertebral collapse on radiographs; the patient was also symptom-free. **(a)** Pre-operative CT scan reformatting showing the hyperflexion type of injury with fracture of posterior elements. **(b)** Intra-operative picture of percutaneous pedicle screw and rod insertion. The fracture was stabilized in situ. No additional fusion was necessary as the lesion was predominantly osseous. **(c)** Post-operative lateral X-ray at 3 months. The patient was symptom-free

increase stability and avoid possible complications from cement resorption [46]. Other cement compositions are being currently tested, for instance, calcium phosphate cements with various amounts of poly-methyl-methacrylate (PMMA) as well as ceramic cements. However, at present, there are not sufficiently clear data in the literature to recommend cement augmentation as a routine procedure for non-osteoporotic thoraco-lumbar fractures.

## Conclusions

Spine injuries in polytrauma patients with cord injury do not usually pose decisional problems for their management strategy. Priority should be given to cardiopulmonary resuscitation with adequate oxygenation and mechanical protection of the cervical spine to avoid additional injury. The treatment decision, non-surgical vs. surgical, is made after vital functions have been stabilized. There is still some controversy as to the optimal timing of surgery, but it is generally accepted that emergency surgical decompression and stabilization should be performed within 6–24 h after injury. Additional specific therapy, in particular the use of steroids, is not to be recommended given the current status of scientific evidence.

Non-surgical (conservative) treatment remains the treatment of choice for the majority of thoraco-lumbar fractures without neurologic deficit and can be applied even in some cases of incomplete and complete neurologic injuries. Surgical treatment of thoraco-lumbar fractures without neurologic deficit remains a controversial issue and care should be taken not to overtreat patients, an upcoming trend to be faced given the recent advent of less invasive and less time-consuming surgical procedures. Precise data are still lacking in the literature, but it has to be recalled that surgical treatment still induces today probably more pain and higher direct medical costs than non-surgical treatment [44, 51]. In non-osteoporotic spine fractures, cement augmentation techniques seems to be a promising alternative to bracing or as an additional technique to a posterior stabilization.

## References

1. Aebi M, Arlet V et al (2007) AOSpine manual. Thieme, New York
2. Anderson S, Biros MH et al (1996) Delayed diagnosis of thoracolumbar fractures in multiple-trauma patients. *Acad Emerg Med* 3(9):832–839
3. Arlet V, Orndorff DG et al (2009) Reverse and pseudoreverse cortical sign in thoracolumbar burst fracture: radiologic description and distinction—a propos of three cases. *Eur Spine J* 18(2):282–287
4. Bernhard M, Gries A et al (2005) Spinal cord injury (SCI)—prehospital management. *Resuscitation* 66(2):127–139
5. Böhler L (1929) Die Technik der Knochenbruchbehandlung Wien, Maudrich
6. Bracken MB, Shepard MJ et al (1998) Methylprednisolone or tirilazad mesylate administration after acute spinal cord injury: 1-year follow up. Results of the third National Acute Spinal Cord Injury randomized controlled trial. *J Neurosurg* 89(5):699–706
7. Burkus JK, Denis F (1994) Hyperextension injuries of the thoracic spine in diffuse idiopathic skeletal hyperostosis. Report of four cases. *J Bone Joint Surg Am* 76(2): 237–243
8. Capen DA (1999) Classification of thoracolumbar fractures and posterior instrumentation for treatment of thoracolumbar fractures. *Instr Course Lect* 48:437–441
9. Chance GQ (1948) Note on a type of flexion fracture of the spine. *Br J Radiol* 21(249):452
10. Court-Brown CM, Caesar B (2006) Epidemiology of adult fractures: a review. *Injury* 37(8):691–697
11. Dai LY, Ding WG et al (2009) Assessment of ligamentous injury in patients with thoracolumbar burst fractures using MRI. *J Trauma* 66(6):1610–1615
12. Dai LY, Jiang LS et al (2008) Conservative treatment of thoracolumbar burst fractures: a long-term follow-up results with special reference to the load sharing classification. *Spine (Phila Pa 1976)* 33(23):2536–2544
13. Denis F (1983) The three column spine and its significance in the classification of acute thoracolumbar spinal injuries. *Spine (Phila Pa 1976)* 8(8):817–831
14. Denis F, Burkus JK (1992) Shear fracture-dislocations of the thoracic and lumbar spine associated with forceful hyperextension (lumberjack paraplegia). *Spine (Phila Pa 1976)* 17(2):156–161
15. Dick W, Kluger P et al (1985) A new device for internal fixation of thoracolumbar and lumbar spine fractures: the ‘fixateur interne’. *Paraplegia* 23(4):225–232
16. Driscoll P, Wardrope J (2005) ATLS: past, present, and future. *Emerg Med J* 22(1):2–3
17. Emery SE, Pathria MN et al (1989) Magnetic resonance imaging of posttraumatic spinal ligament injury. *J Spinal Disord* 2(4):229–233
18. Fehlings MG (2001) Summary statement: the use of methylprednisolone in acute spinal cord injury. *Spine* 26(24 Suppl):S55
19. Fehlings MG, Perrin RG (2006) The timing of surgical intervention in the treatment of spinal cord injury: a systematic review of recent clinical evidence. *Spine (Phila Pa 1976)* 31(11 suppl):S28–S35; discussion S36
20. Fisher CG, Noonan VK et al (2006) Changing face of spine trauma care in North America. *Spine (Phila Pa 1976)* 31(11 suppl):S2–S8; discussion S36
21. Harris MB, Sethi RK (2006) The initial assessment and management of the multiple-trauma patient with an associated

- spine injury. *Spine (Phila Pa 1976)* 31(11 suppl):S9–S15; discussion S36
22. Holdsworth F (1970) Fractures, dislocations, and fracture-dislocations of the spine. *J Bone Joint Surg Am* 52(8): 1534–1551
  23. Kelly RP, Whitesides TE Jr (1968) Treatment of lumbodorsal fracture-dislocations. *Ann Surg* 167(5):705–717
  24. Koller H, Acosta F et al (2008) Long-term investigation of nonsurgical treatment for thoracolumbar and lumbar burst fractures: an outcome analysis in sight of spinopelvic balance. *Eur Spine J* 17(8):1073–1095
  25. Lee HM, Kim HS et al (2000) Reliability of magnetic resonance imaging in detecting posterior ligament complex injury in thoracolumbar spinal fractures. *Spine* 25(16): 2079–2084
  26. Lee JY, Vaccaro AR et al (2007) Assessment of injury to the thoracolumbar posterior ligamentous complex in the setting of normal-appearing plain radiography. *Spine J* 7(4): 422–427
  27. Lindsey RW, Dick W (1991) The fixateur interne in the reduction and stabilization of thoracolumbar spine fractures in patients with neurologic deficit. *Spine (Phila Pa 1976)* 16(3 suppl):S140–S145
  28. Liu S, Li H et al (2009) Monosegmental transpedicular fixation for selected patients with thoracolumbar burst fractures. *J Spinal Disord Tech* 22(1):38–44
  29. Maestretti G, Cremer C et al (2007) Prospective study of standalone balloon kyphoplasty with calcium phosphate cement augmentation in traumatic fractures. *Eur Spine J* 16(5):601–610
  30. Magerl F, Aebi M et al (1994) A comprehensive classification of thoracic and lumbar injuries. *Eur Spine J* 3(4):184–201
  31. McCormack T, Karaikevic E et al (1994) The load sharing classification of spine fractures. *Spine* 19(15):1741–1744
  32. Meves R, Avanzi O (2006) Correlation among canal compromise, neurologic deficit, and injury severity in thoracolumbar burst fractures. *Spine (Phila Pa 1976)* 31(18): 2137–2141
  33. Miller SM (2008) Methylprednisolone in acute spinal cord injury: a tarnished standard. *J Neurosurg Anesthesiol* 20(2): 140–142
  34. Mohanty SP, Venkatram N (2002) Does neurological recovery in thoracolumbar and lumbar burst fractures depend on the extent of canal compromise? *Spinal Cord* 40(6): 295–299
  35. Parker JW, Lane JR et al (2000) Successful short-segment instrumentation and fusion for thoracolumbar spine fractures: a consecutive 41/2-year series. *Spine (Phila Pa 1976)* 25(9): 1157–1170
  36. Patel AA, Dailey A et al (2009) Thoracolumbar spine trauma classification: the Thoracolumbar Injury Classification and Severity Score system and case examples. *J Neurosurg Spine* 10(3):201–206
  37. Patel AA, Vaccaro AR et al (2007) The adoption of a new classification system: time-dependent variation in interobserver reliability of the thoracolumbar injury severity score classification system. *Spine (Phila Pa 1976)* 32(3): E105–E110
  38. Petersilge CA, Pathria MN et al (1995) Thoracolumbar burst fractures: evaluation with MR imaging. *Radiology* 194(1):49–54
  39. Rechten GR (1999) Nonsurgical treatment of thoracic and lumbar fractures. *Instr Course Lect* 48:413–416
  40. Rechten GR II (2006) Nonoperative management and treatment of spinal injuries. *Spine (Phila Pa 1976)* 31(11 suppl): S22–S27; discussion S36
  41. Schmelzer-Schmied N, Cartens C et al (2009) Comparison of kyphoplasty with use of a calcium phosphate cement and non-operative therapy in patients with traumatic non-osteoporotic vertebral fractures. *Eur Spine J* 18(5):624–629
  42. Siebenga J, Lefrink VJ et al (2006) Treatment of traumatic thoracolumbar spine fractures: a multicenter prospective randomized study of operative versus nonsurgical treatment. *Spine (Phila Pa 1976)* 31(25):2881–2890
  43. Vaccaro AR, Lehman RA Jr et al (2005) A new classification of thoracolumbar injuries: the importance of injury morphology, the integrity of the posterior ligamentous complex, and neurologic status. *Spine* 30(20):2325–2333
  44. van der Roer N, de Bruyne MC et al (2005) Direct medical costs of traumatic thoracolumbar spine fractures. *Acta Orthop* 76(5):662–666
  45. Verheyden AP, Hoelzl A et al (2004) The endoscopically assisted simultaneous posteroanterior reconstruction of the thoracolumbar spine in prone position. *Spine J* 4(5): 540–549
  46. Verlaan JJ, Dhert WJ et al (2005) Balloon vertebroplasty in combination with pedicle screw instrumentation: a novel technique to treat thoracic and lumbar burst fractures. *Spine (Phila Pa 1976)* 30(3):E73–E79
  47. Voormolen MH, Mali WP et al (2007) Percutaneous vertebroplasty compared with optimal pain medication treatment: short-term clinical outcome of patients with subacute or chronic painful osteoporotic vertebral compression fractures. The VERTOS study. *AJNR Am J Neuroradiol* 28(3):555–560
  48. Wannamaker GT (1954) Spinal cord injuries; a review of the early treatment in 300 consecutive cases during the Korean Conflict. *J Neurosurg* 11(6):517–524
  49. Watson-Jones R (1938) The results of postural reduction of fractures of the spine. *J Bone Joint Surg Am* 20(3):567–586
  50. Wessberg P, Wang Y et al (2001) The effect of surgery and remodelling on spinal canal measurements after thoracolumbar burst fractures. *Eur Spine J* 10(1):55–63
  51. Wood K, Buttermann G et al (2003) Operative compared with nonoperative treatment of a thoracolumbar burst fracture without neurological deficit. A prospective, randomized study. *J Bone Joint Surg Am* 85-A(5):773–781
  52. Wood KB, Khanna G et al (2005) Assessment of two thoracolumbar fracture classification systems as used by multiple surgeons. *J Bone Joint Surg Am* 87(7):1423–1429

# The Rheumatoid Cervical Spine

Zdenek Klezl, Girish N. Swamy, and Jan Stulik

---

## Introduction

Rheumatoid arthritis (RA) is a progressive, debilitating disease with serious physical, psychological and economic consequences [24, 36]. RA afflicts about 1% of world population, more than 2.9 million Europeans and over two million patients in the United States. The clinical course of RA fluctuates and prognosis is unpredictable [13, 14]. Seventy percent of patients with recent onset of RA show evidence of radiographic changes within 3 years [35]. Fifty percent of RA patients are work-disabled within 10 years of disease onset [1, 30, 37].

Cervical spine involvement is common in RA (up to 90%) with neurological involvement occurring in 7–13% of patients. Neurological deterioration can be irreversible and presence of myelopathy is an indicator of poor prognosis. Up to 10% of patients with RA die of unrecognised spinal cord or brain stem compression. Atlanto-axial instability occurs in up to 40% of patients.

The challenge to the physician is that pain cannot be equated to instability and instability to neurological symptoms [9]. The goals of treatment are to identify patients at risk, to avoid irreversible neurological deficit and alleviate pain.

---

## Pathophysiology

Rheumatoid arthritis of the cervical spine follows the same pathophysiology as that of the peripheral joints; i.e. inflammation of the synovial membrane. Pannus (Fig. 1a, b), overgrowth

of hyaline cartilage and peri-articular inflammation resulting in bony erosion [9, 19] and synovial cysts causing joint laxity and subluxation affecting the atlanto-axial joint, facet joints, uncovertebral joints, retrodental bursa, the transverse ligament of the atlas, the anterior and posterior atlanto-axial ligaments, interspinous ligament and the intervertebral disc if invaded by the synovium.

Conlon et al. [8] demonstrated that 50% of patients with cervical spine involvement had radiological signs of instability. Atlanto-axial subluxation represents two-thirds of rheumatoid cervical subluxations and the majority (65%) are anterior, 20% are lateral and 10% posterior [3, 9, 21]. The rheumatoid involvement of the cervical spine can be divided into three phases. In the early stage of the disease there is an isolated atlanto-axial subluxation [9]. With further progression, destruction of the atlanto-axial joints can lead to vertical instability also referred to as basilar invagination or cranial settling. The involvement of middle and lower cervical spine can cause a sub-axial instability. Sub-axial subluxations are found in 20–25% of patients. Basilar invagination with or without atlanto-axial subluxation occurs in approximately 20% of patients. Neurological deficit varies from 11 to 58% [7, 11, 33], which is due to the difficulty in detecting subtle loss of strength from spinal cord compression in the presence of weakness and disuse atrophy arising from painful peripheral joints (Figs. 2 and 3).

---

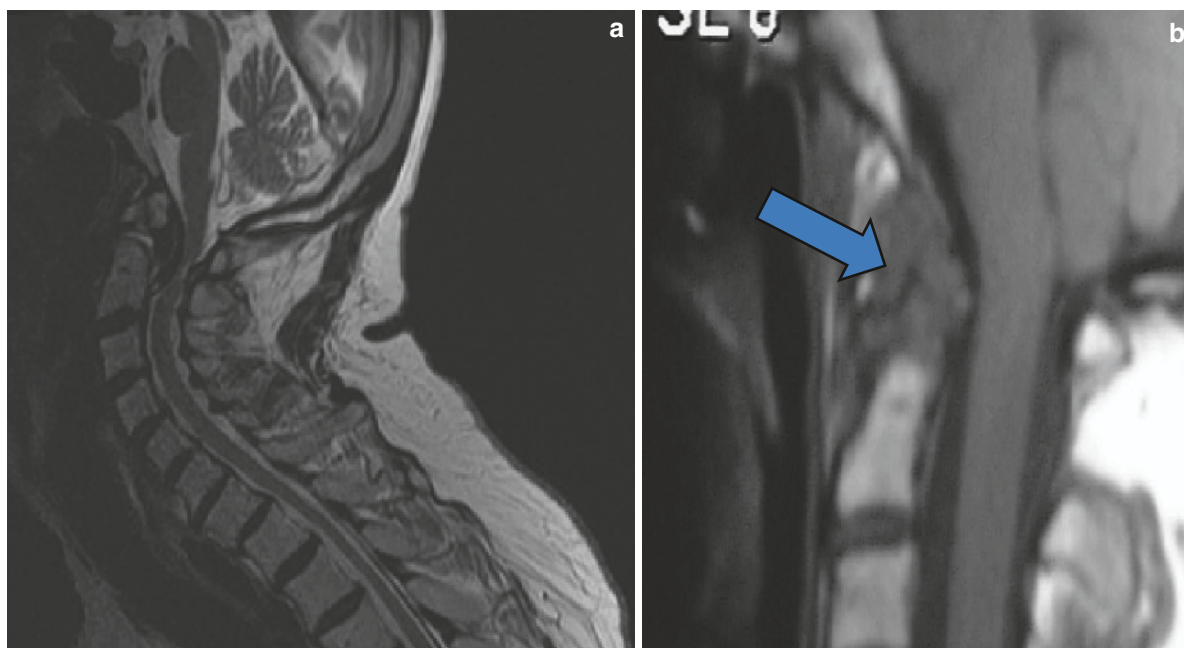
## Natural History

The knowledge of the natural history of the rheumatoid cervical spine is limited by the difficulty in performing long-term clinical follow-up studies.

Rana and co-workers [31] reported on a prospective series of 41 cases of atlanto-axial subluxation followed for a minimum of 10 years or until death. Radiologically, 61% remained unchanged, 27% showed progression of the atlanto-axial subluxation and 12% showed decrease in the amount of

---

Z. Klezl (✉)  
Department of Trauma and Orthopaedics,  
Derby Hospitals NHS Foundation Trust,  
Uttoxeter Road, Derby DE22 3NE, UK  
e-mail: zklezl@aospine.org



**Fig. 1** Sagittal sections of the MRI showing significant pannus formation (**b**-arrow), causing cord compression and myelopathic changes (**a**)



**Fig. 2** Bony deformities and soft tissue atrophic changes in the upper limbs pose a challenge for neurological assessment

atlanto-axial subluxation, including one following surgery. During this 10-year period, 12 patients with atlanto-axial subluxation died, two with evidence of neurologic damage. The remaining 10 patients had died from unrelated causes. Only three patients underwent surgical stabilization. This study concluded that the atlanto-axial subluxation is compatible with life, but some patients require surgical intervention.



**Fig. 3** Bony deformities and soft tissue atrophic changes in the lower limbs pose a challenge for neurological assessment

Boden et al. [2] analyzed 73 patients for rheumatoid involvement of the cervical spine with an average follow-up of 7 years. Neurological deficit did not develop in 31 patients (Ranawat et al. (32), Class I) but paralysis developed in the remaining 42 patients (58%) and 35 (48%) had operative stabilization. Of the seven patients with neurological deficit managed conservatively (due to medical or patient reasons) all seven (100%) had an increase in the severity of the paralysis and all patients died within 4 years, with three deaths within 1 month of onset of paralysis. Five of the seven patients died of cord compression. The posterior atlanto-dental interval (space available for the cord) and the diameter of the subaxial sagittal canal measured on the cervical radiographs demonstrated statistically significant correlations with the presence and severity of paralysis. All the patients who had a Class-III neurological deficit had a posterior atlanto-dental interval or diameter of the subaxial canal that was less than 14 mm. In contrast, the anterior atlanto-dental interval, which has traditionally been reported, did not correlate with paralysis. The prognosis for neurological recovery following the operation was not affected by the duration of the paralysis but was influenced by the severity of the paralysis at the time of the operation. The most important predictor of the potential for neurological recovery after surgery was the pre-operative posterior atlanto-dental interval. In patients who had paralysis due to atlanto-axial subluxation, no recovery occurred if the posterior atlanto-dental interval was less than ten millimetres, whereas recovery of at least one neurological class always occurred when the posterior atlanto-dental interval was at least ten millimetres. If basilar invagination was superimposed, clinically important neurological recovery occurred only when the posterior atlanto-dental interval was at least thirteen millimetres. All patients who had paralysis and a posterior atlanto-dental interval or diameter of the subaxial canal of fourteen millimetres had complete motor recovery after surgery.

Weissman et al. [38, 39] found that spinal cord compression was more common; in males (25% vs. 7.5%), when  $\geq 9$  mm anteroposterior subluxation is present (24% vs. 2%), when there was associated basilar invagination (20% vs. 7.3%) and when there was associated lateral subluxation.

Omura et al. [29] in 2002 compared surgical treatment with posterior long fusion performed for the progressive, mutilating type cervical lesions of rheumatoid arthritis with patients who did not undergo surgical treatment. Out of the 17 seropositive patients with rheumatoid arthritis with mutilating-type joint involvements eleven patients underwent surgical treatments (operated group), whereas six patients did not (non-operative group). All patients in the non-operative group had worsening activities of daily living score and were either completely bedridden or dead by the

time of final follow-up. In contrast, all of the 11 operated patients either improved or maintained the activities of daily living score. Those operated because of neurologic compromise due to myelopathy improved at least one class in the activities of daily living score, and those operated because of severe occipito-cervical pain maintained the activities of daily living with relief of pain.

Matsunaga et al. [25] in 2000 compared the long-term results and advantages of surgical with non-surgical management in 40 patients with rheumatoid arthritis and myelopathy caused by irreducible atlanto-axial dislocation with or without upward migration of the odontoid process. Nineteen were treated by occipito-cervical fusion with C1 laminectomy and 21 matched patients were treated conservatively. All patients had radiological and clinical assessment with follow-up until their death. In the surgical group the atlanto-dental interval improved immediately after surgery, and this result was well maintained at the final follow-up assessment. Neural assessment with the Ranawat classification system [32] showed improvement in 68% patients and survival rate was 84% at 5 years after surgery. In the non-surgical group, atlanto-dental interval was aggravated. These patients showed no neural improvement, and aggravation was found in 76% of cases during the follow-up period. All the patients were bedridden within 3 years after the onset of myelopathy. The survival rate was 0% in the first 8 years. They concluded that occipito-cervical fusion associated with C1 laminectomy for patients with rheumatoid arthritis was helpful in decreasing nuchal pain, reducing myelopathy and improving prognosis.

---

## Clinical Presentation

The clinical presentation is variable with subtle neck and occipital pain, symptoms of cervical subluxation, neurological deficit and sudden death. The most common presentation is atlanto-axial subluxation (which may be asymptomatic) causing various vague neurologic symptoms and neck pain. Typically, patients complain of a deep ache in the upper cervical spine and occiput. Some patients complain of their "head falling off" on flexion of the neck. A sense of clumsiness or paresthesia in the hands is not uncommon. Patients may also find it hard to put their complaints into words. Weakness in the extremities, especially the intrinsic muscles of the hand may be present. L'Hermitte's phenomenon, characterised by an electric shock sensation with flexion of the neck may occur.

Symptoms of vertebro-basilar insufficiency including loss of consciousness, vertigo, dysphagia, convulsions, tinnitus, loss of equilibrium, dysarthria, nystagmus or visual



disturbances are more commonly seen with basilar invagination of the cervical spine.

The morbidity and mortality associated with cervical rheumatoid arthritis is multifactorial [9]. Atlanto-axial subluxation may result in myelopathy, syringomyelia or hydrocephalus. Myelopathy may develop from a direct compressive effect on the spinal cord or due to ischaemia. Rheumatoid involvement of the cervical facet joints can cause vertebral artery stenosis leading to ischaemia of the spinal cord if the anterior spinal branch is involved.

Neurologic assessment in patients with rheumatoid arthritis is difficult, especially because radiographic progression does not coincide with neurologic progression [6, 8, 9, 17, 26, 31]. Furthermore, the weakness and disuse atrophy from painful involvement of peripheral joints (Figs. 2 and 3) can mask findings of subtle neurologic deficits on physical examination. Even hyperreflexia can be masked by the peripheral joint disease [4, 21, 22]. The brain stem compression symptoms from basilar invagination in rheumatoid arthritis mimic those caused by vertebral artery stenosis or insufficiency and cranial nerve palsies are also seen in patients with rheumatoid arthritis [7, 12, 18, 23].

Various classification systems have been described to assess the neurological status in patients with cervical spine involvement including the Frankel's classification grading

**Table 1** Acute spinal cord injury: Frankel classification grading system

Grade A	Complete neurological injury – no motor or sensory function clinically detected below the level of the injury
Grade B	Preserved sensation only – no motor function clinically detected below the level of the injury; sensory function remains below the level of the injury but may include only partial function (sacral sparing qualifies as preserved sensation)
Grade C	Preserved motor non-functional – some motor function observed below the level of the injury, but is of no practical use to the patient
Grade D	Preserved motor function – useful motor function below the level of the injury; patient can move lower limbs and walk with or without aid, but does not have a normal gait or strength in all motor groups
Grade E	Normal motor – no clinically detected abnormality in motor or sensory function with normal sphincter function; abnormal reflexes and subjective sensory abnormalities may be present

**Table 2** Nurick's classification system for myelopathy on the basis of gait abnormalities [31]

Grade	Root signs	Cord involvement	Gait	Employment
0	Yes	No	Normal	Possible
I	Yes	Yes	Normal	Possible
II	Yes	Yes	Mild abnormality	Possible
III	Yes	Yes	Severe abnormality	Impossible
IV	Yes	Yes	Only with assistance	Impossible
V	Yes	Yes	Chair bound or bed ridden	Impossible

**Table 3** Ranawat classification of neurologic deficit [17]

Class I	Pain, no neurologic deficit
Class II	Subjective weakness, hyperreflexia, dysesthesias
Class III	Objective weakness, long tract signs
Class IIIA	Class III, ambulatory
Class IIIB	Class III, nonambulatory

system for acute spinal cord injury (Table 1), Nurick's classification system [27] for myelopathy on the basis of gait abnormalities (Table 2) and Ranawat's classification [32] of neurological deficit (Table 3).

## Radiologic Work-Up

Flexion–extension lateral radiographs (Figs. 4 and 5) are used as a screening tool to rule out atlanto-axial instability (Figs. 6 and 7) and are necessary prior to intubation in elective surgery with general anaesthesia. They are also more commonly available to general practitioners and are deemed cost effective [9]. Measuring the posterior atlanto-dental interval (SAC; Fig. 5) has been shown to be the most reliable way to predict patients at risk for neurological deterioration [2, 9, 28]. Instability is present when anterior atlanto-dental interval (ADI) difference on flexion/extension views is more than 3.5 mm. A 7-mm difference may imply disruption of the transverse ligaments and a difference of >9 mm is associated with an increase in neurologic deterioration. The posterior ADI is also found to be the best predictor for neurological recovery after surgery.



**Fig. 4** Extension lateral radiograph of the upper cervical spine

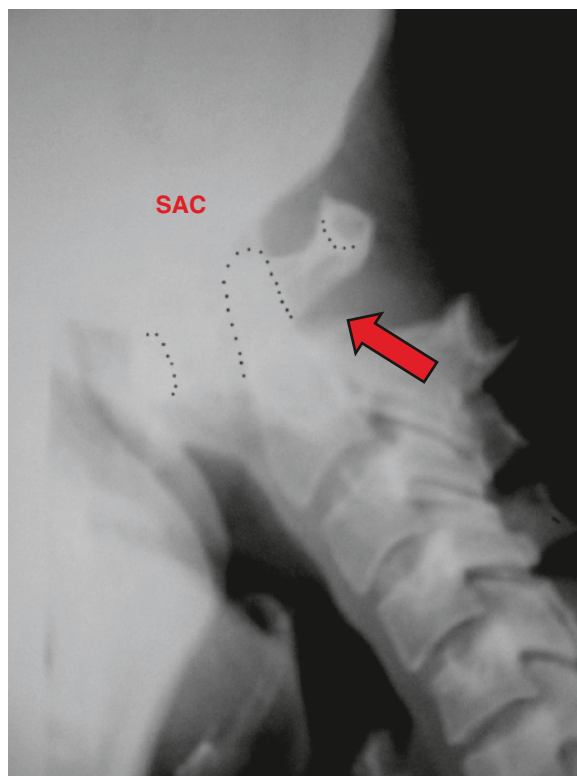
Radiographic evaluation can be carried by measuring

1. McGregor's line from hard palate to posterior occipital curve. The tip of the dens is  $>8$  mm above this line in men and  $>10$  mm in women.
2. Chamberlain's line from anterior foramen to the top of the C1 arch. If the dens is  $>6$  mm above this line, it is consistent with cranial migration.
3. Ranawat's line from the centre of C2 pedicle to the C1 arch. Normal is  $>17$  mm and  $<13$  mm is consistent with cranial migration.

CT scan with myelography can be used [20] but is deemed too invasive and not readily available.

Magnetic resonance imaging (MRI) allows for visualisation of not only the bony changes but also the pannus and the spinal cord.

Dvorak et al. [10] found that in RA patients if the spinal cord diameter is less than 6 mm in flexion, they are at risk of neurologic deficit. Also if the space available for the spinal cord is less than 13 mm, compression of the cord was evident. Pannus overgrowth in patients with atlanto-axial



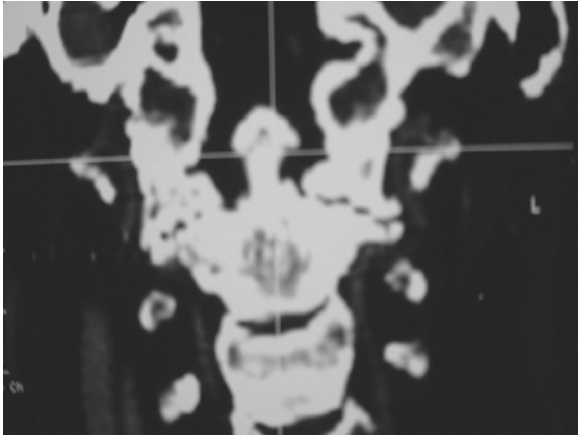
**Fig. 5** Flexion lateral radiograph of upper cervical spine of the same patient demonstrating C1-C2 instability and decreased space available for the cord (SAC) pointed to by arrow



**Fig. 6** Peg view radiograph, arrow pointing at asymmetrical destruction of the left atlanto-axial joint leading to lateral subluxation

instability often exceeds 3 mm which is not visualised on radiographs and approximately 4 mm of anteroposterior distance is needed to encase the 9 mm cord for 13 mm of

canal diameter to avoid compression at C2. MRI can also be used to measure the cervico-medullary angle (Fig. 8). The normal angle is 135–175° and Bundschuh et al. [5] found a strong correlation between cervico-medullary



**Fig. 7** Peg view CT scan demonstrating advanced atlanto-axial joint destruction leading to vertical migration of the dens



**Fig. 8** T1 weighted saggital MRI demonstrating the cervico-medullary angle

angles of less than 135° and myelopathy and paralysis in patients with rheumatoid arthritis. Lateral subluxation is also a well-recognised risk factor which can be diagnosed using the MRI or CT scans.

### Treatment of Cervical Rheumatoid Arthritis

The goals of treatment in patients with RA with cervical spine involvement are to prevent irreversible neurological deficit, to avoid sudden death attributable to unrecognised cord compression and also to alleviate intractable pain.

#### Conservative Treatment

This is reserved for patients with subtle symptoms and non-progressive instability. Regular follow-up is advocated in these patients. In indicated cases a CT-guided peridental steroid injection can be performed (Fig. 9).

#### Surgical Treatment

Indications:

1. Intractable pain.
2. Increasing neurological deficit (both somatosensory evoked potential and motor evoked potential).



**Fig. 9** Axial CT scan demonstrating the trajectory for the peridental steroid injection

3. Posterior atlanto-dental interval (SAC) less than 14 mm.
4. Cervico-medullary angle less than 135°.
5. Lateral subluxation more than 2 mm.

Pre-operative assessment should be elaborative considering the systemic nature of the illness, increased incidence of anaemia of chronic disease, increased risk of both frequency and severity of post-surgical infections (especially associated with methotrexate and TNF blockers) and also the challenges of post-operative physiotherapy and rehabilitation. Atlanto-axial instability with subtle clinical myelopathy, if detected early is best treated by atlanto-axial immobilisation (fusion). This eliminates severe pain, further subluxation and progressive instability. It also prevents progressive tissue destruction and cranial settling.

Currently there are two techniques commonly used:

1. Trans-articular screw fixation (Figs. 10 and 11) as described by Grob and Magerl [15].
2. Lateral mass fixation described by Goel [11] and modified and popularised by Harms and Melcher [16].

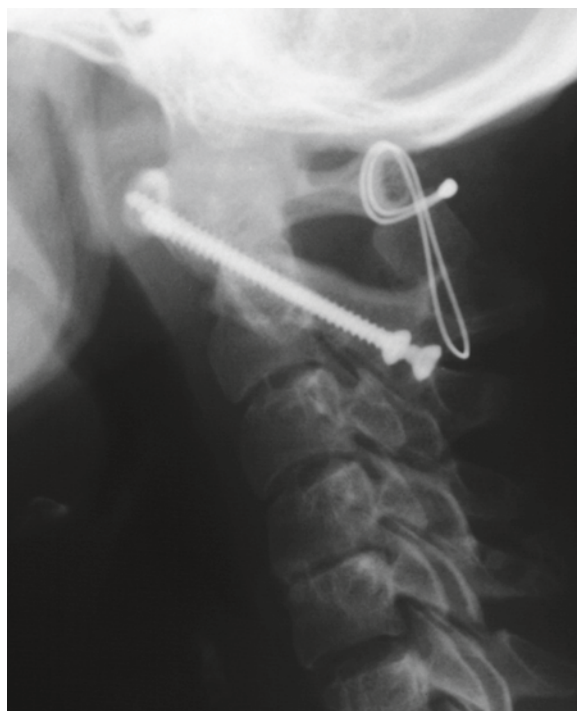
Both these techniques carry the risk of injury to the vertebral artery and require pre-operative imaging to minimise the risk. Solanki and Crockard [34] recommended intra-operative fluoroscopy to measure the vertebral groove depth (22% vertebral artery groove anomalies noted) and plotting of a safe screw trajectory to determine anatomic suitability. In the event of an injury to a vertebral artery, the procedure should be abandoned and posterior wiring (Figs. 12 and 13) technique (Gallie/Brooks-Jenkins fusion) is recommended.

In patients with significant pannus formation, the anterior trans-oral approach with pannus resection is not indicated as the pannus resolves within a few months of an atlanto-axial fusion.

The trans-oral decompression is indicated in severe cases of cranial migration of the dens into the foramen magnum.

If C1-C2 instability occurs together with cranial migration of the dens, occipito-cervical fixation is indicated. Unfortunately atlanto-axial instability with cranial settling can occur along with sub-axial instability and subluxation. In these cases occipito-cervical fixation is extended down to the upper thoracic spine (C0-T1) to avoid junctional instability (Figs. 14 and 15).

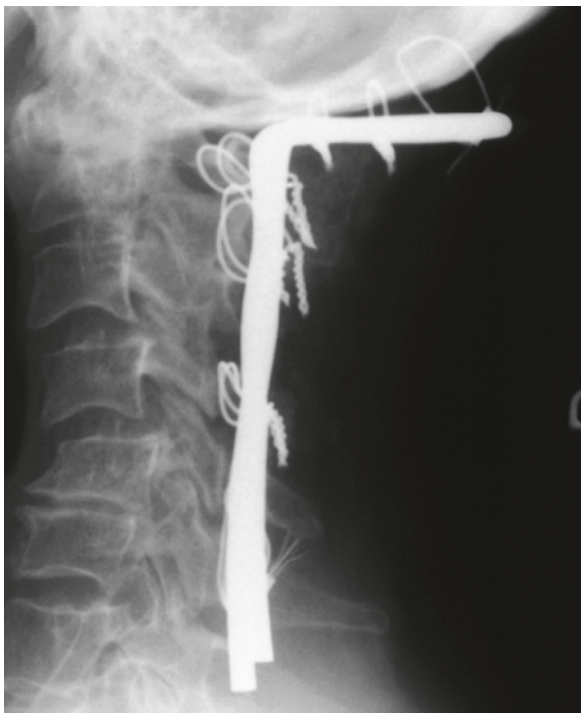
Anchoring individual screws is critical, especially in osteoporotic bones and transpedicular screws and hooks are used to extend the fixation to the thoracic spine.



**Fig. 10** Lateral radiograph of the trans-articular screw fixation



**Fig. 11** Peg view radiograph of the trans-articular screw fixation



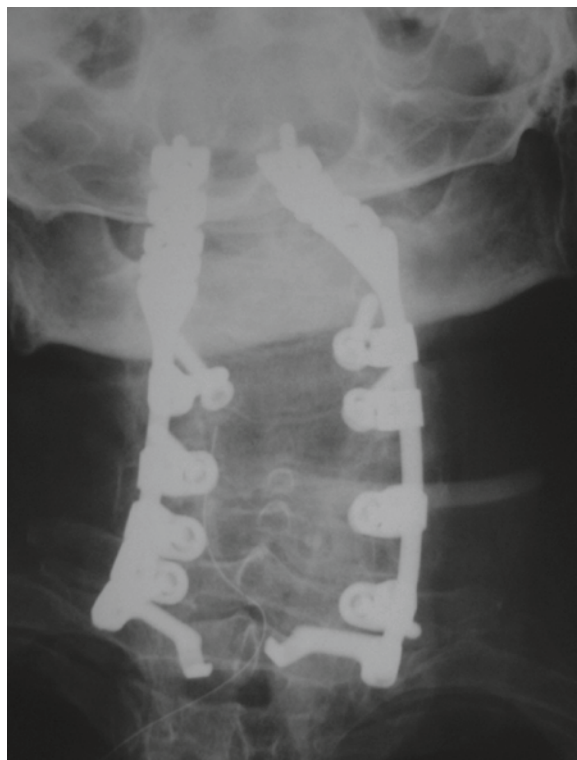
**Fig. 12** Lateral radiograph of the cervical spine demonstrating posterior wiring technique



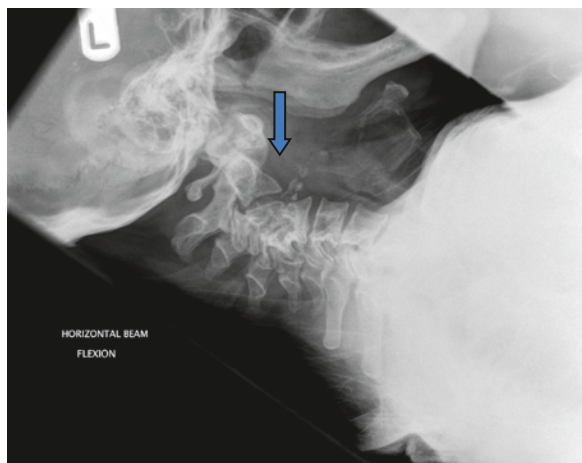
**Fig. 14** Lateral radiograph demonstrating extended fixation from C0-T1 in a patient with combined atlanto-axial and subaxial instability to avoid junctional instability



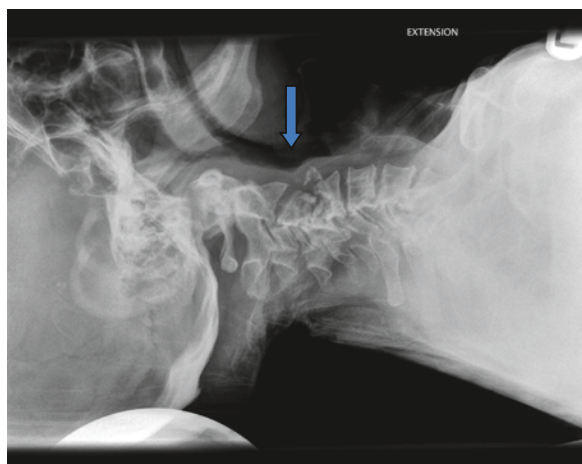
**Fig. 13** A-P radiograph of the cervical spine demonstrating posterior wiring technique



**Fig. 15** Same patient and A-P view



**Fig. 16** Lateral flexion radiograph demonstrating complex deformities. Arrow pointing to significant kyphosis, also note atlanto-axial subluxation

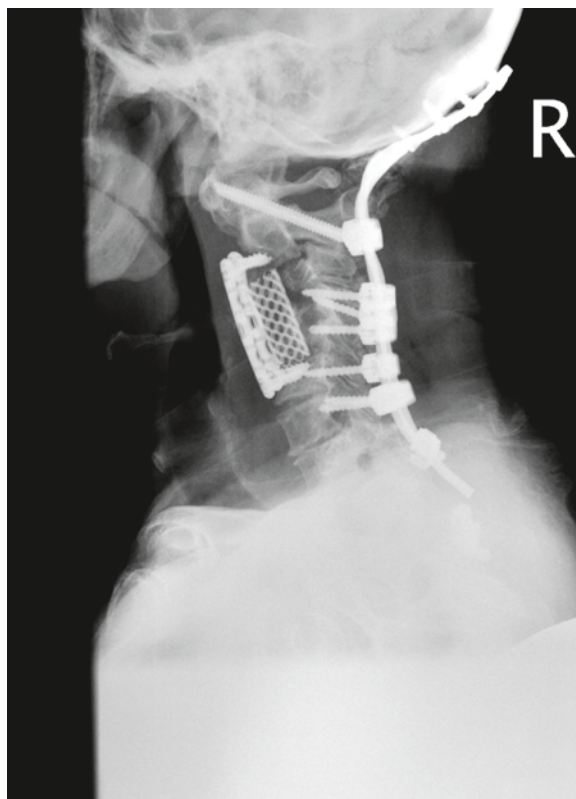


**Fig. 17** Lateral extension radiograph demonstrating reduced atlanto-axial subluxation and minor correction of kyphosis—arrow

Complex deformities (Figs. 16 and 17) require a multidisciplinary approach and a combination of both anterior and posterior stabilisation (Fig. 18). Recent developments in cervical instrumentation, especially the posterior stabilisation system, facilitates effective reduction and fixation of C1-C2 with extension both distally and proximally into the occiput.

## Conclusions

Involvement of the cervical spine by the rheumatoid process is a serious sequel, which may significantly alter patient's pain, quality of life and even survival. At present, there is an increasing tendency to address early stages of atlanto-axial



**Fig. 18** Lateral radiograph demonstrating both anterior and posterior stabilisation in patient with complex deformity, which led to neurological improvement from Ranawat IIIB to IIIA (patient is able to ambulate independently)

instability with surgical treatment to improve pain and prevent or improve neurological deficit and cranial settling.

Surgical treatment of cervical instability in RA represents one of the most challenging areas of spinal surgery because of frequently poor general condition of the patients, advanced peripheral disease, immunological compromise and significant osteoporosis.

Fortunately it seems that the disease is becoming less aggressive which may well be due to the modern disease-modifying anti-rheumatic drug therapy.

## References

1. Allaire SH, Prashker MJ, Meenan RF (1994) The costs of rheumatoid arthritis. *Pharmacoeconomics* 6(6):513–522
2. Boden SD, Dodge LD, Bohlman HH, Rehtine GR (1993) Rheumatoid arthritis of the cervical spine. A long-term analysis with predictors of paralysis and recovery. *J Bone Joint Surg Am* 75(9):1282–1297
3. Bokduk N, Major GA, Carter J (1984) Lateral subluxation of the atlas in rheumatoid arthritis: a case report and post-mortem study. *Ann Rheum Dis* 43(2):341–346

4. Brattström H, Granholm L (1976) Atlanto-axial fusion in rheumatoid arthritis. A new method of fixation with wire and bone cement. *Acta Orthop Scand* 47(6):619–628
5. Bundschuh C, Modic MT, Kearney F, Morris R, Deal C (1988) Rheumatoid arthritis of the cervical spine: surface-coil MR imaging. *AJR Am J Roentgenol* 151(1):181–187
6. Cohen C (1969) Fatal dislocation of cervical spine in rheumatoid disease. *Gerontol Clin (Basel)* 11(4):239–243
7. Conaty JP, Mongan ES (1981) Cervical fusion in rheumatoid arthritis. *J Bone Joint Surg Am* 63(8):1218–1227
8. Conlon PW, Isdale IC, Rose BS (1966) Rheumatoid arthritis of the cervical spine. An analysis of 333 cases. *Ann Rheum Dis* 25(2):120–126
9. Dreyer SJ, Boden SD (1999) Natural history of rheumatoid arthritis of the cervical spine. *Clin Orthop Relat Res* (366):98–106
10. Dvorak J, Grob D, Baumgartner H, Gschwend N et al (1989) Functional evaluation of the spinal cord by magnetic resonance imaging in patients with rheumatoid arthritis and instability of upper cervical spine. *Spine (Phila Pa 1976)* 14(10):1057–1064
11. Goel A, Laheri V (2002) Re: Harms J, Melcher P. Posterior C1-C2 fusion with polyaxial screw and rod fixation. (*Spine* 2001; 26: 2467-71). *Spine (Phila Pa 1976)* 27(14):1589–1590
12. Gow PJ, Gibbon T (1977) Dysphagia due to vertical subluxation of the axis in rheumatoid arthritis. *Rheumatol Rehabil* 16(3):155–157
13. Grassi W, De Angelis R, Cervini C (1998) Corticosteroid prescribing in rheumatoid arthritis and psoriatic arthritis. *Clin Rheumatol* 17(3):223–226
14. Grassi W, De Angelis R, Lamanna G, Cervini C (1998) The clinical features of rheumatoid arthritis. *Eur J Radiol* 27(Suppl 1):S18–S24
15. Grob D, Magerl F, McGowan DP (1990) Spinal pedicle fixation: reliability and validity of roentgenogram-based assessment and surgical factors on successful screw placement. *Spine (Phila Pa 1976)* 15(3):251
16. Harms J, Melcher RP (2001) Posterior C1-C2 fusion with polyaxial screw and rod fixation. *Spine (Phila Pa 1976)* 26(22):2467–2471
17. Hopkins JS (1967) Lower cervical rheumatoid subluxation with tetraplegia. *J Bone Joint Surg Br* 49(1):46–51
18. Jones MW, Kaufmann JC (1976) Vertebrobasilar artery insufficiency in rheumatoid atlantoaxial subluxation. *J Neurol Neurosurg Psychiatry* 39(2):122–128
19. Katz WA, Bland JH (1988) Shoulder, neck and thorax. Diagnosis and management of rheumatoid disease, 2nd edn. JB Lippincott, Philadelphia, pp 88–120
20. Kaufman RL, Glenn WV Jr (1983) Rheumatoid cervical myelopathy: evaluation by computerized tomography with multiplanar reconstruction. *J Rheumatol* 10(1):42–54
21. Lipson SJ (1984) Rheumatoid arthritis of the cervical spine. *Clin Orthop Relat Res* (182):143–149
22. Lipson SJ (1989). Rheumatoid arthritis in the cervical spine. *Clin Orthop Relat Res* (239):121–127
23. Macedo TF, Gow PJ, Heap SW, Wallis WE (1988) Bilateral hypoglossal nerve palsy due to vertical subluxation of the odontoid process in rheumatoid arthritis. *Br J Rheumatol* 27(4):317–320
24. Markenson JA (1991) Worldwide trends in the socioeconomic impact and long-term prognosis of rheumatoid arthritis. *Semin Arthritis Rheum* 21(2 suppl 1):4–12
25. Matsunaga S, Ijiri K, Koga H (2000) Results of a longer than 10-year follow-up of patients with rheumatoid arthritis treated by occipitocervical fusion. *Spine (Phila Pa 1976)* 25(14):1749–1753
26. Mayer JW, Messner RP, Kaplan RJ (1976) Brain stem compression in rheumatoid arthritis. *JAMA* 236(18):2094–2095
27. Nurick S (1972) The pathogenesis of the spinal cord disorder associated with cervical spondylosis. *Brain* 95(1):87–100
28. Oda T, Fujiwara K, Yonenobu K, Azuma B, Ochi T (1995) Natural course of cervical spine lesions in rheumatoid arthritis. *Spine (Phila Pa 1976)* 20(10):1128–1135
29. Omura K, Hukuda S, Katsura A, Saruhashi Y, Imanaka T, Imai S (2002) Evaluation of posterior long fusion versus conservative treatment for the progressive rheumatoid cervical spine. *Spine (Phila Pa 1976)* 27(12):1336–1345
30. Pincus T (1995) Long-term outcomes in rheumatoid arthritis. *Br J Rheumatol* 34(suppl 2):59–73
31. Rana NA (1989) Natural history of atlanto-axial subluxation in rheumatoid arthritis. *Spine (Phila Pa 1976)* 14(10):1054–1056
32. Ranawat CS, O’Leary P, Pellicci P, Tsairis P et al (1979) Cervical spine fusion in rheumatoid arthritis. *J Bone Joint Surg Am* 61(7):1003–1010
33. Sherk HH (1978) Atlantoaxial instability and acquired basilar invagination in rheumatoid arthritis. *Orthop Clin North Am* 9(4):1053–1063
34. Solanki GA, Crockard HA (1999) Perioperative determination of safe superior transarticular screw trajectory through the lateral mass. *Spine (Phila Pa 1976)* 24(14):1477–1482
35. van der Heijde DM, van Riel PL, van Leeuwen MA et al (1992) Prognostic factors for radiographic damage and physical disability in early rheumatoid arthritis. A prospective follow-up study of 147 patients. *Br J Rheumatol* 31(8):519–525
36. van der Heijde DM, van Riel PL, van Rijswijk MH et al (1988) Influence of prognostic features on the final outcome in rheumatoid arthritis: a review of the literature. *Semin Arthritis Rheum* 17(4):284–292
37. Weinblatt ME (1996) Rheumatoid arthritis: treat now, not later! *Ann Intern Med* 124(8):773–774
38. Weissman BN, Aliabadi P, Weinfeld MS et al (1982) Prognostic features of atlantoaxial subluxation in rheumatoid arthritis patients. *Radiology* 144(4):745–751
39. Weissman BN, Sosman JL (1975) The radiology of rheumatoid arthritis. *Orthop Clin North Am* 6(3):653–674

## Part VI

---

# Upper Limb



# Partial Rotator Cuff Ruptures

Antonio Cartucho

## Partial Rotator Cuff Rupture

Partial rotator cuff ruptures are not rare and occur mainly in the supraspinatus tendon and may extend to that of infraspinatus, but rarely to the tendon of subscapularis. Isolated lesions in the tendons of infraspinatus, teres minor or subscapularis are rare. Partial ruptures usually occur before the sixth decade of life and can be a cause of unexplained pain in the shoulder giving considerable disability. We performed a systematic literature review and pooled our experience in order to be able to justify the current concepts presented below on this difficult issue.

## Anatomy of the Supraspinatus Footprint

### Gross Anatomy

In order to classify and to grade partial rotator cuff ruptures we must be aware of the characteristics of supraspinatus insertion on the humerus. The mean antero-posterior dimension of the supraspinatus insertion is 25 mm. The mean superior to inferior thickness at the rotator interval is 11.6 mm, 12.1 at mid-tendon and 12 mm at the posterior edge. The distance from the articular cartilage margin to the bony tendon insertion is 1.5–1.9 mm, with a mean of 1.7 mm. This being said, articular partial-thickness tears with more than 7 mm of exposed bone lateral to the articular margin should be considered significant tears approximating to 50% of the tendon substance [44].

The superficial tendon fibres run longitudinally, while the deep fibres run obliquely. The supraspinatus tendon fuses

with the infraspinatus tendon approximately 15 mm proximal to their insertion on the greater tuberosity. They are not visualised as two individual tendons and cannot be separated by blunt dissection in this region [8]. In direct communication with the supraspinatus is the deep projection of the coracohumeral ligament which runs perpendicular and deep to the supraspinatus tendon but superficial to the joint capsule.

### Microscopic Anatomy

The tendons of the rotator cuff are composed primarily of water (55% of net weight) and type I Collagen (85% of dry weight). Additional constituents include other collagens (III and XII), PGs and GAGs, elastin and fibroblasts. The collagen bundles of the cuff tendons are confluent and form a hood over the humeral head [33].

Near the insertions of the supraspinatus and infraspinatus tendons into the greater tuberosity, a five-layer complex has been described that describes the density and organisation of collagen and its associated elements. Layer one is the superficial coraco-humeral ligament. Layer two represents the main portion of the tendon complex with large closely-packed fascicles. Layer three is also dense, but with smaller fascicles running in a less uniform direction. Layer four is loose connective tissue with thick collagen fibres running perpendicular to the primary fascicle orientation. This layer contains the deep coraco-umeral ligament. Layer five is the true joint capsule. It has been suggested that this intra-tendinous variation of collagen fibre density and orientation may produce shearing forces within the layers during active movement and produce intra-substance tears [18, 50].

### Blood Supply to the Rotator Cuff

The rotator cuff receives its blood supply from several different branches off the axillary artery. The rotator cuff tendons are not encased by a true synovial sheath or paratenon [4].

---

A. Cartucho  
Hospital Cuf Descobertas, R. Mário Botas,  
1998-018 Lisbon, Portugal  
e-mail: antonio.carvalho Pereira@jmellosoaude.pt

They are supplied by the above-named branches that send smaller branches through the periosteum, across the musculo-tendinous junction, via the overlying bursa. A “critical zone” has been described in the supraspinatus tendon, within 1 cm of its insertion into the greater tuberosity [38]. Arm position has been shown to affect the tenuous blood flow pattern in this region with adduction causing compression of the supraspinatus against the humeral head, squeezing the vessels in this critical region [43]. The bursal surface blood flow in the supraspinatus tendon is more robust than its corresponding articular surface flow [31].

Although less robust in some areas, this vascular pattern may be adequate to meet the metabolic needs of a healthy rotator cuff, as histological evidence of hypoperfusion has not been demonstrated [6]. Therefore, the existence of a true critical zone, and its significance relative to pathological changes occurring within the rotator cuff remains in question.

Histologic and immunohistochemical and intra-operative Doppler flowmetry analysis have shown relative hyperperfusion at the area of the critical zone [15, 24]. The hypervascularity in such cases is thought to come from proliferation in the subsynovial layer in response to injury.

---

## Local Biomechanics

Variation in fibre orientation within the cuff/capsule complex from superficial to deep affects its biomechanical properties. The bursal side of the supraspinatus tendon has been demonstrated to have a lower modulus of elasticity with a higher ultimate strain and stress, compared with the articular side of the tendon. This finding suggests that the articular portion of the supraspinatus may be more susceptible to mechanical failure in tension. Indeed, articular-sided tears have been more commonly reported [50].

The bursal layers are composed primarily of tendon bundles which may elongate with a tensile load and are resistant to rupture, whereas the joint-side layers, a complex of tendons, ligaments, and joint capsule, do not stretch and tear more easily. This suggests that intra-tendinous lamination is caused by differential shear stress within the supraspinatus tendon.

In addition, with a simulated partial-thickness tear in one portion of the tendon, the remainder of the tendon demonstrates increased strain. This reflects the supraspinatus tendon’s interconnected five-layer complex and helps to explain why partial-thickness tears may propagate into large full-thickness tears [3]. Other studies support the concept that partial thickness tears could potentially propagate

in the transverse plane, especially with >50% thickness partial tears. From biomechanical data, bursal-sided tears of over 50% thickness should warrant more concern to the surgeon as the results from these studies may imply tear propagation in the transverse plane in the antero-posterior direction [46].

Rotator cuff tears disrupt the force balance in the shoulder and the glenohumeral joint in particular resulting in compromised arm elevation torques. This dynamic instability contributes to further structural damage aggravating the initial lesion.

---

## Definition and Classification

A partial-thickness tear is considered to be a definite disruption of the fibres of the tendon and is not simply fraying, roughening or softening of the surface. The degree of tearing is described more by the depth involved in the thickness of the tendon than by the area of the tear. There are three sub-types:

1. A bursal-side tear (BT) which is confined to the bursal surface of the tendon.
2. An intra-tendinous tear (IT) which is found within the tendon.
3. A joint-side tear (JT) which is present on the side of the tendon adjacent to the joint.

Ellman [11] proposed a classification which included the site and extent of the partial tear, whether its location was adjacent to the articular or bursal surface or whether it was intra-tendinous. The grade was defined in terms of the depth as measured arthroscopically by a probe. Grade-I tears had a depth of less than 3 mm, grade II of 3–6 mm and grade III, involvement of more than half of the thickness of the tendon.

More recently Habermeyer [21] described a 2-dimensional classification of articular-sided supraspinatus tendon tears in the coronal plane as well as the sagittal plane, with regard to the origin of articular-sided partial tears at the tendon insertion. The authors described three types of rupture in the sagittal plane:

Type A tear: tear of coraco-humeral ligament continuing into the medial border of the supraspinatus tendon.

Type B tear: isolated tear within the crescent zone and

Type C tear extending from the lateral border of the pulley system over the medial border of supraspinatus tendon up to the area of crescent zone. This classification combines the classifications of Snyder [48] and Ellman that lack anatomic landmarks with reference to the localization

of the tear at the insertion of the tendon, especially at the border of the tendon insertion, at the rotator cable, or within the crescent zone.

---

## Incidence

The incidence of partial tears is difficult to access, because most lesions can only be identified at arthroscopy, and MRI may demonstrate partial tears in asymptomatic individuals [47]. Cadaver studies have consistently shown that partial-thickness are more common than full-thickness tears [54]. Among the three sub-types of partial tear, JTs are two to three times more common than BTs. Intra-substance tears are less frequent, comprising 7.9–13.6% in the series of Fukuda et al. [14]. Most of the earlier reports did not include intra-tendinous lesions. The apparent lack of the last in published series is due to the difficulty of the diagnosis [17, 27, 37, 41].

---

## Pathogenesis

Probably, rotator cuff tendinopathy is secondary to multiple factors. Combinations of intrinsic and extrinsic factors are responsible for the development of pathology in the rotator cuff. Pathological changes in tendons can lead to reduced tensile strength and a predisposition to rupture [22]. Intrinsic tendinopathy and/or enthesopathy due to changes in vascularity of the cuff or other metabolic alterations associated with ageing, may lead to degenerative tears. Extrinsic factors produce lesions to the rotator cuff through compression of the tendons by bony impingement or direct pressure.

More recently a postero-superior impingement due to repetitive interaction between the undersurface of the supraspinatus tendon and the postero-superior glenoid was found responsible for JT partial tears [50].

The injured tendon has inflammatory changes.

Oxidative stress, tissue remodelling and apoptosis are all important parts of this pathological process [24].

The loss of dynamic, fine-tuned control, due to rotator cuff pathology leads to numerous adaptative changes on a regional and broader scale. Increase of effective moment arms through connections to other tendon sub-regions tend to overload the latter [30]. On a broader scale the alterations in the shoulder muscle firing patterns namely the upper trapezius lead to an increase of scapular contribution to arm elevation [36].

The loss of normal shoulder kinematics leads to further stress not only in the injured tendon but in all rotator cuff

tendons and scapular muscles [41]. This fact may contribute to further aggravation of the structural injury, of the functional problem and of the clinical presentation.

All these inflammatory, degenerative and mechanical factors, contribute to the onset, stabilization propagation and aggravation of the partial rotator cuff rupture.

---

## Natural History

Determining the natural history of partial rotator cuff ruptures is essential to decision-making on treatment strategies. Studies of anatomic findings according to age have established that degenerative rotator cuff tears are exceedingly rare before 40 years of age and that both their prevalence and their extent increase with advancing age. Thus, partial-thickness tears usually occur in the sixth decade of life, full-thickness tears in the seventh decade, and involvement of multiple tendons in the oldest patients [1]. These data support clinical experience regarding the progression of degenerative rotator cuff pathology.

Not all partial tears are symptomatic but more than 50% of patients with partial rotator cuff tears become symptomatic over the years [54], especially in a context of a symptomatic contra-lateral tear.

Although anatomic damage fails to correlate with clinical manifestations, tear progression may be more common in patients with symptoms. Nevertheless, half the patients with symptoms experienced no progression. Pain is far more closely correlated to subacromial bursitis and long biceps tendinopathy than to tear size or site [56].

From the clinical and histological aspects, spontaneous healing of partial tears appears to be unlikely except on rare occasions. Various untoward factors are involved in the healing of the torn tendon include ageing, separation of the tear caused by muscular contraction and the weight of the arm, hypovascularity, inflammatory changes, oxidative stress, augmented apoptosis, shear stress within the tendon, and subacromial impingement. In the same way any process that impairs tissue healing, like smoking, will also contribute to cuff disease and a less effective healing response [24].

---

## Clinical Presentation

There have been few data on the characteristics of asymptomatic rotator cuff tears such as their size, location, involvement of the biceps tendon and bursal or gleno-humeral effusion. Asymptomatic tears are typically limited to the supraspinatus

tendon and are very uncommon in subjects younger than 60 years but the prevalence increases with age [37].

The physical signs and symptoms of rotator cuff disease can be separated in two categories. Firstly, the ones from mechanical impairment due to the structural damage with the adaptative response of the shoulder girdle and secondly, others resulting from inflammatory changes and involvement of the long head of the biceps.

Pain especially at night is the most disturbing symptom. There is evidence that the pain is proportional to the degree of subacromial bursitis, not to the depth or extent of the tear [19]. Impingement signs, painful arc and a positive procaïne test are the result of tendon and bursal inflammatory status. The consequences of the tendon rupture are muscle atrophy, muscle weakness, lack of dynamic control (drop arm sign), crepitus, changes in muscle activation patterns with an early activation of the upper trapezius and changes in the shoulder rhythm with elevation of the scapula in the initial two-thirds of movement [28, 36]. Differential shoulder muscle firing patterns in patients with rotator cuff pathology may play a role in the presence or absence of symptoms. Asymptomatic patients have increased firing of the subscapularis whereas symptomatic subjects continue to rely on torn rotator cuff tendons and periscapular muscle substitution resulting in compromised function. Increased scapular contribution to arm elevation, may allow function at a higher level and can be considered a positive adaptation [28]. At present it is not possible to confirm the direction of these effects in order to be able to design rehabilitation programs to optimize scapular mechanics.

The Neer and Hawkins (Fig. 1) tests have good sensitivity but low specificity for sub-acromial impingement syndrome. For diagnosing tears of the supraspinatus or infraspinatus, the Jobe sign and the “full can” test shows



**Fig. 1** Hawkin’s sign

similar performance characteristics to the Patte test and resisted external rotation with the elbow at the side flexed at 90° [2].

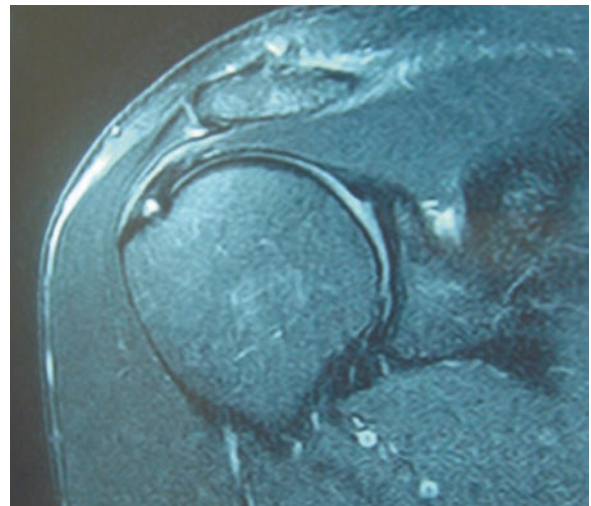
## Diagnostic Imaging

Although it is possible to use shoulder arthrography in the diagnosis of partial rotator cuff tears, MRI and ultrasonography are the most commonly-used. Arthrography of the shoulder allows evaluation of the integrity of the undersurface of the rotator cuff. However, its value in diagnosing JTs. remains uncertain with an accuracy ranging from 15 to 83%.

There has been in recent years substantial improvement of ultrasound technology, which enables higher spatial resolution and superior image quality with modern, high-frequency probes. Recent studies [52] found comparable accuracy for ultrasonography and MRI in the detection of partial tears, with MRI having slightly superior rates for sensitivity in intra-substance ruptures (Fig. 2).

MRI arthrography has been considered superior in detecting rotator cuff pathology, especially partial tears [12, 27]. However, ultrasound scan, unlike MRI, is a dynamic examination that enables the examiner to repeat and re-scan the suspected area. In addition; relationships with other tendons and the presence of secondary signs of impingement may aid correct diagnosis.

MRI should be reserved for doubtful cases and in patients with involvement of multiple anatomical structures of the gleno-humeral joint such as the capsule-labral complex.



**Fig. 2** Intra-substance rupture

## Diagnosis at Surgery

The use of arthroscopy permits a very effective inspection of the cuff. Nevertheless it is essential to correlate the arthroscopic findings with the clinical presentation in order to understand if the structural change present is responsible for the patient's complaints. For confirmation of the diagnosis a systematic inspection and palpation of the joint and bursal sides of the cuff should be performed. Joint side fraying should be debrided, the extent of the lesion measured and a suture marker passed. Then an inspection of the bursal side should be performed, with careful bursectomy and the quality of the bursal side of the tendon assessed. If an intra-tendinous lesion is suspected, thinning of the cuff and bulging and pressing the tendon with a probe on elevation or rotation of the arm can give the location of the lesion. Using the shaver can put the lesion in sight.

## Treatment Options and Indications

It is important to recognise that the choice of treatment depends on the exact cause of the lesion. Treatment of most symptomatic partial tears should be directed towards a primary diagnosis such as an impingement syndrome or instability, with treatment of the partial tear itself being considered a part of a broader problem. Nevertheless in traumatic lesions the rotator cuff lesion is the cause of the dynamic impairment and consequently of the secondary inflammatory process and the repair of the structural problem is the key.

The goal is to achieve a clinical cure. If the signs and symptoms of inflammation are alleviated, and if those due to the mechanical deficiency of the torn cuff are compensated for, by the residual cuff muscles and prime movers, the patient becomes asymptomatic and the benefits of an operation should be carefully assessed, taking into consideration the possibility of tear progression and of recurrence of symptoms based on the quality of the mechanical balance achieved by conservative treatment.

### Conservative Treatment

Patients with degenerative partial-thickness tears due to impingement are treated similarly to those with rotator cuff tendinopathy and sub-acromial bursitis. This involves time, local rest, application of cold or heat, massage, non-steroidal anti-inflammatory medication for a short period of time, modification of activities, gentle exercises for anterior and

posterior capsular stretching, and later, muscle-strengthening for the rotator cuff and the peri-scapular musculature to restore the mechanical balance [29]. Sub-acromial or intra-articular corticosteroid injections can also be used judiciously, depending on the location of the tear for those patients with persistent symptoms unresponsive to other means of pain reduction. Classically no more than two or three injections should be performed but there is no data to show that patients who do not respond to an injection and the described conservative methods, will benefit from the use of more injections.

Fukuda [15] found no evidence of healing occurring in histologic sections obtained from partial-thickness tears. Yamanaka [55] followed 40 articular-sided tears treated non-operatively during a 2-year period and found tear progression in 80% of patients. A decrease in tear size occurred in only 10%, and complete disappearance of the tear occurred in another 10%. Therefore, tear progression is the greatest concern during non-operative management

Pain and loss of active elevation have been identified as poor prognostic factors for successful conservative treatment [53]. Most BTs respond poorly to conservative treatment [23]. Once the circle of sub-acromial impingement has been established and/or the tear is deep, conservative treatment is rarely helpful. Early surgical intervention should be considered when the severe clinical manifestations and positive imaging suggest a BT diagnosis [10].

In most cases, 3 months of conservative treatment are sufficient to assess the clinical gains achievable without surgery. A rapid therapeutic response predicts better outcomes. Among the components of the clinical presentation, strength fails to improve [5, 25]. By contrast, conservative treatment consistently alleviates the pain and improves the range of motion.

### Operative Treatment

The timing of surgical intervention has to be established according to the age and activity of the patient, type of rupture (degenerative/traumatic), the presence of associated pathology and the response to conservative measures.

The surgical management of partial-thickness tears basically involves one of three options:

1. Arthroscopic debridement of the tear.
2. Debridement with acromioplasty.
3. Rotator cuff repair with or without acromioplasty. Surgery may be performed open, arthroscopically-assisted with mini-open approach, or entirely arthroscopic. Although, there is not sufficient data to support one technique over another in the management of partial-thickness tears, arthroscopy permits the articular and

bursal side cuff evaluation which represents a major advantage over an open surgical procedure especially in articular partial tears.

### Arthroscopic Assessment

Arthroscopy can be performed in a “beach chair” or lateral decubitus position depending on the training and preferences of the surgeon. Through a posterior portal an articular side inspection is performed. The quality of the cuff should be accessed, fraying should be debrided and the presence of associated lesions should be noted (Fig. 3). Very often a superior labral lesion is present. Normally a Snyder type one lesion resulting from vertical dynamic instability of the humeral head and only a debridement should be considered (Fig. 4). In other rare cases with type two or three “slap” lesions, the stability of the fragments and of the long head of the biceps should be assessed in order to decide whether to repair the lesion or perform a biceps tenodesis.

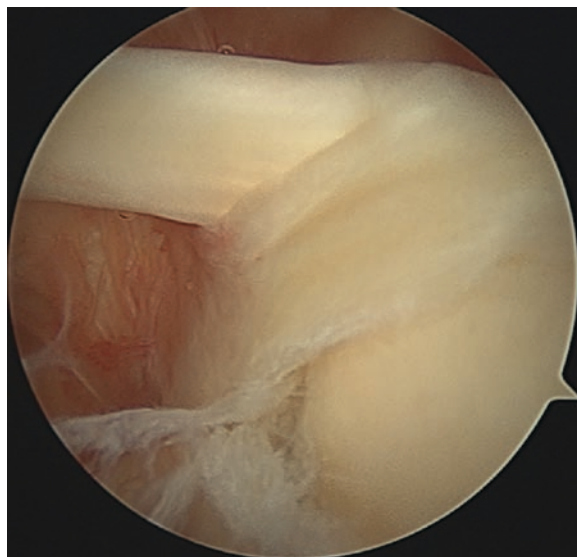
After debriding the lesion, the extent should be measured. Using a bent, preferably calibrated arthroscopic probe the amount of bone footprint uncovered should be measured and a monofilament suture marker should be passed through the tendon (Fig. 5). Care should be taken to access the integrity of the biceps posterior pole and biceps stability (Fig. 6).

Through the same posterior portal the arthroscope is directed to the subacromial space. A careful but complete bursectomy should be performed and the suture marker

identified (Fig. 7). The quality of the tendon on the bursal side should be accessed and indirect signs of impingement like fraying of the coraco-acromial arch should be noted (Fig. 8).

Palpation of the cuff tissue to assess tissue integrity and the injection of saline into the area in question can be used to diagnose intra-tendinous tears.

At this point the surgeon must decide according to his or her experience and the type of rupture, if an all arthroscopic



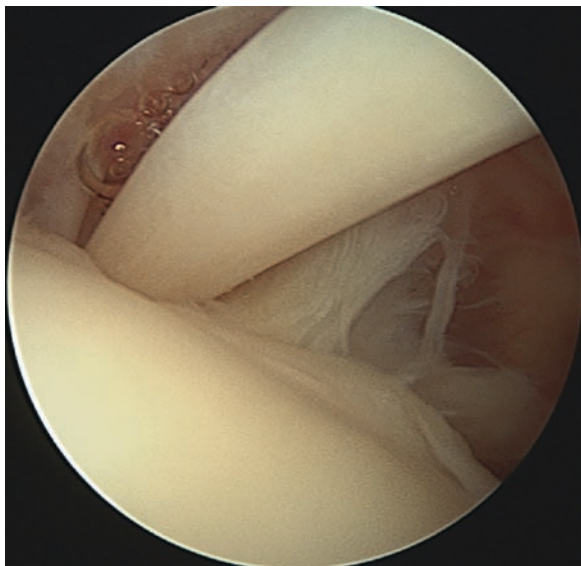
**Fig. 4** Fraying of the superior labrum



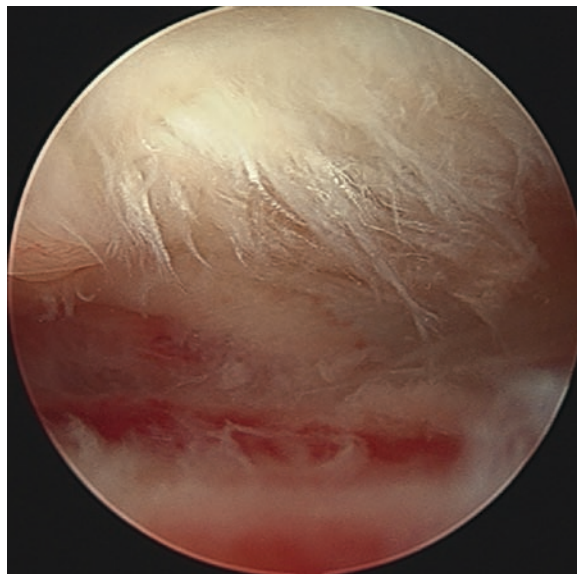
**Fig. 3** Fraying on the articular side



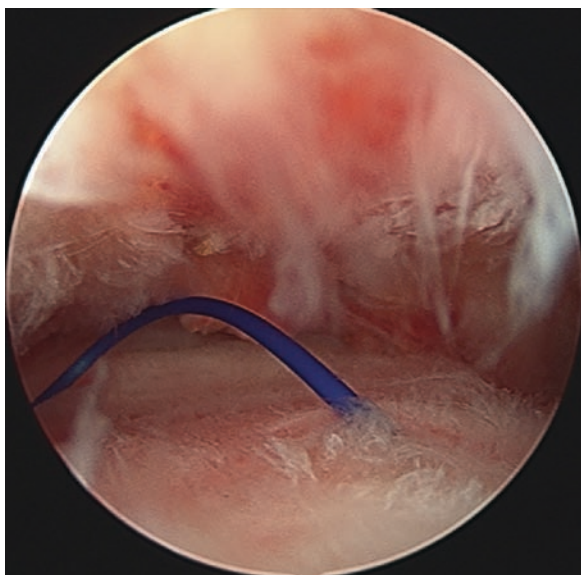
**Fig. 5** Suture marker on the articular side



**Fig. 6** Biceps stability assessment



**Fig. 8** Fraying of the coraco-acromial arch



**Fig. 7** Identification of the suture mark on the bursal side

technique, a mini-open technique or an open procedure is going to be performed.

#### Arthroscopic Debridement Alone

Budoff [7] evaluated 79 shoulders with partial-thickness cuff tears treated with arthroscopic debridement alone with a mean follow-up of 58 months using the (UCLA) Shoulder

Rating Scale. He found the results of debridement alone were good to excellent in 89% in the group of patients with less than 5 years of follow-up and decreased to 81% in those with more than 5 years.

#### Arthroscopic Debridement and Sub-Acromial Decompression

Release of the coraco-acromial ligament and debridement of the undersurface of the acromion with a high-speed burr to remove any acromial or acromioclavicular spurs (co-planning) have been recommended by some authors for the older patient with either articular-side or bursal-side tears due to external cuff impingement [45].

Snyder [49] in a retrospective study of 31 patients with partial thickness tears treated with debridement and decompression reported 84% good to excellent results. However, 13 of the 31 patients did not undergo sub-acromial decompression and no significant difference was found in the outcome, regardless of whether decompression was performed. This fact gives special importance to the mechanical imbalance produced by the injured tendon as a major prognostic factor.

Another study [10] evaluated the clinical outcome of arthroscopic acromioplasty and debridement in 162 patients with normal cuffs and impingement syndrome or partial-thickness tears of the rotator cuff. There was no difference in outcome between those with partial-thickness tears, less than 50% of tendon thickness, compared with those without any tears. However, an increased failure rate in patients

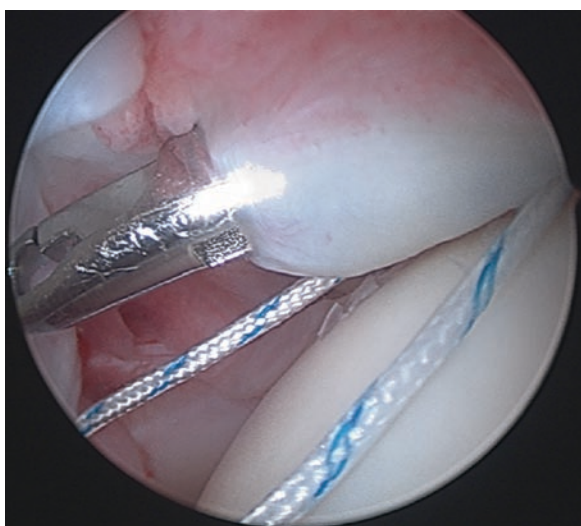
with grade 2B (bursal-sided tears) even affecting less than 50% of tendon thickness was detected.

Arthroscopic debridement should be performed in ruptures that involve less than 50% of the tendon on the articular side. The age and level of activity of the patient should be taken in to account. Bursal side, Ellman type B2 ruptures should be repaired in an early phase. Sub-acromial decompression should be performed if there is evidence of an anterior acromial or acromio-clavicular spur.

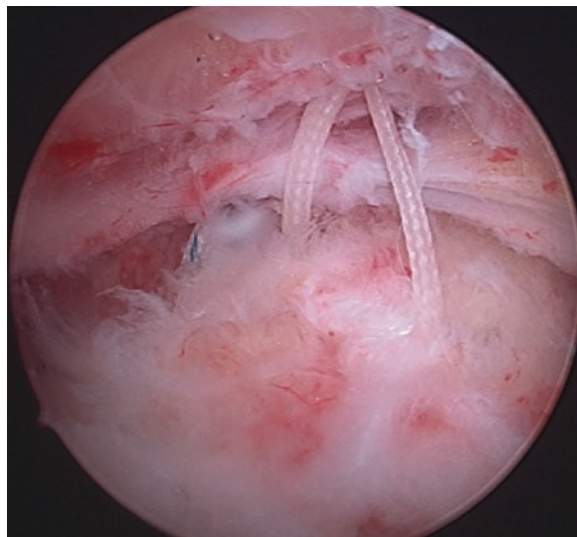
### Cuff Repair

The critical decision is to know which patients will benefit from a repair and the ones that should be treated otherwise. Once a repair has been decided upon, another decision to be made is whether to do a trans-tendon repair or to remove the remaining tissue and treat the rupture as a complete rupture. Some authors believe that the cuff material that remains in the immediate area is of poor quality which increases the possibility of post-operative pain and re-rupture [42]. Besides a 5-mm anchor should pass the remnant tissue and the correct positioning can be difficult to achieve. The procedure implies an articular vision and working through the subacromial space to pass the sutures in the cuff (Fig. 9). After this step the previously “cleaned” subacromial space is accessed in order to collect and tie the sutures (Fig. 10).

In recent work in cadavers by Lomas [32] in-situ trans-tendon repair was biomechanically superior to tear completion in articular-sided supraspinatus tears. If a completion of the rupture is decided the configuration of the fixation



**Fig. 9** Passing the sutures



**Fig. 10** Passed sutures on the bursal side

should be designed according with the extent of the rupture, the tissue quality and elasticity. If a single row technique is used, the sutures of a double-loaded anchor can be passed using a mattress or a modified Matsen-Allen stitch. If more stability and footprint coverage is necessary, a double-row or a suture bridge configuration should be considered. The former can be useful especially with poor quality tendons that won't support the outer stitch. For a double-row repair, medial anchors are placed at the medial margin of the rotator cuff footprint just lateral to the articular surface, and the lateral anchors are placed at the lateral margin of the footprint.

With bursal-side ruptures if maintenance of the articular tissue is decided a fairly external position of the suture anchor is a good solution to achieve a good position of the tendon on the footprint (Fig. 11).

If an intra-tendinous tear is identified, it should be opened on the bursal surface, while viewing from the sub-acromial space. All non-viable tissue is debrided, with care taken not to disrupt the articular surface attachment of the cuff. Through an accessory anterior working portal multiple vertical mattress No. 2 non-absorbable sutures are passed from anterior to posterior along the entire length of the tear.

The results of surgical treatment of partial thickness rotator cuff ruptures have been presented by several authors [9, 40, 42, 50, 52]. Park compared the results of arthroscopic repair of patients who had partial-thickness rotator cuff tears with those of patients who had full-thickness tears. Evaluation showed that 93% of all patients had good or excellent results, and 95% demonstrated satisfactory outcome with regard to pain reduction and functional outcome. A pre-operative





**Fig. 11** External position of the suture anchor

assessment of the acromioclavicular joint as a potential source of pain was recommended in patients with arthritic changes of this joint. Porat in a retrospective study of 51 patients with a minimum follow-up of 2 year presents 83% of excellent/good results and recommends completion to full-thickness tears with an all arthroscopic repair technique.

## Conclusions

Degenerative partial-thickness tears are an important part of pathology of the rotator cuff with an unknown incidence. This condition occurs more often in the population aged over 40 years. Traumatic ruptures occur in a younger and more active population.

Functionally they produce mechanical imbalance responsible for an impingement syndrome. Morphologically, they can be placed between subacromial bursitis/tendinitis, and the full-thickness tear.

Symptoms arise from mechanical impairment with adaptive response of the shoulder girdle and from inflammatory changes and involvement of the long head of the biceps.

The diagnosis is difficult even with MRI and ultrasonography.

With progression of the tear, clinical cure by conservative measures may be impossible to obtain. Surgical treatment with the correct indications has consistent results. The choice of the surgical treatment depends on the type of

rupture, the age and level of activity of the patient and of the degree of pain and functional impairment.

In the future, better understanding of injury mechanism, natural history and risk of tear progression, the fine-tuning of indications for operative intervention, based on prospective, randomized clinical trials and finally the use of growth factors to stimulate healing [20], as have been applied to other areas of Sports Medicine, may contribute to optimize the treatment of this condition.

## References

1. Beaudreil J, Bardin T, Orcel P (2007) Natural History or outcome with conservative treatment of degenerative rotator cuff tears. *Joint Bone Spine* 74:527–529
2. Beaudreuil J, Nizard R, Thomas T, Peyre M, Liotard JP, Boileau P, Marc T, Dromard C, Steyer E, Bardin T, Orcel P, Walch G (2009) Contribution of clinical tests to the diagnosis of rotator cuff disease: a systematic literature review. *Joint Bone Spine* 76:15–19
3. Bey MJ, Ramsey ML, Soslowsky LJ (2002) Intratendinous strain fields of the supraspinatus tendon: effect of a surgically created articular-surface rotator cuff tear. *J Shoulder Elbow Surg* 11(6):562–569
4. Blevins FT, Djurasovic M, Flatow EL et al (1997) Biology of the rotator cuff tendon. *Orthop Clin North Am* 28(1): 1–16
5. Bokor DJ, Hawkins RJ, Huckell GH, Angelo RL, Schickendantz MS (1993) Results of nonoperative management of full-thickness tears of the rotator cuff. *Clin Orthop Relat Res* 294:103–110
6. Brooks CH, Revell WJ, Heatley FW (1992) A quantitative histological study of the vascularity of the rotator cuff tendon. *J Bone Joint Surg* 74B(1):151–153
7. Budoff JE, Nirschl RP, Guidi EJ (1998) Debridement of partial-thickness tears of the rotator cuff without acromioplasty: long-term follow-up and review of the literature. *J Bone Joint Surg Am* 80:733–748
8. Clark JM, Harryman DT II (1992) Tendons ligaments, and capsule of the rotator cuff. *J Bone Joint Surg* 74A(5): 713–725
9. Conway JE (2001) Arthroscopic repair of partial-thickness rotator cuff tears and SLAP lesions in professional baseball players. *Orthop Clin North Am* 32:443–456
10. Cordasco FA, Backer M, Craig EV, Klein D, Warren RF (2002) The partial thickness rotator cuff tear: is acromioplasty without repair sufficient? *Am J Sports Med* 30: 257–260
11. Ellman H (1990) Diagnosis and treatment of incomplete rotator cuff tears. *Clin Orthop* 254:64–74
12. Ferrari FS, Governi S, Burresi F, Vigni F, Stefani P (2002) Supraspinatus tendon tears: comparison of US and MR arthrography with surgical correlation. *Eur Radiol* 12: 1211–1217

13. Fukuda H (2003) The management of partial – thickness tears of the rotator cuff. *J Bone Joint Surg* 85-B(1):2–11
14. Fukuda H, Craig EV, Yamanaka K (1987) Surgical treatment of incomplete thickness tears of rotator cuff: long-term follow-up. *Orthop Trans* 11:237–238
15. Fukuda H, Hamada K, Nakajima T, Tomonaga A (1994) Pathology and pathogenesis of the intratendinous tearing of the rotator cuff viewed from en bloc histologic sections. *Clin Orthop Relat Res* 304:60–67
16. Fukuda H, Mikasa M, Ogawa K, Yamanaka K, Hamada K (1983) The partial thickness tear of the rotator cuff. *Orthop Trans* 7:137
17. Gartsman GM, Milne JC (1995) Articular surface partial-thickness rotator cuff tears. *J Shoulder Elbow Surg* 4: 409–415
18. Gohlke F, Essigkrug B, Schmitz F (1994) The pattern of the collagen fiber bundles of the capsule of the glenohumeral joint. *J Shoulder Elbow Surg* 3:111–128
19. Gotoh M, Hamada K, Yamakawa H, Inoue A, Fukuda H (1998) Increased substance P in subacromial bursa and shoulder pain in rotator cuff diseases. *J Orthop Res* 16:618–621
20. Gulotta VL, Rodeo SA (2009) Growth factors for rotator cuff repair. *Clin Sports Med* 28:13–23
21. Habermeyer P, Krieter C, Tang K, Lichtenberg S, Magosch P (2008) A new arthroscopic classification of articular-sided supraspinatus footprint lesions: a prospective comparison with Snyder's and Ellman's classification. *J Shoulder Elbow Surg* 17:909–913
22. Hashimoto T, Nobuhara K, Hamada T (2003) Pathologic evidence of degeneration as a primary cause of rotator cuff tear. *Clin Orthop Relat Res* 415:111–120
23. Hawkins RH, Dunlop R (1995) Nonoperative treatment of rotator cuff tears. *Clin Orthop* 321:178–188
24. Yadav H, Nho S, Romeo A, John MacGillivray D (2009) Rotator cuff tears: pathology and repair. *Knee Surg Sports Traumatol Arthrosc* 17:409–421
25. Itoi E, Tabata S (1992) Conservative treatment of rotator cuff tears. *Clin Orthop Relat Res* 275:165–173
26. Itoi E, Tabata S (1992) Incomplete rotator cuff tears: results of operative treatment. *Clin Orthop* 284:128–135
27. Kassirjian A, Bencardino JT, Palmer WE (2006) MR imaging of the rotator cuff. *Radiol Clin North Am* 44:503–523, vii–viii
28. Kelly BT, Williams RJ, Cordasco FA, Backus SI, Otis JC, Weiland DE, Altchek D, Craig EV, Wickiewicz T, Warren RF (2005) Differential patterns of muscle activation in patients with symptomatic and asymptomatic rotator cuff tears. *J Shoulder Elbow Surg* 14:165–171
29. Kuhn JE (2009) Exercise in the treatment of rotator cuff impingement: a systematic review and a synthesized evidence based rehabilitation protocol. *J Shoulder Elbow Surg* 18:138–160
30. Langenderfer JE, Patthanacharoenphon C, Carpenter JE, Hughes RE (2006) Variation in external rotation moment arms among subregions of supraspinatus, infraspinatus, and teres minor muscles. *J Orthop Res* 24(8):1737–1744
31. Lohr JF, Uthoff HK (1990) The microvascular pattern of the supraspinatus tendon. *Clin Orthop* 254:35–38
32. Lomas G, Kippe MA, Brown GD, Gardner TR, Ding A, Levine WN, Ahmad CS (2008) In situ transtendon repair outperforms tear completion and repair for partial articular-sided supraspinatus tendon tears. *J Shoulder Elbow Surg* 17:722–728
33. Malcarney HL, Murrell GAC (2003) The rotator cuff – biological adaptations to its environment. *J Sports Med* 33(13): 993–1002
34. Matthew Matava J, Derek Purcell B, Jonas R (2005) Rudzki partial-thickness rotator cuff tears. *Am J Sports Med* 33: 1405–1417
35. McConville OR, Ianotti JP (1999) Partial-thickness tears of the rotator cuff: evaluation and management. *J Am Acad Orthop Surg* 7:32–43
36. Mell AG, Lascalza S, Guffey P, Ray J, Maciejewski M, Carpenter JE, Hughes RE (2005) Effects of rotator cuff pathology on shoulder. *Rhytm J Shoulder Elbow Surg* 14: 58S–64S
37. Moosmayer S, Smith J, Tariq R, Larmo A (2009) Prevalence and characteristics of asymptomatic tears of the rotator cuff. *J Bone Joint Surg (Br)* 91-B:196–200
38. Moseley HF, Goldie I (1963) The arterial pattern of the rotator cuff and the shoulder. *J Bone Joint Surg* 45B: 780–789
39. Olsewski JM, Depew AD (1994) Arthroscopic subacromial decompression and rotator cuff debridement for stage II and stage III impingement. *Arthroscopy* 10:61–68
40. Park JY, Chung KT, Yoo MJ (2004) A serial comparison of arthroscopic repairs for partial- and full-thickness rotator cuff tears. *Arthroscopy* 20:705–711
41. Perry SM, Getz CL, Soslowsky LJ (2009) Alterations in function after rotator cuff tears in an animal model. *J Shoulder Elbow Surg* 18:296–304
42. Porat S, Nottage WM, Fouse MN (2008) Repair of partial thickness rotator cuff tears: a retrospective review with minimum two-year follow-up. *J Shoulder Elbow Surg* 17:729–731
43. Rathbun JB, Macnab I (1970) The microvascular pattern of the rotator cuff. *J Bone Joint Surg* 52B:540–553
44. Ruotolo C, Fow JE, Nottage WM (2004) The supraspinatus footprint: an anatomic study of the supraspinatus insertion. *Arthroscopy* 20(3):246–249
45. Seitz WH, Froimson AI, Sordon TL (1991) A comparison of arthroscopic subacromial decompression for full thickness versus partial thickness rotator cuff tears. Paper #36, ASES Specialty Day, Anaheim, CA; March
46. Yang S, Park H, Flores S, Levin S, Makhosous M, Lin F, Koh J, Nuber G, Zhang L (2009) Biomechanical analysis of bursal-sided partial thickness rotator cuff tears. *J Shoulder Elbow Surg* 18:379–385
47. Sher JS, Uribe JW, Posada A, Murphy BJ, Zlatkin MB (1995) Abnormal findings on magnetic resonance images of asymptomatic shoulders. *J Bone Joint Surg [Am]* 77-A: 10–15
48. Snyder SJ (ed) (2003) Arthroscopic classification of rotator cuff lesions and surgical decision making. In: *Shoulder arthroscopy*, 2nd edn. Lippincott Williams & Wilkins, Philadelphia, pp 201–207

49. Snyder SJ, Pachelli AF, Del Pizzo W, Friedman MJ, Ferkel RD, Pattee G (1991) Partial thickness rotator cuff tears: results of arthroscopic treatment. *Arthroscopy* 7:1–7
50. Soslowsky LJ, Carpenter JE, Bucchieri JS (1997) Biomechanics of the rotator cuff. *Orthop Clin North Am* 28(1):17–30
51. Walch G, Boileau P, Noel E, Donell ST (1992) Impingement of the deep surface of the supraspinatus tendon on the posterosuperior glenoid rim: an arthroscopic study. *J Shoulder Elbow Surg* 1:238–245
52. Weber SC (1999) Arthroscopic debridement and acromioplasty versus mini-open repair in the treatment of significant partial-thickness rotator cuff tears. *Arthroscopy* 15:126–131
53. Vlychou M, Dailiana Z, Fotiadou A, Papanagiotou M, Fezoulidis IV, Malizos KN (2009) Symptomatic partial rotator cuff tears: diagnostic performance of ultrasound and magnetic resonance imaging with surgical correlation. *Acta Radiol* 1:101–105
54. Yamaguchi K, Tetro MA, Blam O, Evanoff BA, Teefey SA, Middleton WD (2001) Natural History of asymptomatic rotator cuff tears: a longitudinal analysis of asymptomatic tears detected sonographically. *J Shoulder Elbow Surg* 10(3):199–203
55. Yamanaka K, Fukuda H (1987) Pathological studies of the supraspinatus tendon with reference to incomplete thickness tear. In: Takagishi N (ed) *The shoulder*. Professional postgraduate services, Tokyo, pp 220–224
56. Yamanaka K, Matsumoto T (1994) The joint side tear of the rotator cuff: a follow-up study by arthrography. *Clin Orthop Relat Res* 304:68–73
57. Zeitoun-Eiss D, Brasseur JL, Goldmard JL (2005) Corrélations entre la sémiologie échographique et la douleur dans les ruptures transfixiantes de la coiffe des rotateurs. In: Blum A, Tavernier T, Brasseur JL et al (eds). *Une approche pluridisciplinaire*. Sauramps médical, Montpellier, p 287e94

# Massive Tears of the Rotator Cuff

Fernando Marco and Carlos García-Fernández

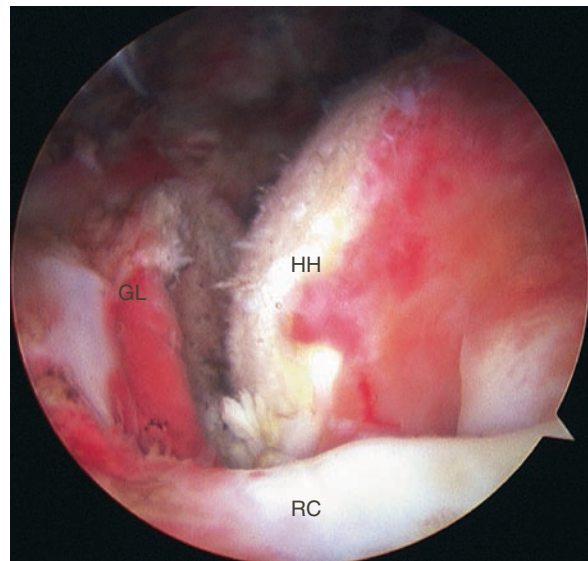
## Introduction

Even though it may seem simple to classify rotator cuff tears only by size, clinical studies tend to confirm that treatment outcomes are directly related to it. Tear size was identified as the single most important factor determining final active motion, strength, satisfaction and need for revision in a series of 105 patients who underwent open rotator cuff repair [11]. When comparing repairs performed in groups of large and massive tears with groups including small and medium sized ones there is a definite tendency to poorer outcomes in those with the bigger defects independent of the surgical technique used [22]. In this unfavourable scenario treatment protocols should be well-established. The surgeon finds himself facing a wide range of options with confusing data supporting each one. There are reports of successful non-operative management; débridement and decompression has been shown to help others; structurally-failed repairs are not unheard of with good clinical outcomes; and partial repairs can restore function [4, 27]. It is only by reviewing pertinent information and matching it with personal experience that the surgeon can make appropriate treatment decisions.

## Anatomy and Biomechanics

The primary function of the cuff is contributing to joint stability in the intermediate positions of range of motion. This is achieved by joint compression and resistance to translation both in the medio-lateral and supero-inferior planes. Massive cuff tears imply dysfunction and loss of humerus head cen-

tering by uncoupling of primary stabilizing and motion forces about the glenohumeral joint. On this principle is based the understanding of why some tears represent disability for patients while others do not, independent of the relative size of the defect. These “functional tears” have been characterized in different clinical series and quoted as a rationale for conservative surgical treatment restricted to débridement as a means of controlling pain [8, 33]. The anatomical corollary of these tears would be lesions disrupting partially the infraspinatus and/or subscapularis tendons without extension below the equator of the humeral head. In these patients the cuff would still provide a fixed fulcrum to counterbalance the eccentric force of the deltoid when it acts to move the shoulder. When the tears exceed this critical anatomical and biomechanical turning point the humeral head begins its upward migration and functional deficits appear (Fig. 1).



**Fig. 1** Arthroscopic view from a posterior standard portal of a rotator cuff massive rotator cuff (RC) tear extending below the equator of humerus head (HH) and exposing also the glenoid (GL)

F. Marco (✉)

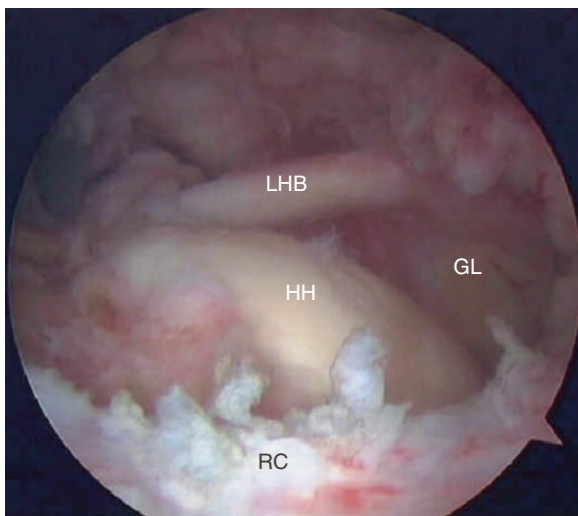
Professor of Orthopaedic Surgery, Chief Shoulder Surgery Service, Department of Orthopaedic Surgery, Clínico San Carlos Hospital, Complutense University of Madrid, Spain  
e-mail: fermarco@hotmail.com

This concept of “functional tears” also supports partial repair of massive lesions when full closure of the defect is not feasible [10] and could help explaining why partially structurally-failed repairs preserve good function [15, 25].

## Evaluation and Classification

Patients and tears should be carefully evaluated as a prerequisite for successful treatment. Sizing the defect is a good starting point taking the 5 cm mark as a minimum requirement for “massive” lesions [32]. However, more parameters need to be established. Location of the tear is one of them and two types can be frequently described: antero-superior and postero-superior [38]. The majority of lesions would be postero-superior including at least supraspinatus and infraspinatus tendons and extending or not into the teres minor tendon. The antero-superior lesions involve supraspinatus and subscapularis tendons with frequently-associated pathology of the biceps tendon and an increased risk of the feared antero-superior escape of the humeral head especially after iatrogenic damage to the coracoacromial arch (Fig. 2). It is also important to ascertain the stability of the humeral head in its static position.

Hamada et al. [20] established a classification based on radiographic changes after rotator cuff lesions (Table 1). This system considers six grades related to the superior migration of the head. The first three of them imply tears without arthropathy and the last two represent stages



**Fig. 2** Arthroscopic view from a posterior standard portal of an antero-superior massive rotator cuff (RC) tear with exposure of the humeral head (HH), glenoid (GL) and long head of biceps (LHB)

showing degenerative changes. In grades II and III the shoulder has lost the balance of forces and this is currently an expression of chronic evolution.

Muscle changes would be the last item to review regarding the cuff. There are two items which need to be reviewed: muscle atrophy and fatty infiltration. Goutallier et al. [19] established five stages of fatty muscle degeneration (Table 2) with the aid of CT scans. Today, MRI has taken the place of CT to evaluate in a reproducible manner atrophy and fatty infiltration of the cuff [14] (Fig. 3). These two changes in muscle appearance have been shown in clinical and imaging studies to correlate with poorer tendon healing, and poorer healing with worse outcomes [5, 17].

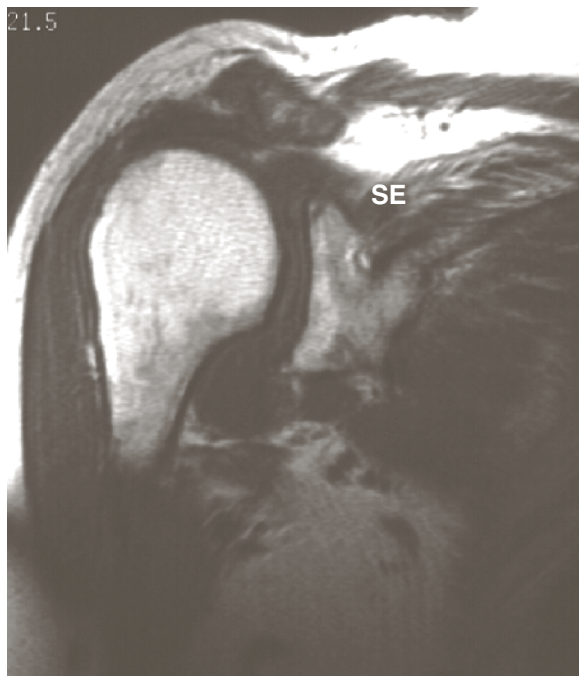
Patient-specific factors as medical co-morbidities should be taken in account in their capability to enhance shoulder dysfunction but paradoxically a recent publication reported greater improvement in shoulder scores for patients with worse pre-operative medical status [35]. It is important however to try to determine the “age” of the tear. Patients can associate significant trauma with the acute onset of pain and dysfunction in an otherwise previously normal shoulder. This modality of massive tear is more common in the younger population. Far more common are the other two

**Table 1** Hamada classification of radiographic changes in the shoulder after rotator cuff tearing

Grade	Acromiohumeral distance (mm)	Characteristics
I	>6	
II	<5	
III	<5	Acetabularization of the glenoid and undersurface of the acromion
IV	<5	Grade III changes, with narrowing of the glenohumeral joint space
V	<5	Grade IV changes, with collapse of the humeral head

**Table 2** Goutallier staging for fatty degeneration of cuff musculature by CT evaluation

Grade	Muscle appearance
0	No fatty deposits
1	Some fatty streaks
2	More muscle than fat
3	As much muscle as fat
4	Less muscle than fat



**Fig. 3** MRI T1 image of a shoulder in the coronal plane showing atrophy and fatty infiltration of the supraspinatus (SE)

clinical presentations, chronic or acute-on-chronic usually occurring in the older population. Patients with a chronic massive tear may have been compensating for the functional deficits or tolerating the lesion by balanced force couples when a minor event puts them into a symptomatic scenario. Acute-on-chronic patients have often been experiencing minor symptoms from a degenerated and torn cuff when an identifiable traumatic event produces a massive defect. It has been observed from experimental and clinical data, comparing acute vs. delayed repair, that results favour the former [4, 27].

## Treatment

Establishing a clear-cut treatment protocol for patients with massive tears involves two issues. In the first place there are many options open ranging from non-surgical management to reconstruction procedures for both partial or complete repairs. But this wide choice cannot always be related directly to individuals due to the variability in results reported [4]. We can observe certain paradoxes such as patients satisfied after an image-proved failure of their repair or good and painless function with just conservative treatment. It is true

that treatment selection needs a careful evaluation both of the patient and the lesion characteristics but a brief although systematic review of choices may help the surgeon to select the most appropriate one for their patients.

### Conservative Management

A combination of medication, physical therapy and activity modification can render a shoulder pain-free and allow for function which is good enough for the patient [1]. This clinical improvement can be sustained over a 4-year period as demonstrated by Zingg et al. [42]. It is true however that the success of this form of management seems to be related to the previous duration of symptoms. Bokor et al. [7] observed significant improvement in pain in patients with symptoms up to 3 months compared with patients whose symptoms had been present for more than 6 months. The last consideration we have to make concerning conservative treatment is when to decide to call it a failure. Oftentimes it is determined by a demand from the patient who seeks a quick recovery through surgery. We believe that, as long as the clinical situation is manageable, a 3-month period of non-surgical treatment can be allowed once it has been started.

### Isolated Débridement and Decompression

The classic paper by Rockwood et al. [33] reported 83% good to excellent results at 6.5 years follow-up in 53 shoulders with massive tears where open débridement with aggressive acromioplasty and complete release of the coracoacromial ligament had been performed. Over the years current techniques and opinion favour an arthroscopic approach for this procedure together with a more conservative surgical approach. The objective is to remove all pain sources while preserving the coracoacromial arch through a limited acromioplasty. Some surgeons exchange acromioplasty for remodelling of the greater tuberosity [13]. This tubero-plasty in combination with débridement provides also a high degree of patient satisfaction and hopefully avoids the devastating complication of antero-superior escape of the humeral head. One other issue in pain reduction is the management of the long head of the biceps. Recent clinical evidence shows that tenodesis or tenotomy can contribute significantly to pain relief without compromising the stability of the humeral head [6]. Altogether these procedures which do not imply attempts at cuff repair can obtain satisfactory results especially in old and low-demand patients. Pre-requisites should include relative

preservation of shoulder function implying balanced force couples. In fact, irreparable ruptures of the subscapularis or teres minor or both have been identified as negative prognostic factors for this treatment [16]. Probably, the main issue to take into account when deciding this treatment over repair is the durability of results. Improvement appears to diminish over time especially when compared with a group of patients who have undergone repair [30].

### Direct Cuff Repair

In view of the excellent results achieved by successful repair of the cuff defects [17], this technique should be the surgeon's preference. There are however reasons that make this objective difficult if not sometimes impossible. Recent pathology studies of torn tendons establish differences in oedema, degeneration, chondroid metaplasia and inflammatory response which reduce healing possibilities in bigger defects [28]. Of course a retracted cuff is difficult to mobilize to its original position safely. Not only will excessive tension of the repair compromise the result but there are also limits to the distance that the suprascapular nerve can be stretched without neurological damage, thus compromising the outcome [40].

There are technical tips and tricks to increase the success of the repair. Careful release of bursal and articular adhesions around the cuff is the first step. If this proves insufficient, interval slides or margin convergence can be performed. Interval slides release the supraspinatus from the subscapularis anteriorly or the supraspinatus from the infraspinatus posteriorly. These intervals are closed after cuff repair to bone [3]. Margin convergence as proposed by Burkhart [9] decreases the strain at the tendon-bone interface of the repair. The U-shaped tears are converted to crescent-shaped by side-to-side suturing beginning at the apex of the defect and progressing from medial to lateral.

Due to the inherent mechanical and biological problems which threaten the repair there are other techniques to increase the quality of it. All elements of the repair have to be taken into account: stronger sutures in combination with better traction sutures; improved suture anchors or suture plate re-inforcement; and recently a current trend favouring double-row fixation. This last technique places a second, more lateral, row of anchors with the intention both of fixation re-inforcement and also of mimicking the natural wide footprint of tendon attachment to bone. Biomechanical and clinical studies seem to support this approach [26].

Two important and related issues should be addressed at this point: tear recurrence and the discrepancy in the correlation between anatomical integrity of the repair and clinical results. An analysis of recent series which have documented re-tear rate give percentages of failure ranging from 34 to 94% [15, 17, 25]. Galatz et al. [15] followed 18 patients with arthroscopically-repaired large and massive tears. After a minimum of 1 year, 17 out of them had a recurrent tear documented by ultrasound. This fact did not preclude clinical improvement in 16 of them although these results deteriorated over time with an average ASES Shoulder Index score which decreased at 2 years follow-up from 84.6 to 79.9%. Jost et al. [25] singled out twenty patients with structural failure of their repair after 3.2 years of follow-up. This group of patients was re-examined at an average of 7.6 years and 19 of them continued to be satisfied. Their Constant scores were equivalent in both clinical exams and no progression of the re-tear size was documented by MRI. From these examples we can appreciate both the inherent difficulties of performing a durable repair but also the rewarding result of setting our surgical goals on it. Even in the groups with a recurrent tears the final configuration of the cuff after surgery can render a functional cuff for the patient.

### Partial Cuff Repair

Although it could be considered a poor objective, lessons learned from the clinical setting support it in situations where a complete repair is not possible. Not only structurally partially-failed repairs show good results as we have seen above [15, 25] but also series of patients where primarily full repair was not achieved. A partial repair was performed in a group of 24 patients and the retrospective review showed a 92% of patient satisfaction, 83% of them with good pain relief and average active elevation, improved from 114 to 154° [12]. Burkhart has led in the support of the biomechanical rationale of this procedure with his "suspension bridge" biomechanical model [10]. In this model the integrity of the anterior and posterior portions of the cuff are sufficient to balance forces across the gleno-humeral joint. With this balance recovered the resultant residual defect becomes functionally less important. Technically, mobilization of the cuff to obtain secure fixation of the anterior and posterior portions of the cuff above the equator of the humeral head is the minimum requisite. In Burkhart's cited series of partial repairs 93% of patients were subjectively satisfied and the UCLA score improved from an average of 9.8 pre-operatively to 27.6 post-operatively. Prospectively followed series with partial

repairs such as the one by Bennett [2], where only 78% of patients the humeral heads were primarily covered, confirm the benefits of attempts at surgical repair.

### **Combination of Cuff Repair and Tissue Substitution or Augmentation**

Cuff repair has not been free of the temptation to solve the problem of mobilization and fixation by bridging the defect with different synthetic or biological substitutes. Conceptually however there are theoretical limitations to these techniques. The durability of the material is one, taking into account problems of graft incorporation and wear. The host's potential undesired local reaction to the substitute is another. But third and most important is the fact that the graft leaves the patient's cuff in its retracted and non-functional situation. Even though the scientific literature historically favourable reports we can use Moore et al. [31] more recent series of 32 patients reconstructed with tendon allograft (patellar, Achilles or quadriceps) to illustrate this point. Two sets of data emerge from it. One hundred percent of the grafts evaluated by MRI arthrogram showed structural failure. Although clinical improvement was experienced by 23 out of 28 patients at 31 month follow-up, the results were comparable to that obtained from isolated débridement and subacromial decompression.

A different approach is the re-inforcement of the repair with different biomaterials. This could offer biological and mechanical advantages in combining partial bearing of loads with the repair and providing a scaffold and enhancement of the repair process. Once again these ideas are not yet proved by clinical practice. Porcine small intestinal submucosa is a well-known bio-absorbable tissue scaffold biomaterial commercially promoted on a sound scientific basis and with previous experience in other body sites. Two studies show failures when used for augmentation of repairs of the rotator cuff. The first one [34] evaluated with MRI eleven patients with augmented repairs of massive tears at 6 months follow-up. Ten out of the eleven patients showed failure of the repairs and furthermore their clinical status had not improved after surgery. The second study of this biomaterial has been carried out in a randomized prospective fashion assigning patients with massive tears to the repair with augmentation or to repair without augmentation [21]. No difference in clinical outcome was found for the two groups and at 1 year follow-up four of the 15 patients with augmentation had healed repairs while nine out of the 15 patients repair without augmentation had healed.

However and although it is not a clinical reality yet, we believe in biological stimulus to the repair site and propose

it as a line of work to be sustained exploiting every path from scaffolds, through cytokines, to cells.

### **Cuff Reconstruction with Tendon Transfers**

Tendon transfers can be used as a primary reconstruction procedure of irreparable tears or as a secondary salvage technique. It seems that these two different clinical settings yield different results for the same technique. Miniaci and MacLeod [29] recommended latissimus dorsi transfer as a revision procedure only after their series of 17 patients with this procedure yielded an 82% satisfaction rate using limited functional goals criteria. In contrast, Warner and Parsons [39] compared the same transfer when used as a primary or salvage operation and concluded that this last indication gives more limited improvement. In their group of primary procedures the average Constant post-operative score reached 70% but in the revision group only 55%.

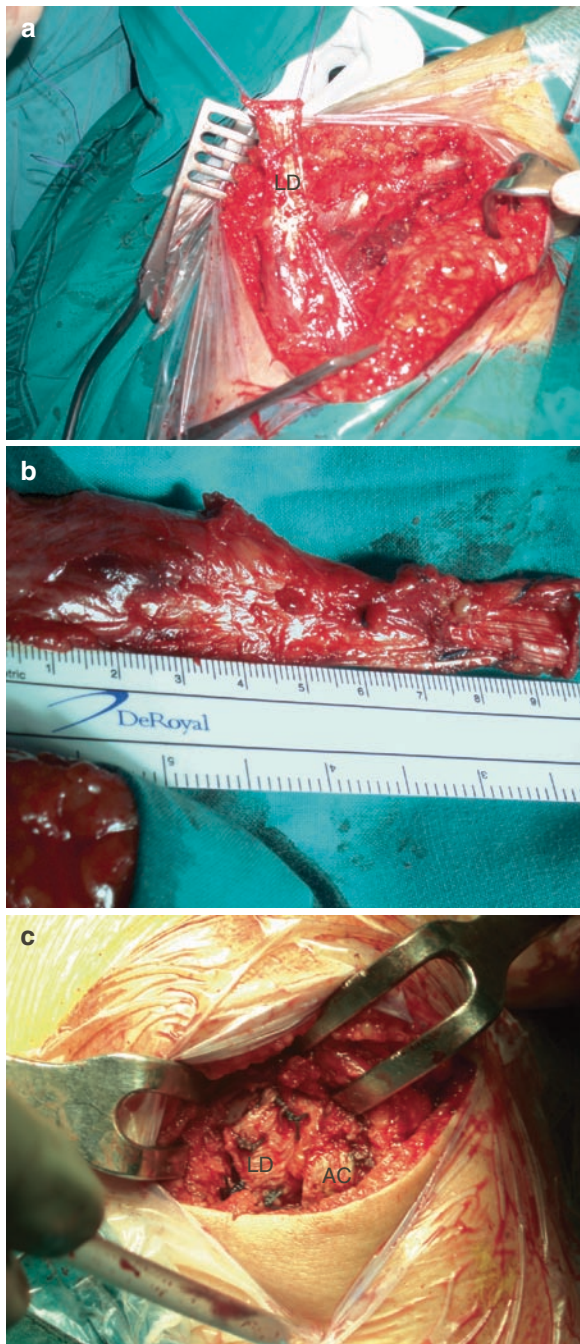
Tendon transfers are not free of complications. Some come from their anatomical relationships as in the latissimus dorsi technique. The proximity of the radial and axillary nerves to the tendon insertion places these two structures at risk with rates of neurapraxia reported as high as 5% [18]. Other complications are secondary to the reconstruction itself with late rupture of the transfer evaluated by Warner [37] reaching 20–30% of patients. They recommended the augmentation of the latissimus dorsi tendon with fascia lata autograft.

Overall, tendon transfers remain techniques to be reserved for young patients with irreparable tears and functional deficits. The results will be subjected to many variables although biomechanical reasoning can guide us in initial selection of the most suitable one for each type of cuff rupture.

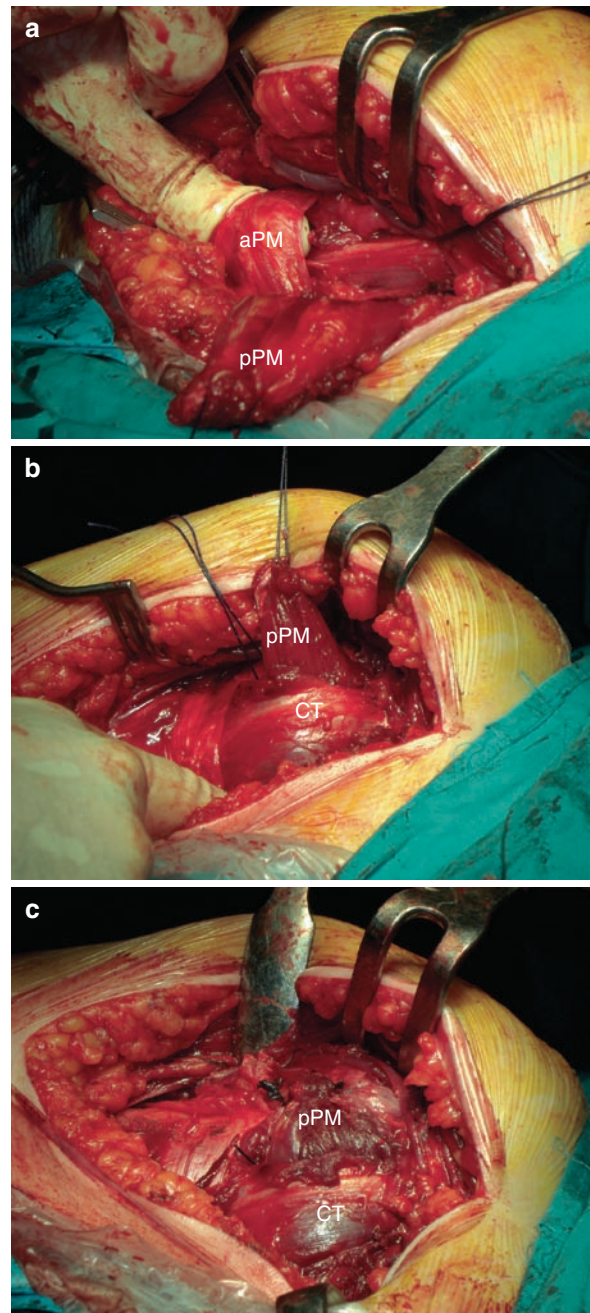
Postero-superior defects are amenable to treatment using a latissimus dorsi tendon transfer (Fig. 4). Although there are other alternatives like teres major transfer this one presents technical problems such as a small tendon and short excursion. Gerber et al. [18] have followed a series of 67 cases for an average of 53 months. Function improved with increase in average flexion from 104 to 123° and pain relief was satisfactory. Some constraints to the technique were stated, mainly those patients with associated subscapularis deficits where the transfer is incapable of restoring balanced biomechanics.

An antero-superior irreparable tear is suitable for pectoralis major transfer as probably the best option nowadays. Biomechanically the subcoracoid re-directioning of the tendon appears to be superior providing improved line of action for the tendon rather than just sliding it over the conjoined tendon (Fig. 5). We favour an anatomical split of the tendon insertion as proposed by Jennings et al. [23]. Instead



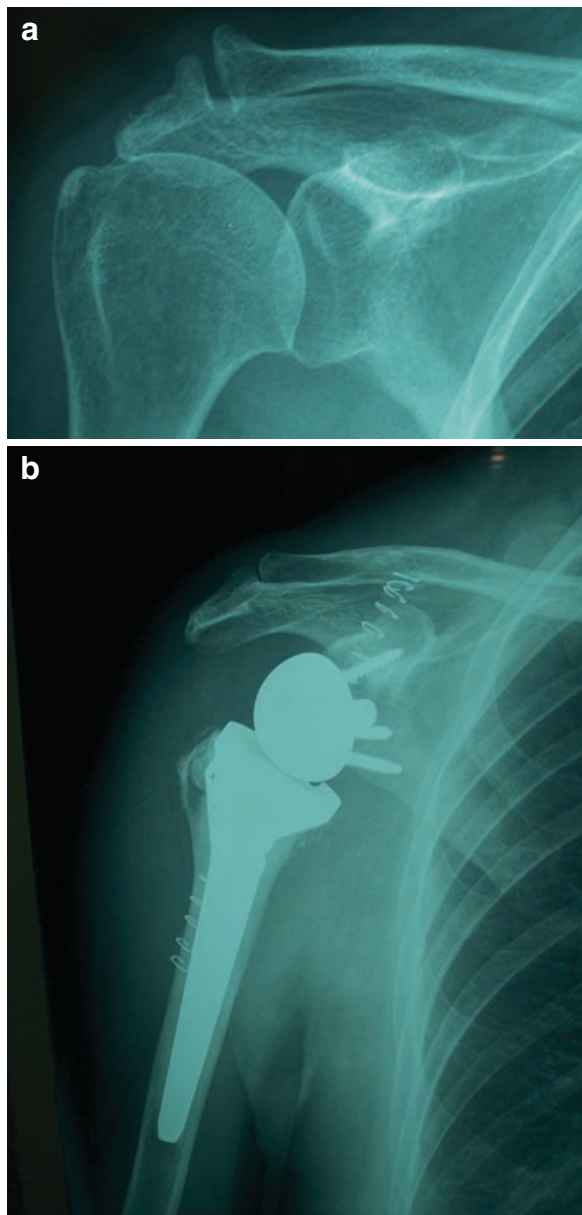


**Fig. 4** (a) Harvesting of the latissimus dorsi tendon (LD) from a dorsal approach. (b) Sizing of the latissimus dorsi tagged tendon. (c) View of the sutured latissimus dorsi (LD) transfer from a superior approach with the patient in a lateral decubitus position (AC Acromion)



**Fig. 5** (a) Harvesting of the posterior lamina of the pectoralis major tendon (pPM) with preservation of the anterior lamina (aPM) performed in a right shoulder in a lateral decubitus position. (b) Re-direction of the pectoralis major (pPM) under the conjoined tendon (CT). (c) Pectoralis Major tendon (pPM) sutured into place after its passage under the conjoined tendon (CT)

of dividing the tendon in superior and inferior portions the pectoralis major insertion is identified in its anterior and posterior laminae. The transference of the posterior lamina carrying the sternal portion of the muscle provides a better force vector to counterbalance the deltoid eccentric action.



**Fig. 6** (a) AP X-ray view of a shoulder with upward humeral head migration – grade II of Hamada’s classification. (b) Post-operative AP X-ray view of the shoulder treated with a delta III reverse arthroplasty

Jost et al. [24] have documented clinical results of this transfer with 82% of patient satisfaction. Better results can be expected if the defect is restricted only to the subscapularis although differences with combined supraspinatus-subscapularis deficiency were not statistically significant.

### Arthrodesis and Arthroplasty

These two techniques should be considered end-stage procedures but while arthrodesis indications are receding, arthroplasty is increasing. Arthrodesis could be considered in informed young patients with a very deficient shoulder which includes loss of deltoid function and whose daily requirements include physical labour.

We will discuss arthroplasty indications for a clinical picture of cuff deficiency in the absence of cuff tear arthropathy. Although this would seem to suggest that the surgeon selects the easy way out, the scientific literature keeps providing papers where results support the use of prostheses (Fig. 6). Of course we will restrict our discussion to reverse shoulder arthroplasties which are able through their unique semi-constrained design to offer a stable fulcrum for deltoid action and consequent arm elevation. Motion with this type of arthroplasty gains in elevation but lacks in external rotation if other surgical procedures such as tendon transfers are not added. Wall et al. [36] have retrospectively reviewed 186 cases with different indications for reverse prostheses. Analysis of results by etiology showed that patients older than 70 years with massive, irreparable cuff tears without arthritis were ones with better outcomes. It is true that this prostheses can produce a significant number of complications which can reach the 50% mark [41] and that their long-term survival is questioned. But surgeons are finding out through personal experience that this is a reproducible procedure that offers selected patients pain relief and functional improvement.

Personal indications include massive cuff defects in elderly patients with intractable pain or pseudoparalysis that does not improve after a period of conservative management.

### References

1. Ainsworth R (2006) Physiotherapy rehabilitation in patients with massive, irreparable rotator cuff tears. *Musculoskeletal Care* 4:140–151

2. Bennet WF (2003) Arthroscopic repair of massive rotator cuff tears: a prospective cohort with 2- to 4-year follow-up. *Arthroscopy* 19:380–390
3. Bigliani LU, Cordasco FA, Melveen SJ, Musso ES (1992) Operative repair of massive rotator cuff tear: long-term results. *J Shoulder Elbow Surg* 1:120–130
4. Boes MT, McCann PD, Dines DM (2006) Diagnosis and management of massive rotator cuff tears: the surgeon's Dilemma. *AAOS Instr Course Lect* 55:45–57
5. Boileau P, Brassart N, Watkinson DJ, Carles M, Hatzidakis AM, Krishnan SG (2005) Arthroscopic repair of full thickness tears of the supraspinatus: does the tendon really heal? *J Bone Joint Surg Am* 87:1229–1240
6. Boileau P, Basque F, Valerio L, Ahrens P, Chuinard C, Trojani C (2007) Isolated arthroscopic biceps tenotomy or tenodesis improves symptoms in patients with massive irreparable rotator cuff tears. *J Bone Joint Surg Am* 89:747–757
7. Bokor DJ, Hawkins RJ, Huckell GH, Angelo RL, Schickendanz MS (1993) Results of nonoperative management of full-thickness tears of the rotator cuff. *Clin Orthop Rel Res* 294:103–110
8. Burkhart SS (1993) Arthroscopic debridement and decompression for selected rotator cuff tears: clinical results, pathomechanics and patient selection based on biomechanical parameters. *Orthop Clin North Am* 24:111–123
9. Burkhart SS (2001) Arthroscopic treatment of massive rotator cuff tears. *Clin Orthop Relat Res* 390:107–118
10. Burkhart SS, Nottage WM, Ogilvie-Harris DJ, Kohn HS, Pachelli A (1994) Partial repair of irreparable rotator cuff tears. *Arthroscopy* 10:363–370
11. Cofield RH, Parvizi J, Hoffmeyer PJ, Lanzer WL, Ilstrup DM, Rowland CM (2001) Surgical repair of chronic rotator cuff tears. *J Bone Joint Surg Am* 83:71–77
12. Duralde XA, Bair B (2005) Massive rotator cuff tears: the result of partial rotator cuff repair. *J Shoulder Elbow Surg* 14:121–127
13. Fenlin JM Jr, Chase JM, Rushton SA, Friedman BG (2002) Tuberooplasty: creation of an acromiohumeral articulation – a treatment option for massive, irreparable rotator cuff tears. *J Shoulder Elbow Surg* 11:136–142
14. Fuchs B, Weishaupt D, Zanetti M, Hodler J, Gerber C (1999) Fatty degeneration of the muscle of the rotator cuff: assessment by computed tomography versus magnetic resonance imaging. *J Shoulder Elbow Surg* 8:599–605
15. Galatz LM, Ball CM, Teefey SA, Middleton WD, Yamaguchi K (2004) The outcome and repair integrity of completely arthroscopically repaired large and massive rotator cuff tears. *J Bone Joint Surg Am* 86-A:219–224
16. Gartsman GM (1997) Massive, irreparable tears of the rotator cuff. Results of operative debridement and subacromial decompression. *J Bone Joint Surg Am* 79:715–721
17. Gerber C, Fuchs B, Hodler J (2000) The results of repair of massive tears of the rotator cuff. *J Bone Joint Surg Am* 82:505–515
18. Gerber C, Fuchs B, Hodler J (2006) Latissimus dorsi transfer for the treatment of irreparable rotator cuff tears. *J Bone Joint Surg Am* 88:113–120
19. Goutallier D, Postel JM, Bernageau J, Lavau L, Voisin MC (1994) Fatty muscle degeneration in cuff ruptures. Pre- and postoperative evaluation by CT scan. *Clin Orthop Relat Res* 304:78–83
20. Hamada K, Fukuda H, Mikasa M, Kobayashi Y (1990) Roentgenographic findings in massive rotator cuff tears: a long-term observation. *Clin Orthop Relat Res* 254:92–96
21. Iannotti JP, Codsi MJ, Kwon YW, Derwin K, Ciccone J, Brems JJ (2006) Porcine small intestine submucosa augmentation of surgical repair of chronic two-tendon rotator cuff tears: a randomized, controlled trial. *J Bone Joint Surg Am* 88:1238–1244
22. Ide J, Maeda S, Takagi K (2005) A comparison of arthroscopic and open rotator cuff repair. *Arthroscopy* 21:1090–1098
23. Jennings GJ, Keereweer S, Buijze GA, De Beer J (2007) Transfer of segmentally split pectoralis major for the treatment of irreparable rupture of the subscapularis tendon. *J Shoulder Elbow Surg* 16:837–842
24. Jost B, Puskas GJ, Lustenberger A, Gerber C (2003) Out-come of pectoralis major transfer for the treatment of irreparable subscapularis tears. *J Bone Joint Surg Am* 85-A:1944–1951
25. Jost B, Zumstein M, Pfirrmann CW, Gerber C (2006) Long-term outcome after structural failure of rotator cuff repairs. *J Bone Joint Surg Am* 88:472–479
26. Lafosse L, Brozka R, Toussaint B, Gobezie R (2007) The outcome and structural integrity of arthroscopic rotator cuff repair with use of the double-row suture anchor technique. *J Bone Joint Surg Am* 89:1533–1541
27. Lin KC, Krishnan SG, Burkhead WZ (2008) Massive rotator cuff tears. In: Galatz LM (ed) *Orthopaedic knowledge update. Shoulder and elbow 3*. American Academy of Orthopaedic Surgeons, Rosemont, IL
28. Matthews TJ, Hand GC, Rees JL, Athanasou NA, Carr AJ (2006) Pathology of the torn rotator cuff tendon: reduction in potential for repair as tear size increases. *J Bone Joint Surg Br* 88:489–495
29. Miniaci A, Macleod M (1999) Transfer of the latissimus dorsi muscle after failed repair of a massive tear of the rotator cuff: a two to five-year review. *J Bone Joint Surg Am* 81:1120–1127
30. Montgomery TJ, Yergler B, Savoie FH III (1994) A comparison of arthroscopic debridement with open surgical repair for full-thickness tears of the rotator cuff. *J Shoulder Elbow Surg* 3:70–78
31. Moore DR, Cain EL, Schwartz ML, Clancy WG Jr (2006) Allograft reconstruction for massive, irreparable rotator cuff tears. *Am J Sports Med* 34:392–396
32. Post M, Silver R, Singh M (1983) Rotator cuff tear: diagnosis and treatment. *Clin Orthop* 173:78–91
33. Rockwood CA Jr, Williams GR Jr, Burkhead WZ Jr (1995) Débridement of degenerative, irreparable lesions of the rotator cuff. *J Bone Joint Surg Am* 77:857–866

34. Selamberg SG, Tibone JE, Itamura JM, Kasraeian S (2004) Six-month magnetic resonance imaging follow-up of large and massive rotator cuff repairs reinforced with porcine small intestinal submucosa. *J Shoulder Elbow Surg* 13:538–541
35. Tashjian RZ, Henn RF, Kang L, Green A (2006) Effect of medical co-morbidity on self-assessed pain, function and general health status after rotator cuff repair. *J Bone Joint Surg Am* 88:536–540
36. Wall B, Nove-Josserand L, O’Conner DP, Edwards TB, Walch G (2007) Reverse total shoulder arthroplasty: a review of results according to etiology. *J Bone Joint Surg Am* 89:1476–1485
37. Warner JJ (2001) Management of massive irreparable rotator cuff tears: the role of tendon transfer. *Instr Course Lect* 50:63–71
38. Warner JJ, Gerber C (1998) Treatment of massive rotator cuff tears: posterior-superior and anterior-superior. *American Academy of Orthopaedic Surgeons*, Rosemont, IL
39. Warner JJ, Parsons IM IV (2001) Latissimus dorsi tendon transfer: a comparative analysis of primary and salvage reconstruction of massive, irreparable rotator cuff tears. *J Shoulder Elbow Surg* 10:514–521
40. Warner JP, Krushell RJ, Masquelet A, Gerber C (1992) Anatomy and relationships of the suprascapular nerve: anatomical constraints to mobilization of the supraspinatus and infraspinatus muscles in the management of massive rotator-cuff tears. *J Bone Joint Surg Am* 74:36–45
41. Werner CML, Steinmann PA, Gilbert M, Gerber C (2005) Treatment of painful pseudoparesis due to irreparable rotator cuff dysfunction with the Delta III reverse-ball-and-socket total shoulder prosthesis. *J Bone Joint Surg Am* 87:1476–1486
42. Zingg PO, Jost B, Sukthankar A, Buhler M, Pfirrmann CW, Gerber C (2007) Clinical and structural outcomes of nonoperative management of massive rotator cuff tears. *J Bone Joint Surg Am* 89:1928–1934

## Part VII

---

### Hip

# Hip Resurfacing

Derek McMinn, Joseph Daniel, Hena Ziaee, and Chandra Pradhan

## Introduction

Hip arthritis, whether primary or secondary, manifests itself through the final common pathway of articular surface loss. Being essentially a surface problem it has always attracted surgeons to apply a surface solution. Sir John Charnley [4] himself developed the first generation resurfacings made of polytetrafluoroethylene. However the early failures experienced by him and the failures with different other materials used in the various second generation hip resurfacings, led to hip resurfacings being rejected as a bad concept altogether in the late 1980s. Early results suggested that the failures occurred as a result of femoral head avascular necrosis [2, 15] or stress-shielding [12, 16]. However, careful analysis showed that this was a failure of materials rather than a failure of the procedure itself. Metal-on-metal resurfacings are not totally new either [14, 30]. Maurice Muller gave up using metal-on-metal hip resurfacings in the 1960s when polyethylene had become a favoured material. Gerard also performed a small series of metal-on-metal resurfacings during the same period but he too stopped using them.

Charnley knew that the high activity levels in young patients would jeopardize long-term success of total hip replacements, and suggested that they should not be offered to young patients unless there were other physical restraining factors [3]. The Swedish National Hip Arthroplasty Register [24], on the strength of the available evidence, considered young patients with hip arthritis to be the “supreme challenge” for hip replacement.

It was for this specific group of young and active patients who perform poorly with a conventional THR that modern M-M hip resurfacing was developed and it is in this group that a hip resurfacing works best.

---

D. McMinn (✉)  
The McMinn Centre, 25 Highfield Road, Edgbaston  
Birmingham, B15 3DP West Midlands, UK  
e-mail: derekmcminn@mcminncentre.co.uk

## The Birmingham Hip Resurfacing

The pioneering work of Derek McMinn on metal-on-metal bearing hip resurfacings at Birmingham UK, in 1991 signalled the beginning of the new era of modern metal-on-metal hip resurfacings [28]. The initial phase included pilot studies [29] to determine the best method of fixation. These studies showed that cemented femoral and uncemented acetabular components gave the best results. The lessons learnt from the first 6 years of implantation during which the results of 500 hip resurfacings were evaluated led eventually to the development of the Birmingham hip resurfacing [27] in July 1997.

Alongside this development, advances were also made by McMinn in the realm of the optimal surgical technique [26] to implant a resurfacing. It had always been obvious that the resurfacing procedure would be technically more challenging than a regular total hip replacement. The retained femoral head would impede good acetabular exposure unless adequate capsular releases were performed and a pocket was created into which the femoral head could escape while the socket was being prepared. A resurfacing procedure also meant that all this had to be achieved without rendering the femoral head and neck avascular. Some of these steps, which are specific to resurfacing include avoidance of femoral neck notching, preservation of femoral neck soft tissues in order to prevent proximal femoral devascularization and negative pressure venting of the metaphysis to prevent systemic fat embolism and femoral neck vessel embolization. Time has proven that these measures are critical to the success of resurfacing.

The Birmingham Hip Resurfacing has a hydroxyapatite-on-porous metal uncemented cup and a cemented femoral component. The porous surface consists of unique cast-in beads which are integral with the substance of the cup and therefore are not easily dislodgeable. The beads also provide an excellent surface for bony in-growth and enduring fixation of the cup.

## Results, Problems and Solutions

Our series of 3,014 consecutive hip resurfacings performed between July 1997 and December 2008 provide the single-surgeon (DJWM) cohort from which we have analyzed the overall results of Birmingham Hip Resurfacings (BHRs) (Table 1).

In addition to our own documentation and follow-up, our patients are independently followed up by two other Outcomes Centres in the UK, and this leads us to believe that we have as far as humanly possible, captured all our revisions. Seventy-four percent of these patients had a diagnosis of primary osteoarthritis. Other diagnoses include avascular necrosis, inflammatory arthritis, sequelae of childhood hip disorders, traumatic and post-septic arthritis. Age at operation ranged from 13 to 86 years. In this series which includes all ages and all diagnoses, with revision of either component for any reason as the end-point, the failure rate is 1.9% and

**Table 1** Patient demographics in the cohort studied

Number of hips	3,014
Max follow-up	12
Min follow-up	0.5
Mean follow-up (years)	7.1
Component years	21,372
Men	72%
Primary osteoarthritis	2,233 (74%)
Mean age at operation (years)	53.6

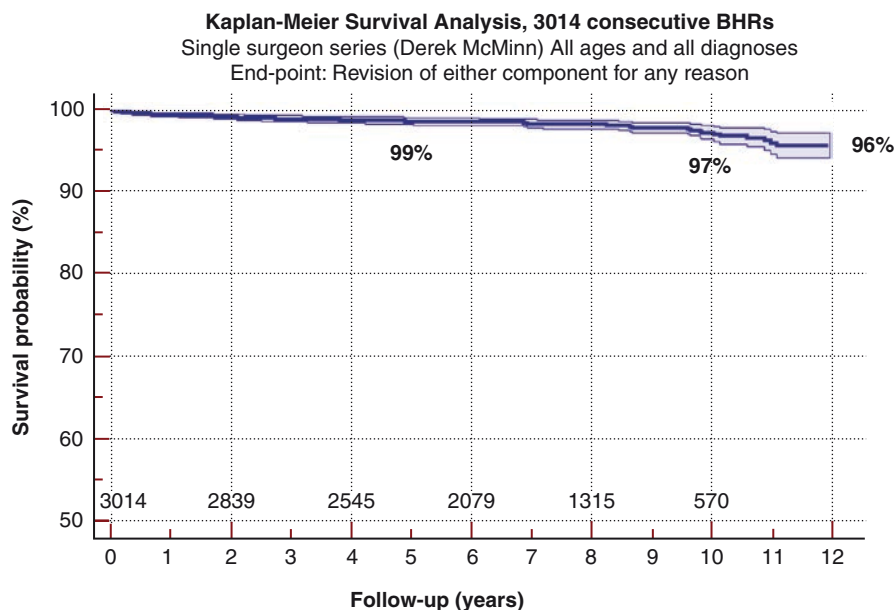
the survivorship is 99% at 5 years, 97% at ten years and 96% at 12 years (Fig. 1). Among patients with osteoarthritis as the primary diagnosis the survivorship in all ages is better than those with other primary diagnoses which include femoral head AVN, hip dysplasia, old Perthes disease, old SCFE and inflammatory arthritis (Fig. 2).

Out of 58 revisions in the entire cohort, 47 were non-bearing-related (1.6%) and include either femoral failures (fractures or femoral head collapse) or infections and one technical failure. The rest include: one failure with osteolysis, one cup loosening, two due to unexplained groin pain and seven pseudotumors.

Previously we published our results [6] in 403 consecutive Birmingham Hip Resurfacings performed between 1997 and 2001 in patients with primary osteoarthritis under the age of 55 years. We have continued to follow these patients up over the years (Fig. 3). There are currently three failures in this group giving a 12-year survivorship of 98.9% (Fig. 4). Furthermore most of these patients report that they have forgotten about their hips and carry on with life as normal. None of these patients had to change their occupation or life style. Nine out of ten men with unilateral hip resurfacings continue to participate in sporting activity and six out of ten continue to participate in impact sports or are involved in heavy or moderately heavy activity at their workplace.

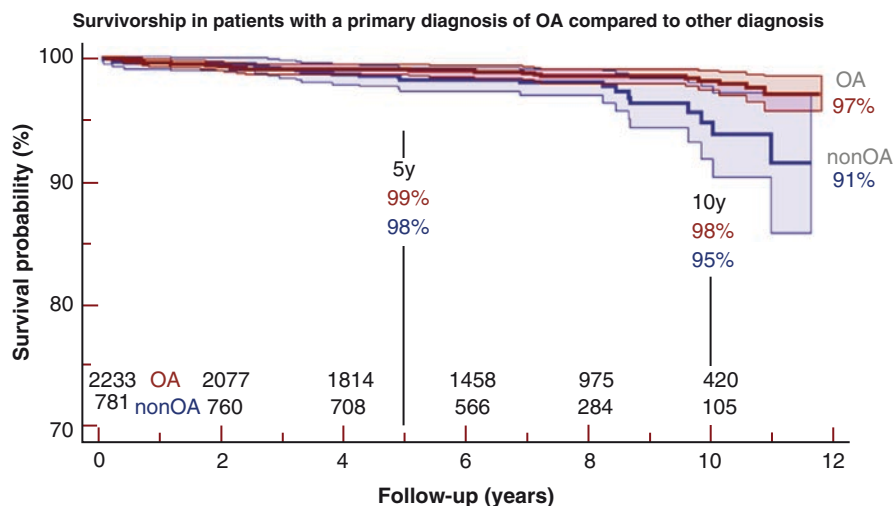
### Femoral Failures

As opposed to a conventional THR, the femoral head and neck are retained in a hip resurfacing procedure. This adds the



**Fig. 1** Implant survivorship of BHRs in 3,014 consecutive hips, using revision of either component for any reason as the end-point

**Fig. 2** Implant survivorship of BHRs in patients with osteoarthritis compared to that in those with other diagnoses



risks of femoral neck fracture in the early months after a resurfacing, and of a femoral head collapse in the early or later years. In our own series mentioned earlier, the incidence of femoral neck fractures is (12/3014) 0.4%. All our fractures occurred in the first 6 months after operation. An Australian multi-centre series [34] reported 50 femoral neck fractures out of 3497 BHRs (1.4%) inserted between 1999 and 2004, by 89 surgeons, most of whom were new to the procedure. Technical errors in positioning the femoral component or intra-operative notching of the femoral neck were responsible for the fracture in 85% of their fractured cases. Peri-prosthetic fractures also occur following a THR. In a large series [35] of 30,000 THRs, there was a prevalence of 1.1% post-operative femoral fractures after primary THRs and 4% after revision THRs.

Femoral neck fracture following hip resurfacing is an easy matter to deal with compared to treatment of a peri-prosthetic fracture around a THR. Furthermore, retention of the head and neck in a resurfacing offers the long-term benefit of more natural load-bearing and therefore better bone density preservation. This is seen clearly from a DEXA scan study [21] of bone mineral density of the proximal femur. An increase in bone density of around 11% was demonstrated in the critical calcar region after a hip resurfacing as compared to a 17% loss of bone density following an uncemented THR 2 years after the operation in age, disease and gender-matched patients.

Femoral head collapse is another failure pattern with hip resurfacing and occurs as a result of the collapse of a previously avascular, osteopenic or cystic segment. Femoral head collapse can be a slowly progressive process leading to a tilt of the femoral component into varus. In a varus position the resurfacing component is rendered mechanically disadvantaged and leads to progressively increasing

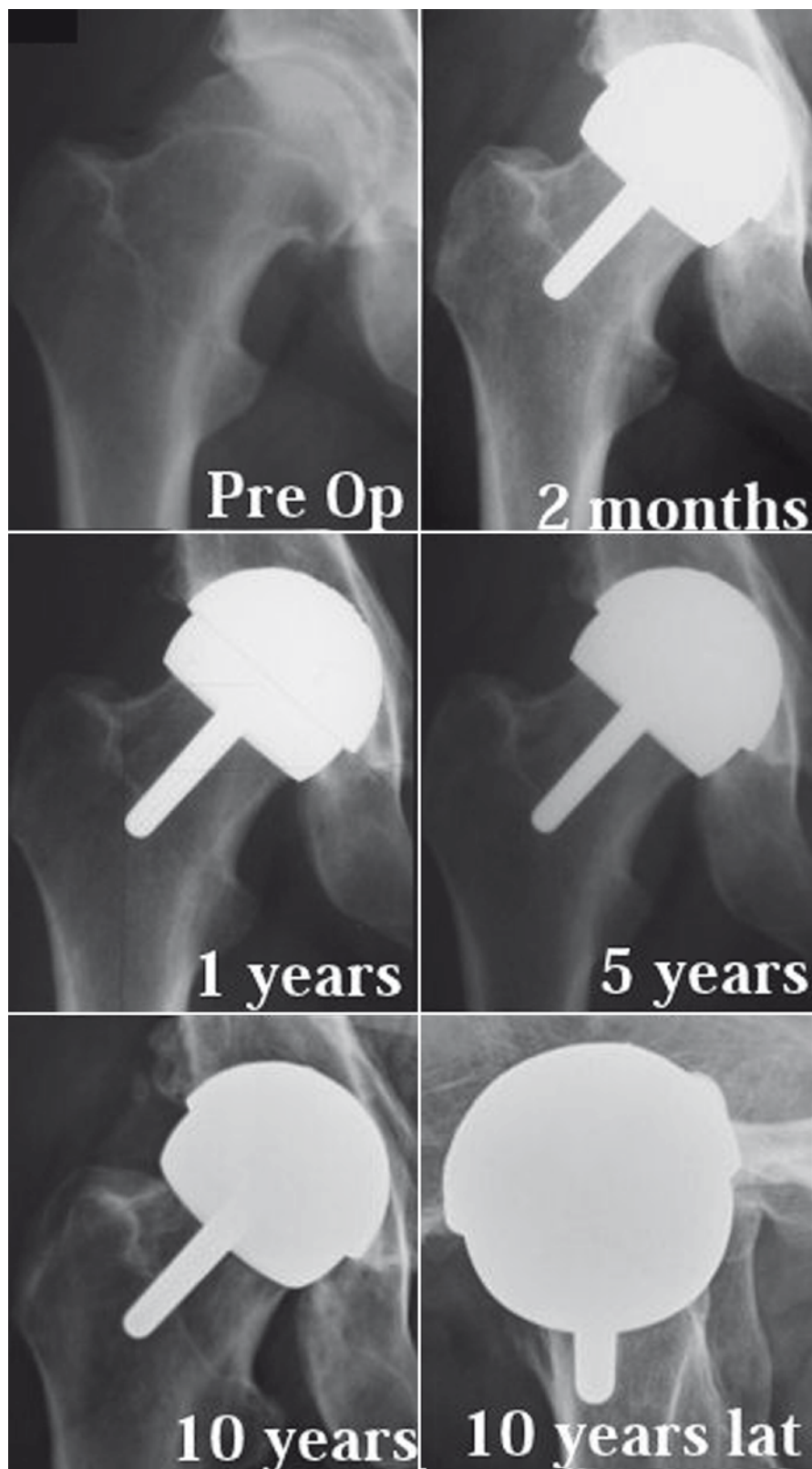
deformity until at some point it fails with or without a precipitating factor such as a trivial injury. This can happen a few weeks after the initial collapse or several years later. Although it is possible that collapse may also occur as a result of operative devascularisation of the femoral head, it has been shown through several studies that if the correct operative precautions are adhered to femoral head devascularization is an extremely rare event [25, 36].

### The Birmingham Mid-Head Resection

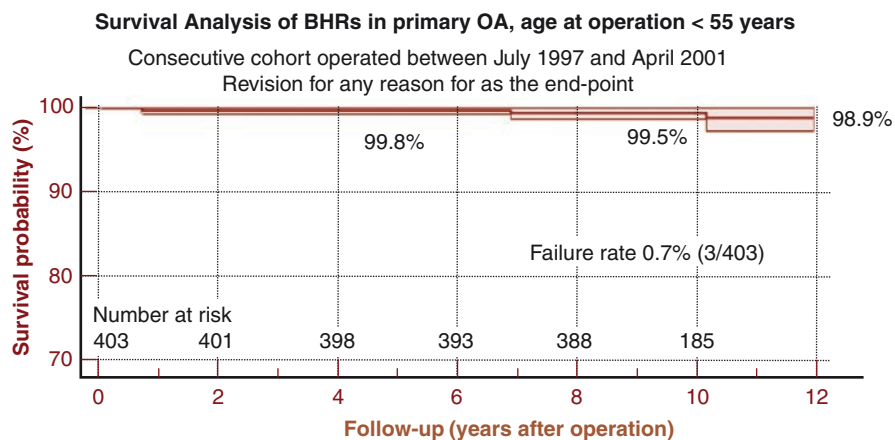
The femoral component of a resurfacing depends on the integrity and quality of the femoral head bone for long-term success. In cases with a pre-existent avascular or osteopenic bone, or cystic segment in the femoral head, a resurfacing can be performed with supplementary bone grafting or cement build-up of the defect. Experience has shown us that even though in some cases that strategy is successful, it does not always give a reproducible result, since there is a high risk of fracture or further collapse of the weakened segment. For young patients who present with such a situation a total hip replacement with a medullary stem was the only option in the past. The Birmingham Mid-Head Resection (BMHR) [Smith and Nephew Orthopaedics, Warwick, UK] offers the prospect of circumventing the need for a more invasive procedure like hip replacement in precisely such young patients who would greatly benefit from a conservative arthroplasty, but lack good femoral head bone quality, which is a prerequisite for a successful hip resurfacing. Furthermore in patients with proximal femoral dysmorphism such as in old Perthes disease or old SCFE (slipped capital femoral



**Fig. 3** The 10-year radiographic series of a 45-year-old patient with primary osteoarthritis treated with a Birmingham hip resurfacing



**Fig. 4** Twelve-year Kaplan-Meier implant survivorship in patients with osteoarthritis treated with a Birmingham hip resurfacing under the age of 55 years at operation



epiphysis) deformity, there is insufficient peripheral support for the femoral component and therefore a tendency to accept a sub-optimal component implantation, such as in varus. The BMHR stem however does not need the peripheral support that a BHR component demands and therefore can be reliably placed in an optimum position even in these conditions. A third area of need for the BMHR includes cases where there is scope for significant leg-length equalization or offset enhancement in order to restore symmetrical biomechanics of the hip.

The BMHR consists of an uncemented short stem made of titanium alloy and a large diameter cobalt-chrome metal-on-metal bearing which is identical to the BHR bearing (Figs. 5 and 6). This short-stemmed device does not violate the medullary canal of the femoral shaft and makes future revisions easier. A unique osteotomy line which passes through the middle of the femoral head exploits the natural internal shape of the femoral head and neck to create a perfect cone for robust stem fixation. This resection level also ensures removal of poor quality bone in the proximal femoral head, while retaining the distal part of the femoral head which contributes to load transfer from the stem. Continued load-bearing through this segment prevents stress-shielding deterioration of the neck which has been a regular feature with other neck-preserving total hip replacements.

The excellent early clinical and radiographic results relating to the BMHR, the absence of stress-shielding on plain radiographs and the absence of significant stem migration as seen from an RSA (Roentgen Stereophotogrammetry Analysis) study offer good reasons to be optimistic that the BMHR device will prove to be a strategic tool in the modern management of end-stage hip arthritis in the young, especially when the femoral head anatomy or bone quality is sub-optimal or suspect. We are seeing a reduction in femoral failures in conservative hip arthroplasty following the judicious usage of the BMHR in selected patients.

### Metal Ion Elevation

Systemic metal ion elevation invariably follows MM (metal-on-metal) bearing usage [5, 7, 8] and there have been concerns relating to the possibility of nephrotoxicity, carcinogenesis and mutagenesis as a sequel to metal ion elevation. Epidemiological studies have not upheld these concerns [38]. Other studies have demonstrated that there are biological regulatory systems [33] and mechanisms that maintain the internal milieu within reasonable limits [10, 41] thereby mitigating some of the effects of clinically-relevant elevations of metal ions. Furthermore some of the biological changes demonstrated in patients with MM bearings have also been found in association with other bearings including metal-on-polyethylene [11, 13] and ceramic bearings [22].

In addition to the long-standing concern relating to the systemic effects of elevated metal ion levels in metal-on-metal (MM) hip resurfacings, there have been recent reports of peri-prosthetic local adverse tissue responses including effusions and what have come to be known as “pseudotumours”. Pseudotumours are not unique to resurfacings or to MM bearings. They have been described in relation to metal-on-polyethylene resurfacings [18] and replacements [1, 17, 37], knee replacements [19], viscosupplementation [20] and even in the absence [40] of any device.

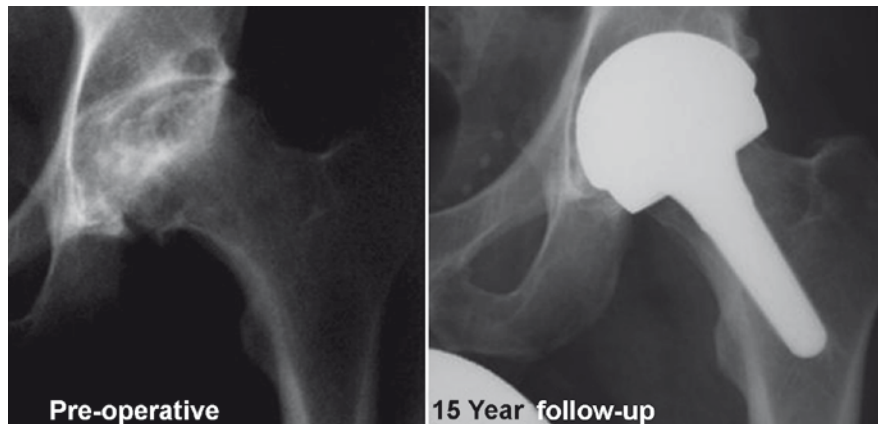
### Pseudotumours

Recent reports from two centres with respect to pseudotumours following the use of metal-on-metal bearings have caused concern. In the Norfolk and Norwich Hospital in England 643 MM THAs (Total hip arthroplasties) (DePuy Ultima TPS femoral component in combination with Ultima metal-on-metal bearing, Depuy Johnson and Johnson, Leeds UK) were implanted. Sixty of these (9.3%) failed with

**Fig. 5** A Birmingham hip resurfacing femoral and acetabular components and a Birmingham mid-head resection device stem and modular head



**Fig. 6** The pre-operative and follow-up radiographs of a 31-year-old-male with steroid-induced AVN of the femoral head treated with a Birmingham mid-head resection device



effusions [31] containing thick pus-like fluid, extensive soft tissue and bony necrosis, peri-prosthetic fractures and recurrent dislocations reportedly due to corrosion from the stem (i.e. Implant-related). Before the introduction of the BHR we too have had a high incidence of implant-related failures (14% failure at 10 years) with double heat-treated resurfacings implanted during 1996 (Corin Medical Ltd, Cirencester, Gloucs, UK) [9]. Although we have found metallosis and osteolysis in most of those failures we did not investigate

these patients specifically with imaging techniques aimed at identifying pseudotumours, because this had not been recognized as a specific pattern of failure then.

In Oxford [32] three different types of metal-on-metal resurfacings were employed with a 1% incidence of cystic or solid masses and in some cases semi-liquid pus-like contents, necrosis, pathological fractures and dislocations. Patients who were revised for pseudotumours in Oxford were reported to have had major complications including

nerve palsies, pathological fractures, recurrent dislocations and a high incidence of re-revisions. We compare our results with those reported from Oxford.

In our series of 3,014 consecutive BHRs we have had eight symptomatic pseudotumours so far (0.27%), three of which have been revised to a non-MM bearing THR at our Centre, four revised at other Centres and another is awaiting revision. In the three patients that were revised at our Centre we found no muscle necrosis, no nerve palsies, no pathological fractures or dislocations. There was straw-coloured fluid in two and metal staining in one. On the basis of patient feedback and hip outcome scoring, none of the other four revised in other centres had major complications, such as pathological fractures, dislocations, nerve palsies or femoral artery stenosis. There were no re-revisions in any of the seven. Current hip function in all the revised hips is good with a mean Oxford hip score of 18 (range 12–25) which is not worse than the score in revisions for all other reasons (18, range 14–22) in our series nor different from the score in the control group in the Oxford series for primary total hip replacements (18).

It is possible that some of the revisions at Oxford were due to edge-loading from sub-optimal cup inclination. Data relating to cup anteversion is not available for those revisions. Although the cup inclination in our failures is acceptable, the combined anteversion of cup and femoral neck are beyond the safe limit of 45° in a majority. It is possible that this has also led to edge-loading and excess wear. With a 10-year survivorship of 99.6% with revision for pseudotumours as the end-point, these are rare events. However periprosthetic pseudotumours remain a big concern and we are currently investigating this issue with a thorough clinico-radiological (including multi-slice CT scanning) and metal ion study of our consecutive series of patients with BHRs who have completed 10-year follow-up. Preliminary findings suggest that component malposition leading to edge loading and excess wear is the primary reason for this phenomenon.

Many of the reported painful failures with or without pseudotumours have been found to display characteristic histomorphological features [39] in the peri-prosthetic tissues suggesting the development of an immunological response to wear debris. Whether the prime mover in the whole process is excess wear leading on to sensitization of the patient to debris from metal wear/corrosion, or an innate hypersensitivity to expected levels of wear/corrosion, is debatable.

Apart from the complex problem of metal hypersensitivity which is not fully understood at this stage, preliminary findings suggest that these wear-debris-related local responses and failures are due to three different factors:

- (a) Implant factors as seen in our 1996 series and also reported from Norfolk.
- (b) Surgeon factors such as sub-optimal orientation of components as seen from the evidence of edge-loading in some of the retrieved explants.
- (c) Patient factors – dysplasia with socket deficiency and femoral torsion deformities creating difficulty in implant positioning both in terms of cup inclination and version of cup and femoral neck. Meticulous attention to both component inclination and version and good implant and patient selection are critical to obtaining good results in the long-term. Every implant is different in its micro-structure, design and geometry. The techniques, results, risks and benefits of the different implants are not mutually transferable. Careful assessment of women with premature arthritis to exclude unrecognized femoral neck anteversion is essential in order to minimize this mode of failure in resurfacing.

These biological concerns should serve as an impetus to surgeons to strive for more precise implantation technique, and to engineers and designers to continue their pursuit of bearings with reduced wear characteristics. We have been exploring the possibility of developing some potential alternative bearing concepts into workable solutions.

---

### Bearings Other than Metal-On-Metal for Resurfacing

There are several potential alternatives to metal-on-metal bearings worth considering. These include surface modifications, compliant bearings, ceramics and composites and newer polyethylenes.

Surface coatings including ceramic and diamond-like carbon (DLC) coatings have been shown to enhance the scratch resistance of a bearing surface in the laboratory. When they were used clinically however, delamination of the coating led to early failures and therefore they were abandoned. Improvements are constantly being made to the processes involved in applying these coatings with the expectation that they would be prove more resilient but none has reached the stage at which they could be re-introduced clinically.

In contrast to coatings, surface modifications have proved more durable. Oxinium, a surface-modified Zirconium has been successfully developed into hip and knee replacement components although this too has been having teething problems. Plasma carburization which is a surface modification process has been successfully used in the manufacture of racing car components for many years. During this process the alloy is heated in the presence of a compound which liberates carbon as it breaks down, thereby enriching the carbon content of the outer layers of the metal and

hardening it, while the core retains its original properties. Since this is not a type of surface coating, the problem of delamination does not arise with this material. Furthermore if the hardened layer is worn through, it only exposes the original metal substrate rather than a completely new surface.

Laboratory tests have shown low temperature plasma carburization substantially increases the carbon content up to a depth of 10  $\mu\text{m}$  from the bearing surface and more than doubles the hardness of the material. Furthermore surface profiling of the carburized cobalt-chrome shows a lower surface-roughness than untreated cobalt-chrome material and is equivalent to the finish obtainable with alumina ceramic. The dual benefits of improved scratch resistance combined with a smoother surface finish potentially result in less metal ion release. Hip simulator results of plasma carburized head BHRs show less wear rates compared to regular BHRs out to three million cycles and the differences are statistically significant through run-in and steady-state phases.

Polyurethane is a compliant bearing which has undergone extensive laboratory testing for possible use in hip arthroplasty devices. This is a material which functions in an elasto-hydrodynamic mode through squeeze film lubrication when it is coupled against a metallic femoral head construct. Friction testing of the material against a metal shows significantly lower friction factors compared to a metal-on-polyethylene bearing. The problem with polyurethane is that it is an elastomer and deforms under load, and therefore requires an extremely large clearance between the polyurethane liner and femoral head. Furthermore fixation of the cup requires a metal shell thereby increasing the thickness of the combined metal-polymer construct. In the past this material has been used as a bearing couple with metal by Charles Townley in his resurfacing device. When a cohort of 26 patients with these resurfacings were subsequently reviewed, they showed a 100% failure rate through polyurethane wear at 24 years.

Carbon-fibre re-inforced composites have the added advantage of being materials which are almost iso-elastic to bone. The Cambridge cup has a carbon-fibre-re-inforced polybutyleneterephthalate (CFR-PBT) backing for its polyethylene bearing surface. The stiffness of the PBT layer approximates to that of the subchondral bony plate allowing it to deform in concert with the surrounding acetabular bone so that micromotion at the fixation interface is minimized and stress shielding is reduced. CFR PEEK (Carbon-fibre re-inforced poly ether-ether ketone) is a carbon-fibre re-inforced composite having superior wear characteristics and can be produced as thin-walled components needed for resurfacing.

Although ceramic components are not suitable for use as thin-walled resurfacing components, ceramic-polyurethane

composites lend themselves to solutions in resurfacing. This material has been used for over a decade in total hip arthroplasty components in Europe and demonstrates excellent wear resistance and strength in clinical usage. It holds promise for usage as resurfacing components, especially in those patients who are hypersensitive to nickel and therefore unsuitable for MM resurfacing, but are still young and therefore would potentially benefit from a conservative hip procedure. We have used these ceramic-composite resurfacings in a small number of such patients and have seen good early results.

Ceramic-on-metal is another bearing which has demonstrated potential in hip simulator testing. Large diameter ceramic-on-metal bearings when tested in the laboratory have shown significant reduction in wear compared to a metal-on-metal bearing of identical design. Early results with this bearing mounted on a Birmingham Mid-head Resection stem in a small number of patients also shows promising outcomes.

The problem with some of the above bearings is that they are new materials and do not have a long history of clinical usage. One material that has almost as long a history of clinical usage as MM is polyethylene. Conventional polyethylenes especially those that had been gamma irradiated in air were subject to oxidation and deterioration leading to early and excessive wear. In order to combat that problem, several improvements have been progressively made to this material. Highly cross-linked polyethylene was developed which improved wear resistance but diminished the mechanical properties of the material resulting in cracking, fissuring and breakage. Furthermore, although hip simulator studies demonstrated almost no wear even after 40 million cycles of testing, in vivo clinical experience using RSA (roentgen stereo-photogrammetric analysis) has shown that wear in these bearings starts increasing after 5 years [23]. The wear plot then starts to run roughly parallel to the wear in conventional polyethylene.

It has been shown that the deterioration in mechanical properties of the polyethylene are due to the presence of residual free radicals created during cross-linkage. Furthermore in vivo oxidation of the material from exposure to oxidizing agents present in the tissues and joint fluid continues to occur after implantation leading to creation of fresh free radicals. In order to eliminate or quench the continuing oxidation and in order to further improve its wear resistance and strength, third generation polyethylenes have been introduced. One of these, E-poly uses vitamin-E doping. We are on the threshold of a further advance in polyethylene in the form of surface modification similar to the modifications described above whereby the surface of polyethylene would be made wear-resistant while the core retains the excellent mechanical properties of untreated polyethylene. The

development of a suitable fixation interface will then allow this material to be used as a thin potential resurfacing cup component for articulation against an advanced metal bearing surface with the potential for low wear.

The increasing use of metal-on-metal bearings was initiated as a solution to the debris induced osteolysis secondary to polyethylene wear. It is ironic that in our attempt to find a solution to MM bearing wear we have gone full circle to look for solutions again in polyethylene or in one of its improved forms.

## Conclusion

Hip resurfacing is not a panacea for all hip arthritis. As noted above it works best in young patients with good quality femoral head bone and a reasonable proximal femoral anatomy which does not need extensive re-adjustment of leg length, offset and hip centre. The Birmingham Hip Resurfacing, when performed well in properly selected patients, continues to demonstrate excellent outcomes. The results however leave room for better patient selection and more precise surgery. There is also a scope for alternative bearing materials especially for those patients who are potentially intolerant to metal-on-metal bearings.

## References

1. Beksaç B, Tözün R, Baktiroglu S et al (2007) Extravascular compression of the femoral vein due to wear debris-induced iliopsoas bursitis: a rare cause of leg swelling after total hip arthroplasty. *J Arthroplasty* 22:453–456
2. Bogoch ER, Fornasier VL, Capello WN (1982) The femoral head remnant in resurfacing arthroplasty. *Clin Orthop* 167:92–105
3. Charnley J (1979) *Low friction arthroplasty of the hip. Theory and practice.* Springer, Berlin, Heidelberg, New York
4. Charnley J (1960) *Surgery of the hip joint. Present and future developments.* *Br Med J* 5176:821–826
5. Daniel J, Ziaee H (2009) Metal ions. In: McMinn DJW (ed) *Modern hip resurfacing.* Springer, UK, London, pp 135–162
6. Daniel J, Pynsent PB, McMinn DJ (2004) Metal on metal resurfacing of the hip in patients under the age of 55 years with osteoarthritis. *J Bone Joint Surg [Br]* 86-B:177–184
7. Daniel J, Ziaee H, Pradhan C et al (2007) Blood and urine metal ion levels in young and active patients after Birmingham hip resurfacing arthroplasty: four-year results of a prospective longitudinal study. *J Bone Joint Surg [Br]* 89:169–173
8. Daniel J, Ziaee H, Pradhan C et al (2009) Six-year results of a prospective study of metal ion levels in young patients with metal-on-metal hip resurfacings. *J Bone Joint Surg [Br]* 91:176–179
9. Daniel J, Ziaee H, Kamali A et al (2010) Ten-year results of a unique first-of-a-kind metal-metal bearing. *J Bone Joint Surg [Br]* 1:20–27
10. Daniel J, Ziaee H, Pradhan C et al (2009) Renal Clearance of Cobalt: Is the renal threshold being breached in patients with metal-on-metal arthroplasty? *J Bone Joint Surg [Am]* Accepted for publication. (In press)
11. Davies AP, Sood A, Lewis AC et al (2005) Metal-specific differences in levels of DNA damage caused by synovial fluid recovered at revision arthroplasty. *J Bone Joint Surg [Br]* 87:1439–1444
12. de Waal Malefijt MC, Huiskes R (1993) A clinical radiological and biomechanical study of the TARA hip prosthesis. *Arch Orthop Trauma Surg* 112:220–225
13. Doherty AT, Howell RT, Ellis LA et al (2001) Increased chromosome translocations and aneuploidy in peripheral blood lymphocytes of patients having revision arthroplasty of the hip. *J Bone Joint Surg [Br]* 83:1075–1081
14. Gerard Y (1978) Hip arthroplasty by matching cups. *Clin Orthop* 134:25–35
15. Head WC (1981) Wagner surface replacement arthroplasty of the hip. Analysis of fourteen failures in forty-one hips. *J Bone Joint Surg [Am]* 63:420–427
16. Head WC (1984) Total articular resurfacing arthroplasty. Analysis of component failure in sixty-seven hips. *J Bone Joint Surg [Am]* 66:28–34
17. Hisatome T, Yasunaga Y, Ikuta Y et al (2003) Hidden intrapelvic granulomatous lesions associated with total hip arthroplasty: a report of two cases. *J Bone Joint Surg [Am]* 85:708–710
18. Howie DW, Cain CM, Cornish BL (1991) Pseudo-abscess of the psoas bursa in failed double-cup arthroplasty of the hip. *J Bone Joint Surg [Br]* 73:29–32
19. Jacobs JJ, Urban RM, Wall J et al (1995) Unusual foreign-body reaction to a failed total knee replacement: simulation of a sarcoma clinically and a sarcoid histologically. A case report. *J Bone Joint Surg [Am]* 77:444–451
20. Jones KB, Patel PP, DeYoung BR et al (2005) Viscosupplementation pseudotumor. A case report. *J Bone Joint Surg [Am]* 87:1113–1119
21. Kishida Y, Sugano N, Nishii T (2004) Preservation of the bone mineral density of the femur after surface replacement of the hip. *J Bone Joint Surg [Br]* 86:185–189
22. Ladon D, Bhamra M, Turner J et al (2000) Changes in chromosome aberrations and metal levels in the peripheral blood of patients after ceramic-on-ceramic hip replacement. *Proceedings 51st Annual Meeting of the Orthopaedic Research Society.* Washington, DC
23. Malchau H (2009) Presidential guest lecture. ‘Highly cross linked polyethylene’. *Proceedings British Hip Society, Annual Scientific Meeting.* Manchester, UK
24. Malchau P, Herberts P, Soderman P (2000) Update and validation of results from the Swedish Hip Arthroplasty Registry 1979–1998. *Department of Orthopaedics University of Goteborg, Sweden. Proceedings 67th Annual Meeting of American Academy of Orthopaedic Surgeons.* Orlando, USA

25. McMahon S, Hawdon G (2009) Vascularity of the femoral head in hip resurfacing. In: McMinn DJW (ed) *Modern hip resurfacing*. Springer, UK, London, pp 117–124
26. McMinn DJW (1997) *Birmingham hip resurfacing: operative technique*. Midland Medical Technologies, Birmingham, UK
27. McMinn DJW (2003) Development of metal-metal hip resurfacing. *Hip Int* 13:S41–S53
28. McMinn DJW (2009) Development perspectives. In: McMinn DJW (ed) *Modern hip resurfacing*. Springer, UK, London, pp 1–41
29. McMinn D, Treacy R, Lin K et al (1996) Metal on metal surface replacement of the hip. Experience of the McMinn prosthesis. *Clin Orthop* 329(S):S89–S98
30. Müller ME, Boitzky X (1968) Artificial hip joints made from Protasul. *Bulletin Association for Study of Problems in Internal Fixation*. pp 1–5
31. Nolan JF, Darrah C, Donell ST et al (2007) Metal on metal hip replacement. *Proceedings British Hip Society Annual Scientific Meeting*. Leeds, UK
32. Pandit H, Glyn-Jones S, McLardy-Smith P et al (2008) Pseudotumours associated with metal-on-metal hip resurfacings. *J Bone Joint Surg [Br]* 90:847–851
33. Reynolds M, Peterson E, Quievryn G et al (2004) Human nucleotide excision repair efficiently removes chromium-DNA phosphate adducts and protects cells against chromate toxicity. *J Biol Chem* 279:30419–30424
34. Shimmin AJ, Back D (2005) Femoral neck fractures following Birmingham hip resurfacing: a national review of 50 cases. *J Bone Joint Surg [Br]* 87:463–464
35. Springer BD, Berry DJ, Lewallen DG (2003) Treatment of periprosthetic femoral fractures following total hip arthroplasty with femoral component revision. *J Bone Joint Surg [Am]* 85:2156
36. Sugano N, Nishii T, Hananouchi T (2009) Femoral head blood supply studies. In: McMinn DJW (ed) *Modern hip resurfacing*. Springer, UK, London, pp 125–128
37. Svensson O, Mathiesen EB, Reinholt FP et al (1988) Formation of a fulminant soft-tissue pseudotumor after uncemented hip arthroplasty. A case report. *J Bone Joint Surg [Am]* 70:1238–1242
38. Visuri T, Pukkala E (2001) Does metal-on-metal hip prosthesis have influence on cancer? A long-term follow-up study. In: Reiker C, Oberholzer S, Wyss U (eds) *World tribology forum in arthroplasty bern*. Hans Huber, Toronto, Seattle, pp 181–188
39. Willert HG, Buchhorn GH, Fayyazi A et al (2005) Metal-on-metal bearings and hypersensitivity in patients with artificial hip joints. A clinical and histomorphological study. *J Bone Joint Surg [Am]* 87:28–36
40. Yoon TR, Song EK, Chung JY (2000) Femoral neuropathy caused by enlarged iliopsoas bursa associated with osteonecrosis of femoral head—a case report. *Acta Orthop Scand* 71:322–324
41. Ziaee H, Daniel J, Datta AK et al (2007) Transplacental transfer of cobalt and chromium in patients with metal-on-metal hip arthroplasty: a controlled study. *J Bone Joint Surg [Br]* 89:301–305

## Part VIII

---

### Knee



# Total Knee Arthroplasty in Extra-Articular Deformities

José A. Hernández-Hermoso

## Introduction

Total knee arthroplasty (TKA) in extra-articular deformity is uncommon, 1 per 1,000 TKA procedures [23], but when it is present it can be challenging to achieve correct alignment. At TKA the surgeon usually confronts deformities due to intra-articular bone erosion or ligamentous laxity, with the centre of the deformity located at the knee. In contrast, with extra-articular deformity the centre of the deformity is outside the knee adding complexity to the basic principles of TKA.

Such a deformity can be located in the femur, in the tibia or in both bones and may be secondary to a mal-united or non-united fracture, a previous osteotomy, physeal trauma, osteomyelitis, metabolic bone disease (rickets, osteomalacia), Paget's disease or congenital bowing [15, 19, 23]. Extra-articular deformity can be uni-planar, bi-planar or tri-planar [14, 27] depending on isolated or combined affection of the coronal (varus-valgus), sagittal (flexion-extension) or transverse (rotational) plane by the deformity and may or may not be associated with intra-articular instability.

There are no large clinical series of TKA in patients with associated extra-articular deformities. Therefore, review of the literature does not reveal which is the most appropriate method of performing TKA in this situation. The primary goal of the surgeon will be to obtain a well-aligned and stable TKA, without pain and with a good range of motion. To correct the extra-articular deformity the surgeon has two choices. It can be corrected by an extra-articular osteotomy performed before or simultaneously with the TKA and located at the site of the deformity

or at a distance from it. An alternative is to correct the extra-articular bone deformity by an off-setting intra-articular femoral or tibial bone resection and soft-tissue balancing of the resultant instability. Intra-articular correction of an extra-articular deformity is limited by the magnitude, location and multi-planar orientation of the deformity.

## Extra-Articular Correction

Correction of the deformity by osteotomy allows bony resections more similar to those routinely performed at the time of arthroplasty and less complex soft-tissue releases [14]. Only an osteotomy at the site of the deformity will correct completely all the aspects of the deformity [7, 15, 16, 19]. Bi-planar and tri-planar deformities are more likely to require extra-articular correction than uni-planar ones. A femoral extra-articular deformity is more likely to require extra-articular correction than a tibial one of the same magnitude, because soft-tissue imbalance created after intra-articular correction of tibial extra-articular deformity is easier to compensate than femoral [15]. An angular extra-articular deformity close to the joint-line is more likely to require extra-articular correction because it requires compensating the deformity by more off-setting of the intra-articular cut than distant ones of the same magnitude [9, 10].

Osteotomy at the site of the deformity can be performed simultaneously [11, 14, 24] with TKA or in a staged fashion [28, 29]. The use of a retrograde intra-medullary nail for fixation involves less tissue dissection than a plate or blade-plate fixation, and rehabilitation may be facilitated [11, 14]. In a staged procedure, the retrograde nail can be removed through the same incision when the TKA is performed, although with navigation techniques it may not be necessary. Stemmed implants have some disadvantages; they force the use of a revision-TKA design component,

J.A. Hernández-Hermoso  
Orthopaedic Department, Terrassa Hospital,  
Carretera Torre Bonica s/n, 08227 Barcelona, Spain  
e-mail: jahernandez@cst.cat

compromising future hip arthroplastic surgery and if a non-union develops it is easier to handle with a nail exchange than with a stemmed component in place. The decision on cement or cementless fixation of the extended stem is unclear because cement will provide a rigid fixation but has the danger of cement extrusion at the osteotomy site which could lead to a non-union.

Although rare, prior osteotomy correction at the site of the deformity may yield satisfactory results avoiding the need of subsequent TKA [28]. Performing sequential correction of the deformity may have the potential advantage of avoiding or delaying the TKA procedure. TKA after prior osteotomy at the site of the deformity, has given good results [5, 14, 19, 23, 24, 28] but complication rates increase [5, 14, 19] and can be as high as 55% [23].

When an osteotomy away from the deformity site is planned, it should be a supracondylar osteotomy [14, 15, 26]. The angle of correction of the supracondylar osteotomy will be a function of the magnitude of the deformity and the distance of the osteotomy from the deformity. The objective is to obtain a joint line perpendicular in relation to the mechanical axis. The disadvantage of this type of osteotomy is that it will create a Z-form of the bone making the use of a stemmed prosthesis difficult, but when possible, the advantage is that it can be done simultaneously through the same incision of the TKA and fixed with the stem or with a plate.

### Intra-Articular Correction

It is technically demanding to correct an extra-articular deformity by means of intra-articular resection of bone and soft-tissue balancing. Technical difficulties increase when the surgeon tries to correct a bi-planar or tri-planar deformity affecting the femur or tibia or when a combined femoral and tibial deformity is addressed at the same time (Figs. 1 and 2).

Intra-articular correction of a uni-planar deformity of the femur or tibia has different effects depending on if it corrects the coronal, sagittal or transverse plane. It is important to know what will be affected in order to perform the TKA procedure properly. The same basic principles are applied to correct a multi-planar deformity or a combined femoral and tibial deformity.

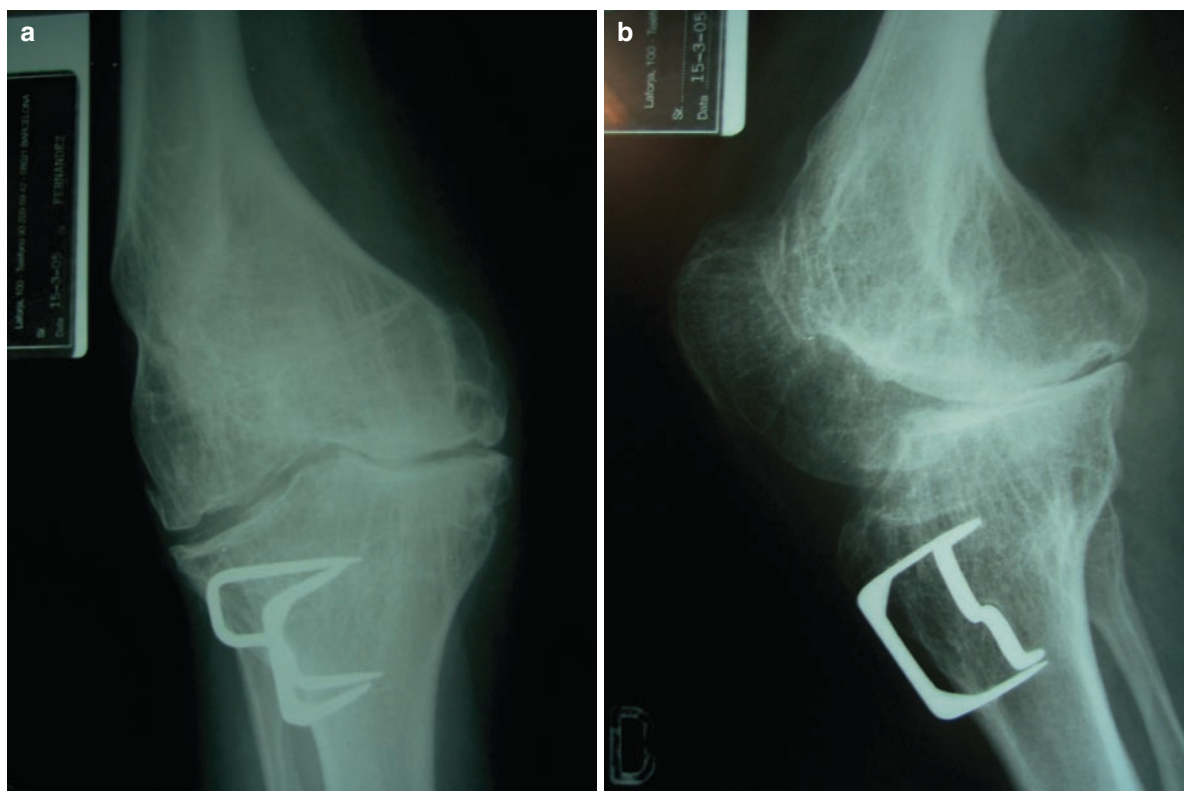
Severe extra-articular femoral and tibial deformities have been treated with this technique, up to 22° in the coronal and 38° in the sagittal plane with 10° external and 20° internal rotation in the femur and 30° in the coronal plane in the tibia [16, 27]. The major advantage of intra-articular correction of the deformity is the avoidance of an



**Fig. 1** Full-length standing anteroposterior radiograph of the lower extremity of a 74-year-old woman demonstrated significant post-osteotomy varus deformity of the right distal femur and valgus deformity of the tibia, in a patient with hip arthrodesis, corrective abduction proximal femur osteotomy and lower limb shortening

additional surgical procedure for the osteotomy and also there are no concerns or complications with healing of the osteotomy site such as non-union, infection or arthrofibrosis [20, 27].

Satisfactory short-term clinical results have been obtained after intra-articular correction of extra-articular deformity in TKA with no complications [15, 19, 23, 27]. However the deformity is not always fully corrected and non-progressive radiolucent lines can be observed [15] and further follow-up is needed to evaluate the long-term effectiveness. The mayor concern of this technique is that



**Fig. 2** (a) Anteroposterior radiograph of a right osteoarthritic knee with severe ( $25^\circ$ ) angular varus deformity of the distal femur secondary to supracondylar osteotomy and valgus deformity of the

proximal tibia secondary to proximal osteotomy. (b) Lateral radiograph showing flexion deformity of the distal femur

ligament releases can lead to instability in flexion [19, 25], although this instability is probably not a problem unless the flexion gap is big enough to create prosthesis dislocation [25]. Extra-articular deformity due to prior supracondylar varus femoral osteotomy does not affect the result of a primary TKA, although the distal femur is off-set medially on the femoral diaphysis [4].

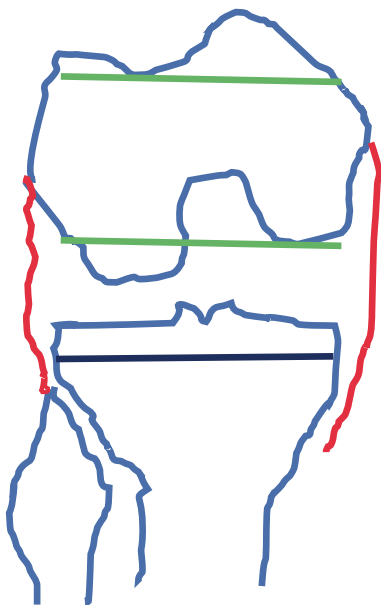
## Effects of Intra-Articular Femoral Correction

### Effects in the Coronal Plane

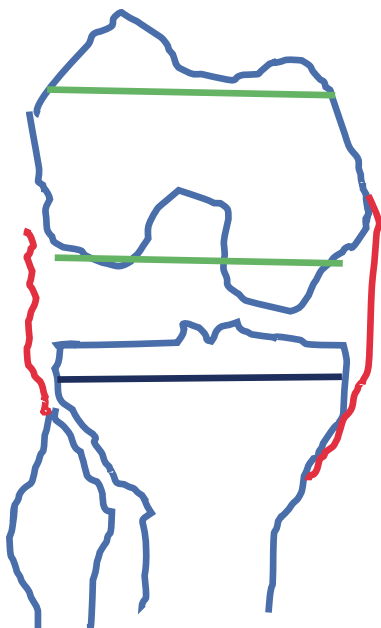
Intra-articular correction of extra-articular femoral malalignment will require an off-set and compensatory distal femoral cut, perpendicular to the mechanical axis (centre of the knee to the centre of the hip). The magnitude of the off-set will depend on the magnitude of the angular deformity and the distance of the angular deformity to the centre of the

knee [9, 10]. An angular deformity closer to the joint-line will require greater off-set than distant ones. The off-set of the distal femoral cut to correct a varus or valgus extra-articular deformity will affect the extension gap creating an asymmetric gap with a more or less lateral or medial laxity, respectively. Minor degrees of lateral laxity are better tolerated than medial laxity due to dynamic stabilization by the ilio-tibial tract, the biceps tendon, the popliteus, and the lateral head of the gastrocnemius muscle. Therefore, valgus deformities are more likely to require extra-articular correction than varus ones of similar magnitude.

Extensive medial or lateral release in extension to balance the extension gap will affect the flexion gap [22], increasing relative external or internal rotation of the femur when tensing the ligaments to balance the flexion gap. If a posterior femoral cut parallel to the proximal tibial cut is performed to compensate the asymmetry of the flexion gap, while maintaining tension in the ligaments it will produce internal or external rotation of the femoral component, respectively (Figs. 3 and 4). The rotational alignment



**Fig. 3** Extensive medial release in extension to balance the extension gap will produce asymmetry of the flexion gap due to increasing relative external rotation of the femur. If the proximal tibial cut and the balanced flexion gap method is used to compensate the asymmetry, the posterior femoral cut will produce internal rotation of the femoral component



**Fig. 4** Extensive lateral release in extension to balance the extension gap will produce asymmetry of the flexion gap due to increasing relative internal rotation of the femur. If the proximal tibial cut and the balanced flexion gap method is used to compensate the asymmetry, the posterior femoral cut will produce external rotation of the femoral component

of the femoral component affects the balance of the collateral ligaments in flexion and the tracking of the patella.

### Effects in the Sagittal Plane

Femoral flexion or extension deformity can be attenuated increasing the extension or flexion of distal femoral cut. Anterior or posterior femoral cortex notching limits the degree of flexion or extension of the distal femoral cut. Increasing the flexion or extension of distal femoral cut may produce difficulty in the use of a stemmed prosthesis, so that a short cemented stem may be necessary.

### Effects in the Transverse Plane

Intra-articular correction of internal or external rotational femoral deformity is limited because it means increasing the external or internal rotation of the femoral component and that may produce patellar mal-tracking and imbalance of the flexion gap. Changes in component rotation mean changes in the posterior femoral cut orientation that may cause asymmetry of the flexion gap. When there is an associated femoral deformity in the coronal and in the transverse plane, the change in femoral component rotation may be useful to correct the flexion gap asymmetry produced by the soft tissue releases performed to compensate the extension soft-tissue imbalance created by the off-set distal femoral cut, as has been mentioned.

## Effects of Intra-Articular Tibial Correction

### Effects in the Coronal Plane

The option of intra-articular correction of a varus or valgus extra-articular deformity in the coronal plane will depend on the amount of offset and compensatory proximal tibial cut that will be required to compensate the extra-articular mal-alignment. The proximal tibial cut will affect the extension and flexion gap in contrast with the femoral distal compensatory intra-articular cut that only will affect the extension gap.

Hungerford [9] established that the magnitude of this compensatory cut will be influenced by two factors: the absolute magnitude of the axial deformity and the distance of the deformity from the centre of the knee. A distal tibial metaphyseal deformity may have little influence on the orientation of the proximal tibial cut compared to a proximal tibial metaphyseal angular deformity.

The compensatory off-set of the proximal tibial cut to compensate an extra-articular deformity in varus or valgus will create a more or less severe lateral or medial laxity, respectively. A contra-lateral ligamentous release to compensate this laxity will affect the flexion and extension gaps and will balance the offset of the proximal tibial cut both in flexion and extension. The proximal tibial cut and the balanced flexion gap method can be used to establish the femoral component rotation. Extensive releases will alter joint line position, increase tension on the posterior cruciate ligament and change patello-femoral contact stress. A posterior stabilized (PS) or condylar constrained (CCK) TKA design is recommended to increase constraint after ligamentous releases, but if mayor instability persist a more constrained hinge prosthesis is needed.

In theory, ligament advancement to compensate the laxity created after a proximal tibial offset cut is more straightforward than after a distal femoral offset cut, because in the latter, the femoral centre of rotation may be altered; otherwise in the tibial deformity the isokinetic point for the femur is unchanged [9]. To my knowledge there are no reports of the results obtained after the use of this technique in TKA in extra-articular deformities.

A tibial deformity produces alterations at the knee and the ankle, requiring varus or valgus hind-foot alignment to compensate them. An intra-articular tibial osteotomy will correct the alignment at the knee but may leave alignment at the ankle incompletely corrected.

### Effects in the Sagittal Plane

The flexion or extension deformity can be corrected by an intra-articular proximal tibial cut performed in the opposite direction of the deformity. The limit of flexion or extension of the proximal tibial cut is established by the length of the tibial stem that can contact the posterior or anterior cortices of the tibia.

### Effects in the Transverse Plane

When the level of the rotational deformity is proximal to the anterior tibial tubercle, internal and external rotational deformities can be simply corrected by the alignment of the tibial implant with the anterior tibial tubercle. Nevertheless, when the centre of the rotational deformity is distal to the anterior tibial tubercle complete correction with an intra-articular procedure can cause mal-alignment of the extensor apparatus. In both circumstances, it is probably better to let the trial tibial component self-align to the femoral component rotation after flexing and extending the knee several times.

## Pre-Operative Planning

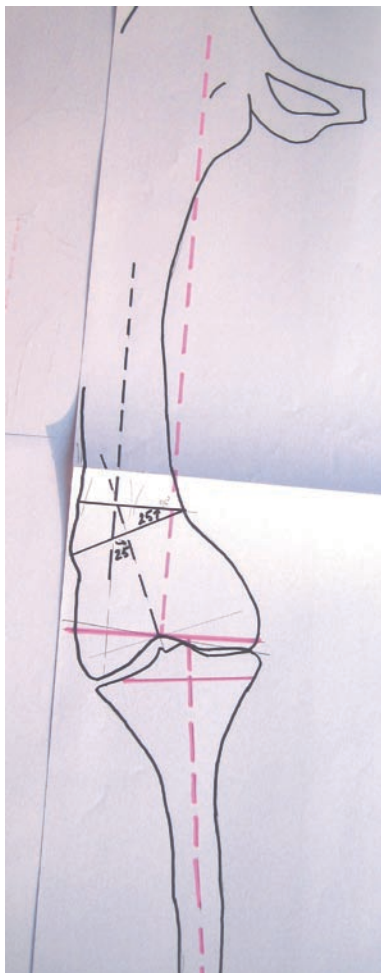
Individual evaluation of each case is necessary because of several factors affecting the type of treatment such as age, activity level, underlying diagnosis, other joint disease, quality of the skin, range of motion, and neurovascular status [20].

To plan the correction of the deformity in the coronal plane, the mechanical axis of the knee is measured on a full weight-bearing radiograph from the hip to the ankle in neutral rotation of the affected limb pre-operatively (Fig. 1). To determine the level of the corrective osteotomy at the knee associated with extra-articular deformity of the femur a line is drawn from the centre of the femoral head to the centre of the knee and a second line is drawn perpendicular to the first line through the femoral condyle on the pre-operative full-length weight-bearing radiograph. The level of resection is moved further distally to avoid the origin of the collateral ligaments but if the anticipated femoral condylar resection violates the integrity of the insertion of either the medial or the lateral collateral ligament an extra-articular corrective osteotomy is planned.

Drawings, cut-outs (Fig. 5) and templating is done to ensure proper alignment and to ensure that the femoral component will sit securely, especially the position of the intercondylar box of constrained condylar prosthesis (CCK) and the position of the stem extension when it is needed, because the box or the stem may impinge with the femoral cortex. In this situation an offset stem may have some value or a custom femoral component is needed with the appropriate stem angle.

The level of the offset proximal tibial cut to correct a tibial coronal extra-articular deformity is determined by drawing a line from the centre of the knee to the center of the ankle and a second line perpendicular to the first line is drawn through the tibial plateau. The level of resection can be moved distal to the joint line no more than 1 cm and depending on the medial or lateral resultant bone defect and the amount of soft tissue release needed to compensate the extension and flexion gap, the surgeon must decide if an extra-articular corrective osteotomy is planned.

The limit of extra-articular tibial deformity that can be corrected by an intra-articular procedure can be established by the Wang method [27]. In this method a line is drawn from mid-point of the distal part of the medullary canal of the tibia to the knee joint. If the line passes within the tibial condyle an intra-articular procedure can be planned but if not, an extra-articular osteotomy is planned. Templating helps to ensure that the tibial component and the stem sit properly. When a stem is needed in a severe deformity, an offset stem or a custom tibial stem may play a role.



**Fig. 5** Pre-operative planning of the different options to correct the femoral deformity by an extra-articular supracondylar osteotomy at the site of the angular deformity or intra-articular off-setting distal femoral cut

To plan the correction of the deformity in the sagittal plane, a non-weight-bearing lateral radiograph of the femur or tibia is done. A line is drawn in the mid-point of the medullary canal of the proximal and distal segment of the femur or tibia and the angle between these lines is measured. To determine the level of the corrective osteotomy, a line perpendicular to the line of the medullary canal is drawn through the knee joint surface and a second line is drawn, reproducing with the latter the extension or flexion deformity angle measured before in opposite directions. The flexion-extension limit of the distal femoral cut, is established by femoral template to avoid excessive anterior or posterior cortical notching and to assure perfect setting of the inter-condylar box and stem. The flexion-extension

limit of the proximal tibial cut is established by the tibial stem to avoid contact with the anterior or posterior tibial cortices.

The rotational deformity is measured on a computerized tomography scan [17]. The femoral torsion is determined by the angle formed between lines drawn in the middle of the neck of the femur with another drawn in the clinical epicondylar axis of the distal femur. The tibial torsion is determined by the angle formed by a line parallel to the posterior tibial cortex at the joint surface with a line drawn through the medial and lateral malleoli. The femoral and tibial torsion is compared with the values of the contralateral extremity whenever possible to establish the rotational deformity.

### Surgical Technique

The Surgical approach is performed through a standard mid-line incision or through a previous surgical scar close to it. Limited soft tissue flaps are developed and a medial parapatellar arthrotomy is performed to allow patellar subluxation. When subluxation is difficult proximal extension of quadriceps tendon arthrotomy combined or not with an oblique snip helps to obtain subluxation in most cases. Soft tissue balance is performed after distal femoral and proximal tibial cuts.

### Extra-Articular Correction

Corrective extra-articular osteotomy is performed prior to TKA, depending on the location of the osteotomy site. A separate, small lateral or anterior approach to the femoral or tibial diaphysis respectively will be necessary. When a supracondylar femoral or proximal metaphyseal tibial osteotomy is planned, exposure of this region is obtained by extending the knee arthrotomy proximally or distally. When a plate or a blade-plate is preferred to fix the supracondylar femoral osteotomy, frequently a lateral-based approach is necessary [14]. Bone grafting of the osteotomy should be considered [19].

Fluoroscopic assistance helps to ensure proper location and correction at the osteotomy site. Also, guided introduction and reaming of an intra-medullary nail. An intra-medullary nail or a plate, are preferred to a blade-plate or stemmed implant to fix the diaphyseal or supracondylar femoral osteotomy [14], but a plate or stemmed implant are preferred for the tibia [20, 24]. A small plate will provide additional rotational control when a stemmed implant

fixes the osteotomy [26]. If a plate is used, initial fixation is obtained with uni-cortical screws to allow the introduction of the intra-medullary jig. Otherwise, an extra-medullary alignment system is needed [14]. With a retrograde nail the intra-medullary alignment jig is introduced into the nail or a specially threaded alignment rod can be connected to the nail [11].

### Intra-Articular Correction

#### Extra-Articular Deformity of the Femur

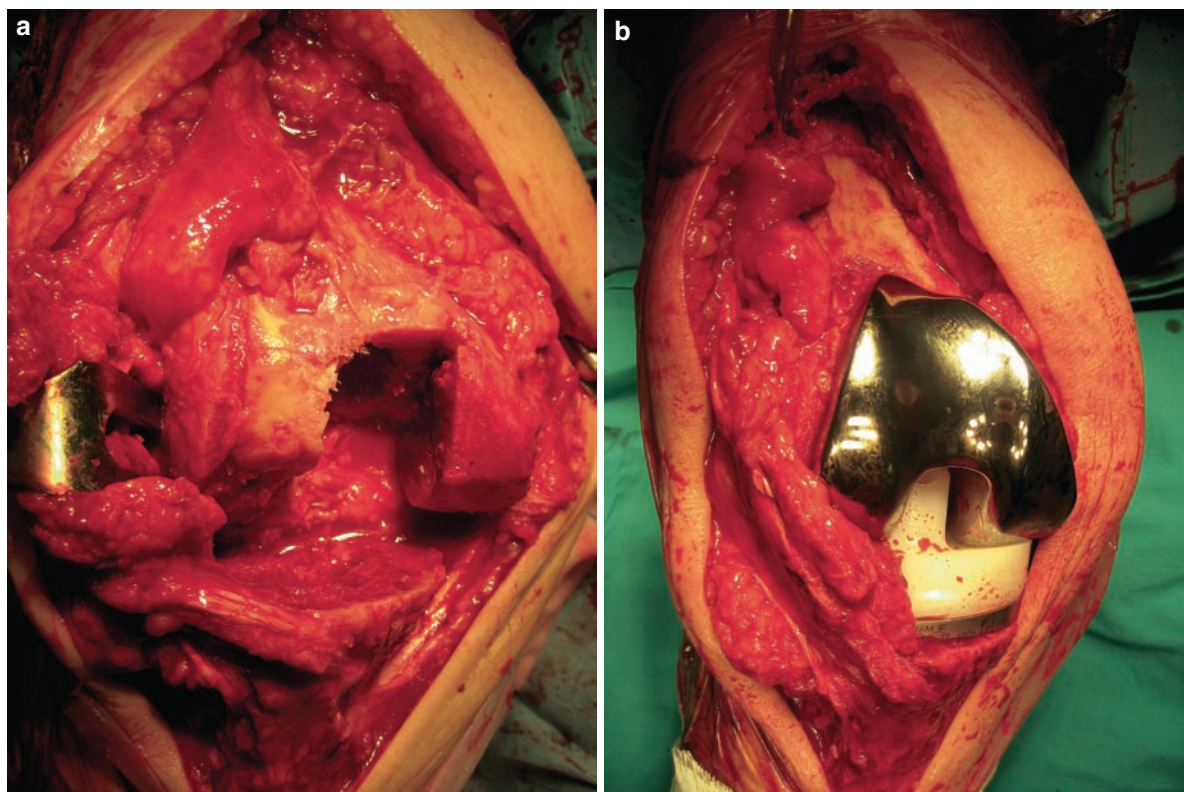
To perform the distal femoral cut, an intra-medullary guided system is preferred and can be used when the intra-medullary rod passes through the angular deformity. If this fails an extra-medullary guided system or computer-guided navigation system may be used. Passing the rod through the angular deformity is more easily accomplished when the deformity is close to the joint. The entry point of the intra-medullary guide is placed in the lateral femoral condyle for a varus deformity (Figs. 6–8) and in the medial

femoral condyle for a valgus deformity. The distal femoral cut is done perpendicular to the mechanical axis.

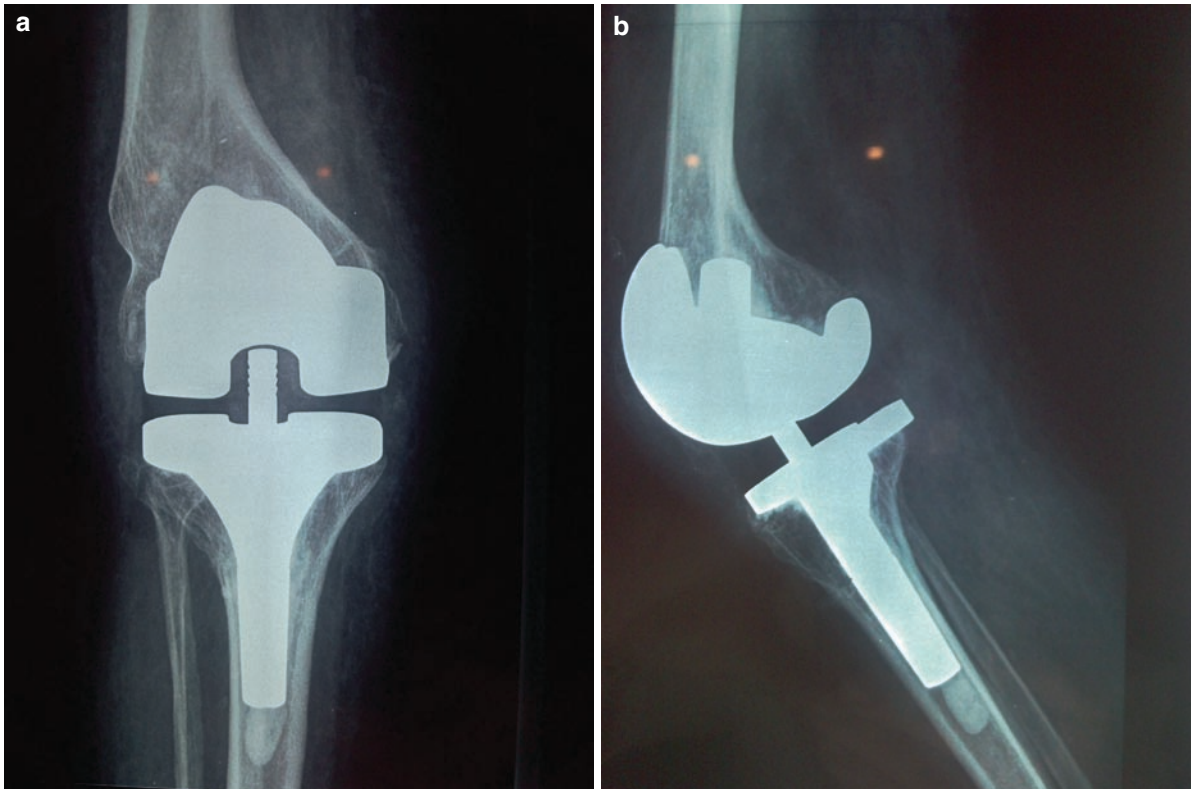
When using an extra-medullary guided system the external alignment rod is attached to the distal femoral cutting bloc and the proximal part of the guide is positioned 2 cm medial to the anterior superior iliac spine crest. It should correspond to the centre of the femoral head. A skin marker positioned previously in the centre of the femoral head under radiographic control can also be used as a reference.

#### Extra-Articular Deformity of the Tibia

The intra-medullary guided system can be used when the intra-medullary rod passes the angular deformity, this is more easily accomplished when the angular deformity is closer to the joint line; if not an extra-medullary guide or navigation system may be used. The intra-medullary rod is inserted into the lateral tibial condyle for a varus deformity and into the medial tibial condyle for a valgus deformity. The proximal tibial cut is done perpendicular to the



**Fig. 6** Intra-operative image showing autograft used to fill the bone defect created by the intramedullary guide introduced through the lateral femoral condyle (a) and the medial offset position of the femoral component (b)



**Fig. 7** Anteroposterior (a), lateral (b) radiograph after total knee arthroplasty at the latest follow-up, showing medial offset of the femoral component. Opposite intra-articular off-setting cut of

the femur and tibia compensate each other creating less soft tissue imbalance

mechanical axis and the level of resection is limited to 1 cm below the joint line (Figs. 9–11).

The remaining defect of the medial or lateral plateau is filled with a cortico-cancellous autograft from the femoral or tibial condyle or a metal augment when it is bigger than 5 mm, or with morsellized autograft. The use of a stem extension with modular tibial augments may not be possible because of the tibial deformity. If there are difficulties with the short stem of the tibial tray an all-polyethylene tibial component allows shaving of the stem to fit the tibial canal [15].

Reverse tibial flip autograft technique [18] has been described to correct the extra-articular valgus knee secondary to overcorrected high tibial osteotomy by an intra-articular technique. It increases the lateral compartment with a wedge of bone resected from the medial tibia and then flipped. The flipped bone wedge is fixed provisionally with K-wires during the TKA and finally, the tibial plate and the cement provide a strong fixation for the wedge autograft. The lateral soft tissue tightens after wedge transplantation

hence lateral release is performed to balance soft tissue. This technique is only indicated for knees in which the angle of the tibial shaft to the tibial plateau is more than 15° because harvesting a small bone wedge of less than 8 mm thick is technically very difficult.

### Soft-Tissue Balancing

The offsetting of the distal femoral cut creates soft-tissue imbalance only in the extension gap, in contrast to the offsetting proximal tibial cut that creates extension and flexion gap imbalance. Extensive releases required to compensate the imbalance will affect extension and flexion gaps [22]. In extra-articular femoral deformity medial or lateral releases performed to compensate the extension gap, cause asymmetry in the flexion gap when the ligaments are tensed, increasing femoral external or internal rotation, respectively. If the epicondylar axis or anteroposterior troclear line is used for determining femoral





**Fig. 8** Full-length standing radiograph after total knee arthroplasty demonstrating improvement of the alignment of the lower extremity

component rotation, asymmetry of the flexion gap is maintained causing flexion instability. If the long axis of the tibia or the proximal tibial cut are used to determine the femoral component rotation while tensing the flexion gap, a symmetric flexion gap will be obtained but excessive and abnormal femoral component rotation may result. Careful evaluation of femoral component rotation is necessary to avoid patellar mal-tracking and to equalize the flexion gap. Depending on the magnitude and complexity of the soft-tissue imbalance the surgeon must decide whether to solve it by soft tissue reconstruction or by a constrained prosthesis.

In extra-articular deformity of the tibia the offsetting proximal tibial cut affects the flexion and extension gap and soft tissue releases will compensate both gaps without the necessity to change femoral component rotation.

In a varus deformity where more lateral femoral or tibial condyle would have to be removed, it will result in a relative lengthening of the lateral soft-tissue structures. To compensate this, an extensive medial release will be required that includes meticulous sub-periosteal release of



**Fig. 9** Full-length standing radiograph of the lower extremity of a 63-year-old man with a left valgus deformity post tibial fracture mal-union

the deep and superficial medial collateral ligament and pes anserinus tendons from their tibial insertions. The semi-membranosus and posterior capsule may be also released from the postero-medial corner [7].

In a valgus deformity the resection of more medial femoral or tibial condyle produces a relative lengthening of the medial structures that will require extensive lateral release to balance the extension gap. Lateral release includes sequential lengthening of the postero-lateral aspect of the capsule and fascia lata, lateral collateral ligament and popliteus tendon [7].

Femoral chamfer cuts are made once correct alignment and soft-tissue balance have been obtained and trial



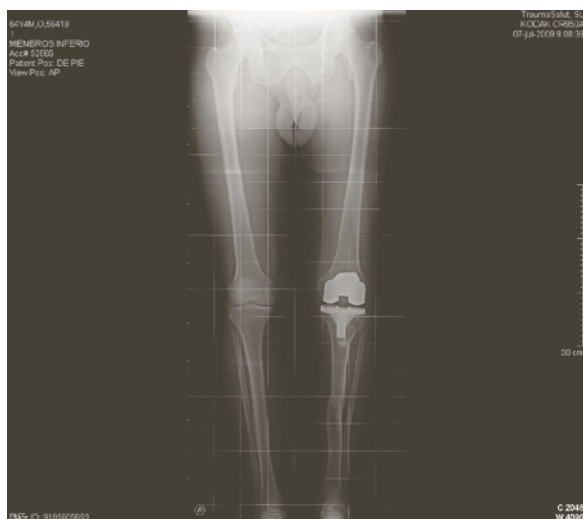
**Fig. 10** Anteroposterior (a), lateral (b) and axial (c) radiograph showing a well aligned total knee arthroplasty without radiolucencies after 2 year follow-up

components are inserted. If augmentation or increased constraint is necessary, it is preferable to use a stemmed component. This may not be possible with a severe deformity unless a custom stem has been made pre-operatively. If it is not available, you may choose to cement the component without a stem extension or with a shorter stem extension.

Wound closure can be difficult after correcting a severe deformity and to avoid skin tension pre-operative plastic surgery advice will be useful, especially in the multi-scarred knee. It may be necessary to consider using a pre-operative tissue expander or gastrocnemius rotational flap.

## The Role of Navigation

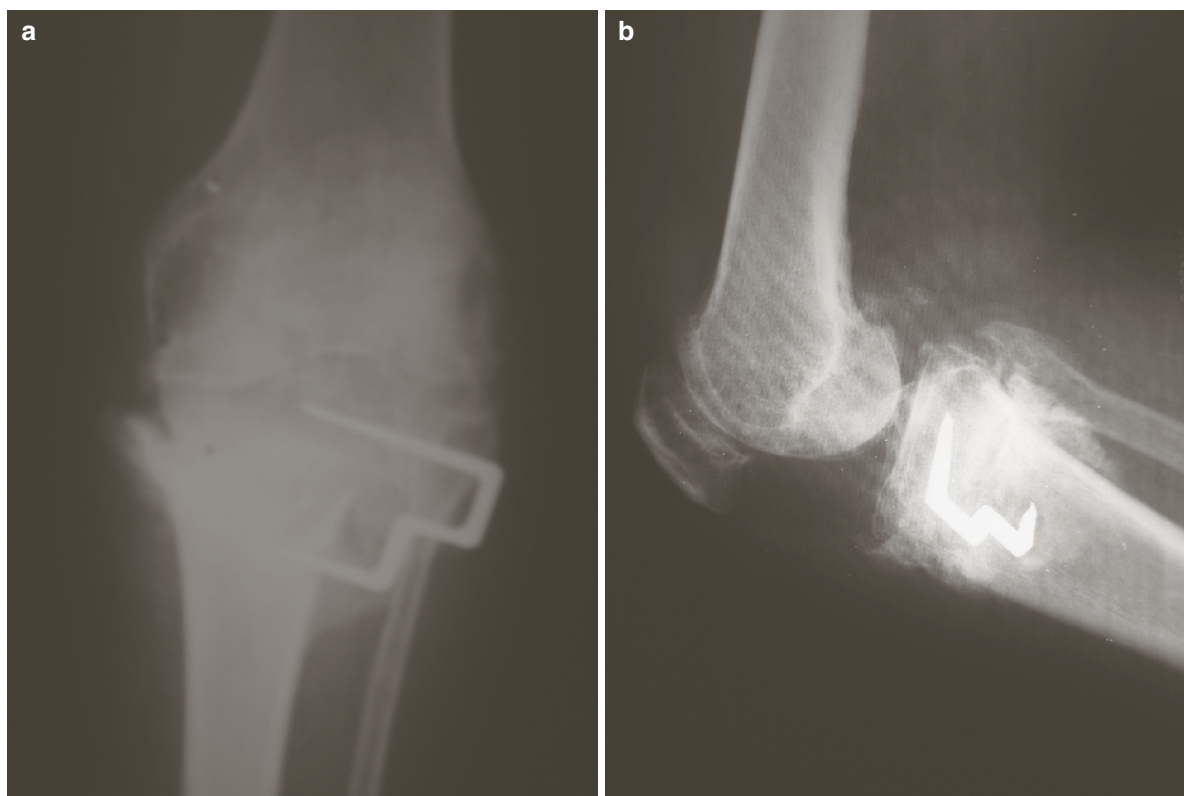
Navigated TKA has demonstrated better alignment than does the conventional technique in coronal and sagittal planes [1]. The computer-assisted navigation system directly measures the mechanical axis irrespective of local bone morphology or deformity and allows the performance of an accurate offset femoral or tibial cut perpendicular to the mechanical axis (Figs. 12 and 13). A navigation system permits better adjustment of the desired flexion or extension in the distal femoral or proximal tibial cut than the



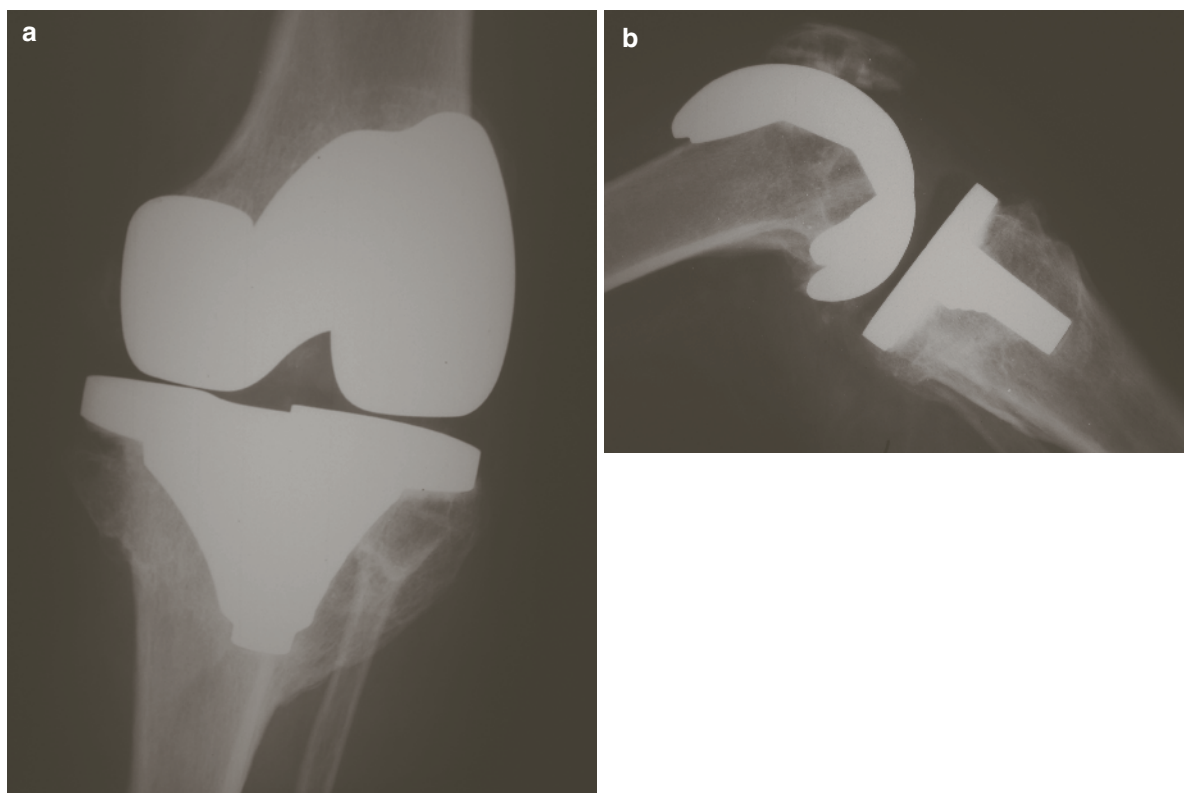
**Fig. 11** Improvement of the mechanical axis after total knee arthroplasty. The mechanical axis of the lower extremity passes 3 mm from the centre of the knee

conventional technique. No better accuracy has been proven in rotational alignment with navigation compared to conventional technique, and some authors [2, 6] prefer to base the femoral rotation on balancing the flexion gap to the tibial cut surface instead of the epicondylar or troclear antero-posterior axis that can be altered due to rotational mal-alignment. Navigation can also be used in second stage, after corrective osteotomy at the site of the deformity, avoiding the need for intra-medullary nail or plate extraction.

Comparable accuracy in coronal alignment can be obtained with navigation compared with osteotomy at the site of the deformity and in intra-articular bone resection with a standard guide approach [2, 6, 8, 13, 21]. Navigation is a feasible and less-invasive alternative method for TKA surgery in extra-articular deformities that obtains good and reproducible clinical results without increasing complications and with the potential advantage of less blood loss [12] and decreased risk of fat embolism [3].



**Fig. 12** (a) Anteroposterior radiograph of a 58-year-old woman with a failed high tibial osteotomy and important lateral shift of the tibial plateau. (b) Lateral radiograph showing inversion of the proximal tibial tilt



**Fig. 13** (a) Anteroposterior radiograph of a total knee arthroplasty performed using a navigation-guided system that permits accurate proximal tibial cut perpendicular to the mechanical axis. (b) Lateral radiograph showing correction of the tibial tilt

## Conclusions

Extra-articular femoral or tibial deformity increases technical difficulties in TKA and may adversely affect the results. This deformity can be addressed with an extra-articular correctional osteotomy performed prior to or at the time of primary TKA or with an intra-articular offset bone resection and soft-tissue balance. Careful pre-operative planning is mandatory on an individual basis and will obviate potential problems. Intra-articular correction may be appropriate when the insertion of the collateral ligament is not violated by the intra-articular bone resection and when the resultant bone defect and soft tissue imbalance may be solved. Most complex deformities of great magnitude or those located close to the joint line or multi-planar, may require an extra-articular corrective osteotomy.

Femoral intra-articular offset cuts affect the extension gap whilst, in contrast, tibial offset cuts affect the extension and flexion gap. Ligament releases performed to balance the extension gap after a femoral intra-articular offset cut produce asymmetry in the flexion gap; if it is balanced with the

posterior femoral cut; it may produce abnormal femoral component rotation. Nevertheless, ligament releases performed after the offsetting proximal tibial cut balance the flexion and extension gap. Navigation systems directly measure the mechanical axis irrespective of local bone morphology and are less invasive.

Satisfactory short-term clinical results have been obtained after intra-articular and extra-articular correction of extra-articular deformity in TKA, but complication rates increase with extra-articular correction. However, by an intra-articular technique further follow-up is needed to evaluate the long-term effectiveness of both methods.

## References

1. Anderson KC, Buehler KC, Markel DC (2005) Computer assisted navigation in total knee arthroplasty: comparison with conventional methods. *J Arthroplasty* 20:132–138
2. Bottros J, Klika AK, Lee HH et al (2008) The use of navigation in total knee arthroplasty for patients with extra-articular deformity. *J Arthroplasty* 23:74–78

3. Caillouette JT, Anzel SH (1990) Fat embolism syndrome following the intramedullary guide in total knee arthroplasty. *Clin Orthop* 251:198–199
4. Cameron HU, Park YS (1997) Total knee replacement after supracondylar femoral osteotomy. *Am J Knee Surg* 10:70–71
5. Cameron HU, Welsh RP (1988) Potential complications of total knee replacement following tibial osteotomy. *Ortho Rev* 17:39–43
6. Chou W-Y, Ko J-Y, Wang Ch-J et al (2008) Navigation-assisted total knee arthroplasty for a knee with malunion of the distal femur. *J Arthroplasty* 23:1239e 13–19
7. Cuckler JM (2007) Correcting extra-articular deformity of the knee: acting in tandem. *Orthopedics* 30(9):774–776
8. Fehring TK, Mason JB, Moskal J et al (2006) When computer-assisted knee replacement is the best alternative. *Clin Orthop* 452:132–136
9. Hungerford DS (2002) Effect of extra-articular deformity on the tibial component. In: Scuderi GR, Tria AJ (eds) *Surgical techniques in total knee arthroplasty*. Springer, New York
10. Hungerford DS (2002) The effect of extra-articular deformity on the femoral component. In: Scuderi GR, Tria AJ (eds) *Surgical techniques in total knee arthroplasty*. Springer, New York
11. Incavo SJ, Kapadia C, Torney R (2007) Use of an intramedullary nail for correction of femoral deformities combined with total knee arthroplasty. A technical tip. *J Arthroplasty* 22:133–135
12. Kalairajah Y, Simpson D, Cossey AJ et al (2005) Blood loss after total knee replacement: effects of computer-assisted surgery. *J Bone Joint Surg* 87-B:1480–1482
13. Klein GR, Austin MS, Smith EB et al (2006) Total knee arthroplasty using computer-assisted navigation in patients with deformities of the femur and tibia. *J Arthroplasty* 21:284–288
14. Lonner JH, Siliski JM, Lotke PA (2000) Simultaneous femoral osteotomy and total knee arthroplasty for treatment of osteoarthritis associated with severe extra-articular deformity. *J Bone Joint Surg* 70-A:547–555
15. Mann JW III, Scuderi GR, Insall JN (2002) Total knee replacement with associated extra-articular angular deformity of the femur. In: Scuderi GR, Tria AJ (eds) *Surgical techniques in total knee arthroplasty*. Springer, New York
16. Mann JW, Insall JN, Scudery GR (1997) Total knee arthroplasty in patients with associated extra-articular angular deformity. *Orthop Trans* 21:59
17. Moussa M (1994) Rotational malalignment and femoral torsion in osteo-arthritic knees with patellofemoral joint involvement: a CT scan study. *Clin Orthop* 304:176–183
18. Naguno A, Ishibashi Y, Tesuda E et al (2006) A reversed tibial flip autograft technique for correcting over-valgus knee after high tibial closing-wedge osteotomy in total knee arthroplasty. *J Arthroplasty* 21:771–774
19. Papadopoulos EC, Parvizi J, Lai CH et al (2002) Total knee arthroplasty following prior femoral distal femoral fracture. *Knee* 9:267–274
20. Paspagelopoulos PJ, Karachalios T, Themistocleous GS et al (2007) Total knee arthroplasty in patients with pre-existing fracture deformity. *Orthopedics* 30:373–378
21. Patai J, Janositz G, Mécs L et al (2007) Navigated total knee arthroplasty in a patient with severe diaphyseal deformities. *Acta Orthop Bel* 73:536–540
22. Peters CL, Mohr RA, Bachus KN (2001) Primary total knee arthroplasty in the valgus knee. Creating a balanced soft tissue envelope. *J Arthroplasty* 16:721–729
23. Rand JA, Franco MG (1991) Revision considerations for fractures about the knee. In: Goldberg V (ed) *Controversies of total knee arthroplasty*. Raven Press, New York
24. Radke S, Radke J (2002) Total knee arthroplasty in combination with a one-stage tibial osteotomy: a technique for correction of a gonarthrosis with severe (>15°) tibial extra-articular deformity. *J Arthroplasty* 17:533–537
25. Ritter MA (2003) Total knee replacement following extra-articular deformities. *Orthopedics* 26:969–970
26. Vince KG, Cameron HU, Hungerford DS et al (2005) What would you do? Case challenges in knee surgery. *J Arthroplasty* 20:44–50
27. Wang J-W, Wang Ch-J (2002) Total knee arthroplasty for arthritis of the knee with extra-articular deformity. *J Bone Jt Surg* 84-A:1769–1774
28. Wolf AM, Hungerford DS, Pepe CL (1991) The effect of extra-articular varus and valgus deformity on total knee arthroplasty. *Clin Orthop* 271:135–151
29. Yagi K, Matsui Y, Nakano S et al (2006) Treatment of knee osteoarthritis associated with extraarticular varus deformity of the femur: staged total knee arthroplasty following corrective osteotomy. *J Orthop Sci* 11:386–389

# Unicompartmental Knee Arthroplasty

Simon Thomas Donell

## Articular Cartilage

All biological tissues contain matrix, cells, and water. In articular cartilage the matrix is a complex structure within which the chondrocytes are distributed in a recognisable pattern. The matrix contains principally type II collagen bundles with attached and unattached proteoglycan complexes. The latter contain aggrecan (proteoglycan) and glycosaminoglycan (GAG). Aggrecan comprises a hyaluronan molecule which is linked through keratan and chondroitin sulphate molecules to form a series of “bottle-brushes” on either side of the hyaluronan chain. Aggrecan is extremely hydrophilic, attracting water. This increases tissue tension and the water is contained by the large-diameter collagen bundles. The arrangement of the bundles is shown schematically in Fig. 1 [8].

Because articular cartilage is under continuous load it has neither a nerve supply nor blood supply. The lack of these means that there is no perception of injury, and limited capacity for repair. Nutrients are gained from the synovial fluid and enter through the fluid fluxes that occur under load. As a result the chondrocytes can be viable even after death. The cells make up 5% of the volume, 70% is water, and 25% organic matrix. The cells are interspersed in the matrix and have territorial areas around within which they react chemically. Because they are sparse there are interterritorial areas between the cells that are independent of direct cellular control.

Cartilage homeostasis depends on the balance between synthesis of the protein molecules stimulated by growth factors, and the degradation by cytokines by the production of enzymes such as collagenases. Homeostasis also depends

on the synovial membrane and the properties of synovial fluid.

The main functions of articular cartilage are to distribute loads evenly across the joint to reduce stress, and to allow movement between the opposing surfaces with the minimum of friction and wear. The complex structure and the way that water moves in and through it means that articular cartilage is viscoelastic, like a water-soaked sponge. The water moves when a pressure gradient under load is applied. It behaves differently depending on the loading conditions; known as anisotropy. Since the loads differ throughout the articular cartilage (see Fig. 2) the resulting mechanical behaviour varies depending on the depth. When normal, articular cartilage can withstand loads of 10,000 N. But this diminishes when there is proteoglycan loss and even more so when the collagen network is damaged.

When a biomechanical insult is applied to a chondrocyte it releases degrading enzymes that remove aggrecans and collagen II. This results in fragments forming that leach into the synovial fluid. This in turn mounts an inflammatory response from the synovial fibroblasts that includes metalloproteinases. These further destroy the articular cartilage. Wear debris also has a direct effect through wear.

The articular cartilage damaged may be classified as:

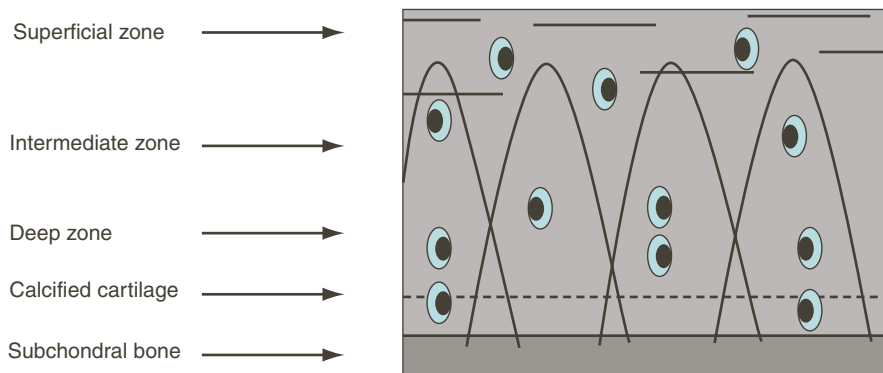
- Partial thickness
- Full thickness (Focal)
  - Chondral
  - Osteochondral
- Degenerate
  - Ageing
- Arthritis

Only if there is bleeding through the subchondral bone plate can fibroblasts and therefore scar tissue form. It follows from this that lost bearing surface is not naturally replaced. This loss can alter the mechanical alignment adversely leading to further loss. The problem then is that

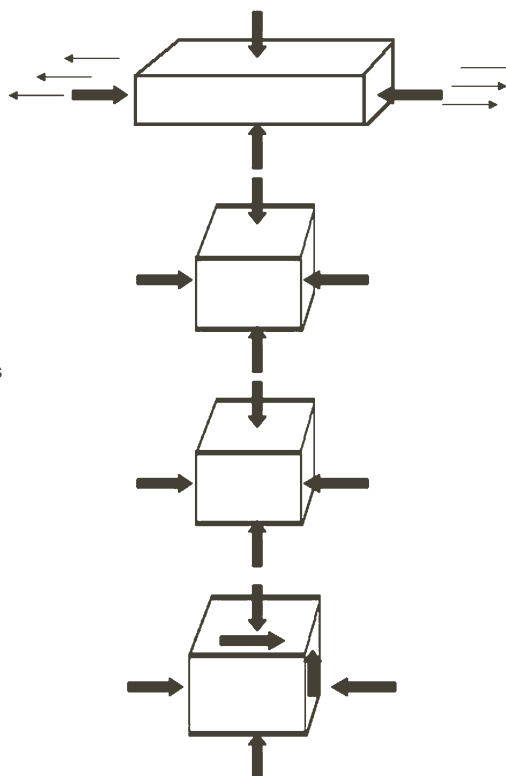
---

S.T. Donell  
Norfolk and Norwich University Hospital,  
Colney Lane, Colney, Norwich NR4 7UY, UK  
e-mail: simon.donell@nnuh.nhs.uk

**Fig. 1** Schematic of the structure of articular cartilage



- Superficial zone
  - High flow In/Out
  - Fluid pressure/tension
  - Large compressive strains
  - Tensile surface strains
- Intermediate zone
  - Low fluid flow
  - Mainly fluid pressure
  - Moderate compressive strains
- Deep zone
  - Little fluid flow
  - Fluid pressure
  - Small compressive strains
- Calcified cartilage
  - No fluid flow
  - Fluid pressure
  - No compressive strains
  - Interface shear strains



**Fig. 2** Schematic of the in vivo mechanical behaviour of articular cartilage

both the mechanical and biological environments may need correcting in symptomatic patients.

- Direct trauma (chondral or osteochondral damage)
- Osteochondritis dissecans
- Osteonecrosis
- Meniscectomy
- Malalignment
- Dysplasia (e.g. trochlear dysplasia in the patellofemoral joint)

**The Knee**

The knee is composed of three compartments; medial and lateral tibiofemoral, and patellofemoral. Focal or isolated unicompartmental articular cartilage loss can occur from:

Where there is an inflammatory arthropathy, or the primary problem is biological rather than mechanical, then the articular cartilage loss is likely to extend beyond one

compartment to all three. Progression of osteoarthritis is well-recognised where there is instability secondary to ligament rupture, most notable with the ACL.

Loss of the bearing surface in the knee can be replaced by biological material such as osteochondral grafts, or autologous chondrocyte implantation, or with artificial materials (metal and plastic). Total knee replacement is the treatment of choice where the loss is beyond one compartment or likely to progress in the future. Where the loss is focal then unicompartmental knee replacement (UKR) can be undertaken provided one assumes that:

- The ligaments and soft tissues are intact.
- The UKR replaces only the missing bearing surface so that the tissue tensions are restored to normal.
- The remaining articular cartilage behaves normally biologically because the mechanical environment has been restored to normal.

---

## Unicompartmental Knee Replacement

### Tibiofemoral

#### History

Initial pioneers of UKR were Marmor in the USA and Cartier in France in the 1970s and 1980s [4]. Marmor defined the minimum thickness of polyethylene at 6 mm. Initially the results were very variable with poor results from wrong patient selection and imperfect surgical technique, although these were not specifically recognised initially. For instance, Insall overcorrected the medial UKRs and undercorrected the lateral ones [7]. More recently a distinction has been made between early and late failures with the late failures (over 10 years) being due to polyethylene wear. Many series published in the literature have reported survivorship of over 90% [1–3, 5, 9, 13, 14, 16, 17].

#### Indications

UKR should be considered in the younger active and older sedentary patients with osteoarthritis confined to one tibiofemoral compartment. The ligaments must be intact. In these conditions the deformity will be correctable on stressing, there will be no excessive fixed flexion deformity, and the patient will flex beyond 90°. There is some debate about how active the patient can be, and how heavy, although most are reluctant to perform a UKR on someone with a BMI above 35.

### Introduction

Tibiofemoral UKRs may have a number of different designs. They may be fixed-bearing or mobile-bearing. The fixed-bearing may be metal-backed or all polyethylene. The femoral component may be resurfacing or resection. The tibial component may be different for a lateral UKR compared to a medial UKR. UKR is an attractive technique for managing unicompartmental arthritis of the knee since it can be performed through a minimal access approach which allows for a shorter inpatient stay. The recovery is quicker and the complications rates lower than a total knee replacement (TKR). However two points need to be considered:

- Does the UKR have the same functional results and survival rates as a TKR?
- Is the UKR selected as an alternative to a TKR or to an osteotomy (upper tibial or distal femoral)?

If the latter then the outcomes in terms of function and longevity are less strict than those for a TKR. However a UKR runs the risk of disease progression in the other compartments that does not occur with a TKR. A recent systematic review recommended that the short- and long-term function and longevity of all these treatments need to be assessed in order to evaluate the success of UKA in comparison to HTO and TKA for the treatment of unicompartmental OA [10].

As with TKR, UKR also runs the risk of long-term polyethylene wear. This is well-recognised if the polyethylene is point- or line-loaded due to incongruity between the femoral metal component and the upper surface of the tibial insert. In the 1980s, Goodfellow and O'Connor addressed this problem by developing the Oxford UKR (Biomet, Swindon, UK) with a spherical femoral surface and a flat tibial surface a mobile polyethylene bearing was designed for perfect congruence. Not only should this minimise wear it also should allow normal kinematics. This is because the bearing can behave like the meniscus by moving forward and backwards during knee motion. The Oxford UKR is the most popular in the UK with reported excellent long-term results of 95% survivorship rates at 15 years [1, 12, 15] without significant polyethylene wear and component loosening. These results support Goodfellow & O'Connor's concept of perfect congruence minimising wear of the polyethylene (Fig. 3).

However, the Oxford UKR's results, particularly early failure rates, depend on surgical experience. This has been borne out by auditing the results from our own unit. Although in experienced hands the results can be remarkable, it is not the implant of choice for the occasional UKR surgeon. The most notable failure is bearing dislocation.

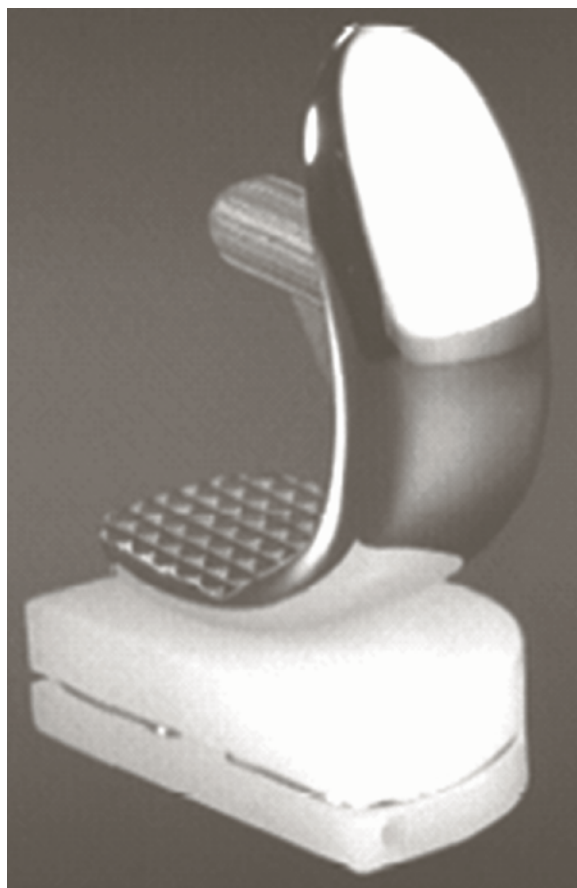




**Fig. 3** The Oxford mobile-bearing UKR

This is typically due to an imbalance between the flexion and extension gaps. Early failure is almost always due to surgical error. In addition, on the lateral side, the tibiofemoral joint is much more mobile naturally. Lateral mobile UKR is especially prone to bearing dislocation. It also requires a different technique to achieve balance since wear of the posterior condyle means that it cannot be used to define the flexion gap joint line. Fixed bearing designs therefore can be easier to insert. However they run a greater risk of bearing failure in the medium- to long-term.

The fixed bearing design with which I am familiar is the HLS (Tornier, Grenoble, France) UKR. This is a resurfacing design where there is no milling of the femoral extension surface as only the missing articular cartilage is replaced. It also has an all-polyethylene tibial component (Fig. 4). The 10-year survivorship outcome has been reported as 94%. The femoral component is polycentric and the tibial component is flat. Partially-congruent designs fail from early wear and it is surprising that a flat design is better. The reason for this is that the point of contact of the femoral component on the tibial surface is impossible to predict intra-operatively. After implantation of the components, the femur rests on the flat tibia as the soft tissues allow. Subsequent mobilisation results in creep of the plastic in the contact area of the polyethylene. This in turn leads to congruence and thus, theoretically, minimises



**Fig. 4** Uni-HLS Evolution® prosthesis, Tornier

wear. Survivorship analysis based on component loosening and revision showed a 99% survival for the meniscal-bearing implant and 93% survival for the fixed-bearing implant at 11 years [6, 13]. However although the 10-year outcomes are similar for the Oxford UKR and the HLS Tornier, this may not be the case at 20-years when polyethylene wear may become apparent in the fixed-bearing design. Despite this, since a fixed-bearing cannot dislocate, this design may have advantages for surgeons with a lower volume practice.

## Principles

### Soft Tissues

There is no intrinsic stability in any UKR design. Therefore everything depends on the knee ligaments being intact, notably the anterior cruciate ligament. Therefore any intra-articular deformity secondary to ligament insufficiency, such

as lateral translation in genu varum, is a contra-indication to UKR. This is assessed by screening radiographs.

### Bony Alignment

In addition the UKR cannot be used to correct an extra-articular malalignment, unlike a TKR. Its purpose is only to replace the missing bearing surface and restore the patient to their original structural alignment, whether that is varus, valgus, or neutral. This means that a patient with a pre-existing extra-articular varus is corrected back to that original varus. This is not an “undercorrection”, which is a misnomer and comes from considering the principles of TKR. Using this term leads to confusion and should be avoided when discussing UKR. The problem is usually “over-correction” in an attempt to correct alignment as for a TKR. This results in overload of the opposite compartment which leads to progression of the osteoarthritis.

The axial alignment is varus for medial UKAs and valgus for lateral UKAs; although patients with no structural bone deformity may have final alignment of 180° without this being overcorrection. Conversely, a post-operative mechanical femorotibial angle of 178° in a patient whose structural alignment was originally 172° (before wear) is a case of overcorrection by 6°. To avoid this type of error, the ligaments on the concave side must never be released. These ligaments are the sole reference for defining the thickness of the bearing. The purpose of UKR is to achieve proper ligament balance without excessive tension (or even with 2 or 3 mm residual laxity as a safety margin). This reference ensures proper correction for the wear in relation to the patient’s own anatomy. The need to keep a safety margin of 2 or 3 mm laxity is to avoid any overcorrection.

### Restoration of the Joint Line

Although the implant is a filler in the tibio-femoral joint space, it is important to restore the prosthetic joint line to that of the original in both flexion and extension, and to keep its correct orientation (slight varus and posteriorly-sloped). This requires knowledge of the thicknesses of the implant being used, and an understanding of what is the reference point when deciding on the bony cuts. At least 6 mm of polyethylene is needed (less for the fully congruent Oxford) plus the thicknesses of the metal. To obtain the correct joint level in extension the distal femur must be replaced by metal of the same thickness as any cartilage and/or bone loss. The depth of the tibial resection depends on the total implant thickness (plus 1–2 mm. to allow for some ligament laxity).

### Medial Tibiofemoral UKR

The tibia is usually cut first. Since the arthritis has an antero-medial pattern of wear the posterior tibial articular cartilage is intact. This can then act as the reference for the joint line. In flexion the posterior femoral condylar cartilage is also intact and so removing the amount of posterior condyle to match the thickness of the metal on the posterior condyle of the femoral component defines the joint level accurately. However intra-operatively it can be difficult to define the posterior tibial slope because of the anterior wear. The slope should follow the line of the medial meniscal rim. If the flexion gap is first defined then balance of the extension gap is referenced from the tibia, and equals the pre-determined flexion space with implants plus some residual laxity. The distal femoral cut can be made.

Alternatively the distal femur can be used as a reference, when there has been cartilage but not bony loss. The distal femur is the reference in extension, with valgus correction. From this the amount of tibia to be cut is defined by the total implant thickness plus 1–2 mm. for laxity. The tibia is then cut, and the flexion gap defined from this.

The danger is overcutting the tibia. The surface area diminishes as the cut is made more distal. This significantly affects the load and increases the risk of tibial implant failure. This can occur when residual articular cartilage is present on the femur, and has not been removed before referencing.

The femoral component needs to be aligned with the femoral condyles and the anatomical axis of the femur. In fixed-bearing designs malrotation can lead to edge loading on the polyethylene and therefore increased wear. The spherical design of the Oxford avoids this problem. Malrotation can also cause an overhang and lead to soft tissue impingement and subsequent pain. The tibial component needs to rest on the posterior and medial cortex of the tibial plateau otherwise it is liable to sink under load.

### Lateral Tibiofemoral UKR

The lateral UKR is different from the medial in a number of respects. The lateral plateau is smaller and domed. In flexion the femur naturally glides off the back. The flexion gap is normally larger than the extension gap opening up in flexion and valgus (as in the figure-four position). The patella is difficult to retract in a minimal access approach. The sagittal alignment of the femoral component when lined up with the centre of the hip in flexion appears malrotated, and the tibial component sagittal alignment is best achieved by a cut made through the patella ligament.

Because of the laxity in flexion there is a much greater risk of overstuffing the space. However there is also the problem

of lateral condylar hypoplasia and that lateral tibiofemoral osteoarthritis is a disease in flexion with the wear occurring in the posterior condyles. It is therefore very difficult to define the flexion gap properly at operation, and therefore restore the joint line correctly. Mobile-bearings are at particular risk of dislocating. However the results of lateral UKR are often better than medial UKR provided there are no technical errors.

### Conclusion

Tibiofemoral UKR can be an extremely satisfying procedure with very happy patients. When it fails it tends to be within the first 2 years. Early failure is usually due to surgical error i.e. poor patient selection, or poor surgical technique. The best results occur with surgeons who perform UKR regularly. Failures at around 5 years tend to be because of disease progression. By 10 years the survival rates match those of TKR. The complication rates are much lower in UKR than TKR. Revision is also easier.

### Patellofemoral Joint

The patellofemoral joint needs to be considered when undertaking tibiofemoral UKR. It may be replaced in its own right, and is technically another UKR. However here we are considering only whether disease in this compartment precludes UKR in the tibiofemoral joint. Most authors consider patellofemoral arthritis as a contra-indication to UKR. In medial tibiofemoral osteoarthritis with anterior wear, patellofemoral chondral lesions are common and include exposed bone. If they involve the medial patellofemoral compartment, then re-alignment by a medial UKR can be expected to alter the patellofemoral joint alignment and offload the affected area. The lesions can even affect the medial ridge of the patella and the trochlear groove without affecting the clinical result [9]. In the Swedish Arthroplasty registry only one patient out of 50 revised was for patellofemoral disease [11]. In patients with severe changes including bone loss and overhanging osteophytes then almost all would agree that this would be a contra-indication, but mild changes can probably be ignored.

### Concluding Remarks

Unicompartmental knee replacement is a worthwhile procedure in selected patients. It replaces the missing bearing surface only, and in the tibiofemoral joint should not be

used to correct extra-articular alignment. When successful it can allow patients to return to a near-normal existence for their age. Caution needs to be taken though as a number have early failures and some (particularly the patellofemoral replacements) have persistent pain. Revising when there is no obvious reason to a TKR frequently does not improve the symptoms. In my experience beware the overweight female in her fifties as they have a much higher dissatisfaction rate than any other group. My strategy then is to insist on loss of weight before considering UKR (or even TKR). The principle is that, by replacing the missing bearing and keeping the correct alignment of the knee, the mechanical environment for the remaining articular cartilage is restored, thus preserving its normal homeostasis.

### References

1. Ashraf T, Newman JH, Evans RL et al (2002) Lateral unicompartmental knee replacement. Survivorship and clinical experience over 21 years. *J Bone Joint Surg Br* 84-B: 1126–1130
2. Berger R, Meneghini RM, Jacobs JJ (2005) Results of unicompartmental arthroplasty at a minimum of 10 year follow-up. *J Bone Joint Surg Am* 87-A:999–1006
3. Berger R, Nedeff D, Barden R et al (1999) Unicompartmental Knee Arthroplasty: clinical experience at 6 to 10 year follow-up. *Clin Orthop* 367:50
4. Cartier P, Khefacha A (2005) Fixed-bearing unicompartmental knee arthroplasty. In: Bellemans J, Ries M, Victor JMK (eds) *Total knee arthroplasty: a guide to better performance*. Springer, Berlin, pp 317–321
5. Cartier P, Sanouillet JL, Grelsamer RP (1996) Unicompartmental knee arthroplasty after 10-year minimum follow-up period. *J Arthroplasty* 11:782–788
6. Deschamps G (2005) Results at 6 years minimum follow-up of a continuous series of 122 HLS Uni. Communication ISAKOS meeting, Miami, FL
7. Deshmukh RV, Scott RD (2001) Unicompartmental knee arthroplasty: long-term results. *Clin Orthop* 392:272–278
8. Donell ST (2006) Alternatives to knee replacement in unicompartmental disease. In: *La Prothèse du Genou. 12èmes Journées Lyonnaises de Chirurgie du Genou*. Sauramps Médical, Montpellier France, pp 341–347
9. Goodfellow J, O' Connor J, Dodd C, Murray D (eds) (2006) *Unicompartmental arthroplasty with the Oxford knee*. Oxford University Press, USA
10. Griffin TT et al (2005) Unicompartmental knee arthroplasty for unicompartmental osteoarthritis: a systematic review. ASERNIP-S Report No. 44. Adelaide, ASERNIP-S, South Australia
11. Lewold S, Goodman S, Knutson K, Robertsson O, Lidgren L (1995) Oxford meniscal bearing knee versus the Marmor knee in unicompartmental arthroplasty for arthrosis. A Swedish multicenter survival study. *J Arthroplasty* 10:722–731

12. Murray DG (2005) Mobile-bearing unicompartmental knee arthroplasty. In: Bellemans J, Ries M, Victor JMK (eds) *Total knee arthroplasty: a guide to better performance*. Springer, Berlin, pp 322–325
13. Murray DW, Goodfellow JW, O'Connor JJ (1998) The Oxford medial unicompartmental arthroplasty. A ten year survival study. *J Bone Joint Surg Br* 80-B:983–989
14. Pennington DW, Swienckowski JJ, Lutes WB et al (2006) Lateral unicompartmental knee arthroplasty. Survivorship and technical considerations at an average follow-up of 12.4 years. *J Arthroplasty* 21:13–17
15. Price AJ, Waite JC, Svard U (2005) Long-term clinical results of the Oxford unicompartmental knee arthroplasty. *Clin Orthop* 435:171–180
16. Tabor OB Jr, Tabor OB (1998) Unicompartmental knee Arthroplasty: long-term follow-up study. *J Arthroplasty* 13:373
17. Weale AE, Murray DW, Crawford R et al (1999) Does arthritis progress in the retained compartments after Oxford medial UKA? A clinical and radiological study with a minimum 10 year follow-up. *J Bone Joint Surg Br* 81-B: 783–789

# Isolated Patellofemoral Osteoarthritis

Ferran Montserrat

The patellofemoral joint is a complex articulation that remains a relatively uncommon subject in the Orthopaedic literature. This lack of interest is surprising given the fact that patellofemoral symptoms are relatively common and can be extremely debilitating. Currently, “Isolated PatelloFemoral OsteoArthritis” (IPFOA) is recognized to be more common than previously thought. However, the evidence-base for managing patellofemoral osteoarthritis is behind that for the tibiofemoral joint. The prevalence of this pathology has been shown by different authors. Davies et al. [32] found in a series of 206 knees 9.2% of IPFOA. In this series IPFOA was found in 13.4% of women and 15.4% of men older than 60 years. McAlindon et al. [84], in a series of 273 symptomatic patients older than 55 years, documented that 11% of men and 24% of women showed evidence of isolated patellofemoral osteoarthritis. In asymptomatic patients this figure was 4%. The prevalence of IPFOA was 8% in women and 2% in men.

In a cadaver study of 100 individuals who were more than 65 years-old at the time of death, 79% had evidence of patellofemoral osteoarthritis, although it was not in isolation [96].

The incidence of cartilage lesions of the patella is surprisingly high. Authors have shown some degree of cartilage wear in 40–60% of patients at autopsy [21] and in 20–50% of patients at the time of knee arthroscopy for other diagnoses [123]. In a review of 31,516 arthroscopies of the knee, 4% of knees had Grade IV lesions, within this 4%, 21% had patellar chondral wear, and 15% had wear in the trochlear groove [31].

---

F. Montserrat  
IMAS, Passeig Sant Gervasi 16-20,  
08022 Barcelona, Spain  
e-mail: drfmontserrat@pulso.com

---

## Biomechanics

The flexion-extension pathway of the patellofemoral joint is a complex and dynamic cycle.

Most studies have been of cadavers, and there have been very few in vivo or clinical measurements [5]. Patellar motion can be described in terms of six-degrees-of-freedom, commonly described as three mutually perpendicular translations, plus three mutually perpendicular rotations. The method described by Grood and Suntay [55] has been used widely to describe knee motion and may be the most useful system [17]. This is a three cylindrical open-chain system in which the patellar co-ordinate system is tethered to the femoral mediolateral axis by a floating axis which also serves as the axis about which patellar rotation takes place.

The movements of the patellofemoral joint are complex and have been reported by Goodfellow et al. [51]. In full extension, the patella does not come into contact with the trochlear groove. As knee flexion is initiated, the inferior pole of articular surface of the patella comes into contact with the trochlea. As knee flexion continues from 0 to 90°, the area of patellofemoral contact moves proximally on the patella, from the inferior pole toward the central portion, and finally toward the superior pole. At 90° of flexion, only the superior region of the patella is in contact with the distal aspect of the trochlear groove [4]. After 120° of flexion, only the most medial and lateral aspects of the patella come into contact with the femoral condyles. The articular cartilage of the patella is the thickest of any in the body, an adaptation to the great pressures throughout the patellofemoral joint during knee flexion [54].

---

## Aetiology

The causes of patellofemoral osteoarthritis are not always known. In the majority of cases we can find extensor mechanism disorders. Other documented causes are trauma and

obesity and recently there are some studies on the relationship between genetics and cartilage degeneration.

Abnormal mechanics of the patellofemoral articulation lead to abnormal pressures on the articular surface, cartilage breakdown, and severe functional limitations secondary to anterior knee pain. Patellar tracking is defined as the motion of the patella relative to the femur and femoral groove on knee flexion and extension. Abnormalities of tracking (maltracking) are thought to relate to many disorders of the patellofemoral joint [44, 48]. In a study of 72 patellofemoral arthroplasties, 61 of the 72 knees (75%) needed some form of re-alignment procedure [19]. Other series of patellofemoral arthroplasty have found that patellofemoral arthritis associated with patella malpositioning was the most common clinical presentation in these patients groups [6, 14].

There are a lot of terms used in the patellofemoral disorders. In the literature we can find different words trying to define similar concepts. Extensor mechanism includes quadriceps muscle and tendon, patella, patellar tendon with its tibial insertion and patellofemoral joint (PFJ). PFJ includes articular surface of patella with its specific cartilage and the trochlea of femur which is probably the most important part of this issue. Malalignment represents different anatomical disorders in the extensor mechanism well documented by clinical and radiological explorations but not necessarily associated with a subjective clinical instability. Malalignment represents an objective description of the anatomical extensor mechanism situation. This concept can be present with or without clinical symptoms of instability. Patellofemoral instability is the condition in which there are some subjective symptoms such as giving way, dislocation, pain and reduction of Activities of Daily Living (ADL) or sport practice. In some cases there is a long history of subjective symptoms (many times it starts in childhood) with or without previous patellar dislocation. PF instability implies some malalignment or anatomical alteration that leads the patella maltracking and, in some cases, patella dislocation. Nevertheless, we can find different clinical forms of presentation. In both malalignment without instability and true PF instability, the PFJ evolution is to the degenerative disease as an isolated form. Because the main cause of IPFOA is an extensor mechanism disorder, especially dysplasia of trochea, the diagnosis of the different causes is critical to choose the treatment form.

It is generally agreed that Extensor mechanism disorders cause IPFOA. These include: Patella alta, Patellar tilt and Patellar shift, TT-GT distance, Dysplasia of patella and Trochlear dysplasia.

Post et al. [102] defined patellofemoral malalignment as “when bony alignment, joint geometry, soft tissue restraints, neuromuscular control, and functional demands combine to

produce symptoms as a result of abnormally directed loads which (sic) exceed the physiological threshold of the tissues.”

## Extensor Mechanism Causes

### Patella Alta

Patella alta represents a stress between the lower and posterior part of patella and the proximal edge of trochlea (some cases with bump form). This is not a common pathology but it is present in some cases. Nevertheless, patella alta as an isolated cause was not found to lead to PF osteoarthritis [34].

### Patellar Tilt and Patellar Shift

Ficat et al. [42] and Merchant et al. [87] were the first to postulate that the increased patellar tilt associated with a tight lateral retinaculum would lead to excessive pressures at the lateral facet of the patellofemoral compartment. Iwano et al. [64] in isolated patellofemoral arthrosis series have reported lateral changes in 92% of cases (59 of 64 knees).

Patellar tilt was previously considered a direct consequence of a vastus medialis dysplasia [61, 97]. More recently, however, it has been shown that there is a high statistical correlation between the type of trochlear dysplasia and the patellar tilt: the greater the trochlear dysplasia, the higher the patella tilt [119].

Nevertheless, Galtier et al. [49] have studied the vastus medialis in 44 knees of cadavers, finding two types of nerve pedicles from the femoral nerve. In cases with few or no patellar cartilage injury they found two nerve pedicles, while the knees with severe cartilage lesions in the lateral facet of the patella had a single nerve pedicle for the vastus medialis. This fact would support the opinion that the patellar tilt and the vastus medialis dysplasia not only are interrelated, but that may be a cause of IPFOA.

In a study of 31 patients, Cohen et al. [27] reported the average cartilage thickness distribution in 33 patellofemoral joints in patients with arthrosis and compared them with the normal templates. The resulting “difference” maps provided an intuitive visual assessment of the distribution of cartilage loss across the articular surfaces of the patellofemoral joint. These results confirmed that, on average, cartilage loss in patellofemoral joint arthritis occurs predominantly on the lateral facet of the patella, and to a lesser extent, on the lateral trochlea.

Iwano et al. [64] found in a series of PFA that in cases with a history of dislocation or subluxation of the patella,

the patella tends to tilt laterally but in cases without dislocation, the patella tends to shift laterally (Fig. 1).

The medial patellofemoral ligament is the primary passive soft-tissue restraint to lateral patellar displacement. It provides 50–60% of lateral restraint from 0 to 30° of knee flexion [39]. A study of cadavers showed that cutting the medial structures results in a 50% decrease in the force required to move the patella 10 mm laterally [110]. After patellar dislocation, the most important factor causing patellar tilt and lateral instability is the torn medial patellofemoral ligament. We must keep in mind that the lesion of MPFL is a consequence of PF joint dislocation, but it is not the cause of the first dislocation.

#### TT-TG Distance/Q Angle (Extensor Mechanism Alignment)

In order to quantify the extensor mechanism alignment Goutallier and Bernageau described the distance between the Tibial Tubercle and the Trochlear groove (TT-TG) [53].

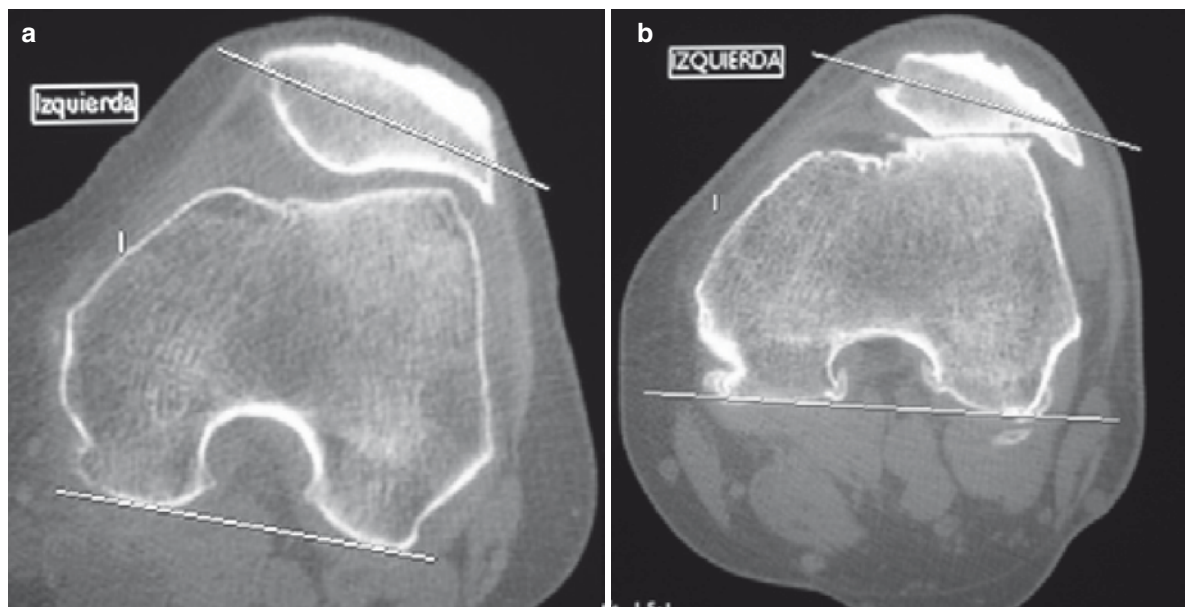
The extensor mechanism alignment is usually analyzed with the CT scan [119] which is more reliable than the Q angle. The CT scan protocol superimposes two cuts, the first through the more proximal point on the trochlea (reference cut, where the notch is like a Roman arch) and the second cut through the more proximal point on the tibial tubercle. The two points are projected on the posterior bicondylar line; the distance between them is the tibial tubercle-

trochlear groove (TT-TG). This measurement allows the surgeon to propose a perfect correction and prevent a hypocorrection or hypercorrection. The normal value in full extension is between 10 and 20 mm [37] (Fig. 2).

There are many publications concerning the relationship between the TT-TG distance and patellofemoral instability but no studies on this parameter in patellofemoral arthrosis.

A greatly increased (or decreased) Q angle could be expected to lead to increased pressures between the patella and the lateral (or medial) wall of the trochlea. Indeed, Goutallier and Bernageau [52] have found increased pain in patients whose normally-positioned tibial tuberosity is transposed medially. They also have introduced the concept of the Q angle as it relates to the trochlea. A patient who has a steep trochlea is more susceptible to increased patellar-trochlear pressures if the Q angle is significantly high or low. In their study the patients who experienced the greatest pain following tibial tuberosity transfers were those who had a steep trochlea (<140°) and low Q angles.

In most of the literature related to PFOA the parameter more commonly-used has been the Q angle, but it is an imprecise measure. The normal Q angle ranges from 10 to 20°, [62] but a number of variations have been described. Freeman [45] described a normal Q angle of 10–20° in females and 8–10° in males. Aglietti et al. [2] described a normal Q angle of 17° in females and 14° in males. Hughston [59] believed that a Q angle of >10° in either gender was abnormal and should be corrected. We believe



**Fig. 1** In cases with a history of dislocation or subluxation the patella tends to tilt laterally (a) but in cases without dislocation, the patella tends to shift laterally (b)



**Fig. 2** TT-GT distance: The CT scan protocol superimposes two cuts, the first through the more proximal point on the trochlea (reference cut, where the notch is like a roman arch) and the second cut through the more proximal point on the tibial tubercle. The two points are projected on the posterior bicondylar line; the distance between them is the tibial tubercle-trochlear groove (TT-TG)

that TT-TG is a more reproducible measure than Q angle and we propose its routine use.

### Dysplasia of the Patella

There are a few works that studied specifically the relationship between the patellar dysplasia and IPFOA. Dejour and Allain found that 42% of patients with IPFOA had a patellar dysplasia Wiberg type II ( $p < 0.001$ ) [34].

### Trochlear Dysplasia

Trochlear dysplasia is defined by a sulcus angle of more than  $145^\circ$  [80] and it is well defined on the true lateral view by the “crossing sign” [37] (Fig. 3a). In this view we can find a line representing the deepest part of the trochlear groove. When this line is in the same plane of the anterior border of both condyles the groove is flat in this point. In the same way, when the sulcus line is ahead of anterior condylar borders the trochlea becomes convex (Fig. 3). So, the trochlear groove may become flat or convex. Trochlear dysplasia has been

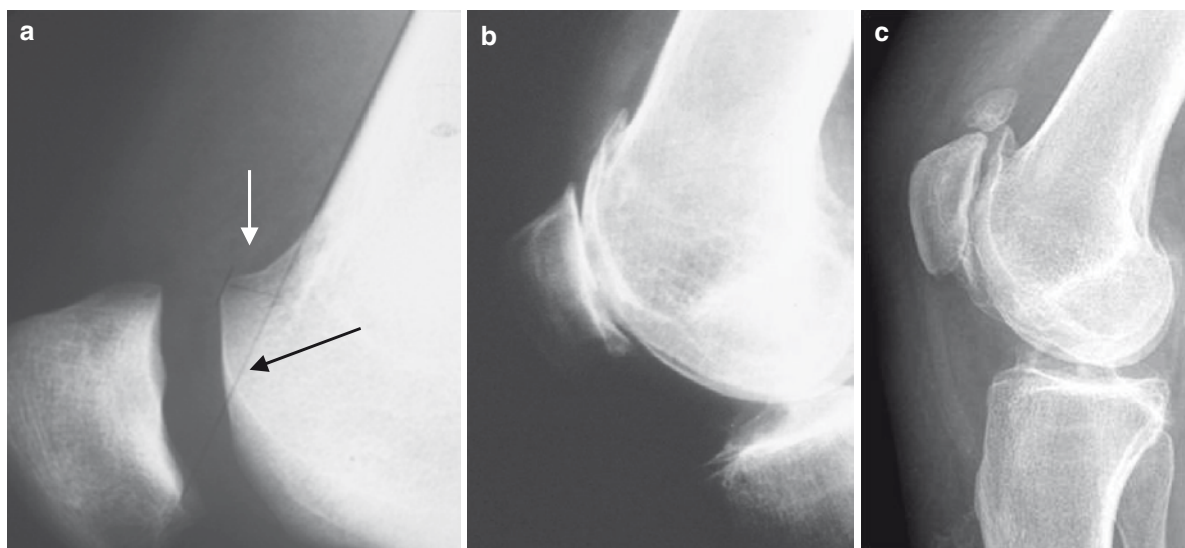
classified by David Dejour into four categories based on the sulcus line in the lateral X-ray view [35, 120] (Fig. 4).

Trochlear dysplasia is strongly linked with patellofemoral arthritis (Fig. 3b, c). In a study of 367 patients who had IPFOA, Dejour and Allain [34] noted that 78% of patients had a positive “crossing sign” on the lateral radiograph. In the same work the authors found a direct correlation ( $p = 0.0046$ ) between the Iwano’s stage of PF arthrosis and the severity of dysplasia according to Dejour’s classification. The trochlear dysplasia represents the most common predisposing factor in the IPFOA. The patients with IPFOA secondary to the trochlear dysplasia start the clinical symptoms earlier than in other cases because of other causes (54 year-old vs. 56 year-old). In this series the high grade dysplasia is more prevalent in the instability-arthritis category (66%) than in the no instability category (38%). Even trochlear dysplasia is the most important cause of patellofemoral instability, including true PF dislocation; also it is one of more frequent causes of IPFA in patients without PF history of instability.

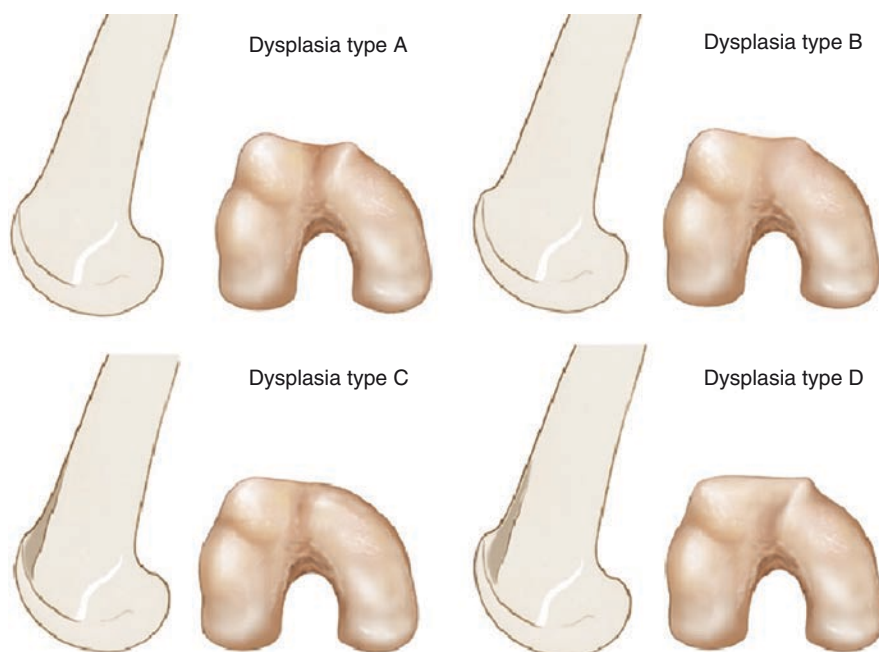
### PF Dislocation

In the group of patients with IPFA secondary to the extensor mechanism disorders there is a sub-group which, because the severity of the anatomical alteration or because combined number of disorders, presents with patellofemoral dislocation. This sub-group has a specific behaviour and only few studies have been dedicated to analyze the relationship between PF dislocation and IPFOA [77, 78, 95]. Mäempää and Lehto [78] studied a series of 85 patients (52 females) treated conservatively for a PF acute dislocation. Seventeen patients were treated with late surgery because of different clinical symptoms (pain, swelling, instability). The series was divided in two groups: Group I include 56 patients (66%) with predisposing dislocation factors and Group II with 29 patients (34%) without predisposing factors. The incidence of osteoarthritic changes in the patellofemoral joint in the whole study population was 22% in the affected knee and 11% in the unaffected knee. This study shows that the results after conservative treatment are comparable in the long-term with those of operative treatment. Patients suffering from recurrence had a lower incidence of degenerative changes in the patellofemoral joint than did those without occurrence (14% vs. 29%). It seems that degeneration of the joint is due much more to the abnormal tracking of the patella than to the occasional single recurrence of patellar dislocation. In this study the authors found a higher rate of arthritis in the series of 17 patients who had undergone late surgical treatment than in 68 patients without late surgery. However, it must be noted that the surgical techniques





**Fig. 3** (a) Trochlea dysplasia is well defined on the true lateral view by the “crossing sign” (black arrow). In dysplasia type D we can see the “supratrochlear spur” (white arrow). (b) Patellofemoral arthrosis in dysplasia type B. (c) Patellofemoral arthrosis in dysplasia type C



**Fig. 4** Dejour's classification of trochlear dysplasia classification. Type A: crossing sign (flat or convex trochlea). Type B: crossing sign and supratrochlear spur. Type C: crossing sign and double contour. Type D: crossing sign, supratrochlear spur, double contour, and sharp step-off of the trochlea (With permission [35])

performed in this series are not used currently [78]. Iwano et al. [64] used the same criteria in their assessment and found that lesions in the patellofemoral joint have a history of patellar dislocation or subluxation in 28% of cases.

Two factors are important in PF dislocation. Trochlear dysplasia, as a cause of instability, that was noted in 96% of

patients who had a history of one or more documented dislocations but was present in only 3% of a control population [36, 37]. Secondly, the clinical importance of the medial patellofemoral ligament in the stability of the patellofemoral joint against lateral patellofemoral dislocation has been well established [7].

### No Extensor Mechanism Causes

The IPFOA can be the result of a lot of causes, some of them are outside the knee. Thus we can find this pathology with normal anatomy and patellar alignment. All series in the literature present about 70–85% of extensor mechanism disorders but none presents 100% of malalignment.

### Trauma

There are two types of PF trauma that can be the cause of PFOA. The blunt closed trauma with silent lesions of PF cartilage that progress slowly with cracking and knee pain. And the patella fracture with surgical or conservative treatment. Argenson et al. [8] found, in a series of 66 patients whom were performed PF arthroplasty, that 20 knees had a patella fracture as cause of IPFOA. Dejour and Allain [34] found 9% of articular fractures as the cause of patellofemoral arthrosis.

### Obesity

Obesity has been well known as a main cause of mono-, bi- or tri-compartmental knee osteoarthritis [25, 33, 85]. The IPFA is also linked with obesity in some series [29, 85].

### Gender

There are no prospective trials about the relationship between gender and patellofemoral arthrosis, but many series in the literature have shown a large number of women involved in this pathology.

Felson et al. [40] found that gender is a major risk factor for knee osteoarthritis (OA), with women being affected more than aged-matched men. Hanna et al. [57] in a recent study of 271 participants (169 women) aged between 50 and 79 years with no clinical history of knee pain or pathology were examined using magnetic resonance imaging at baseline and 2.3 years later. The authors found that at the patella, the average annual percentage loss of cartilage volume was significantly greater in women (2.3% [95% CI, 1.7–2.8]) than in men (0.8% [95% CI, 0.1–1.6]) ( $p=0.02$ ).

McAlindon et al. [84], in a series of 273 symptomatic patients older than 55 years, found IPFA in 11% of men and 24% in women. They found that, in the female series, IPFA is more common and tending to increase in frequency with age. Iwano et al. [64] reviewed 66 patients with patellofemoral osteoarthritis of whom 61 were women. Dejour and Allain [34] found in their series of 367 patients 72% of women.

### Genetic

A classic twin study undertaken in the UK has estimated the heritability of knee OA – that is, the proportion of the variance in occurrence of knee OA that may be explained by genetic factors, to be 39% [114].

Some patients may have a genetic predisposition to deterioration of the articular cartilage. Spector and MacGregor [115], for example, have noted that the ability of collagen to withstand high stresses has a genetic component. Neame et al. [94] studied a series of 490 knee OA index cases listed for total knee replacement and 737 of their siblings aged >40 years and 1,729 community subjects aged >40 years. The age, sex, and knee pain-adjusted odds ratios in siblings were 2.9 (95% confidence interval 2.3–3.7) for tibiofemoral OA and 1.7 (1.4–2.2) for patellofemoral OA. This difference remained after adjustment for important environmental risk factors. The heritability estimate for knee OA was 0.62. The authors concluded that siblings are at increased risk of knee OA in comparison with the general population. This is likely to be due to genetic factors.

---

### Clinical Presentation

Pain is the primary symptom that should be addressed but, in the patellofemoral joint, pain is a common sign in a large number of conditions such as: Anterior knee pain syndrome, Patellofemoral instability with or without dislocation, tendinitis, focal osteochondral lesions, complex regional pain syndrome and patellofemoral OA. Other exceptional causes can be associated with a patellar tumour, infection, or stress fracture. Consequently, the surgeon must first correctly identify the source of this pain. It is tempting to attribute pain to physical changes that are clearly visible on a radiograph, on a magnetic resonance imaging scan, or to the naked eye. However, the major source of a patient's pain may in fact be abnormal intraosseous pressures, abnormal levels of biochemical mediators such as pro-inflammatory cytokines or substance P19, posterior chains syndrome or other factors that are not readily apparent. All of them are many times included in the so-called anterior knee pain syndrome.

A patient with isolated patellofemoral arthritis typically describes anterior knee pain when rising from a seated position and/or ascending stairs. However the patients typically state that going downstairs is more difficult than going upstairs. The pain is diminished or absent when the subject walks on level ground. This is typically worse with exercise, kneeling and squatting. It is associated with a grinding or crackling sensation (crepitus). The knee may be stiff. There may be pseudolocking

due to “kissing” lesions between the patella and trochlear groove, when exposed bone rubs on exposed bone.

Examination of the knee includes assessing lower limb alignment noting femoral version and tibial torsion. Quadriceps power should be documented with special reference to the vastus medialis obliquus. There may be an effusion (patellofemoral OA may produce hemarthrosis). The patella may be tight with little medial-to-lateral play (due to tightness of the lateral retinaculum), tracking abnormally with crepitus, and it may be large from formation of osteophytes. Palpating the undersurface may elicit tenderness. Hip and spine pathology should be excluded, as well as polyarthropathies.

There is not a key sign of patellofemoral arthritis on the physical examination because tenderness and pain at the lateral or occasionally medial facet of the patella are common signs in the majority of knee conditions. We must be remembered that when applying pressure to the facet, the examiner is simultaneously applying pressure to all of the soft tissues between the skin and bone, including the retinaculum and the synovium. The specific source of the pain can therefore be debated.

Iwano et al. [64] reviewed 66 patients with patellofemoral osteoarthritis of whom 42 were bilateral and 61 were women. The study group was 108 knees of which 64 had IPFOA (PF alone group) and the other 44 knees had tibiofemoral osteoarthritis associated (combined group). In this study they looked at seven clinical features and recorded seven activities of daily living (ADL) (Table 1). Each activity scored two points if performed easily, one point if there was some difficulty, and zero points if with great difficulty, giving a maximum ADL score of 14 points. All knees displayed crepitation by movement or grinding, 45% had peripatellar pain, abnormalities of the other activities were positive in only a few cases. In the isolated patellofemoral arthritis group the mean ADL score was 9.4 compared to 4.1 if the tibiofemoral joint was involved as well. In the isolated group zero scores were found on squatting, running with short steps, and sitting with a fully flexed knee. No patients had problems getting up from a low chair. More patients had some difficulty going downstairs than upstairs. The ADL score did not correlate with the severity of arthritis on X-ray.

### Evaluating the Outcome of Patellofemoral Disorders

Few outcome instruments were designed specifically for the assessment of the patellofemoral joint [104]. The development of such an instrument is difficult because of the potentially different expectations for the function of this joint among patients in different age groups. An outcome instrument that targets older patients with arthritis, such as WOMAC instrument, may be favourable to patients with

**Table 1** Iwano et al. [65] studied the series with two groups of features

<i>Seven clinical features</i>
Pain on grinding the patella (medio-laterally and infero-superiorly)
Crepitation on grinding the patella
Crepitation during knee movement
Peripatellar tenderness
Pain on compression of the patella
Limitation of patellar mobility
Clarke’s test
<i>Seven activities of daily living (ADL)</i>
Sitting with full knee flexion in the Japanese manner
Standing up from a low chair
Running with short steps
Squatting down
Going upstairs
Going downstairs
Standing on one leg with the knee semi-flexed

Clarke’s test is positive when patients complained of pain during knee extension with patella compression

patellofemoral arthritis, but many patients with PF disorders are younger. Two instruments that measures sports activity and instability, such as the Lysholm and Kujala scores [69], may be excessively unfavourable to such older patients with evolved disease. The Knee Society Score (KSS) Clinical Rating System has been used for evaluating patients with knee arthroplasty and its Knee score includes tibiofemoral joint evaluation. The International Knee Documentation Committee (IKDC) [63] instrument has shown good validity and reliability in the evaluation of patellofemoral disorders, but it has not been assessed in the context of arthritis [63, 128]. The Short Form-36 (SF-36) has been validated for evaluation of quality of life [11, 128] but it requires computerized analysis. The Knee Injury and Osteoarthritis Outcome Score (KOOS) [105] is a 42-question tool consisting of the reliable WOMAC instrument with the addition of items pertaining to younger, active patients, but it has not yet been utilized with any degree of frequency.

Paxton and Fithian [99] recently published a review of general health instruments (Medical Outcomes Study (MOS) SF-36 and SF-12), knee scales (KSS, Knee Outcome Survey, IKDC, KOOS) and a disease specific scale (WOMAC) for patellofemoral arthroplasty outcome assessment. Based on this review of the literature, they recommend the SF-36 and KOOS for evaluation of patellofemoral arthroplasty outcomes and provide recommendations for implementation of these instruments in a clinical setting.

Unfortunately, none of these instruments has been uniformly accepted by surgeons experienced in the treatment of patellofemoral disease

## Radiology and Imaging

Because the clinical signs and symptoms of IPFOA are common with other clinical syndromes in the knee, as we commented previously, the Surgeon must look for the imaging information that shows most exactly the extensor mechanism anatomy. The conventional X-ray, the CT scan and MRN are good tools for this task.

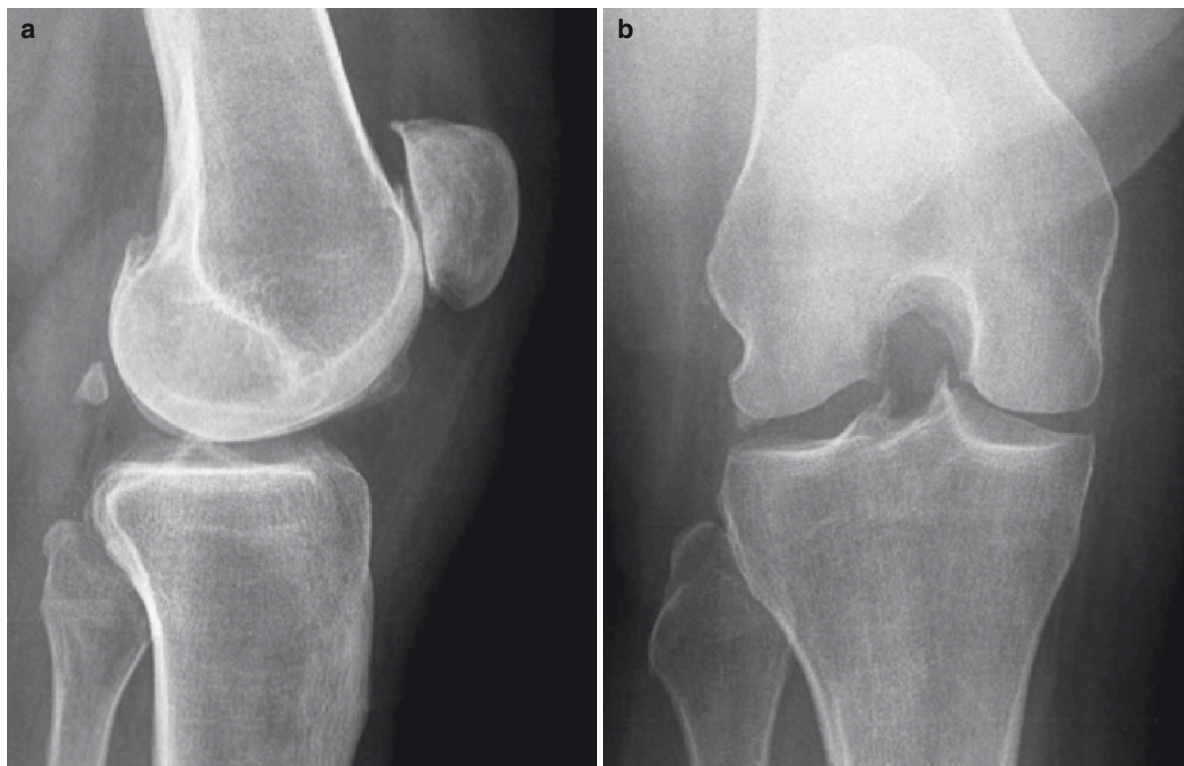
### X-Ray

The conventional X-ray is the first examination which should be used for PF pathology. The set of radiographs we recommend is: AP weight-bearing plain radiograph, Rosenberg view, true lateral view at 30° and skyline PF view.

The weight-bearing plain X-ray is necessary due to the different treatment necessary if the TF joint is involved (bi- or tri-compartmental disease). It is critical to know accurately what the tibiofemoral axis and morphotype the leg has. The Rosenberg view (named Schuss view in EU) [106] is also imperative to know the narrowing of TF joint line (Fig. 5). In order to know if the TF joint is involved in the knee arthrosis these two projections are imperative.

The true lateral view has been used for many authors to evaluate the grade of OA in PF joint [16, 23, 79, 84, 117]. The true lateral view is necessary to know the trochlea type [37] and measure the patellar height [22].

There are two classical radiological classifications of osteoarthritis [3, 65] but only the Ahlbäck's study includes a specific PF joint study with lateral and axial views. In this work the author comments "Radiographic examination of the patellar articulation should actually include both lateral and axial projections. During this investigation it was found that a diagnosis of narrowing was not definitive from lateral projections alone". Although the original article by Merchant et al. [88] described a knee flexion angle of 45°, lesser angles, such as 30°, are more desirable as they allow imaging of a



**Fig. 5** In the IPFA is imperative to know the narrowing of TF joint line. (a) Patellofemoral osteoarthritis secondary to trochlear dysplasia type A in true lateral view. (b) Indemnity of tibiofemoral joint in Rosenberg view

more proximal portion of the patellofemoral joint. Since trochlear dysplasia is most commonly proximal, it is imperative that this portion of the compartment be visualized. In the same way, Lanyon et al. [70] studied 108 osteoarthritic knees to compare two plain radiographic methods for sensitivity to detect progression of patellofemoral osteoarthritis. The authors concluded that it is possible to detect significant joint space loss with time on the skyline view that is not apparent on the lateral view and that the skyline view should be the method of choice to detect progression of patellofemoral osteoarthritis. Cicuttini et al. [24] agreed in a study in 504 knees. We believe that it is necessary to perform a skyline view to see clearly what the situation of the patella is.

However, some authors do not agree with this opinion. Bhattacharya et al. [13], recently, comparing the sensitivity and specificity of both lateral and skyline views found similar results and they did not recommend the routine use of the radiological skyline view.

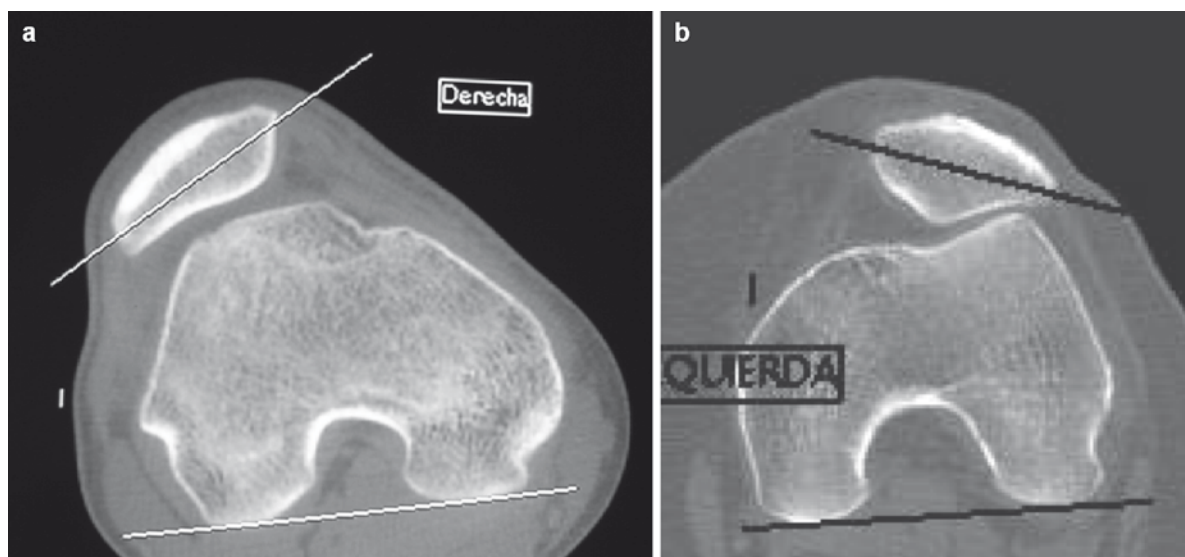
There is no discussion about both, plain weight-bearing and Schuss views, but there are different points of view regarding the other two exams. We believe that true profile is imperative to know the trochlear type but also to know the grade of osteoarthritis and the skyline view is very useful to know exactly the extent of narrowing of PF joint.

Iwano et al. [64] showed four different stages of PFOA using the Merchant's view. Currently, this classification is the most frequently used in the IPFOA.

## CT Scan

Cross-sectional imaging with transverse computed tomography slices at different positions along the lower limb can provide a three-dimensional view of the patellofemoral joint and be used to assess the lateral offset of the tibial tuberosity from the deepest point in the trochlear groove. A distance between the tibial tuberosity and the trochlear groove (TT-TG) exceeding 20 mm is nearly always associated with patellar instability [37].

Also, CT scan is the best investigation to measure the patellar tilt and patellar shift. The patellar tilt is measured on the CT scan superimposing two cuts, the first through the centre of the patella and the second through the reference trochlear cut. Two lines are drawn, the first going through the patellar axis and the second line going through the posterior bicondylar line. The tilt is the angle between those two lines (Fig. 6). The tilt is measured with and without quadriceps contraction. Eighty-three percent of patellofemoral instabilities with dislocation have a patellar tilt higher than  $20^\circ$  [36, 37]. The patellar shift can be measured in CT-scan with the distance between the trochlear groove and patella apex. However, because the cartilage and bone apex are not coincident in 85% of knees [116], we believe that the congruence angle of Merchant [89] is a good option to know if the patella is medial-, centrally- or laterally-placed.



**Fig. 6** (a) Patella tilt in dysplasia type D. (b) Patellar tilt in normal trochlea

## MRI

MRI is useful, especially in PF instability with patellar dislocation because it is a good tool to evaluate the medial-side soft tissue and osteochondral lesions that are present usually in patellar dislocation. When operative findings were correlated with magnetic resonance imaging findings, MRI was found to be 85% sensitive and 70% accurate in detecting disruption of the medial patellofemoral ligament [108]. MRI in the majority of cases is not useful because in later isolated patellofemoral osteoarthritis it has a very low rate of cost-effectiveness.

## Treatment

### General Considerations

If the aetiology, clinical presentation and diagnosis of IPFOA are very complex, the treatment of this pathology represents one of the most important challenges in knee surgery.

In the literature patellofemoral arthrosis has been treated for a long time by many techniques with different results. The challenge is that we are treating a complex pathology that shows different presentations over time. We find PF OA in knees with and without extensor mechanism disorder, with instability and patellar dislocation, and in knees without dislocation but with extensor mechanism disorders. We are treating PF instability early in the disease in young patients, but in the later degenerative period, we must treat a true arthrosis. Thus we believe there should be different treatment for different IPFOA presentation.

There are two main groups: The first one includes young patients with predominance of clinical instability and the second group has evolved degenerative disease predominantly. The first group should be treated with surgical techniques for PF instability and the second group should be treated with specific techniques for degenerative joint disease. The surgeon is the person who should decide in which group each patient belongs.

### Non-Operative Management of Patellofemoral Arthritis

Although PFOA is one of the most prevalent degenerative diseases of the knee we only have found two Randomized Clinical Trials (RCT), with different results, related to physiotherapy treatment.

Quilty et al. [103] published a study in which 87 patients (44 in the control group, 43 in the experimental group) were included in a RCT to evaluate physiotherapy treatment for

PF OA. The authors concluded that the treatment package produced small improvements in knee pain scores and quadriceps muscle strength 10 weeks after the end of the treatment period. And there was no difference between the two groups at 1 year.

Crossley et al. [30] evaluated, in a series of ninety people with lateral PFJ OA, whether a physiotherapy treatment, targeted to the PFJ, resulted in greater improvements in pain and physical function than a physiotherapy education intervention in people with symptomatic and radiographic PFJ OA. They concluded that “The project’s outcome will influence PFJ OA rehabilitation, with the potential to reduce the personal and societal burden of this increasing public health problem”.

The classical challenge of physical therapy is to strengthen and stretch the structures about the knee without eliciting pain. But currently the physical therapy ideal is centred on limb control and body positioning. In that respect, so-called core-strengthening is an excellent approach to the treatment of a patient with patellofemoral arthritis. Core-strengthening focusses on abdominal muscle control, trunk balance, and limb control. Exercises designed to improve limb balance focus on the hip and the knee to maximize the efficiency of the limb. One key benefit of using core-strengthening principles during rehabilitation is the avoidance of excessive repetitive strengthening exercises about the knee, which may exacerbate symptoms.

Taping has been shown to increase quadriceps muscle torque and to activate the vastus medialis obliquus earlier than the vastus lateralis during stair ascent and descent. There is increasing evidence that weight-bearing or closed-chain training is more efficacious than open-chain exercises. Closed kinetic training allows training of the vastus muscles simultaneously with gluteal and trunk-muscle strengthening to control limb position.

## Surgical Procedures

### Role of Arthroscopy

Arthroscopic debridement includes loose body and instable cartilage lesion removal but the surgeon must realize that these measures may have only temporary effects on symptoms. In general there is no place for the arthroscopy in the IPFOA.

### Soft-Tissue Re-Alignment of the Extensor Mechanism

Soft-tissue re-alignment surgery has been described in the literature on patellar instability but the outcome of

soft-tissue patellar re-alignment for the treatment of patellofemoral pain in the absence of instability is largely unknown. More than 150 variations of patellar re-alignment have been reported. Most reports are retrospective and typically involve mixed groups that have both pain and instability. Few have utilized a valid pain scale or a validated functional scale.

### ***Isolated Lateral Retinacular Release***

Studies have shown that the lateral retinacular release relieves anterior knee pain when there is radiographic documentation of pathologic lateral patellar tilt in the absence of patellar instability [1, 111]. One study revealed that cutting the lateral retinaculum resulted in a 10% decrease in the lateral restraining force [39]. Other authors [107] believe that the isolated lateral retinacular release in the treatment of patellofemoral arthritis may have some applicability when the following factors are present: (1) lateral patellofemoral arthritis on one or both sides of the patellofemoral joint, (2) radiographic evidence of tilt without subluxation, and (3) no clinical history of instability. Combining this release with a partial lateral facetectomy when there is a large osteophyte may increase its clinical effectiveness. However, there is no evidence that isolated lateral release provides long-term benefit for patients with patellofemoral arthritis [28].

### ***Lateral Patellar Facetectomy***

In cases with evolved patellofemoral disease, excessive lateral tilt and/or shift may lead to the formation of a large lateral osteophyte visible on the skyline view. Some authors [83] advocate excision of the lateral facet overgrowth (a so-called “partial lateral patellar facetectomy”) and re-tensioning of the lateral tissues. Lateral patellar facetectomy may provide pain relief and decrease the lateral overload in the patellofemoral compartment. Yercan et al. [126] followed eleven patients for an average of 8 years after such a procedure and noted a significant increase in the average pain score ( $p=0.04$ ) and in the average KSS ( $p=0.02$ ) (based on the findings of a physical examination). The average KSS functional score (based on walking and stair-climbing) also improved. Partial lateral patellar facetectomy may provide pain relief and decrease the lateral overload in the patellofemoral compartment, and it leave the bone stock necessary for future replacement.

More recently, Becker et al. [12] have published a series of 51 knees in 50 patients with isolated patellofemoral

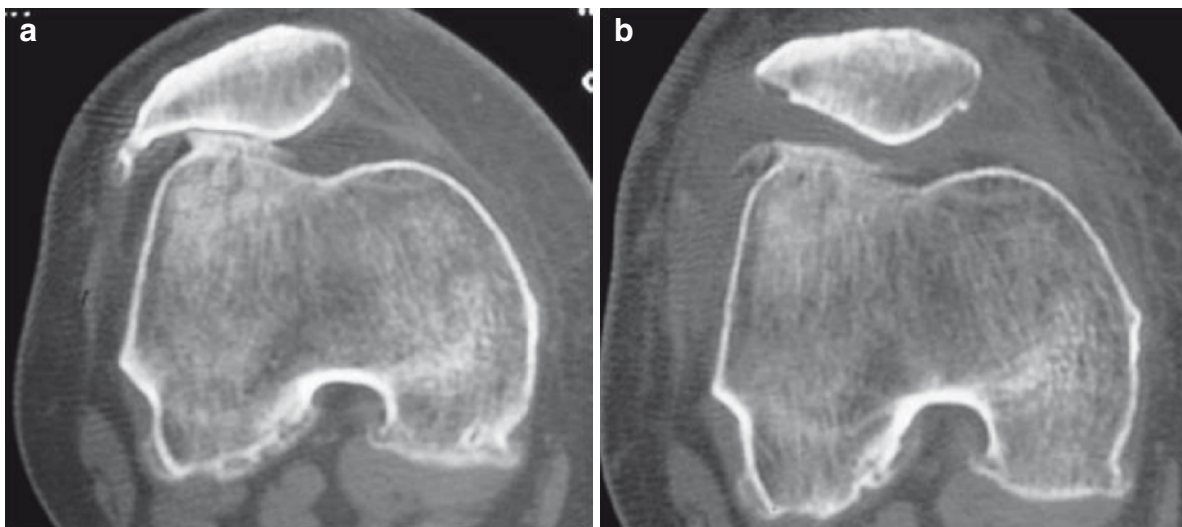
osteoarthritis treated by partial lateral facetectomy, lateral release, and medialization of the tibial tubercle. The mean follow-up was 20.2 months (range, 7–32 months). The majority of these patients experienced improvement in their patellofemoral symptoms. However, the clinical outcome was no better than other surgical procedures. After the short follow-up, they did not recommend combined lateral facetectomy, lateral release, and medialization of the tibial tubercle until longer-term results are available.

The author reported a series of 83 knees (77 female) with IPFOA that never suffered dislocation, which underwent lateral facetectomy, lateral release and proximal re-alignment by Insall’s technique [61], in the period 1992–2001. In 2009, 56 knees were revised (74% of original series) 26 knees were lost and six had died. The successful series (63% of 2009 revised series) has a follow-up of 12 years with no other treatment. The rest of knees (37% of 2009 revised series) were considered failures and needed TKA at a mean follow-up of 8.6 years. These results showed that the lateral partial facetectomy plus Insall’s proximal re-alignment is a safe and stable procedure for a long period [92] (Fig. 7).

### ***Proximal Soft-Tissue Re-Alignment***

Classically, these procedures are focussed on arthritis affecting the lateral facet. Also, they have been used in the PF instability [60, 61]. Other authors have emphasized that this procedure is specially indicated when the patellar tilt is increased ( $>20^\circ$ ) [37]. The mature patient with a high congruence angle and minimal or moderate arthrosis may benefit from proximal soft-tissue re-alignment if other therapeutic measures have failed. A Q-angle of  $<10^\circ$  has also been associated with better outcomes [61].

This technique, described by Insall et al. [61], starts with a medial parapatellar incision from just above the superior pole of the patella to the medial aspect of the tibial tubercle involving the medial capsular tissue that includes MPFL. A release of the lateral patellofemoral ligament and a retinacular release are performed. On both medial and lateral sides it should leave the synovial tissue intact to isolate the joint and the capsular tissue release is carried down to the level of the tubercle. The incision must respect the most lateral fibres of vastus medialis and distally it remains in continuity with the medial capsule tissue. Medially, the vastus medialis is elevated from the underlying synovium about 10 cm from its insertion. It is then advanced to the middle free edge of the patella, creating a sleeve around the patella. Passive knee motion and weight-bearing are begun at 24 h after the surgery. Insall et al. [61] reported 91% of



**Fig. 7** Lateral patellar facetectomy plus Insall procedure. Preoperative (a) and postoperative (b) CT-scan views. The patellar tilt and tracking were corrected

satisfactory results at 4 years after this procedure. Other techniques involving a lateral release with a medial imbrication have also been described [109]. They involve a similar lateral retinacular release with imbrication of the medial retinacular tissue from the medial aspect of the quadriceps tendon to the proximal aspect of the tibial tubercle. Scuderi et al. [109] reported an 81% rate of good or excellent results after 3.5 years of.

It is also not known how far medial displacement must be for the operation to be successful.

The patella is innervated by multiple superficial sensory nerves [38] and this procedure could denervate the patella to a certain extent. It is not known to what extent the denervation contributes to the success of the operation when it works. To our knowledge, no formal study of surgical denervation has been carried out in the specific setting of patellofemoral arthritis.

## Surgery to the Patella

### *Patellar Thinning*

In order to diminish the stresses on the patellofemoral joint, Vaquero and Arriaza [122] used a double saw to remove 7 mm of bone from the centre of the patella and noted a diminution of patellofemoral stresses when they applied Fuji film to the patellofemoral joint. The authors measured the patellar thickness before surgery and found that the remaining bone was enough to place a patellar component if one is needed at a later time.

### *Patellar Osteotomy*

In order to improve the congruence of the patello-trochlear joint, Morscher [93] described an anterior closing-wedge osteotomy fixed with trans-osseous sutures, designed to make a second facet in the patella with dysplasia type Wiberg III [125]. The procedure is technically demanding, because the patella is a small, poorly-vascularized structure with a high proportion of cortical bone. Also, it is difficult to determine the amount of articulation for each facet and exactly where the ridge is to be placed. There is a major risk of necrosis and non-union. This Patellar osteotomy is indicated in patellar dysplasia in which the articular surface of patella is flat. If there is also a flat trochela, this patellar re-shaping is complementary to trochleoplasty. This is a demanding surgery and we recommend its use only by experienced surgeons in few selected cases.

### *Patellectomy*

Patellectomy is an old procedure and its effect on knee function has been a matter of controversy. Although indications for a patellectomy have been narrowed, patellectomy still is recommended as the last choice of treatment and almost all authors advise patellectomy in severe cases in which all other treatment options are not suitable, such as comminuted fractures, advanced osteoarthritis, infections and tumoural conditions[66].

Patellectomy was indicated because it can be reasoned that removal of the patellar half of a painful patellofemoral joint



will cause the pain to disappear. However, the pain from an arthritic trochlea cannot be relieved by a patellectomy, and removal of the patella greatly diminishes the lever arm of the extensor mechanism [46]. This can lead to extensor weakness or a so-called extensor lag, whereby the patient is incapable of completely straightening the knee. Also, excision of the patella results in alteration of the forces acting on the tibiofemoral joint and the instant centre of motion, limitation of range of motion, anterior instability, loss of protection of the trochlea from injury, and poor cosmesis. Several techniques have been described to re-inforce the defect produced by the absence of the excised patella. In these techniques, after closure of the defect transversely [124, 127] or longitudinally [56] or with a strip of proximally-attached quadriceps tendon [10], some parts of the quadriceps muscle are advanced over the site of the excised patella. The technique basically involves a midline incision with a sharp dissection of the patella. Care should be taken to repair the retinaculum to prevent an extensor lag after surgery, and tracking of the quadriceps tendon should be checked to make sure that a proximal re-alignment procedure is not necessary. The patient then is allowed early continuous passive motion and weight-bearing after surgery. Baker and Hughston [10] reported long-term (mean, 13.8 years) results using the Miyakawa technique of pallectomy. In this technique, a strip of quadriceps tendon is pulled distally to fill the void that was left by the removal of the patella; the vastus medialis and lateralis then are advanced over the site of the excised patella. Eighteen of 20 patients were rated as having good or excellent results by objective criteria [10].

Lennox et al. [73] found the poorest results in their patients who underwent a patellectomy for arthritis (Not all of their patients underwent the patellectomy for that indication). They reported a good result in only 54% (21) of 22 patients with arthritis, with 27% (6) of the 22 patients reporting that they felt worse than they had pre-operatively.

Günal et al. [56] did patellectomy by combining it with the vastus medialis obliquus advancement technique. In this technique, the defect is closed longitudinally and the vastus medialis obliquus is advanced distally and laterally and is plicated to increase the angle of insertion in the sagittal plane. The main advantage of this technique is the use of the vastus medialis obliquus muscle, the only part of the quadriceps that has no function in knee extension. However, this technique only has been compared with simple longitudinal repair in a prospective randomized trial.

However, in the majority of these studies it is not clear what percentage of knees had arthritis limited to the patella or how often arthritis was present on both the patella and the trochlea, a point which has not been studied, to our knowledge. The Surgeon must bear in mind that the major drawback of a patellectomy is that it is irreversible.

### Autologous Chondrocyte Implantation

Autologous chondrocyte implantation was first described in 1994 [15]. Initially, in PF arthrosis results were poor [15]. However, in later reports [100, 101], in which patellar tracking was also addressed with re-alignment of the extensor mechanism at the time of transplantation, eleven of seventeen patients had a good or excellent result at 2 years [101] and this improved to thirteen of the seventeen patients at 10 years [100]. The importance of correcting the underlying cause of the chondral injury cannot be underestimated.

Minas and Bryant [90] investigated a group of 45 patients who had patellofemoral arthritis, with or without femorotibial arthritis. Eight patients had isolated patellar arthritis, nine had trochlear arthritis, four demonstrated patellar and trochlear arthritis, and the remainder exhibited arthritis in two or more compartments. The authors noted that, at an average of 2 years post-operatively, the patients with patellar arthritis, trochlear arthritis, and “patella plus trochlea plus weight-bearing condyles” (involvement of both the patellar and the trochlear side of the patellofemoral articulation as well as of the weight-bearing portion of one or both femoral condyles) “all had marked improvement in pain relief and function.” There were eight graft failures in the patellofemoral compartment. In this series osteotomy was added in 64% of knees and also proximal alignment and trochleoplasty were performed in some cases. In a recent study, Gobbi et al. [50] investigated a group of 34 patients treated for full-thickness patellofemoral chondral lesions with second-generation ACI with final follow-up at 5 years. Results were evaluated using the International IKDC 2000 subjective and objective scores, EuroQol visual analogue scale (VAS), and Tegner score. All the scores used demonstrated a statistically significant improvement ( $p < 0.0005$ ) at 2 and 5 years follow-up. In this series, 15 (44%) patients, associated procedures were performed during cartilage harvest or implantation, including patellar re-alignment (10), meniscectomy (5), and ACL reconstruction (1).

Most series are not prospective randomized trials and do not have a comparative series control. Clar et al. [26] compared the cost-effectiveness of autologous chondrocyte implantation procedures in the knee with microfracture and mosaicplasty operations, and they found the results to be inconclusive because of a lack of sufficient long-term data. Also there are a lot of procedures performed at the same time as cartilage transplantation. This makes it difficult to find reliable outcome data. Longer follow-up and clinical trials comparing autologous chondrocyte implantation with other options for treatment of patellofemoral arthritis are needed before this option can be recommended as a standard procedure.

## Osteotomies of the Tibial Tubercle

There are basically three different procedures involving the TT: Anterior tibial tubercle transfer, Medial tibial tubercle transfer and Distal tibial tubercle transfer. These procedures are contra-indicated for patients with an open proximal tibial physis.

### Anterior Tibial Tubercle Transfer

This technique was first described by Maquet [82] developing a procedure that moves the tibial tuberosity anteriorly 2.5 cm. The theory is that to re-locate the tibial tuberosity to a more anterior position allows a diminution of those loads which could lead to pain relief. However, the size of the contact area on which these loads are applied has to be considered when judging subchondral stresses. Indeed, as noted by Lewallen et al. [74], a diminution of forces may not necessarily lead to a reduction of the stresses. Therefore, studies that only evaluate forces [112] may not provide clinically relevant data. Ferrandez et al. [41] noted that



**Fig. 8** The elevation of the tibial tuberosity can paradoxically increase stresses on the proximal portion of the patella

elevation of the tibial tuberosity can paradoxically increase stresses on the proximal portion of the patella, particularly when it is elevated >1 cm (Fig. 8).

Surgical technique: a central longitudinal incision is carried out which is extended below the tubercle. The osteotomy can be performed with an osteotome or a thin oscillating saw-blade from the superior aspect of the tubercle distally in the coronal plane leaving a distal bony hinge. Once mobilized, the proximal segment is displaced anteriorly, allowing plastic deformation of the bone at the distal attachment. An iliac crest graft is then fashioned and placed to allow 1.5–2 cm of anteriorization at the tubercle. More than 1.5 cm of anteriorization, however, is associated with a higher prevalence of skin problems post-operatively. A screw can be utilized for supplemental fixation through the tubercle and the graft, into the metaphyseal aspect of the tibia. Post-operative care involves use of crutches and partial weight-bearing and passive motion. Full weight-bearing is allowed at 6 weeks, when the osteotomy site is usually healed. Because Maquet's procedure has been indicated for different varieties of patellofemoral conditions and its results have not always been correlated with the etiology of patellofemoral pain or arthritis, the clinical outcome can be difficult to evaluate. The Maquet's operation presented a number of complications, most notably skin necrosis, cracking of the osteotomy fragment, patellar tendinitis, and the development of a painful prominence at the level of the tibial tuberosity. Over the last 20 years, Maquet's operation has diminished in popularity in Europe. Therefore, this technique is not recommended.

The antero-medial tibial tuberosity osteotomy described by Fulkerson et al. [47], is a modification of the Maquet operation. The procedure is carried out with an oblique osteotomy of the tibial tuberosity and the tibial tuberosity is transferred both medially and anteriorly, with the relative proportion of each transfer being determined by the slope of the cut. A 45° cut leads to equal medial and vertical displacements. If the tibial tuberosity were displaced 11 mm along a 45° plane, this would lead to 8 mm of medial displacement and 8 mm of anterior displacement. Ateshian and Hung [9] calculated that this would result in only a 10% reduction in stress. This operation has the disadvantage of not allowing the same amount of anterior displacement as Maquet's procedure and it displaces the tibial tuberosity medially even in patients in whom the need for such medialization is unclear. Also, it requires two cuts: The first cut involves all but the most proximal portion of the tibial tuberosity. That cut is angled in an anteromedial-to-posterolateral direction, with care taken to protect both the anterior tibial artery and the deep peroneal nerve posteriorly. The second cut begins at the most proximal and lateral aspect of the first cut and is oriented toward the most

proximal portion of the tibial tuberosity. If the surgeon fails to carry out this second cut and chooses instead to carry the first cut all of the way to the top of the tuberosity, the cut will involve a large part of the proximal tibial metaphysis. Complications associated with this operation include post-operative fracture of the tibial shaft and non-union of the osteotomy site. It is recommended that patients remain none weight-bearing for 8 weeks.

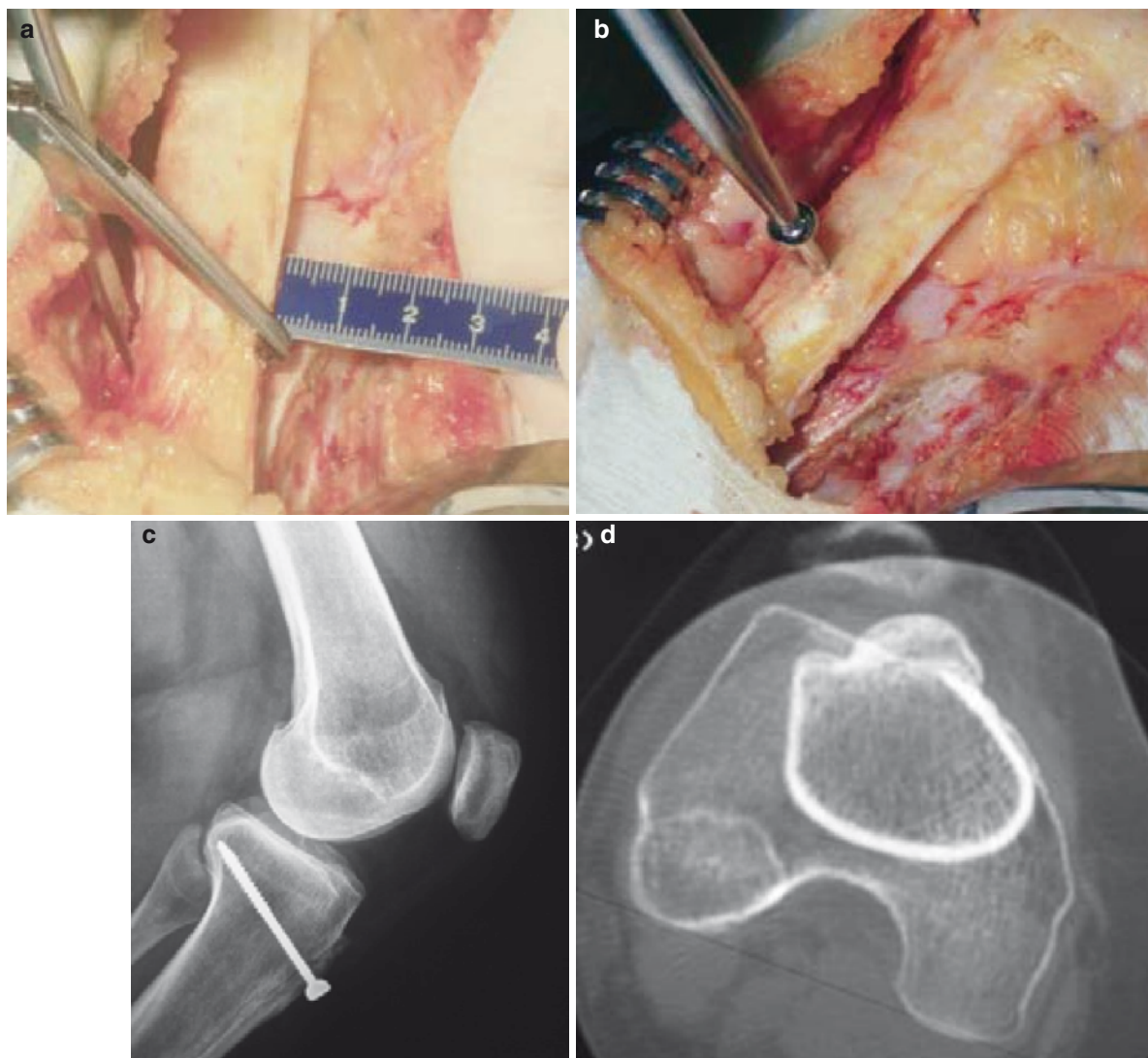
#### *Medial Tibial Tubercle Transfer*

This operation, known as the Elmslie-Trillat procedure [121], is a direct medial tibial tuberosity transfer. It is

effective for controlling instability and lateral tracking. The operation is mainly indicated for patients with an excessive TT-TG distance in a context of PF instability.

**Surgical technique:** The tibial tubercle is fully detached leaving a distal bony hinge as in Maquet's procedure. The block must be trimmed to ensure that it can be medialized with sufficient ease and that it will not sit proud of the tibia. The proximal portion of the tubercle is medialized checking the position with a ruler. Finally it is fixed with a single screw (Fig. 9). The post-operative care is the same as for the Maquet's procedure.

The goal of tibial tubercle transfer is to reduce the TT-TG to between 10 and 15 mm. However, Goutallier and



**Fig. 9** Elmslie procedure: (a) Medialization of Tibial tuberosity. (b) Fixation with one screw. (c) X-ray Lateral view. (d) CT scan view with the TT-TG distance corrected

Bernageau [52] stressed that the shape of the trochlea must also be taken into account in the correction of the TT-TG: the deeper the trochlea, the greater the risk of overmedialization, which would result in patellar impingement on the medial facet of the trochlea, and pain.

### **Distal Tibial Tubercle Transfer**

This technique is indicated only in cases of patella alta and consists of transferring distally the tibial tuberosity with a bone block 5 cm long. This block is fixed with two compression screws. The post-operative care is the same as in the Maquet's and Elmslie's procedures.

To avoid the risk of creating an iatrogenic patella infera, pre-operatively, the surgeon must calculate the patellar height by the Insall-Salvati or Caton-Deschamps index. This procedure is not useful in evolved cases of patellofemoral arthrosis.

Currently, the anterior transfer of tibial tuberosity is not recommended for patellofemoral arthrosis. And the distal and medial transfer of tibial tuberosity are mainly indicated for PF instability, but not for PF osteoarthritis.

### **Patellofemoral Arthroplasty**

McKeever, in 1955 [86], was the first to report such an operation when he described fixing a metallic implant (Vitallium) to the undersurface of the patella by way of a transverse screw. In 1979, Lubinus [76] and Blazina et al. [14] introduced separately the concept of a patellofemoral replacement whereby both the patella and the trochlea were re-surfaced. At this moment, patellofemoral replacement surgery is not been widely accepted. The original descriptions of the procedure did not include strict criteria, technical pitfalls were not fully appreciated, and little emphasis was placed on re-alignment of the extensor mechanism of the knee. The Lubinus prosthesis was reported to have a 50% failure rate at 8 years in a study of 76 cases [118]. Recently, the literature has shown more encouraging results. In 1995, Argenson et al. [8] reported the results of 66 replacements reviewed at an average of 5.5 years. Dividing their results by etiology, they noted a good result in 20 of 22 patients with mal-alignment and dysplasia, 19 of 20 patients with post-traumatic arthritis, and 17 of 24 patients with arthritis of unknown aetiology. They recommend the use of PF arthroplasty especially in arthrosis secondary to patellar fracture. In 1996, Krajca-Radcliffe and Coker [68] in a series of 16 patellofemoral replacements, followed for an average of 5.8 years, reported good-to excellent results in

fifteen cases. Kooijman et al. [67] followed 45 patients for 10–21 years after patellofemoral arthroplasty. Two-thirds of the 45 patients still had the patellofemoral replacement at the time of final follow-up (average of 17 years). However, fifteen patients required another operation (most commonly a soft-tissue operation such as a lateral retinacular release) soon after the index procedure, and 12 underwent either a tibial osteotomy or a total knee replacement at an average of 15 years. In a study of 72 patellofemoral arthroplasties, 61 of these 72 knees (75%) needed some form of re-alignment procedure [19]. Other series of patellofemoral arthroplasty have found that patellofemoral arthritis associated with patella mal-positioning was the most common clinical presentation in these patient groups [6, 14].

Recently, Cartier et al. [20] retrospectively reviewed 79 patellofemoral arthroplasties carried out between 1975 and 1991. The author found that 75% of the prostheses were still functioning at a minimum of six and average follow-up of 10 years after implantation and the most frequent cause of failure was secondary femorotibial osteoarthritis (eight knees). They concluded that “the main indication for this type of implant is advanced patellofemoral osteoarthritis secondary to a constitutional trochlear dysplasia occurring in a correctly-aligned knee for which no other therapeutic procedure allows the same type of result to be achieved”.

In 2005, Leadbetter et al. [72] presented a study based in a complete Medline review of all published articles of patellofemoral arthroplasty between 1979 and 2005. They found 12 articles in which were analyzed the indications, contra-indications, and poor results. They found six consensus criteria indications to perform PF arthroplasty that Sisto and Sarin [113] refined as:

- Degenerative or post-traumatic osteoarthritis limited to the patellofemoral joint.
- Severe symptoms affecting daily activity referable to patellofemoral joint degeneration and unresponsive to 6 months of non-operative treatment and failed prior conservative procedures.
- Failure of a previous surgical procedure to unload the extensor.
- Patellofemoral malalignment or dysplasia-induced degeneration with or without instability.

In this study [72], the most important contra-indications found in the literature were:

- No attempt at non-operative care or exclusion of other sources of pain.
- Arthritis involving the tibiofemoral articulation greater than Kellgren Grade I.
- Systemic inflammatory arthropathy.
- Patella infera.

- Uncorrected patellofemoral instability/mal-alignment; Uncorrected tibiofemoral mechanical mal-alignment (valgus  $>8^\circ$ ; varus  $>5^\circ$ ).
- Active infection.
- Evidence of chronic regional pain syndrome and fixed loss of knee range of motion ( $-10^\circ$  extension  $-110^\circ$  flexion minimum).

The authors added some additional factors that may adversely affect patellofemoral arthroplasty outcome:

- Multiple antecedent procedures or extensive soft tissue trauma associated with residual quadriceps atrophy.
- History of previous arthrofibrosis in the same joint.
- Ligamentous tibiofemoral instability.
- Post-meniscectomy situation.
- Chondrocalcinosis.
- High patient activity.
- Age younger than 40 years.
- Obesity.
- Patella alta.
- Primary osteoarthritis.
- Male gender.

Also, they included unrealistic patient expectations and a surgeon with lack of experience in arthroplasty or extensor mechanism re-alignment.

The poor results reported after patellofemoral replacement have two common themes: failure to appreciate present or incipient femorotibial arthritis and extensor mechanism disorders not diagnosed before or corrected at the time of arthroplasty surgery.

### Total Knee Arthroplasty

The use of total knee arthroplasty for the treatment of isolated patellofemoral arthritis has been confirmed as an effective method of managing this condition in the older patients [71, 91]. The exact age at which total knee arthroplasty becomes a viable option for the treatment of patellofemoral arthritis is debatable and case-dependent, but certainly an age younger than 55 years should be considered a relative contra-indication [91]. Probably, it can be properly used with reliable results in patients in their eighth decade of life.

In a study of 53 patients who had been followed for an average of 7.4 years after total knee replacement with re-surfacing of the patella to treat isolated patellofemoral arthritis, Laskin and van Steijn [71] noted an average range of motion of  $122^\circ$ , compared with  $117^\circ$  in a separate cohort of patients with tricompartmental disease, with 81% (43) of the 53 patients having a good-to-excellent result.

Mont et al. [91] in a series of 33 replacements for primary patellofemoral arthritis reported 28 excellent results, one good result, and one poor result at a mean of 81 months post-operatively. The mean Knee Society objective score increased from 50 points pre-operatively to 93 points post-operatively.

If the complications involving the extensor mechanism and the patellofemoral joint remain the primary non-infectious indications for revision total knee arthroplasty, in these patients the particular attention directed toward the correction of extensor mechanism is mandatory. This is evidenced by the reported rates of retinacular release in these patients, which are as high as 68% [71, 75, 81], a threefold increase compared with the rates associated with standard total knee arthroplasty. Parvizi et al. [98] evaluated the results of 31 knee replacements in 24 patients at an average of 5 years. They noted that 21 patients required a lateral retinacular release and three more required a more extensive realignment.

There are some critical points in the TKA procedure in which surgeon must pay attention to avoid extensor mechanism problems in the post-operative results:

- Avoid an internal position of Tibial insert that will mean an external position of the tibial tubercle.
- Avoid internal rotation of femoral insert that may result a hyperpression between the patella component and the lateral condyle. Internal rotation of the femoral component with respect to the femur will rotate the trochlear groove medially, increase tension on the lateral retinaculum, and cause lateral patellar mal-tracking.
- Avoid medialization of the femoral insert that may produce a conflict between the lateral flange of the femoral component and the lateral border of the patellar component, resulting in patellar subluxation.
- Avoid lateral release that can produce damage to the vascular supply and consequent patellar necrosis and fractures.
- Avoid laterally-placed patellar component that will increase the tension in the lateral retinaculum and cause lateral patellar mal-tracking. The central position requires more lateral release than the medial position.
- Avoid “overstuffing” of the patellofemoral joint. This can occur by use of femoral component that is too large (in the anteroposterior dimension) or by resecting an inadequate amount of the patella increasing tension on the lateral retinaculum

Re-surfacing of the patella in total knee replacement is a controversial issue, even when the patella is arthritic. The current literature seems to favour patellar re-surfacing.

Multiple studies have demonstrated success with patellar re-surfacing, with good relief of pain and good overall outcomes [43, 58]. However, other studies have shown success without insertion of a patellar component. Burnett et al. [18] showed in a RCT the same results with re-surfacing/no re-surfacing patella in a series of 64 knees after 10 years of follow-up. Ideally, a patient treated without patellar re-surfacing should have no patellar arthritis, and a femoral component that was designed to accommodate the native patella should be utilized. In PF arthrosis, where frequently there is a severe deformity on the articular side of patella, the use of patellar component has an advantage because it allows improvement the PF tracking, placing the patellar insert medially.

Three reports on this issue have noted a high rate of residual post-operative patellar tilt, asymmetrically re-surfaced patellae, and residual subluxation [72, 91, 97] reflecting the technical complexity inherent in these cases.

Despite the success of total knee arthroplasty, many surgeons consider it to be excessive sacrifice of healthy tissue and too great a surgical dissection for a patient with disease involving mainly one compartment. However, total knee arthroplasty currently remains the most proven and predictable single procedure for this specific population of older patients with patellofemoral disease.

Total Knee arthroplasty is also the best procedure to rescue patellofemoral surgery performed previously. Basically, there are two situations in which TKA must be performed as a revision: Failure of previous surgical treatment which could not achieve the expected results with permanence of pain and discomfort in the PF joint, and cases in which the degenerative disease involves the tibiofemoral joint. In both cases, the previous procedure may make the surgery of TKA difficult. The surgeon must plan carefully the revision surgery in order to avoid pitfalls that may increase the risk of a new failure. This is important in two situations: previous PF arthroplasty and previous Osteotomy of Tibial tuberosity.

---

## Overview

The surgeon must make an accurate diagnosis of Isolated Patellofemoral Osteoarthritis and its cause extensor mechanisms mal-alignment, Patellofemoral joint trauma or no articular cause.

Assess the extent of Tibiofemoral degenerative disease. Reject global OA with inflammatory synovium.

In Patellofemoral Instability with moderate degenerative disease treat the patellar instability through proximal and/or distal re-alignment.

In later Patellofemoral evolved disease with a collapsed joint, patellofemoral arthroplasty may play a useful role in selected cases.

In cases with global inflammatory synovium, Tibiofemoral joint involvement or older patients the Total Knee Arthroplasty is probably the most effective treatment.

---

## References

1. Aderinto J, Cobb AG (2002) Lateral release for patellofemoral arthritis. *Arthroscopy* 18:399–403
2. Aglietti P, Insall JN, Cerulli G (1983) Patellar pain and incongruence. I. Measurements of incongruence. *Clin Orthop Relat Res* 176:217–224
3. Ahlbäch S (1968) Osteoarthritis of the knee: a radiographic investigation. *Acta Radiologica Suppl* 277:1–61
4. Amis AA, Farahmand F (1996) Biomechanics of the knee extensor mechanism. *Knee* 3:73–81
5. Amis AA, Senavongse W, Darcy P (2005) Biomechanics of patellofemoral joint prostheses. *Clin Orthop Relat Res* 436:20–29
6. Arciero RA, Toomey H (1988) Patellofemoral arthroplasty: a three-to nine-year follow-up study. *Clin Orthop Relat Res* 236:60–71
7. Arent EA, Fithian DC, Cohen E (2002) Current concepts of lateral patella dislocation. *Clin Sports Med* 21:499–519
8. Argenson JN, Guillaume JM, Aubaniac JM (1995) Is there a place for patellofemoral arthroplasty? *Clin Orthop Relat Res* 321:162–167
9. Ateshian GA, Hung CT (2005) Patellofemoral joint biomechanics and tissue engineering. *Clin Orthop Relat Res* 436:81–90
10. Baker CL, Hughston JC (1988) Miyakawa patellectomy. *J Bone Joint Surg [Am]* 70:1489–1494
11. Bartlett W, Gooding CR, Carrington RW, Briggs TW, Skinner JA, Bentley G (2005) The role of the Short Form 36 Health Survey in autologous chondrocyte implantation. *Knee* 12:281–285
12. Becker R, Röpke M, Krull A, Musahl V, Nebelung W (2008) Surgical treatment of isolated patellofemoral osteoarthritis. *Clin Orthop Relat Res* 466:443–449
13. Bhattacharya R, Kumar V, Safawi E, Finn P, Hui AC (2007) The knee skyline radiograph: its usefulness in the diagnosis of patello-femoral osteoarthritis. *Inter Orthop* 31:247–252
14. Blazina ME, Fox JM, Del Pizzo W, Broukhim B, Ivey FM (1979) Patellofemoral replacement. *Clin Orthop Relat Res* 144:98–102
15. Brittberg M, Lindahl A, Nilsson A, Ohlsson C, Isaksson O, Peterson L (1994) Treatment of deep cartilage defects in the knee with autologous chondrocyte transplantation. *N Engl J Med* 331:889–895
16. Buckland-Wright JC (1995) Protocols for precise radio-anatomical positioning of the tibiofemoral and patellofemoral compartments of the knee. *Osteoarthritis Cartilage* 3 Suppl A: 71–80

17. Bull AMJ, Katchburian MV, Shih Y-F, Amis AA (2002) Standardisation of the description of patellofemoral motion and comparison between different techniques. *Knee Surg Sports Traumatol Arthrosc* 10:184–193
18. Burnett RSJ, Boone JL, McCarthy KP, Rosenzweig S, Barrack RL (2007) A prospective randomized clinical trial of patellar resurfacing and nonresurfacing in bilateral TKA. *Clin Orthop Relat Res* 464:65–72
19. Cartier P, Sanouiller JL, Grelsamer RP (1990) Patellofemoral arthroplasty: 2-12-year follow-up study. *J Arthroplasty* 5:49–55
20. Caton Ph, Sanouiller JL, Khefacha A (2005) Long-term results with the first patellofemoral prosthesis. *Clin Orthop Relat Res* 436:47–54
21. Casscells SW (1978) Gross pathologic changes in the knee joint of the aged individual: a study of 300 cases. *Clin Orthop Relat Res* 132:225–232
22. Caton J, Deschamps G, Chambat P, Lerat JL, Dejour H (1982) Patella infera. Apropos of 128 cases. *Rev Chir Orthop* 68:317–325
23. Chaisson CE, Gale DR, Gale E, Kazis L, Skinner K, Felson DT (2000) Detecting radiographic knee osteoarthritis: what combination of views is optimal? *Rheumatology (Oxford)* 39:1218–1221
24. Cicuttini FM, Baker J, Hart DJ, Spector TD (1996) Choosing the best method for radiological assessment of patellofemoral osteoarthritis. *Ann Rheum Dis* 55:134–136
25. Cicuttini F, Baker JR, Spector TD (1996) The association of obesity with osteoarthritis of the hand and knee in woman: a twin study. *J Rheumatol* 23:1221–1226
26. Clar C, Cummins E, McIntyre L, Thomas S, Lamb J, Bain L, Jobanputra P, Waugh N (2005) Clinical and cost-effectiveness of autologous chondrocyte implantation for cartilage defects in knee joints: systematic review and economic evaluation. *Health Technol Assess* 9: iii–iv, ix–x, 1–82
27. Cohen ZA, Mow VC, Henry JH, Levine WN, Ateshian GA (2003) Templates of the cartilage layers of the patellofemoral joint and their use in the assessment of osteoarthritic cartilage damage. *Osteoarthritis Cartilage* 11:569–579
28. Colvin AC, West RV (2008) Patellar instability: current concepts review. *J Bone Joint Surg [Am]* 90:2751–2762
29. Cooper C, McAlindon T, Snow S, Vines K, Young P, Kirwan J, Dieppe P (1994) Mechanical and constitutional risk factors for symptomatic knee osteoarthritis: differences between medial tibiofemoral and patellofemoral disease. *J Rheumatol* 21:307–313
30. Crossley KM, Vicenzino B, Pandey MG, Schache AG, Hinman RS (2008) Targeted physiotherapy for patellofemoral joint osteoarthritis: a protocol for a randomised, single-blind controlled trial. *BMC Musculoskeletal Disorders* 9:122–130
31. Curl WW, Krome J, Gordon ES, Rushing J, Smith BP, Poehling GG (1997) Cartilage injuries: a review of 31, 516 knee arthroscopies. *Arthroscopy* 13:456–460
32. Davies AP, Vince AS, Shepstone L, Donell ST, Glasgow MM (2002) The radiologic prevalence of patellofemoral osteoarthritis. *Clin Orthop Relat Res* 402:206–212
33. Dawson J, Juszcak E, Thorogood M, Marks S, Dodd C, Fitzpatrick R (2003) An investigation of risk factors for symptomatic osteoarthritis of the knee in women using a life course approach. *J Epidemiol Community Health* 57:823–830
34. Dejour D, Allain J (2004) Histoire naturelle de l'arthrose fémoro-patellaire isolée. *Rev Chir Orthop* 90 (suppl 5):1S69–1S129 [in French]
35. Dejour D, Le Coultre B (2007) Osteotomies in patellofemoral instabilities. *Sports Med Arthrosc* 15:39–46
36. Dejour H, Walch G, Neyret P, Adeleine P (1990) La dysplasie de la trochlée fémorale. *Rev Chir Orthop* 76:45–54 [in French]
37. Dejour H, Walch G, Nove-Josserand L, Guier Ch (1994) Factors of patellar instability: an anatomic radiographic study. *Knee Surg Sports Traumatol Arthrosc* 2:19–26
38. Dellon AL, Mont MA, Mullick T, Hungerford DS (1996) Partial denervation for persistent neuroma pain around the knee. *Clin Orthop Relat Res* 329:216–222
39. Desio SM, Burks RT, Bachus KN (1998) Soft tissue restraints to lateral patellar translation in the human knee. *Am J Sports Med* 26:59–65
40. Felson DT, Naimark A, Anderson J, Kazis L, Castelli W, Meenan RF (1987) The prevalence of knee osteoarthritis in the elderly. The Framingham Osteoarthritis Study. *Arthritis Rheum* 30:914–918
41. Ferrandez L, Usabiaga J, Yubero J, Sagarra J, de No L (1989) An experimental study of the redistribution of patellofemoral pressures by the anterior displacement of the anterior tuberosity of the tibia. *Clin Orthop Relat Res* 238:183–189
42. Ficat P, Ficat C, Bailleux A (1975) [External hypertension syndrome of the patella. Its significance in the recognition of arthrosis]. *Rev Chir Orthop Reparatrice Appar Mot* 61:39–59 [in French]
43. Forster MC (2004) Patellar resurfacing in total knee arthroplasty for osteoarthritis: a systematic review. *Knee* 11:427–430
44. Fox JM, Del Pizzo W (1993) The patellofemoral joint. McGraw-Hill, New York, pp 123–175
45. Freeman BL III (1987) Recurrent dislocations. In: Crenshaw AH (ed) Campbell's operative orthopaedics, 7th edn. CV Mosby, St. Louis, pp 2173–2218
46. Fulkerson JP (2005) Alternatives to patellofemoral arthroplasty. *Clin Orthop Relat Res* 436:76–80
47. Fulkerson JP, Becker GJ, Meaney JA, Miranda M, Folcik MA (1990) Anteromedial tibial tubercle transfer without bone graft. *Am J Sports Med* 18:490–497
48. Fulkerson JP, Shea KP (1990) Disorders of patellofemoral alignment. *J Bone Joint Surg [Am]* 77:1424–1429
49. Galtier B, Buillot M, Vanneville G (1995) Anatomical basis of the role of vastus medialis muscle in patellofemoral degenerative arthropathy. *Surg Radiol Anat* 17:7–11
50. Gobbi A, Kon E, Berruto M, Filardo G, Delcogliano M, Boldrini L, Bathan L, Marcacci M (2009) Patellofemoral full-thickness chondral defects treated with second-generation autologous chondrocyte implantation. *Am J Sports Med* 37:1083–1092

51. Goodfellow J, Hungerford DS, Zindel M (1976) Patellofemoral joint mechanics and pathology. Functional anatomy of the patello-femoral joint. *J Bone Joint Surg [Br]* 58:287–290
52. Goutallier D, Bernageau J (1999) Le point sur la TA-GT. In: Goutallier D (ed) *La pathologie femoropatellaire*, vol. 71. Expansion Scientifique Publications, Paris, pp 175–182
53. Goutallier D, Bernageau J, Lecudonnet B (1978) The measurement of the tibial tuberosity. Patella groove distanced technique and results (author's transl). *Rev Chir Orthop Reparatrice Appar Mot* 64:423–428
54. Grelsamer RP, Weinstein CH (2001) Applied biomechanics of the patella. *Clin Orthop Relat Res* 389:9–14
55. Grood ES, Suntay WJ (1983) A joint coordinate system for the clinical description of three-dimensional motions: application to the knee. *J Biomech Eng* 105:136–144
56. Günal I, Taymaz A, Köse N, Göktürk E, Seber S (1997) Patellectomy with vastus medialis obliquus advancement for comminuted patellar fractures: a prospective randomised trial. *J Bone Joint Surg [Br]* 78:13–16
57. Hanna F, Teichtahl AJ, Wluka AE, Wang Y, Urquhart DM, English DR, Giles GG, Cicuttini FM (2009) Women have increased rates of cartilage loss and progression of cartilage defects at the knee than men: a gender study of adults without clinical knee osteoarthritis. *Menopause* 16:624–625
58. Holt GE, Dennis DA (2003) The role of patellar resurfacing in total knee arthroplasty. *Clin Orthop Relat Res* 416:76–83
59. Hughston JC (1968) Subluxation of the patella. *J Bone Joint Surg [Am]* 50:1003–1026
60. Hughston JC, Walsh WM (1979) Proximal and distal reconstruction of the extensor mechanism for patellar subluxation. *Clin Orthop Relat Res* 144:36–42
61. Insall J, Bullough PG, Burstein AH (1979) Proximal “tube” realignment of the patella for chondromalacia patellae. *Clin Orthop Relat Res* 144:63–69
62. Insall JN, Scott WN (2001) *Surgery of the knee*, 3rd edn. Churchill Livingstone, Philadelphia, pp 161
63. Irrgang JJ, Anderson AF, Boland AL, Harner CD, Kurosaka M, Neyret P, Richmond JC, Shelborne KD (2001) Development and validation of the International Knee Documentation Committee Subjective Knee Form. *Am J Sports Med* 29:600–613
64. Iwano T, Kurosawa H, Tokuyama H, Hoshikawa Y (1990) Roentgenographic and clinical findings of patellofemoral osteoarthritis. *Clin Orthop Relat Res* 253:190–197
65. Kellgren JH, Lawrence JS (1957) Radiological assessment of osteoarthritis. *Ann Rheum Dis* 16:494–502
66. Kelly MA, Insall JN (1986) Patellectomy. *Orthop Clin North Am* 17:289–295
67. Kooijman HJ, Driessen AP, van Horn JR (2003) Long-term results of patellofemoral arthroplasty. A report of 56 arthroplasties with 17 years of follow-up. *J Bone Joint Surg [Br]* 85:836–840
68. Krajca-Radcliffe JB, Coker TP (1996) Patellofemoral arthroplasty. A 2- to 18-year followup study. *Clin Orthop Relat Res* 330:143–151
69. Kujala UM, Jaakkola LH, Koskinen SK, Taimela S, Hurme M, Nelimarkka O (1993) Scoring of patellofemoral disorders. *Arthroscopy* 9:159–163
70. Lanyon P, Jones A, Doherty M (1996) Assessing progression of patellofemoral osteoarthritis: a comparison between two radiographic methods. *Ann Rheum Dis* 55:875–879
71. Laskin RS, van Steijn M (1999) Total knee replacement for patients with patellofemoral arthritis. *Clin Orthop Relat Res* 367:89–95
72. Leadbetter WB, Ragland PS, Mont MA (2005) The appropriate use of patellofemoral arthroplasty: an analysis of reported indications, contraindications and failures. *Clin Orthop Relat Res* 436:91–99
73. Lennox IA, Cobb AG, Knowles J, Bentley G (1994) Knee function after patellectomy. A 12- to 48-year follow-up. *J Bone Joint Surg [Br]* 76:485–487
74. Lewallen DG, Riegger CL, Myers ER, Hayes WC (1990) Effects of retinacular release and tibial tubercle elevation in patellofemoral degenerative joint disease. *J Orthop Res* 8:856–862
75. Lewonowski K, Dorr LD, McPherson EJ, Huber G, Wan Z (1997) Medialization of the patella in total knee arthroplasty. *J Arthroplasty* 12:161–167
76. Lubinus HH (1979) Patella glide bearing total replacement. *Orthopedics* 2:119–127
77. Maenpaa H, Lehto MU (1997) Patellar dislocation. The long-term results of nonoperative management in 100 patients. *Am J Sports Med* 25:213–217
78. Maenpaa H, Lehto MU (1997) Patellofemoral osteoarthritis after patellar dislocation. *Clin Orthop Relat Res* 339:156–162
79. Maldague B, Malghem J (1985) Apport du cliché de profil du genou dans le dépistage des instabilités rotuliennes. Rapport préliminaire. [Significance of the radiograph of the knee profile in the detection of patellar instability. Preliminary report]. *Rev Chir Orthop Reparatrice Appar Mot* 71 (suppl 2):5–13 [in French]
80. Malghem J, Maldague B (1989) Depth insufficiency of the proximal trochlear groove on lateral radiographs of the knee: relation to patellar dislocation. *Radiology* 170:507–510
81. Malo M, Vince KG (2003) The unstable patella after total knee arthroplasty: etiology, prevention, and management. *J Am Acad Orthop Surg* 11:364–371
82. Maquet P (1963) Considerations biomechaniques sur l'arthrose du genou. Un traitement biomechanique de l'arthrose femoropatellaire: l'avancement du tendon rotulien. [A biomechanical treatment of femoro-patellar arthrosis: advancement of the patellar tendon]. *Rev Rhum Mal Osteoartic* 30:779–783 [French]
83. Martens M, De Rycke J (1990) Facetectomy of the patella in patellofemoral osteoarthritis. *Acta Orthop Belg* 56:563–567
84. McAlindon TE, Snow S, Cooper C, Dieppe PA (1992) Radiographic patterns of osteoarthritis of the knee joint in the community: The importance of the patellofemoral joint. *Ann Rheum Dis* 51:844–849
85. McAlindon T, Zhang Y, Hannan M, Naimark A, Weissman B, Castelli W, Felson D (1996) Are risk factors for patellofemoral and tibiofemoral knee osteoarthritis different? *J Rheumatol* 23:332–337
86. McKeever DC (1955) Patellar prosthesis. *J Bone Joint Surg [Am]* 37:1074–1084



87. Merchant AC, Mercer RL (1974) Lateral release of the patella. A preliminary report. *Clin Orthop Relat Res* 139:40–45
88. Merchant AC, Mercer RL, Jacobsen RH, Cool CR (1974) Roentgenographic analysis of patellofemoral congruence. *J Bone Joint Surg [Am]* 56:1391–1396
89. Merchant AC, Mercer RL, Jacobsen RH, Cool CR (1974) Roentgenographic analysis of patellofemoral congruence. *J Bone Joint Surg [Am]* 56:1391–1396
90. Minas T, Bryant T (2005) The role of autologous chondrocyte implantation in the patellofemoral joint. *Clin Orthop Relat Res* 436:30–39
91. Mont MA, Haas S, Mullick T, Hungerford DS (2002) Total knee arthroplasty for patellofemoral arthritis. *J Bone Joint Surg [Am]* 84:1977–1981
92. Montserrat F (2009) Non prosthetic treatment of isolated patellofemoral arthrosis. In Symposium: Patellofemoral arthrosis at the 10th Congress of EFFORT; Vienna
93. Morscher E (1978) Osteotomy of the patella in chondromalacia. Preliminary report. *Arch Orthop Trauma Surg* 92:139–147
94. Neame RL, Muir K, Doherty S, Doherty M (2004) Genetic risk of knee osteoarthritis: a sibling study. *Ann Rheum Dis* 63:1022–1027
95. Nikku R, Nietosvaara Y, Kallio PE, Aalto K, Michelsson JE (1997) Operative versus closed treatment of primary dislocation of the patella. Similar 2-year results in 125 randomized patients. *Acta Orthop Scand* 68:419–423
96. Noble J, Hamblen DL (1975) The pathology of the degenerate meniscus lesion. *J Bone Joint Surg [Br]* 57:180–186
97. Nove-Josserand L, Dejour D (1995) Quadriceps dysplasia and patellar tilt in objective patellar instability. *Rev Chir Orthop Reparatrice Appar Mot* 81:497–504
98. Parvizi J, Stuart MJ, Pagnano MW, Hanssen AD (2001) Total knee arthroplasty in patients with isolated patellofemoral arthritis. *Clin Orthop Relat Res* 392:147–152
99. Paxton EW, Fithian DC (2005) Outcome instruments for patellofemoral arthroplasty. *Clin Orthop Relat Res* 436:66–70
100. Peterson L, Brittberg M, Kiviranta I, Akerlund EL, Lindahl A (2002) Autologous chondrocyte transplantation. Biomechanics and long-term durability. *Am J Sports Med* 30:2–12
101. Peterson L, Minas T, Brittberg M, Nilsson A, Sjogren-Jansson E, Lindahl A (2000) Two- to 9-year outcome after autologous chondrocyte transplantation of the knee. *Clin Orthop Relat Res* 374:212–234
102. Post WR, Teitge R, Amis A (2002) Patellofemoral malalignment: looking beyond the view box. *Clin Sports Med* 21:521–546
103. Quilty B, Tucker M, Campbell R, Dieppe P (2003) Physiotherapy, including quadriceps exercises and patellar taping, for knee osteoarthritis with predominant patellofemoral joint involvement: randomized controlled trial. *J Rheum* 30:1311–1317
104. Remy F, Besson A, Gougeon F, Migaud H, Duquenoy A (1999) Evaluation fonctionnelle de l'instabilité femoro-patellaire par un score 100 points. [Functional evaluation of patello-femoral instability using a 100 point scale]. *Rev Chir Orthop 85(suppl III):92–93* [in French]
105. Roos EM, Roos HP, Ekdahl C, Lohmander LS (1998) Knee injury and Osteoarthritis Outcome Score (KOOS) – validation of a Swedish version. *Scand J Med Sci Sports* 8:439–448
106. Rosenberg TD, Paulos LE, Parker RD, Coward DB, Scott SM (1988) The forty-five degree posteroanterior flexion weight-bearing radiograph of the knee. *J Bone Joint Surg [Am]* 70:1479–1483
107. Saleh KJ, Arendt EA, Eldridge J, Fulkerson JP, Minas T, Mulhall KJ (2005) Operative treatment of patellofemoral arthritis. *J Bone Joint Surg [Am]* 87:659–671
108. Sanders TG, Morrison WB, Singleton BA, Miller MD, Cornum KG (2001) Medial patellofemoral ligament injury following acute transient dislocation of the patella: MR findings with surgical correlation in 14 patients. *J Comput Assist Tomogr* 25:957–962
109. Scuderi G, Cuomo F, Scott WN (1988) Lateral release and proximal realignment for patellar subluxation and dislocation. A long-term follow-up. *J Bone Joint Surg [Am]* 70:856–861
110. Senavongse W, Amis AA (2005) The effects of articular, retinacular, or muscular deficiencies on patellofemoral joint stability. *J Bone Joint Surg [Br]* 87:577–582
111. Shea KP, Fulkerson JP (1992) Preoperative computed tomography scanning and arthroscopy in predicting outcome after lateral retinacular release. *Arthroscopy* 8:327–334
112. Singerman R, White C, Davy DT (1995) Reduction of patellofemoral contact forces following anterior displacement of the tibial tubercle. *J Orthop Res* 13:279–285
113. Sisto DJ, Sarin VK (2007) Custom patellofemoral arthroplasty of the knee surgical technique. *J Bone Joint Surg [Am]* 89 Suppl2:214–225
114. Spector TD, Cicuttini F, Baker J, Loughlin J, Hart DJ (1996) Genetic influences on osteoarthritis in women: a twin study. *BMJ* 312:940–943
115. Spector TD, MacGregor AJ (2004) Risk factors for osteoarthritis: genetics. *Osteoarthr Cartil* 12(suppl A): S39–S44
116. Staubli HU, Durrenmatt U, Porcellini B, Rauschnig W (1999) Anatomy and surface geometry of the patellofemoral joint in the axial plane. *J Bone Joint Surg [Br]* 81: 452–458
117. Szebenyi B, Hollander AP, Dieppe P, Quilty B, Duddy J, Clarke S, Kirwan J (2006) Associations between pain, function, and radiographic features in osteoarthritis of the knee. *Arthritis Rheum* 54:230–235
118. Tauro B, Ackroyd CE, Newman JH, Shah NA (2001) The Lubinus patellofemoral arthroplasty. A five- to ten-year prospective study. *J Bone Joint Surg [Br]* 83:696–701. Erratum in: *J Bone Joint Surg [Br]* 2002; 84:308
119. Tavernier T, Dejour D (2001) Knee imaging: what is the best modality. *J Radiol* 82(3 Pt 2):387–405; 407–408
120. Tecklenburg K, Dejour D, Hoser C, Fink C (2006) Bony and cartilaginous anatomy of the patellofemoral joint. *Knee Surg Sports Traumatol Arthrosc* 14:235–240
121. Trillat ADH, Couette A (1964) Diagnostic et traitement des subluxations récidivantes de la rotule. *Rev Chir Orthop Reparatrice Appar Mot* 50:813–824 [French]
122. Vaquero J, Arriaza R (1992) The patella thinning osteotomy. An experimental study of a new technique for reducing patellofemoral pressure. *Int Orthop* 16:372–376

123. Vuorinen O, Paakkala T, Tuuturii T (1985) Chondromalacia patellae: results of operative treatments. *Arch Orthop Trauma Surg* 104:175–181
124. West F, Soto-Hall R (1985) Recurrent dislocation of the patella in the adult: end results of patellectomy with quadricepsplasty. *J Bone Joint Surg [Am]* 40:386–394
125. Wiberg G (1941) Roentgenographic and anatomic studies on the femoropatellar joint. *Acta Orthop Scand* 12:319–410
126. Yercan HS, Ait Si Selmi T, Neyret P (2005) The treatment of patellofemoral osteoarthritis with partial lateral facetectomy. *Clin Orthop Relat Res* 436:14–19
127. Zaricznyj B (1972) Reconstruction of the quadriceps mechanism after patellectomy. *J Bone Joint Surg [Am]* 54:1583–1584
128. Zarins B (2005) Are validated questionnaires valid? *J Bone Joint Surg [Am]* 87:1671–1672

## Part IX

---

# Foot and Ankle

# Achilles Tendinopathy

Nicola Maffulli and Francesco Oliva

## Introduction

Achilles tendinopathy mainly arises from overuse, but is often present in middle-aged overweight patients with no history of increased physical activity [71]. Its natural history in patients who try and continue to exercise is of gradually increasing load-related localized pain coinciding with increased activity. Examination should include thorough inspection to assess for swelling and asymmetry of the involved tendons, range of motion testing, palpation for tenderness, and examination manoeuvres that simulate tendon loading and reproduce pain [125].

In the past three decades, the incidence of overuse injury in sports has risen enormously [107], with more people undertaking recreational and competitive sports, but training for longer and more intensely [55, 93]. Excessive repetitive overload of the Achilles tendon is regarded as the main pathological stimulus that leads to tendinopathy [6]. Achilles tendinopathy is also common among athletes participating in racquet sports, track and field, volleyball and soccer [42, 126]. Achilles tendinopathy is not always associated with excessive physical activity, and in a series of 58 Achilles tendinopathy patients, 31% did not participate in sports or vigorous physical activity [101].

Also, the use of quinolone antibiotics is associated with Achilles tendinopathy and rupture [17, 103]. The management of Achilles tendinopathy lacks evidence-based support, and tendinopathy sufferers are at risk of long-term morbidity with unpredictable clinical outcome [45].

---

N. Maffulli (✉)

Centre Lead and Professor of Sports and Exercise Medicine, Consultant Trauma and Orthopaedic Surgeon, Queen Mary University of London, Barts and The London School of Medicine and Dentistry, Institute of Health Sciences Education, Centre for Sports and Exercise Medicine, Mile End Hospital, 275 Bancroft Road, London E1 4DG, England  
e-mail: n.maffulli@qmul.ac.uk

## Anatomy

The confluence of the gastrocnemius and soleus muscles forms the Achilles tendon. The gastrocnemius is more superficial and originates from two heads above the knee. The soleus is anterior to the gastrocnemius, and originates below the knee [96]. The plantaris muscle, present in approximately 90% of the population, has a short muscle belly of 7–10 cm, arises just below the lateral head of gastrocnemius, and has a long slender tendon that runs medial to the Achilles tendon [67]. The Achilles tendon inserts into the middle part of the posterior surface of the calcaneus, a bursa being interposed between the tendon and the upper part of this surface. The tendon spreads out somewhat at its lower end, so that its narrowest part is about 4 cm above its insertion. It is covered by the fascia, and stands out prominently behind the bone; the gap between the anterior aspect of the tendon and the fascia covering the muscle belly of the flexor hallucis longus is filled with areolar and adipose tissue, Kager's triangle. Along its lateral aspect, but superficial to it, is the small saphenous vein [28]. The Achilles tendon derives its sensory nerve supply from the nerves of the attaching muscles and cutaneous nerves, in particular the sural nerve [44, 45, 112].

## Histology

Healthy tendons are brilliant white with a fibroelastic texture. Tendons demonstrate marked variation in form; they can be rounded cords, straplike bands, or flattened ribbons [12]. Within the extracellular matrix network, tenoblast and tenocytes constitute about 90–95% of the cellular elements of tendons. Tenoblasts are immature tendon cells. They are spindle-shaped and have numerous cytoplasmic organelles, reflecting their high metabolic activity. As they mature, tenoblasts become elongated and transform into tenocytes. Tenocytes have a lower nucleus-to-cytoplasm ratio than

tenoblasts, with decreased metabolic activity [47]. The remaining 5–10% of the cellular elements of tendons consists of chondrocytes at the bone attachment and insertion sites, synovial cells of the tendon sheath, and vascular cells, including capillary endothelial cells and smooth muscle cells of arterioles. Tenocytes are active in energy generation through the aerobic Krebs cycle, anaerobic glycolysis, and the pentose phosphate shunt, and they synthesize collagen and all components of the extracellular matrix network [56, 86]. Collagen constitutes about 90% of tendon protein, or approximately 70% of the dry weight of a tendon [44]. Collagen type I accounts for 65–80% and elastin accounts for approximately 2% of the dry mass of tendons [39, 86, 116]. Tenocytes and tenoblasts lie between the collagen fibres along the long axis of the tendon [51]. Collagen is arranged in hierarchical levels of increasing complexity, beginning with tropocollagen, a triple-helix polypeptide chain, which unites into fibrils; fibers (primary bundles); fascicles (secondary bundles); tertiary bundles; and the tendon itself [6, 85]. Aging significantly decreases tendon glycosaminoglycans and increases collagen concentration [118]. Acute exercise increases type I collagen formation in peritendinous tissue [58]. The essence of tendinopathy is a failed healing response, with haphazard proliferation of tenocytes, some evidence of degeneration in tendon cells and disruption of collagen fibres, and subsequent increase in non-collagenous matrix [44, 61, 63, 71, 84]. Tendinopathic lesions affect both collagen matrix and tenocytes [43, 62]. Normally, collagen fibres in tendons are tightly bundled in a parallel fashion. In tendinopathic samples, there is unequal and irregular crimping, loosening and increased waviness of collagen fibres, with an increase in Type III (reparative) collagen [38, 43, 69, 70, 122].

At electron microscopy, various types of degeneration have been described, namely (a) hypoxic degeneration, (b) hyaline degeneration, (c) mucoid or myxoid degeneration, (d) fibrinoid degeneration, (e) lipoid degeneration, (f) calcification, (g) fibrocartilaginous and bony metaplasia [44, 64, 94]. All can co-exist, depending on the anatomical site and the nature of their causal insult. Therefore, tendinopathy can be considered the end result of a number of etiologic processes with a relatively narrow spectrum of histopathological features [71–73].

---

## Physiopathology

The aetiology of Achilles tendinopathy is still unclear [6, 55]. Tendinopathies have been linked to overuse, poor vascularity, lack of flexibility, genetic make up, sex, and endocrine or metabolic factors [50]. Excessive loading of

the tendon during vigorous training activities is regarded as the main pathological stimulus [42]. The Achilles tendon may respond to repetitive overload beyond physiological threshold by either inflammation of its sheath or degeneration of its body, or by a combination of the two [11]. Damage to the tendon can occur even if it is stressed within its physiological limits when the cumulative microtrauma applied to it do not leave enough time for repair [107]. Microtraumas can result from non-uniform stress within the Achilles tendon from different individual force contributions of the gastrocnemius and soleus, producing abnormal load concentrations within the tendon and frictional forces between the fibrils, with localised fibre damage [5]. Tendinopathy has been attributed to a variety of intrinsic and extrinsic factors, which, alone or in combination, has been put forward to try and explain overuse injuries [65]. Extrinsic factors such as increased frequency and duration of training has been associated with Achilles tendinopathy [29]. However, a retrospective study of partially ruptured Achilles tendons demonstrated that tendinopathy was not necessarily associated with the level of physical activity [7]. Poor technique, footwear, hard, slippery or uneven surfaces can predispose to Achilles tendinopathy [42]. Intrinsic factors can be further classified into alteration in lower limb function, biomechanics, gender, age and genetics. Weakness of the gastrocnemius and quadriceps muscles can alter the coordination of movements of the hip, knee and ankle joints during the kinetic chain, and not surprising are found in patients with tendinopathy, although it is unclear whether this is the cause or effect [54]. Higher ground-reaction forces are incurred with decreased ankle dorsiflexion, and have been associated with both Achilles and patellar tendinopathies [48, 77]. In a prospective study, decreased strength of the plantar flexors but increased dorsiflexion of the ankle was associated with greater risk of developing Achilles tendon overuse injury in male officer cadets [76]. A higher dorsiflexion range may subject the Achilles to greater strain. The over-pronated hindfoot and varus forefoot have been classically linked to Achilles tendinopathy [42, 48], but there is no modern evidence of a cause-effect relationship, and only an association relationship has been demonstrated. These studies are however dated, and have marked methodological flaws.

Gender and genetics have a role in tendinopathies. Large cohort studies of patellar tendinopathy demonstrated that more males than females had asymptomatic tendon pathology [22]. Oestrogen may have a protective effect on tendon pathology [21]. Polymorphism of the guanine-thymine dinucleotide repeat in the tenascin-C (an elastic extracellular matrix protein) gene is associated with Achilles tendinopathy [82]. Changes in the expression of genes such as

the down-regulation of metalloprotease 3 mRNA has been found in tendinopathic Achilles tendons with a resultant increase in the levels of type I and III collagens [40].

### Metalloproteases

Degradation of collagen and other ECM compounds is initiated by MMPs [97]. These are zinc- and calcium-dependent endopeptidases that are secreted from cells in pro-enzyme form [14]. MMPs are the major enzymes that are involved in remodelling of ECM because of their efficacy at neutral pH and their broad proteolytic capability against the ECM. The MMP family contains 23 members that have some characteristics in common [87]. MMPs are subdivided into four main classes: collagenases, gelatinases, stromelysins, and membrane-type MMPs (MT-MMPs) [18]. MMPs are involved in many physiologic remodelling processes, including wound healing, menstruation, uterine involution, bone growth and development, and angiogenesis [104, 123]. Also, MMPs play a role in pathologic processes, such as tumor invasion and metastasis [34, 117], multiple sclerosis [20], periodontal disease [13, 80], hypertension [99], and arthritis [16, 128]. The activity of MMPs is inhibited by tissue inhibitors of metalloproteases (TIMPs) [35, 36]. The balance between the activities of MMPs and TIMPs regulates tendon remodelling. An imbalance in MMPs and TIMPs is associated with collagen disturbances in tendons [26]. Cytokines, such as interleukin (IL)-1 and tumor necrosis factor (TNF)- $\alpha$  enhance the production of MMPs [27, 78], whereas transforming growth factor (TGF)- $\beta$  and IL-6 enhance the production of TIMP-1 [66, 127]. Physical exercise can influence local MMP and TIMP activities in the human Achilles tendon [52], with a pronounced increase in local levels of pro-MMP-9 after exercise. This study suggests that MMP-9 has a role in a potential inflammation reaction in human Achilles tendon that is induced by intensive exercise. Also, exercise causes a rapid increase in serum MMP-9 [53], probably as a result of increased leukocytes in the circulation [79]. Koskinen et al. [52] also observed that the MMP-2 inhibitory activity of TIMP-1 and TIMP-2 increased in response to exercise.

---

### Clinical Examination

Pain is the main symptom of Achilles tendinopathy. However, it should be understood that, even though patients may present acutely, it is likely that the histopathology, even in these instances, is already of a failed healing

response nature, bearing witness to the long standing process which eventually causes clinically relevant symptoms. Generally, pain occurs at the beginning and end of a training session, with a period of diminished discomfort in between. As the pathological process progresses, pain may occur during exercise, and, in severe cases, it may interfere with activities of daily living. In the acute phase, the tendon is diffusely swollen and oedematous, and on palpation tenderness is usually greatest 2–6 cm proximal to the tendon insertion. Sometimes, fibrin precipitated from the fibrinogen-rich fluid around the tendon can cause palpable crepitation. In chronic cases, exercise-induced pain is still the cardinal symptom, but crepitation and effusion diminish. A tender, nodular swelling is usually present in chronic cases, and is believed to signify tendinopathy.

The diagnosis of Achilles tendinopathy is mainly based on a careful history and detailed clinical examination. Diagnostic imaging may be required to verify a clinical suspicion or to exclude other musculoskeletal disorders, such as os trigonum syndrome, tenosynovitis or dislocation of the peroneal tendons, tenosynovitis of the plantar flexors, an accessory soleus muscle, tumours of the Achilles tendon (xanthomas), and neuroma of the sural nerve [124].

Clinical examination is the best diagnostic tool. Both legs are exposed from above the knees and the patient examined while standing and prone. The foot and the heel should be inspected for any malalignment, deformity, obvious asymmetry in the tendon size, localized thickening, Haglund heel and any previous scars. The Achilles tendon should be palpated for tenderness, heat, thickening, nodule and crepitation<sup>63</sup>. The tendon excursion is estimated to determine any tightness. The “painful arc” sign helps to distinguish between tendon and paratenon lesions. In paratenonopathy, the area of maximum thickening and tenderness remains fixed in relation to the malleoli from full dorsi- to plantar-flexion, whereas lesions within the tendon move with ankle motion. There is often a discrete nodule, whose tenderness significantly decreases or disappears when the tendon is put under tension [10].

---

### VISA-A

The VISA – A is a questionnaire based instrument to measure the severity of Achilles tendinopathy [100]. To develop the questionnaire, we performed item generation, item reduction, item scaling, and pretesting. We then tested its validity and reliability in clinical and control populations. The VISA-A questionnaire contained eight questions that covered the three domains of pain (questions 1–3), function

(questions 4–6), and activity (questions 7 and 8.) Questions 1–7 are scored out of 10, and question 8 carries a maximum of 30. Scores are summed to give a total out of 100. An asymptomatic person would score 100. For question 8, participants must answer only part A, B, or C. If the participant has pain when undertaking sport, he or she automatically loses at least 10, and possibly 20, points.

The VISA-A questionnaire provides a valid, reliable, and user friendly index of the severity of Achilles tendinopathy. Although the limited time for consultation in routine clinical practice means that the main role of this tool is likely to be as an outcome measure in treatment studies, the VISA-A scale can be easily administered in clinical practice. The VISA-A has been cross-cultural adapted to Swedish [108] and Italian [60].

---

## Management

### Conservative

Following the “overuse” theory, a first step can be to encourage a sensible training programme. The aim of rehabilitation is to abolish pain and restore function. Anti-inflammatory medications have been used with mixed results although usually with adjunctive physiotherapy. A randomized controlled study where the treatment group was given piroxicam and control group given placebo, both in combination with stretching and strengthening exercises, produced similar results [8]. Orthotics have been used to alter the biomechanics of the foot and ankle [81]. Cryotherapy has an analgesic effect, probably reduces the metabolic rate of tendons and decreases the extravasations of blood and proteins from neo-vessels [98]. The mean VISA score (normal score is 100) in the treatment group rose from 42 to 92 in the treatment group compared to 39–41 in the control group. The authors also demonstrated a decrease in tendon thickness and increased vascularity on ultrasound [121]. Eccentric loading has produced promising results for Achilles tendinopathy [120]. Fifteen recreational athletes with chronic Achilles tendinopathy on a 12-week programme of heavy-load. After a 12-week painful eccentric muscle training programme for chronic Achilles tendinopathy, there was a significant decrease in tendon thickness, normalization of tendon structure [90] and even decrease in neo-vascularity shown on Doppler ultrasound [89]. A randomized controlled trial comparing conventional surgical treatment to a 12-week eccentric training programme in patients with chronic patellar tendinopathy produced similar results for both groups at 12-month follow-up [9].

Non-steroidal anti-inflammatory drugs (NSAIDs) effectively relieve tendinopathy pain and may offer additional benefit in acute inflammatory tendinopathy because of their anti-inflammatory properties. Topical NSAIDs also reduce tendon pain and eliminate the increased risk of gastrointestinal hemorrhage associated with systemic NSAIDs [83]. However, because the majority of chronic tendinopathies are not inflammatory, few data exist to support the use of NSAIDs over analgesics without anti-inflammatory effects [4]. One systematic review [37] found insufficient evidence to recommend or discourage the use of oral NSAIDs in the short-term treatment of tendinopathies.

NSAIDs inhibit tissue inflammation by repressing cyclooxygenase (COX) activity, with a reduction in the synthesis of proinflammatory prostaglandins [119]. Management of an anatomically defined medical condition is ideally based on an understanding of its pathophysiology. Although, as noted earlier, tendinopathy has a non-inflammatory basis, NSAIDs are widely used in attempts at treatment [59]. Ironically, the analgesic effect of NSAIDs [3] allows patients to ignore early symptoms, possibly imposing further damage on the affected tendon and delaying definitive healing.

Cryotherapy is effective for short-term pain relief. Icing may slow the release of blood and proteins from the surrounding vasculature by reducing tissue metabolism. Ice may be effective for reducing swelling and pain in cases of acute inflammatory tendinopathies by blunting the inflammatory response. Although specifics about cryotherapy are not well studied, a recent systematic review of cryotherapy for soft-tissue injuries concluded that applications of ice through a wet towel for 10-min periods are most effective [15].

Eccentric muscle training for treatment of Achilles tendinopathy has been recommended since the mid-1980s [111]. Regardless of the mechanism, significant improvement in patient satisfaction and decreased pain were seen in 60–90% of patients, and it has been demonstrated to be superior to controls [2, 75]. Eccentric training was first advocated in the management of tendinopathy a quarter of a century ago by Stanish et al. [111] Their program involves three sets of 10 repetitions performed once per day, and is progressed by adding load and speed. In their protocol, Stanish and Curwin [110] first recommend that patients should perform the eccentric exercises with no pain. Almost a decade ago, Alfredson et al. [2] introduced another eccentric training program for the Achilles tendon. The Alfredson et al. [2] programme involves three sets of 15 repetitions performed twice a day. In contrast to the Stanish and Curwin program, the Alfredson program involves a greater number of repetitions, isolation of the eccentric muscle contraction, more pain during training, and progression with load and not speed so exercise remains slow; this model consists of two

types of eccentric exercises: (1) with the knee straight, to maximise the activation of the gastrocnemius muscle, and (2) with the knee bent, to maximise the activation of the soleus muscle. Alfredson et al. [2] emphasize the need for patients to complete the exercise protocol despite pain in the tendon. If patients experience no tendon pain doing this programme, the load should be increased until the exercises provoke pain. Although the literature is limited, there is some evidence for the use of painful eccentric training in Achilles [46]. In other two investigations, Sayana et al. [105] and Maffulli et al. [75] used an even more gradual approach than the one advocated by Roos et al. [102] and explained to their patients that eccentric exercises may result in increase in pain in first few weeks. To reduce the impact of this initial inconvenience and to ensure compliance with the exercise regimen, suggested the use of analgesic. To monitor clinical progress of Achilles Tendinopathy has been successfully used the VISA-A Questionnaire [100, 108]. The optimum exercise “dose” is unknown, both in terms of numbers of repetitions and also the speed of movement; neither is the optimum duration of treatment known. Even though the relevance of eccentric training to the conservative management of Achilles tendinopathy is understood, studies regarding its mechanism are needed as a first step to understand and improve the current management regimens [33].

Low dose heparin has been used in the management of peritendinous and intratendinous pathology but an experimental study showed increased tendon degeneration in rats given intratendinous injection of heparin [114]. Peritendinous injection of corticosteroids remains controversial. A randomized controlled trial found that peritendinous injection of methyl prednisolone did not improve the cure rate or shorten the healing time in patients with Achilles tendinopathy [24]. Intratendinous injection of corticosteroid can adversely affect tendon integrity and decrease the amount of load that can be taken before failure [109]. Although intratendinous injection of steroid is discouraged, Read found that the treatment group with peritendinous injection of hydrocortisone in his cohort (n/4 64) with achilodynia did not have a higher rupture rate than the control group [95]. Blind peritendinous injections without the use of imaging guidance may inadvertently be delivered intratendinously. A randomized placebo-controlled double-blind study in patients with Achilles and patellar tendinopathy, using ultrasound for the diagnosis and guided peritendinous injection of triamcinolone, found that the treatment group had reduced pain and tendon thickness [31]. Ultrasound guided injection of a sclerosing agent, polidocanol, to the neovessels in ten patients with Achilles tendinopathy showed reduced pain in eight patients with a corresponding reduction in neovascularity on colour Doppler [88]. The

same authors have also published their results using this procedure in patellar tendinopathy [1]. Several new techniques have been described, including ultrasound guided dry needling and autologous blood injection into the patella tendon [41] with good preliminary results in a small series. The procedure is performed on two occasions, 4 weeks apart. High volume image guided injection (HVIGI) combined with a strict eccentric rehabilitation program has been performed in both chronic recalcitrant Achilles and patella tendinopathy [19, 24] with extremely promising results. Under ultrasound guidance, a needle is inserted just deep in to the affected tendon, and a large volume of fluid (40–60 mL) is injected at the site of maximum neovascularisation. The technique aims to disrupt the neovessels and their accompanying nerve supply. The patients then follow a very strict rehabilitation programme, and return to full activity within 2 weeks of the injection, thus making it very attractive to professional sportsmen.

### Surgical Procedures

Some 24–45.5% of patients with chronic Achilles tendinopathy who fail a 6-month trial of conservative therapy proceed to surgery [57, 68, 106]. It is beyond the scope of this paper to review individual surgical techniques, but the procedures can be categorized as open tenotomy, removal of abnormal tissue without stripping of the paratenon; open tenotomy, removal of abnormal tissue with stripping of the paratenon; open tenotomy with longitudinal tenotomy with or without stripping of the paratenon; percutaneous longitudinal tenotomy; and procedures aiming at interfering with or disrupting neo-vascularity and neo-innervation [57, 68, 106]. Percutaneous longitudinal tenotomy can be used when there is no peritendinous involvement and when the intratendinous lesion is less than 2.5 cm long. Ultrasound guided percutaneous longitudinal tenotomy is able to confirm the precise location of intratendinous lesions and produced similar results to open tenotomy [30, 115]. The aim of surgery is to divide adhesions, excise degenerate nodules and stimulate neo-angiogenesis [32] and the healing response [113]. Over 70% success rates have been reported for operative management for Achilles tendinopathy [32, 68, 92, 106, 115]. Paavola followed 432 consecutive patients for 5 months after surgery and reported 46 complications with 14 of these requiring re operation [91]. Complications include wound breakdown, damage to the sural nerve and deep vein thrombosis [74].

We have recently developed a novel management modality whereby a minimal invasive technique of stripping of neo-vessels from the Kager’s triangle of the AT is performed.



This achieves safe and secure breaking of neo-vessels and the accompanying nerve supply. Four longitudinal skin incisions each 0.5 cm long are made. Two incisions are made just medial and lateral to the origin of the tendon; the other two incisions are made just medial and lateral to the distal end of the tendon close to its insertion. A mosquito is inserted in the incisions, and the proximal and distal portions of Achilles tendon are freed of all the peritendinous adhesions. A Number 1 unmounted Ethibond (Ethicon, Somerville, NJ) suture thread is inserted proximally, passing through the two proximal incisions over the anterior aspect of the Achilles tendon. The Ethibond is retrieved from the distal incisions, over the anterior aspect of the Achilles tendon. The Ethibond is slid on the tendon, which in this way is stripped and freed from adhesions. The procedure is repeated for the posterior aspect of the Achilles tendon. Classically, open surgery for mid-substance tendinopathy of the Achilles tendon, though providing acceptable clinical results [93], has been associated with a relatively high rate of wound complications [93]. This minimally invasive technique reduces the risks of infection, is technically easy to master, and inexpensive. It may provide greater potential for the management of recalcitrant Achilles tendon by breaking neo-vessels and the accompanying nerve supply to the tendon. It can be associated with other minimally invasive procedures to optimize results.

## Conclusions

Achilles tendinopathy may produce marked morbidity, but only limited scientifically-proven management modalities exist. Its management remains a challenge, especially in athletes, in whom physicians may try to be innovative. Many interesting techniques are being pioneered [13, 14, 61, 72]. Although these emerging technologies may develop into substantial clinical management options, their full impact needs to be evaluated critically in a scientific fashion. Future trials should use validated functional and clinical outcomes, adequate methodology, and be sufficiently powered. Clearly, studies of high levels of evidence, for instance large randomized trials, should be conducted to help answer many of the unsolved questions in this field.

## References

1. Alfredson H, Öhberg L (2005) Neovascularisation in chronic painful patellar tendinosis – promising results after sclerosing neovessels outside the tendon challenge the need for surgery. *Knee Surg Sports Traumatol Arthrosc* 13:74–80
2. Alfredson H, Pietila T, Jonsson P et al (1998) Heavy-load eccentric calf muscle training for the treatment of chronic Achilles tendinosis. *Am J Sports Med* 26:360–366
3. Almekinders LC (1990) The efficacy of non-steroidal anti-inflammatory drugs in the treatment of ligament injuries. *Sports Med* 9:137–142
4. Almekinders LC, Temple JD (1998) Etiology, diagnosis, and treatment of tendonitis: an analysis of the literature. *Med Sci Sports Exerc* 30:1183–90
5. Arndt AN, Komi PV, Bruggemann GP et al (1998) Individual muscle contributions to the in vivo achilles tendon force. *Clin Biomech* 13:532–541
6. Astrom M (1997) On the nature and etiology of chronic achilles tendinopathy. PhD thesis, Lund University, Sweden.
7. Astrom M (1998) Partial rupture in Achilles tendinopathy. A retrospective analysis of 342 cases. *Acta Orthop Scand* 69(4):404–407
8. Aström M, Westlin N (1992) No effect of piroxicam on Achilles tendinopathy. A randomized study of 70 patients. *Acta Orthop Scand* 63:631–634
9. Bahr R, Fossan B, Løken S et al (2006) Surgical treatment compared with eccentric training for patellar tendinopathy (Jumper's knee). A randomized controlled trial. *J Bone Joint Surg Am* 88(8):1689–1698
10. Barnes SJ, Gey van Pettius D, Maffulli N (2003) Angioleiomyoma of the Achilles tendon. *Bull Hosp Joint Dis* 61(3–4): 137–139
11. Benazzo F, Maffulli N (2000) An operative approach to Achilles tendinopathy. *Sports Med Arthrosc Rev* 8:96–101
12. Benjamin M, Ralphs J (1995) Functional and developmental anatomy of tendons and ligaments. In: Gordon SL, Blair SJ, Fine LJ (eds) Repetitive motion disorders of the upper extremity. American Academy of Orthopaedic Surgeons, Rosemont, IL, pp 185–203
13. Birkedal-Hansen H (1993) Role of matrix metalloproteinases in periodontal diseases. *J Periodontol* 64(5 suppl):474–484
14. Birkedal-Hansen H, Moore WGI, Bodden MK et al (1993) Matrix metalloproteinase: a review. *Crit Rev Oral Biol Med* 4:197–250
15. Bleakley C, McDonough S, MacAuley D (2004) The use of ice in the treatment of acute soft-tissue injury: a systematic review of randomized controlled trials. *Am J Sports Med* 32:251–261
16. Brinckerhoff CE (1992) Regulation of metalloproteinase gene expression: implication for osteoarthritis. *Crit Rev Euk Gene Expr* 2:145–164
17. Casparian JM, Luchi M, Moffat RE et al (2000) Quinolones and tendon ruptures. *South Med J* 93:488–491
18. Cawston TE, Billington C (1996) Metalloproteinases in rheumatic diseases. *J Pathol* 180:115–117
19. Chan O, O'Dowd D, Padhiar N, Crisp T et al (2008) High volume image guided injections in chronic Achilles tendinopathy. *Disabil Rehabil* 30:1697–1708
20. Chandler S, Miller KM, Clements JM et al (1997) Matrix metalloproteinases, tumor necrosis factor and multiple sclerosis: an overview. *J Neuroimmunol* 72:155–161
21. Cook J, Bass S, Black J (2007) Hormone therapy is associated with smaller Achilles tendon diameter in active post-menopausal women. *Scan J Med Sci Sports* 17(2): 128–132

22. Cook JL, Khan KM, Kiss ZS, Griffiths L (2000) Patellar tendinosis in junior basketball players: a controlled clinical and ultrasonographic study of 268 tendons in players aged 4–18 years. *Scand J Med Sci Sports* 10(4):216–230
23. Cook JL, Kiss ZS, Khan KM, Purdam CR, Webster KE (2004) Anthropometry, physical performance, and ultrasound patellar tendon abnormality in elite junior basketball players: a cross-sectional study. *Br J Sports Med* 38(2): 206–209
24. Crisp T, Khan F, Padhiar N, Chan O et al (2008) High volume ultrasound guided injections at the interface between the patellar tendon and Hoffa's body are effective in chronic patellar tendinopathy. A pilot study. *Disabil Rehabil* 30:1625–1634
25. DaCruz DJ, Geeson M, Allen MJ et al (1988) Achilles paratenonitis: an evaluation of steroid injection. *Br J Sports Med* 22(2):64–65
26. Dalton S, Cawston TE, Riley GP et al (1995) Human shoulder tendon biopsy samples in organ culture produce procollagenase and tissue inhibitor of metalloproteinases. *Ann Rheum Dis* 54(7):571–577
27. Dayer JM, Burger D (1994) Interleukin-1, tumor necrosis factor and their specific inhibitors. *Eur Cytokine Netw* 5:563–571
28. Ebinesan AD, Sarai BS, Walley GD et al (2008) Conservative, open or percutaneous repair for acute rupture of the Achilles tendon. *Disabil Rehabil* 30:1721–1725
29. Ferretti A (1986) Epidemiology of jumper's knee. *Sports Med* 3(4):289–295
30. Ferretti A, Conteduca F, Camerucci E et al (2002) Patellar tendinosis. A follow-up study of surgical treatment. *J Bone Joint Surg (Am)* 84(12):2179–2185
31. Fredberg U, Pfeiffer-Jensen M, Stengaard Pederson K (2004) Ultrasonography as a tool for diagnosis, guidance of local steroid injection, and together with pressure algometry, monitoring of the treatment of athletes with jumper's knee and chronic Achilles tendonitis: a randomized, double-blind, placebocontrolled study. *Scand J Rheumatol* 33: 94–101
32. Friedrich T, Schmidt W, Jungmichel D et al (2001) Histopathology in rabbit Achilles tendon after operative tenolysis (longitudinal fibre incisions). *Scand J Med Sci Sports* 11(1):4–8
33. Gomez DE, Alonso DF, Yoshiji H et al (1997) Tissue inhibitors of metalloproteinases: structure, regulation and biological functions. *Eur J Cell Biol* 74:111–122
34. Goldberg GI, Eisen AZ (1991) Extracellular matrix metalloproteinases in tumor invasion and metastasis. *Cancer Treat Res* 53:421–440
35. Gomez DE, Alonso DF, Yoshiji H et al (1997) Tissue inhibitors of metalloproteinases: structure, regulation and biological functions. *Eur J Cell Biol* 74:111–122
36. Goupille P, Jayson MIV, Valat J et al (1998) Matrix metalloproteinases: the clue to intervertebral disc degeneration? *Spine* 23(14):1612–1626
37. Green S, Buchbinder R, Barnsley L et al (2001) Non-steroidal anti-inflammatory drugs (NSAIDs) for treating lateral elbow pain in adults. *Cochrane Database Syst Rev* (4):CD003686
38. Hamilton B, Remedios D, Loosemore M et al (2008) Achilles tendon rupture in an elite athlete following multiple injection therapies. *J Sci Med Sport* 11:566–568
39. Hess GP, Cappiello WL, Poole RM, Hunter SC (1989) Prevention and treatment of overuse tendon injuries. *Sports Med* 8:371–384
40. Ireland D, Harrall R, Curry V et al (2001) Multiple changes in gene expression in chronic human Achilles tendinopathy. *Matrix Biol* 20(3):159–169
41. James LJ, Ali K, Pocock C, Robertson C, Walter J, Bell J, Connel D (2007) US guided dry needling and autologous blood injection for patellar tendinosis. *Br J Sports Med* 41:518–521
42. James SL, Bates BT, Osternig LR (1978) Injuries to runners. *Am J Sports Med* 6(2):40–50
43. Jarvinen M, Jozsa L, Kannus P et al (1997) Histopathological findings in chronic tendon disorders. *Scand J Med Sci Sports* 7(2):86–95
44. Jozsa L, Kannus P (1997) Human tendon: anatomy, physiology and pathology. *Hum Kinet* 100(3):289–317
45. Kader D, Saxena A, Movin T et al (2002) Achilles tendinopathy: some aspects of basic science and clinical management. *Br J Sports Med* 36(4):239–249
46. Kaeding CC (2004) Surgical treatment of recalcitrant patellar tendinosis. *Techniques Knee Surg* 3(1):29–35
47. Kannus P, Jozsa L, Jarvinen M (2001) Basic science of tendons. In: Garrett WE, Jr SKP, Kirkendall DT (eds) *Principles and practice of orthopaedic sports medicine*. Lippincott Williams and Wilkins, Philadelphia, pp 37–2000
48. Kaufman K, Brodine S, Shaffer R, Johnson C, Cullison T (1999) The effect of foot structure and range of motion on musculoskeletal overuse injuries. *Am J Sports Med* 27(5):585–593
49. Khan KM, Cook JL, Maffulli N et al (2000) Where is the pain coming from in tendinopathy? It may be biochemical, not only structural, in origin. *Br J Sports Med* 34:81–83
50. Khan KM, Maffulli N (1998) Tendinopathy: an Achilles' heel for athletes and clinicians. *Clin J Sport Med* 8:151–154
51. Kirkendall DT, Garrett WE (1997) Function and biomechanics of tendons. *Scand J Med Sci Sports* 7:62–66
52. Koskinen SOA, Heinemeier KM, Olesen JL et al (2004) Physical exercise can influence the levels of matrix metalloproteinases and their inhibitors in tendon related connective tissue. *J Appl Physiol* 96:861–864
53. Koskinen SOA, Höyhty M, Turpeemniemi-Hujanen T et al (2001) Serum concentrations of collagen-degrading enzymes and their inhibitors after downhill running. *Scand J Med Sci Sports* 11:9–15
54. Kountouris A (2003) Rehabilitation of Achilles and patellar tendinopathies. *Best Pract Res Clin Rheum* 21:295–316
55. Kvist M (1991) Achilles tendon overuse injuries. PhD thesis, University of Turku, Finland
56. Kvist M, Jozsa L, Jarvinen MJ, Kvist H (1987) Chronic Achilles paratenonitis in athletes: a histological and histochemical study. *Pathology* 19:1–11
57. Kvist H, Kvist M (1980) The operative treatment of chronic calcaneal paratenonitis. *J Bone Joint Surg (Br)* 62: 352–357

58. Langberg H, Skovgaard D, Petersen LJ et al (1999) Type I collagen synthesis and degradation in peritendinous tissue after exercise determined by microdialysis in humans. *J Physiol* 521 Pt 1:299–306
59. Leadbetter WB (1995) Anti-inflammatory therapy and sports injury: the role of non-steroidal drugs and corticosteroid injection. *Clin Sports Med* 14:353–410
60. Lippi G, Banfi G, Favalaro EJ et al (2008) Updates on improvement of human athletic performance: focus on world records in athletics. *Br Med Bull* 87:7–15
61. Longo UG, Franceschi F, Ruzzini L et al (2007) Light microscopic histology of supraspinatus tendon ruptures. *Knee Surg Sports Traumatol Arthrosc* 15(11):1390–1394
62. Longo UG, Franceschi F, Ruzzini L et al (2007) Light microscopic histology of supraspinatus tendon ruptures. *Knee Surg Sports Traumatol Arthrosc* 15:1390–1394
63. Longo UG, Franceschi F, Ruzzini L et al (2008) Histopathology of the supraspinatus tendon in rotator cuff tears. *Am J Sports Med* 36:533–538
64. Longo G, Ripalda P, Denaro V et al (2006) Morphologic comparison of cervical, thoracic, lumbar intervertebral discs of cynomolgus monkey (*Macaca fascicularis*). *Eur Spine J* 15:1845–1851
65. Lorentzon R (376) Cause of injuries: intrinsic factors. In: Dirix A, Knuttgen HG, Titel K (eds) *The Olympic book of sports medicine*. Blackwell Scientific, Boston, pp 390–1988
66. Lotz M, Gurene PA (1991) Interleukin-6 induces the synthesis of tissue inhibitor of metalloproteinase-1/ erythroid potentiating activity (TIMP-1/EPA). *J Biol Chem* 266:2017–2020
67. Maffulli N, Ajis A, Longo UG et al (2007) Chronic rupture of tendo Achillis. *Foot Ankle Clin* 12(4):583–596
68. Maffulli N, Binfield PM, Moore D (1999) Surgical decompression of chronic central core lesions in the Achilles tendon. *Am J Sports Med* 27(6):747–752
69. Maffulli N, Kader D (2002) Tendinopathy of tendo achillis. *J Bone Joint Surg Br* 84(1):1–8
70. Maffulli N, Kenward MG, Testa V et al (2003) Clinical diagnosis of Achilles tendinopathy with tendinosis. *Clin J Sport Med* 13(1):11–15
71. Maffulli N, Khan KM, Puddu G (1998) Overuse tendon conditions: time to change a confusing terminology. *Arthroscopy* 14(8):840–843
72. Maffulli N, Reaper J, Ewen SW et al (2006) Chondral metaplasia in calcific insertional tendinopathy of the Achilles tendon. *Clin J Sport Med* 16(4):329–334
73. Maffulli N, Sharma P, Luscombe KL (2004) Achilles tendinopathy: aetiology and management. *J R Soc Med* 97(10):472–476
74. Maffulli N, Testa V, Capasso G, Oliva F, Regine R, King JB et al (2006) Surgery for chronic Achilles tendinopathy yields worse results in non-athletic patients. *Clin J Sports Med* 16(2):123–128
75. Maffulli N, Walley G, Sayana MK et al (2008) Eccentric calf muscle training in athletic patients with Achilles tendinopathy. *Disabil Rehabil* 30:1677–1684
76. Mahieu NN, Witvrouw E, Stevens V et al (2006) Intrinsic risk factors for the development of Achilles tendon overuse injury. A prospective study. *Am J Sports Med* 34(2):226–235
77. Malliaras P, Cook J (2000) Reduced ankle dorsiflexion range may increase the risk of patellar tendon injury among volleyball players. *J Sci Med Sports* 9(4):304–309
78. Mauviel A (1993) Cytokine regulation of metalloproteinase gene expression. *J Cell Biochem* 53:288–295
79. McCarthy DA, Dale MM (1988) The leucocytosis of exercise. A review and model. *Sports Med* 6:333–363
80. Meikle MC, Hembry RM, Holley J et al (1994) Immunolocalization of matrix metalloproteinases and TIMP-1 in human gingival tissues from periodontitis patients. *J Periodontol Res* 29:118–126
81. Mohr RN (1997) Achilles tendonitis: rationale for use and application of orthotics. *Foot Ankle Clin* 2:439–456
82. Mokone GG, Gajjar M, September AV et al (2005) The guanine-thymine dinucleotide repeat polymorphism within the tenascin-C gene is associated with Achilles tendon injuries. *Am J Sports Med* 33(7):1016–1021
83. Moore RA, Tramer MR, Carroll D et al (1998) Quantitative systematic review of topically applied non-steroidal anti-inflammatory drugs [published correction appears in *BMJ* 1998;316:1059]. *BMJ* 316:333–338
84. Movin T, Gad A, Reinholt FP et al (1997) Tendon pathology in long-standing achillodynia. Biopsy findings in 40 patients. *Acta Orthop Scand* 68(2):170–175
85. Movin T, Kristoffersen-Wiberg M, Shalabi A, Gad A, Aspelin P, Rolf C (1998) Intratendinous alterations as imaged by ultrasound and contrast medium-enhanced magnetic resonance in chronic achillodynia. *Foot Ankle Int* 19:311–317
86. O'Brien M (1997) Structure and metabolism of tendons. *Scand J Med Sci Sports* 7:55–61
87. Olander SA, Somerville RPT, Apte SS (2003) An update on metalloproteinases in the musculoskeletal system. *Curr Opin Orthop* 14:322–328
88. Öhberg L, Alfredson H (2002) Ultrasound guided sclerosis of neovessels in painful chronic Achilles tendinosis: pilot study of a new treatment. *Br J Sports Med* 36:173–177
89. Öhberg L, Alfredson H (2004) Effects on neovascularisation behind the good results of eccentric training in chronic midportion Achilles tendinosis? *Knee Surg Sports Traumatol Arthrosc* 12:465–470
90. Öhberg L, Lorentzon L, Alfredson H (2004) Eccentric training in patients with chronic Achilles tendinosis: normalized tendon structure and decreased thickness at follow-up. *Br J Sports Med* 38:8–11
91. Paavola M, Kannus P, Orava S et al (2002) Surgical treatment for chronic Achilles tendinopathy: a prospective seven month follow-up study. *Br J Sports Med* 36(3):178–182
92. Paavola M, Orava S, Leppilahti J et al (2000) Chronic Achilles tendon overuse injury: complications after surgical treatment. An analysis of 432 consecutive patients. *Am J Sports Med* 28(1):77–82
93. Perry J (1997) Achilles tendon anatomy. *Foot Ankle Clin* 2:363–370

94. Perugia L, Postacchini F, Ippolito E (1986) The tendons. Biology, pathology, clinical aspects. Editrice Kurtis s.r.l, Milano
95. Read MT (1999) Safe relief of rest pain that eases with activity in Achillodynia by intrabursal or peritendinous injection: The rupture rate was not increased by these steroid injections. *Br J Sports Med* 33:134–139
96. Richards PJ, Braid JC, Carmont MR et al (2008) Achilles tendon ossification: pathology, imaging and aetiology. *Disabil Rehabil* 30:1651–1665
97. Riley GP, Curry V, DeGroot J et al (2002) Matrix metalloproteinase activities and their relationship with collagen remodelling in tendon pathology. *Matrix Biol* 21:185–195
98. Riverburgh DW (1992) Physical modalities in the treatment of tendon injuries. *Clin Sports Med* 11:645–659
99. Robert V, Besse S, Sabri A et al (1997) Differential regulation of matrix metalloproteinases associated with aging and hypertension in the rat heart. *Lab Invest* 76:729–738
100. Robinson JM, Cook JL, Purdam C et al (2001) The VISA-A questionnaire: a valid and reliable index of the clinical severity of Achilles tendinopathy. *Br J Sports Med* 35:335–341
101. Rolf C, Movin T (1997) Etiology, histopathology, and outcome of surgery in achillodynia. *Foot Ankle Int* 118:565–569
102. Roos EM, Engstrom M, Lagerquist A et al (2004) 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* 14:286–295
103. Royer RJ, Pierfitte C, Netter P (1994) Features of tendon disorders with fluoroquinolones. *Therapie* 49:75–76
104. Saarialho-Kere UK, Vaalamo M, Airola K et al (1995) Interstitial collagenase is expressed by keratinocytes that are actively involved in re-epithelialization in blistering skin diseases. *J Invest Derm* 104:982–988
105. Sayana MK, Maffulli N (2007) Eccentric calf muscle training in non-athletic patients with Achilles tendinopathy. *J Sci Med Sport* 10:52–58
106. Schepsis AA, Leach RE (1987) Surgical management of Achilles tendinopathy. *Am J Sports Med* 15:308–315
107. Selvanetti A, Cipolla M, Puddu G (1997) Overuse tendon injuries: basic science and classification. *Oper Tech Sports Med* 5:110–117
108. Silbernagel KG, Thomee R, Karlsson J (2005) Cross-cultural adaptation of the VISA-A questionnaire, an index of clinical severity for patients with Achilles tendinopathy, with reliability, validity and structure evaluations. *BMC Musculoskelet Disord* 6:12
109. Speed CA (2001) Corticosteroid injections in tendon lesions. *BMJ* 323:382–386
110. Stanish WD, Curwin S, Rubinovich M (1985) Tendinitis: the analysis and treatment for running. *Sports Med Clin* 4(4):593–609
111. Stanish WD, Rubinovich RM, Curwin S (1986) Eccentric exercise in chronic tendinitis. *Clin Orthop Relat Res* (208):65–68
112. Stilwell DL, Jr (1957) The innervation of tendons and aponeuroses. *Am J Anat* 100:289–311
113. Tallon C, Coleman BD, Khan KM (2001) Outcome of surgery for chronic Achilles tendinopathy: a critical review. *Am J Sports Med* 29:315–320
114. Tatari H, Kosay C, Ulukus C (2001) Effect of heparin on tendon degeneration: an experimental study on rats. *Knee Surg Sports Traumatol Arthrosc* 9:247–253
115. Testa V, Capasso G, Benazzo F et al (2001) Management of Achilles tendinopathy by ultrasound-guided percutaneous tenotomy. *Med Sci Sports Exe* 34:573–580
116. Tipton CM, Matthes RD, Maynard JA et al (1975) The influence of physical activity on ligaments and tendons. *Med Sci Sports* 7:165–175
117. Tryggvason K, Hoyhtya M, Pyke C (1993) Type IV collagenases in invasive tumors. *Breast Cancer Res Treat* 24:209–218
118. Vaillas AC, Pedrini VA, Pedrini-Mille A et al (1985) Patellar tendon matrix changes associated with aging and voluntary exercise. *J Appl Physiol* 58(5):1572–1576
119. Vane JR (1996) Introduction: mechanism of action of NSAIDs. *Br J Rheumatol* 35:1–3
120. Visnes H, Bahr R (2007) The evolution of eccentric training as treatment for patellar tendinopathy (jumper's knee): a critical review of exercise programmes. *Br J Sports Med* 41:217–223
121. Wang CJ, Ko JY, Chan YS et al (2007) Extracorporeal shockwave for chronic patellar tendinopathy. *Am J Sports Med* 35:972–978
122. Waterston SW, Maffulli N, Ewen SW (1997) Subcutaneous rupture of the Achilles tendon: basic science and some aspects of clinical practice. *Br J Sports Med* 31(4):285–298
123. Werb Z, Alexander CM, Adler RR (1992) Expression and function of matrix metalloproteinases in development. In: Birkedal-Hansen H, Werb Z, Welgus HG et al (eds) *Matrix metalloproteinases and inhibitors*. Stuttgart (Germany) 7 Gustav Fischer, pp 337–343
124. Williams JG (1986) Achilles tendon lesions in sport. *Sports Med* 3(2):114–135
125. Wilson JJ, Thomas M et al (2005) Common overuse tendon problems: a review and recommendations for treatment. *Am Fam Physician* 72:811–818
126. Winge S, Jorgensen U, Lassen Nielsen A (1989) Epidemiology of injuries in Danish championship tennis. *Int J Sports Med* 10(5):368–371
127. Wright JK, Cawston TE, Hazleman BL (1991) Transforming growth factor b stimulates the production of the tissue inhibitor of metalloproteinase by human synovial and skin fibroblasts. *Biochem Biophys Acta* 1094:207–210; metalloproteinases in tendinopathy 275
128. Yasuda T, Poole AR, Shimizu M et al (2003) Involvement of CD44 in induction of matrix metalloproteinases by a COOH-terminal heparin-binding fragment of fibronectin in human articular cartilage in culture. *Arthritis Rheum* 48(5):1271–1280

# The Painful Flatfoot

Victor Valderrabano and Martin Wiewiorski

---

## Introduction

Increased awareness of the acquired adult flatfoot deformity over the past decade has resulted in intensive study of the basic science and pathophysiology behind this common entity. Posterior tibial tendon insufficiency (PTTI), though accepted as the most common aetiology, is frequently missed. This slowly progressive disease requires prompt diagnosis to allow treatment to be started at an earlier, more easily-managed stage. The hallmarks of PTTI are a valgus hindfoot, flattening of the midfoot (longitudinal arch), abduction and supination of the forefoot [36]. This occurs most commonly in women older than 40 years of age. Since the first description of a tendon rupture by Key et al. in 1953 [15], methods of treatment have evolved and the pathology and function of the tendon have been extensively investigated. This article will review the current concepts with regard to the patho-mechanism, clinical evaluation, and treatment of this progressive, debilitating condition.

---

## Anatomy and Biomechanics of the Tibialis Posterior Muscle

The tibialis posterior muscle arises from the posterior surface of the interosseous membrane, the lateral portion of the posterior surface of the shaft of the tibia, and from the upper medial surface of the fibula. It is the most deeply-seated muscle of the deep posterior compartment of the leg. In the lower fourth of the leg its tendon passes in front of that of the flexor digitorum longus and lies with it in a groove behind the medial malleolus in a separate tendon sheath.

---

V. Valderrabano (✉)  
Universitätsspital Basel, Spitalstrasse 21/ Petersgraben 4,  
4031 Basel, Switzerland  
e-mail: vvalderrabano@uhbs.ch

It then passes under the flexor retinaculum, over the deltoid ligament into the foot, and then beneath the spring ligament (calcaneo-navicular ligament) inserting into the tuberosity of the navicular bone. Finally, it inserts with multiple expansions spreading out across the plantar aspect of the hind- and mid-foot (sustentaculum tali of the calcaneus, forward to the medial cuneiform and first metatarsal bone, and laterally to the other cuneiform bones and the bases of the second, third, and fourth metatarsal bones). Since it lies posterior to the axis of the ankle and medial to the axis of the subtalar joint, its function is to plantarflex the ankle, while it inverts the subtalar and oblique axis of the mid-tarsal joints [2]. Due to its anatomical location it provides dynamic support along the plantar aspect of the foot. By passing underneath the spring ligament it stabilizes the osseous configuration at the talonavicular joint and prevents collapsing of the medial longitudinal arch. Loss of the medial longitudinal arch results in dorsiflexion of the first metatarsal. Insufficiency of the first ray may be found in early stages of PTTI [16].

The foot is a complex mechanism acting as a mobile adapter during weight acceptance and a rigid lever arm during propulsion [4]. The talonavicular and calcaneocuboid joints (the midtarsal joint) seem to play an important role in this transition [20]. Blackwood et al. demonstrated significantly less range of motion in the mid-tarsal joint when the calcaneus was maximally inverted compared to when the calcaneus was maximally everted [4]. During normal gait, contraction of posterior tibial muscle limits sub-talar eversion caused by the gastrocsoleus complex. Without the inverting force of the posterior tibial muscle in the stance phase there is less intrinsic osseous stability at the mid-tarsal joint and the forward propulsive force of the complex of gastrocsoleus acts at the mid-foot instead of at the metatarsal heads (push-off phase) [30].

Due to its large cross-sectional area the relative strength of posterior tibial muscle is more than twice that of peroneus brevis, its primary antagonist [21]. With loss of an antagonist force due to PTTI, the unopposed pull from the peroneal tendons forces the heel into eversion.

---

## Epidemiology

The prevalence of PTTI and posterior tibial tendon rupture parallel the degenerative processes of ageing. Hypertension, diabetes mellitus, seronegative arthropathies and obesity have all been identified as risk factors for PTTI [18]. Additionally, the effects of corticosteroids and local surgical procedures may further be associated with local vascular impairment and eventual rupture [13].

---

## Patho-Mechanism of the Adult Flatfoot

A posterior tibial tendon dysfunction evolves from degeneration of the tendon due to repetitive micro-trauma or chronic overuse [22].

The posterior tibial tendon is acutely angled where it passes posterior to the medial malleolus. This area of increased stress on the tendon is the most common site of degeneration of the posterior tibial tendon. The blood supply of the tendon can be divided into the proximal and distal areas. The proximal tendon is supplied by branches of the posterior tibial artery and the distal, which is at the bone-tendon interface, by branches of the posterior tibial and dorsalis pedis arteries [10]. Whether degeneration of the PTTI is due to physiological hypovascularity of the tendon at the degeneration site remains controversial. Frey et al. showed that there is an area of hypovascularity in the tendon about 1 cm distal to the medial malleolus [10]. Contrary to these findings Prado et al. did not find decreased vascularization in the mid-portion of the tendon [26]. Histological examinations of surgical posterior tibial tendons from patients with PTTI revealed a degenerative tendinosis characterized by increased mucin content, fibroblast hypercellularity, chondroid metaplasia, and neo-vascularization. Those result in disruption of the linear orientation of the collagen bundles [9, 22]. Although PTTI is widely accepted as a significant contributor to this deformity, the pathology and deformity involve more than the tendon itself. Basmajian and Stecko concluded from their electromyographic measurement that the ligaments and osseous configuration of the foot form the primary stabilizers of the foot arch [3]. Muscular involvement in stabilization is merely called upon during increased load (take-off phase in walking). MRI studies confirmed the association of spring ligament failure with PTTI [29, 35]. The superomedial calcaneonavicular ligament is most commonly involved, followed by the inferomedial calcaneonavicular and talocalcaneal interosseous ligaments. In patients with surgically proven spring ligament tear. Toye et al. demonstrated an abnormal spring ligament

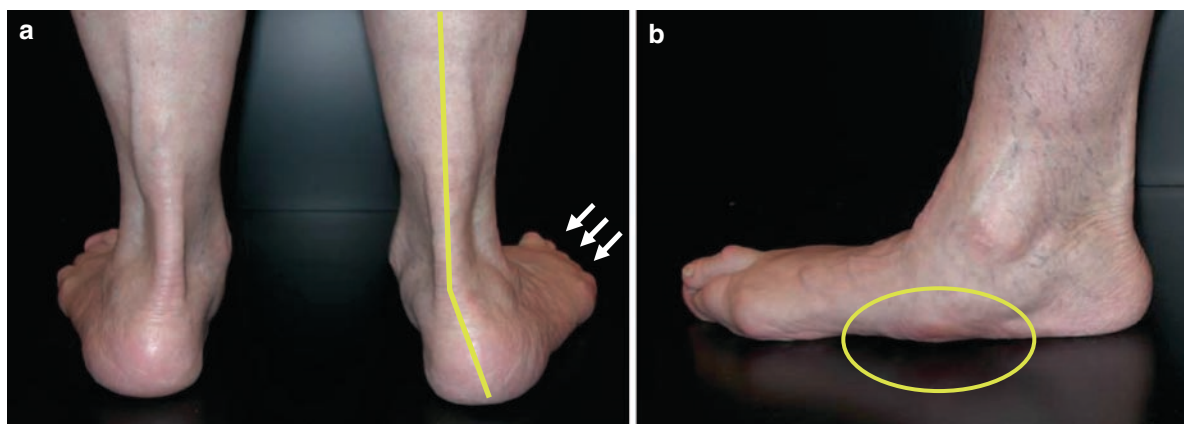
thickening, a full-thickness gap and an abnormal increased signal on pre-operative T2-weighted MRI images [29].

The involvement of the deltoid ligament is being discussed (for clinical differentiation see Diagnostics). Hintermann found PTTI in 22% of patients with medial ankle instability [11]. However, it remains unclear if medial ankle instability may cause a secondary PTTI with elongation and/or rupture of the tendon, or vice versa [11]. Patients with a pre-existing flatfoot deformity show increased gliding resistance at the tendon-sheath interface of the posterior tibial tendon. The findings indicate a possible vicious circle of deformity and tendon dysfunction [31].

---

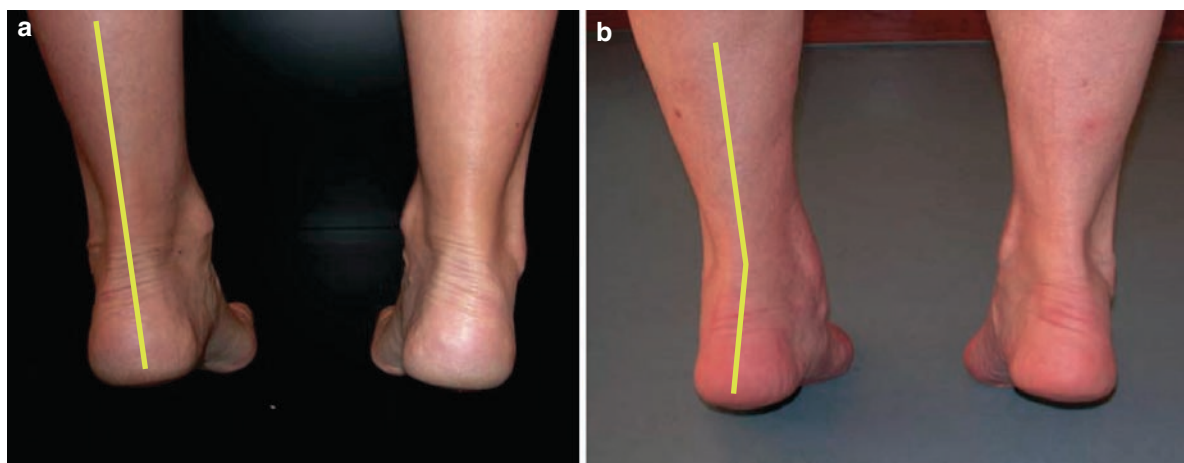
## Clinical Examination

The diagnosis of PTTI is essentially clinical. Systematic examination of the soft tissues needs to consider the potentially unaffected contra-lateral side. Both feet are inspected in a standing position with parallel feet, shoulder width apart. Inspection includes hindfoot alignment, deformity, and swelling. The medial longitudinal arch is inspected for the presence of flattening (Fig. 1b). Hindfoot alignment is best inspected from behind the patient. Valgus mal-alignment presents as an asymmetrical planus and pronation deformity of the affected hindfoot [11]. Valgus angulation of more than 10° (or valgus in bilateral comparison) is typically found in PTTI in stage II and above (Fig. 1a). Forefoot abduction is indicated by the “too-many-toes sign” (Fig. 1a). The test is positive when more of the toes are visible lateral to the ankle joint of the involved side than on the contra-lateral side, when viewed from behind [14]. The dynamic function of the posterior tibial tendon is determined by the single-limb heel-rise test. The patient is asked to rise onto the ball of one foot while the other foot is suspended in the air. As the patient rises off the floor, the posterior tibial tendon inverts and stabilizes the hindfoot (Fig. 2a). In patients with PTTI the heel fails to invert and remains in valgus (Fig. 2b). If unable to achieve the tiptoe position on one foot the patient should perform a bilateral heel-rise. In late stage PTTI patients often will not be able to perform either test. This test is also helpful to differentiate PTTI from elongation or rupture of the deltoid ligament (medial ankle ligament instability). In cases of isolated medial ankle instability without involvement of the posterior tibial tendon, the hindfoot valgus deformity is corrected by activity of the posterior tibial muscle when in tiptoe position. Ankle joint stability must be assessed (drawer test and talar tilt test). Still, clinical differentiation between PTTI and medial ankle instability is challenging and diagnosis frequently



**Fig. 1** *PTTI Stage IV.* A patient with stage IV posterior tibial tendon insufficiency. Viewed from behind, a distinct valgus of the hindfoot with a positive “too-many-toes”-sign (arrows) is

revealed (a). The side view shows a collapse of the medial longitudinal arch and a protruding talus head due to subluxation at the talonavicular joint (b)



**Fig. 2** *Heel-rise test.* Physiological inversion of the hindfoot in tip-toe position (a). In patients with posterior tibial tendon insufficiency, the hindfoot fails to invert and remains in eversion (b). Patients with late stage PTTI often will not be able to perform this test

needs to be further clarified by MRI or arthroscopy [12]. Progressing PTTI is often associated with an abduction and supination deformity of the forefoot. In a patient standing fully weight-bearing on both feet, external rotation of the tibia is applied to provoke varus movement of the hindfoot. In a patient with supination deformity, the first metatarsal will rise off the floor (first metatarsal rise sign).

In flexible stage I and II of PTTI the patients show pain and tenderness in the course of the posterior tibial tendon, especially at the medial malleolar groove, the spring ligament and the tarsal insertion. In later stages, with fixed deformity and frequently found chronic rupture of the posterior tibial tendon, the pain maximum may shift to the lateral ankle due to fibulo-calcaneal impingement. Retro-malleolar pain can

be a sign of posterior ankle joint impingement. Other differential diagnoses include: tarsal coalition, osteoarthritis, and other post-traumatic, neurologic, diabetogenic or iatrogenic pathologies [19].

## Imaging

Posterior tibial tendon dysfunction is essentially a clinical diagnosis. Plain radiography helps to confirm the extent of the deformity and presence of osteoarthritis. Conventional radiological imaging consists of anteroposterior and lateral weight-bearing radiographs of the whole foot and an

anteroposterior radiograph of the ankle joint. If the deformity is present, the anteroposterior radiograph shows a pathological a.p. talar-first metatarsal angle (normal angle, 0–10°) with an abduction of the forefoot at the transverse tarsal joint, with the navicular sliding laterally on the talar head (Fig. 3a). In patients who have an advanced deformity, subluxation or dislocation of the talonavicular joint may occur in association with degenerative osteoarthritis of the posterior facet of the subtalar joint. The lateral radiograph shows a decrease in the lateral talar-first metatarsal angle (normal angle, 0–10°) and flattening of the longitudinal arch (Fig. 3b). The anteroposterior radiograph of the ankle joint reveals potential deformity and narrowing of the fibulo-calcaneal space (Fig. 3c). Several authors emphasize that magnetic resonance imaging (MRI) is not required to make the diagnosis and does not assist in the planning of treatment. Some authors have suggested MRI to be useful in the evaluation of PTTI [27, 28]. By contrast other authors state that it is used too frequently in clinical practice and that its clinical usefulness is questionable [13, 23].

### Classification

There is a continuum of PTTI ranging from tenosynovitis to fixed deformity. In 1989 Johnson and Strom described three clinical stages of dysfunction [14]. Myerson et al. added a fourth to describe the most severe deformity with valgus

collapse of the talus within the ankle [23] (Table 1). Stage I incorporates tenosynovitis. In this stage, the tendon is of normal length and symptoms are usually mild to moderate. Pain and swelling are present on the medial aspect of the foot. Mild weakness and minimal deformity are present. In stage II there is elongation or tearing of the tendon. The limb is weak and the patient is unable to stand on tiptoe on the affected side. There is secondary deformity as the midfoot pronates and the forefoot abducts at the transverse tarsal joint. The subtalar joint remains mobile. Stage III is characterized by a more severe deformity and a fixed hindfoot. Stage IV involves a valgus deformity and degeneration of the ankle joint.

### Treatment

Treatment of PTTI is aimed to stop progression of the tendon dysfunction and to protect the longitudinal arch-stabilizing soft tissues (e.g. spring ligament, deltoid). This can be achieved by reconstruction of the anatomical alignment and recovery of physiological biomechanics. PTTI with no or beginning deformity characterized by a flexible hindfoot foot can be treated in a joint-preserving way by conservative and tendon reconstructive methods. Treatment of rigid hindfoot deformities in later stage III and IV intends to reconstruct the painful deformity. Osseous reposition and subsequent arthrodesis are often inevitable.



**Fig. 3** Conventional radiographs of stage IV posterior tibial tendon insufficiency. The anteroposterior view shows abduction of the forefoot at the transverse tarsal joint, with the navicular sliding laterally on the talar head (a). The lateral

radiograph shows a decrease in the talometatarsal angle (b). Pain at the lateral ankle is often due to fibulo-calcaneal impingement (circle). Additional valgus tilt of the ankle joint is present (dashed lines) (c)



**Table 1** Classification and treatment recommendation for posterior tibial tendon insufficiency

Stage	Tendon	Deformity	Pain	Single limb heel rise	Too many toes sign	Therapy
I	Peritendinitis and/or tendon degeneration	Mobile hindfoot, normal alignment	Medial: focal, mild to moderate	Normal inversion of hindfoot	-	Conservative: RICE, NSAR, orthosis, tendosynovectomy in advanced cases
II	Elongation and marked tendon degeneration	Mobile hindfoot, valgus alignment	Medial: along TPT, moderate	No or reduced inversion of hindfoot	+	Tendon reconstruction <i>and</i> osteotomy of the calcaneus
III	Elongation and marked tendon degeneration	Fixed deformity, valgus position	Medial: possibly lateral, moderate	Unable to perform test, no inversion	+	Varisating subtalar arthrodesis or triple arthrodesis
IV	Marked tendon degeneration	Additional angulation of the talus and early degeneration of the ankle joint	Medial and lateral, distinct pain	Unable to perform test, no inversion	+	Triple arthrodesis with supramalleolar osteotomy Triple arthrodesis with total ankle replacement Pantalar arthrodesis

RICE rest, ice, compression, elevation; NSAR non-steroidal anti-inflammatory drugs

## Stages I and II: The Flexible Foot

### Stage I

A period of 4–8 weeks of immobilisation in a plaster cast below the knee or a walking boot may be required to control accompanying inflammation. Complementary measures are RICE (rest, ice, compression, and elevation) and anti-inflammatory drugs. Footwear plays an important role, and patients should be encouraged to wear flat lace-up shoes, or even lace-up boots, which accommodate orthoses. Stage I patients may be able to manage with an casted insoles. The various casted, semi-rigid insoles support the medial longitudinal arch of the foot and either hold the heel in a neutral alignment (stage I) or correct the outward bent heel to a neutral alignment (stage II). This approach is meant to serve several functions: to alleviate stress on the posterior tibial tendon and muscle; to make gait more efficient by holding the hindfoot fixed; and thirdly, to prevent progression of deformity. When this approach has been used, two thirds of patients have good to excellent results [18]. However, from our experience, conservative therapy shows poor results in the long-term. Some authors propose tenosynovectomy for patients who have advanced stage I disfunction. Good results have been reported for either open or tendoscopic techniques [5, 33].

### Stage II

No soft-tissue reconstructive surgical technique by itself can sufficiently contain the forces of a mal-aligned hindfoot. Therefore, consensus is growing that surgical treatment of stage II PTTI should include a tendon transfer in combination with corrective osteotomy [23, 25]. The rationale behind this approach is that the osteotomy is required to correct the bony architecture of the foot in order to optimize the biomechanics of the reconstructed posterior tibial tendon and protect other foot-stabilizing ligaments and tendons [11]. According to Valderrabano et al., only about 60% of force regeneration can be expected after surgical reconstruction of a dysfunctional or ruptured posterior tibial tendon. The magnitude of regained strength is primarily dependent on the duration of the dysfunction and amount of atrophy [32]. Therefore PTTI needs to be surgically addressed at an early stage after failure of conservative treatment.

The two recommended tendon reconstruction techniques are flexor digitorum longus (FDL) tendon transfer and transfer of a split anterior tibial tendon (Cobb procedure). Re-routing a part of the anterior tibial tendon to the plantar aspect of the cuneiform allows the posterior tibial

tendon to pull at its physiological insertion site. Additionally the Cobb procedure decreases the tension of the anterior tibial tendon [16], thus preventing occasionally anterior tibial tendon ruptures. This may add to correct the deformity, by reduction of the pull of the anterior tibial tendon, which is usually increased in PTTI. This dynamic correction may allow the patient to adapt the forefoot to the ground as required. Additionally, this procedure does not sacrifice the FDL tendon.

Another method is the transfer of flexor digitorum longus. The FDL tendon is detached proximal to the juncture with the flexor hallucis longus (Henry's knot). The periosteum over the navicular is then dissected and a drill-hole is made in the tuberosity from the dorsal to plantar aspects. The tendon is sutured side-to-side to the posterior tibial tendon and passed through the drill hole from plantar to dorsal. The use of the flexor hallucis longus tendon is not recommended due to its important role in the push-off phase off the foot [30]. Intra-operative exploration of the spring ligament in PTTI is mandatory, because of its frequent concomitant degeneration or rupture [35]. If found ruptured, reconstruction has to consider both components of the spring ligament complex [7]. The authors suggest that if an accompanying medial ankle instability is suspected upon clinical examination, an initial ankle arthroscopy needs to be performed to rule out deltoid ligament involvement. If ligament instability is found, it needs to be addressed during the following surgery.

Various osteotomies of the calcaneus can correct the pathological bony alignment. During surgery, these osteotomies should be performed prior to finalizing the medial soft tissue reconstruction. In PTTI with pronounced hindfoot valgus and no or minimal foot abduction a medial sliding osteotomy is recommended [23]. The lateral hindfoot incision extends from the superior border of the calcaneal tuberosity anterior to the retrocalcaneal space to the inferior border of the calcaneus superficial to the plantar fascia. An oblique transverse osteotomy is made in the calcaneus in line with the incision in the skin by use of an oscillating saw. The cut is made at a right angle to the lateral border of the calcaneus and is inclined posteriorly at an angle of approximately 45° to the plane of the sole of the foot. The posterior fragment of the calcaneal tuberosity is translated medially ten or more millimeters and is secured with a cannulated headless compression screw.

In PTTI with pronounced hindfoot valgus and distinct forefoot abduction a lateral calcaneus-lengthening osteotomy is recommended. By lengthening the lateral column, the medial longitudinal arch is restored secondarily to the induced adduction movement of the forefoot that supinates the foot at the subtalar and talonavicular joint. An osteotomy

of the anterior calcaneus was originally described by Evans et al. [8]. Here an osteotomy of the neck of the calcaneus is performed and a tri-cortical bone graft impacted. Several authors have reported good results with this procedure [1, 24]. Myerson et al. proposed a lengthening through the calcaneocuboid joint itself, with use of a tri-cortical bone graft for arthrodesis of the joint [23]. We favour an alternative method proposed by Hintermann et al. [12]. Here an osteotomy is performed from the lateral hindfoot approximately 12–20 mm. proximal to the calcaneocuboid joint at the “floor” of the sinus tarsi. The oscillating saw passes between the posterior and middle facet of the subtalar joint. The medial cortex is kept intact. The gap is widened with a Casper spreader and a tri-cortical graft or alternatively allograft bone is inserted. The amount of widening can be adjusted until the medial arch is restored sufficiently. The graft is fixed with one 3.5 mm cortical screw. Preservation of the subtalar and calcaneocuboideal joint offers certain advantage. One has to keep in mind that fusing the calcaneocuboid joint significantly reduces hindfoot motion [6]. Maintaining hindfoot motion prevents overload on adjacent joints which may lead to osteoarthritis. Additionally, according to Knupp et al. it is easier to reduce the abducted foot if the lateral column is not further shortened by arthrodesis of the calcaneocuboid joint [17].

A recently-presented surgical method for stage II PTTI is the subtalar arthroereisis. Here, the sinus tarsi is emptied and an expanding endorthesis is inserted following prior correction of the deformity and tendon repair. Good results were shown for the subtalar arthroereisis, especially in younger patients [34]. However, one has to keep in mind that the fatty tissue in the sinus tarsi contains abundant nerve cell which are essential for proprioception of the hindfoot (“cerebellum pedis”) and therefore should be dissected carefully and not removed.

---

## Stages III and IV: The Rigid Foot

### Stage III

The goal of surgical treatment of stage III PTTI is correction of the deformity and pain relief. Because at this stage hindfoot deformity cannot be passively reduced, joint-preserving surgery frequently fails. Depending on the extent of the deformity, correction can be achieved through a varisating subtalar arthrodesis or triple arthrodesis of the subtalar, calcaneocuboid, and talonavicular articulations. In our opinion, calcaneocuboidal arthrodesis can be omitted to sustain residual motion of the lateral column.

### Stage IV

Stage IV PTTI has been reached when additional degenerative changes are present in the ankle joint. In such cases, a varisating triple arthrodesis together with a medial closing wedge supramalleolar osteotomy and deltoid ligament reconstruction may solidly address the deformity. In very selected cases a varisating triple arthrodesis may be combined with total ankle replacement. However, the salvage treatment at this stage is usually a pantalar arthrodesis (ankle, subtalar, calcaneocuboid, and talonavicular articulations).

---

## Rehabilitation

Bradytrophic tendon tissue requires sufficient healing time. To reach adequate stability, approximately 12 weeks for the tendon and 8–12 weeks for the bone are required. In the initial 6 weeks immobilization in a pneumatic walking brace with partial weight-bearing (heel-to-toe pattern 15–20 kg with crutches) is mandatory. The load can be then increased gradually until full weight-bearing is reached after 12 weeks post-operatively. Physiotherapeutic care needs to address postural hindfoot stability. From our own experience it takes 3–6 months until the rehabilitation is finished. This time span is needed to adapt the cerebellar control of balance and locomotion to the new anatomic configuration of the hindfoot.

---

## Conclusion

The tibialis posterior muscle is the key dynamic support of the medial longitudinal arch of the foot. When it fails – typically in women older than 40 years of age-progressively, the arch slowly collapses, the heel drifts into valgus, the midfoot flattens, and the forefoot gradually abducts and supinates, resulting in painful acquired pes planovalgus abductus and supinatus. Posterior tibial tendon insufficiency (PTTI) is often misdiagnosed as a chronic ankle sprain, osteoarthritis, or collapsed arch as a result of aging or obesity, and it leaves the patient debilitated. Prompt diagnosis prevents frustration for the patient and allows treatment to be started at an earlier, more easily managed stage. The diagnosis of PTTI is largely a clinical one. An increased awareness of the existence of PTTI should serve to help patients with earlier referral and treatment and by limiting the amount of disability.

### Take Home Pearls

- ▶ Posterior tibial tendon insufficiency (PTTI) is a progressive entity leading to painful pes planovalgus abductus and supinatus.
- ▶ If conservative treatment fails, early surgical intervention slows further progression of the disease.
- ▶ Reconstruction of the posterior tibial tendon and concomitant medial ligamentous lesions need to be accompanied by a corrective calcaneal osteotomy:
  - Distinct forefoot abduction → lateral calcaneus lengthening osteotomy
  - No or minimal forefoot abduction → calcaneus medial sliding osteotomy
- ▶ If additional medial ankle instability is suspected upon clinical examination, an ankle arthroscopy needs to rule out deltoid ligament involvement. If accompanying ligament instability is verified, an additional medial ligamentoplasty is necessary.

### References

1. Anderson AF, Fowler SB (1984) Anterior calcaneal osteotomy for symptomatic juvenile pes planus. *Foot Ankle* 4(5):274–283
2. Baravarian B (2002) Use of the Cobb procedure in the treatment of posterior tibial tendon dysfunction. *Clin Podiatr Med Surg* 19(3):371–389
3. Basmajian JV, Stecko G (1963) The Role of Muscles in Arch Support of the Foot. *J Bone Joint Surg Am* 45:1184–1190
4. Blackwood CB et al (2005) The midtarsal joint locking mechanism. *Foot Ankle Int* 26(12):1074–1080
5. Chow HT, Chan KB, Lui TH (2005) Tendoscopic debridement for stage I posterior tibial tendon dysfunction. *Knee Surg Sports Traumatol Arthrosc* 13(8):695–698
6. Deland JT et al (1995) Lateral column lengthening with calcaneocuboid fusion: range of motion in the triple joint complex. *Foot Ankle Int* 16(11):729–733
7. Deland JT et al (2005) Posterior tibial tendon insufficiency: which ligaments are involved? *Foot Ankle Int* 26(6):427–435
8. Evans D (1975) Calcaneo-valgus deformity. *J Bone Joint Surg Br* 57(3):270–278
9. Fowble VA et al (2006) Neovascularity in chronic posterior tibial tendon insufficiency. *Clin Orthop Relat Res* 450:225–230
10. Frey C, Shereff M, Greenidge N (1990) Vascularity of the posterior tibial tendon. *J Bone Joint Surg Am* 72(6):884–888
11. Hintermann B (2003) Medial ankle instability. *Foot Ankle Clin* 8(4):723–738
12. Hintermann B, Valderrabano V, Kundert HP (1999) Lengthening of the lateral column and reconstruction of the medial soft tissue for treatment of acquired flatfoot deformity associated with insufficiency of the posterior tibial tendon. *Foot Ankle Int* 20(10):622–629
13. Holmes GB Jr, Mann RA (1992) Possible epidemiological factors associated with rupture of the posterior tibial tendon. *Foot Ankle* 13(2):70–79
14. Johnson KA, Strom DE (1989) Tibialis posterior tendon dysfunction. *Clin Orthop Relat Res* 239:196–206
15. Key JA (1953) Partial rupture of the tendon of the posterior tibial muscle. *J Bone Joint Surg Am* 35-A(4):1006–1008
16. Knupp M, Hintermann B (2007) The Cobb procedure for treatment of acquired flatfoot deformity associated with stage II insufficiency of the posterior tibial tendon. *Foot Ankle Int* 28(4):416–421
17. Knupp M et al (2009) Subtalar and talonavicular arthrodesis through a single medial approach for the correction of severe planovalgus deformity. *J Bone Joint Surg Br* 91(5):612–615
18. Kohls-Gatzoulis J et al (2004) Tibialis posterior dysfunction: a common and treatable cause of adult acquired flatfoot. *Bmj* 329(7478):1328–1333
19. Lee MS et al (2005) Diagnosis and treatment of adult flatfoot. *J Foot Ankle Surg* 44(2):78–113
20. Manter J (1941) Movements of the subtalar and transverse tarsal joints. *The Anatomical Record* 80(4):397–410
21. Mizel MS et al (1999) Role of the peroneal tendons in the production of the deformed foot with posterior tibial tendon deficiency. *Foot Ankle Int* 20(5):285–289
22. Mosier SM et al (1998) Pathology of the posterior tibial tendon in posterior tibial tendon insufficiency. *Foot Ankle Int* 19(8):520–524
23. Myerson MS (1997) Adult acquired flatfoot deformity: treatment of dysfunction of the posterior tibial tendon. *Instr Course Lect* 46:393–405
24. Phillips GE (1983) A review of elongation of os calcis for flat feet. *J Bone Joint Surg Br* 65(1):15–18
25. Pinney SJ, Lin SS (2006) Current concept review: acquired adult flatfoot deformity. *Foot Ankle Int* 27(1):66–75
26. Prado MP et al (2006) Vascular density of the posterior tibial tendon: a cadaver study. *Foot Ankle Int* 27(8):628–631
27. Rosenberg ZS et al (1988) Rupture of posterior tibial tendon: CT and MR imaging with surgical correlation. *Radiology* 169(1):229–235
28. Rosenberg ZS et al (1988) Rupture of the posterior tibial tendon: CT and surgical findings. *Radiology* 167(2):489–493
29. Toye LR et al (2005) MRI of spring ligament tears. *AJR Am J Roentgenol* 184(5):1475–1480
30. Trnka HJ (2004) Dysfunction of the tendon of tibialis posterior. *J Bone Joint Surg Br* 86(7):939–946
31. Uchiyama E et al (2006) Gliding resistance of the posterior tibial tendon. *Foot Ankle Int* 27(9):723–727
32. Valderrabano V et al (2004) Recovery of the posterior tibial muscle after late reconstruction following tendon rupture. *Foot Ankle Int* 25(2):85–95
33. van Dijk CN, Kort N, Scholten PE (1997) Tendoscopy of the posterior tibial tendon. *Arthroscopy* 13(6):692–698

34. Viladot R et al (2003) Subtalar arthroereisis for posterior tibial tendon dysfunction: a preliminary report. *Foot Ankle Int* 24(8):600–606
35. Yao L, Gentili A, Cracchiolo A (1999) MR imaging findings in spring ligament insufficiency. *Skeletal Radiol* 28(5): 245–250
36. Yeap JS, Singh D, Birch R (2001) Tibialis posterior tendon dysfunction: a primary or secondary problem? *Foot Ankle Int* 22(1):51–55